

JYU DISSERTATIONS 660

Suvi Ravi

Low Energy Availability-Related Conditions in Female Athletes

**Prevalence Rates and Relationships with
Health, Injuries, and the Sports Career**



UNIVERSITY OF JYVÄSKYLÄ
FACULTY OF SPORT AND
HEALTH SCIENCES

JYU DISSERTATIONS 660

Suvi Ravi

**Low Energy Availability-Related
Conditions in Female Athletes
Prevalence Rates and Relationships with
Health, Injuries, and the Sports Career**

Esitetään Jyväskylän yliopiston liikuntatieteellisen tiedekunnan suostumuksella
julkisesti tarkastettavaksi yliopiston vanhassa juhlasalissa S212
kesäkuun 21. päivänä 2023 kello 12.

Academic dissertation to be publicly discussed, by permission of
the Faculty of Sport and Health Sciences of the University of Jyväskylä,
in building Seminarium, Old Festival Hall S212, on June 21, 2023, at 12 o'clock.



JYVÄSKYLÄN YLIOPISTO
UNIVERSITY OF JYVÄSKYLÄ

JYVÄSKYLÄ 2023

Editors

Anne Viljanen

Faculty of Sport and Health Sciences, University of Jyväskylä

Päivi Vuorio

Open Science Centre, University of Jyväskylä

Copyright © 2023, by the author and University of Jyväskylä

ISBN 978-951-39-9648-2 (PDF)

URN:ISBN:978-951-39-9648-2

ISSN 2489-9003

Permanent link to this publication: <http://urn.fi/URN:ISBN:978-951-39-9648-2>

ABSTRACT

Ravi, Suvi

Low energy availability-related conditions in female athletes: Prevalence rates and relationships with health, injuries, and the sports career

Jyväskylä: University of Jyväskylä, 2023, 153 p. + original articles

(JYU Dissertations

ISSN 2489-9003; 660)

ISBN 978-951-39-9648-2 (PDF)

Physical activity provides many health benefits, but some female athletes suffer from problems related to eating and menstrual function. This dissertation study investigated the prevalence of low energy availability (EA)-related conditions, such as clinical and subclinical eating problems, body weight perceptions, and menstrual dysfunction (MD), among Finnish female athletes and evaluated whether athletes and non-athletes or athletes of different ages and those competing at different levels and sports differ from each other regarding these issues. Furthermore, this study explored the associations of low EA-related conditions and age at menarche with injuries, sports career, and health.

Data were drawn from four research projects: the Finnish Health Promoting Sports Club study baseline (n=283) and follow-up (n=211) studies, the Estrogenic Regulation of Muscle Apoptosis study (n=1098), the Female Athlete 2.0 study (n=846), and the Menstrual Function, Eating Attitudes, Body Image, and a Sports Career study (n=198). Participants were 14- to 55-year-old female athletes and non-athletes. Data on menstrual function, eating attitudes, and body weight perceptions were measured using questionnaires.

The prevalence rates of eating disorders, disordered eating behaviors, body weight dissatisfaction, and MD in athletes varied between 7–18%, 16–25%, 18–24%, and 18–39%, respectively, depending on the data used. MD was more common in athletes than in non-athletes. Athletes did not differ from non-athletes in terms of eating problems, but body weight dissatisfaction was more common in non-athletes. Participation in endurance, weight class, jumping, and aesthetic sports was associated with an increased risk of eating problems and MD. A younger age was associated with higher rates of MD. Later age at menarche was associated with a healthier midlife body composition, but also a lower bone mineral density. Problems with eating behavior were associated with a higher risk of injury and a shorter sports career, while MD, specifically secondary amenorrhea, was associated with higher rates of injury-related harm during the sports career and career termination due to injury.

It is crucial to recognize the high rates of low EA-related conditions in female athletes. Athletes and those working with them should be aware of the negative link of low EA-related conditions to health and the sports career.

Keywords: athletes, eating behavior, menstruation disturbances, athletic injuries

TIIVISTELMÄ (ABSTRACT IN FINNISH)

Ravi, Suvi

Alhaiseen energiansaatavuuteen liittyvät tilat naisurheilijoilla: Esiintyvyys ja yhteydet terveyteen, vammoihin ja urheilu-uraan

Jyväskylä: Jyväskylän yliopisto, 2023, 153 s. + alkuperäisartikkelit

(JYU Dissertations

ISSN 2489-9003; 660)

ISBN 978-951-39-9648-2 (PDF)

Liikunnalla on monia terveyshyötyjä, mutta osalla naisurheilijoista esiintyy syömiseen ja kuukautiskiertoon liittyviä haasteita. Tämä väitöskirjatutkimus selvitti alhaiseen energiansaatavuuteen liittyvien tilojen (syömisen haasteet, kehon painoon liittyvät käsitykset ja kuukautiskierron häiriöt) esiintyvyyttä ja sitä, eroavatko urheilijat ja ei-urheilijat tai eri-ikäiset, eri tasoilla ja eri lajityypeissä kilpailevat urheilijat toisistaan näiden tilojen esiintyvyydessä. Lisäksi tutkittiin alhaiseen energiansaatavuuteen liittyvien tilojen ja kuukautisten alkamisiän yhteyttä vammoihin, urheilu-uraan ja terveyteen.

Tutkimuksessa hyödynnettiin neljän tutkimusprojektin aineistoa: Terveyttä edistävä liikuntaseura -tutkimuksen ensimmäisen ja toisen mittauskerran aineistoa (n = 283 ja n = 211), Estrogeeni, vaihdevuodet ja toimintakyky -aineistoa (n = 1098), Naisurheilija 2.0 -aineistoa (n = 846) sekä Kuukautiskierron häiriöiden sekä syömiseen ja kehonkuvaan liittyvien asenteiden yhteys urheilu-uraan -aineistoa (n = 198). Tutkittavat olivat 14–55-vuotiaita urheilijoita ja ei-urheilijoita. Kuukautiskiertoon ja syömiseen liittyvät tiedot kerättiin kyselylomakkeilla.

Syömishäiriöiden esiintyvyys urheilijoilla oli aineistosta riippuen 7–18 % ja häiriintyneen syömiskäyttäytymisen esiintyvyys 16–25 %. Tyytymättömyyttä kehon painoon esiintyi 18–24 %:lla ja kuukautishäiriöitä 18–39 %:lla urheilijoista. Kuukautishäiriöt olivat yleisempiä urheilijoilla kuin ei-urheilijoilla, mutta ryhmät eivät eronneet toisistaan syömisen haasteiden suhteen. Tyytymättömyys kehon painoon oli yleisempää ei-urheilijoilla. Kestävyys-, painoluokka- ja hyppylajien sekä esteettisten lajien urheilijoilla esiintyi enemmän syömisen ja kuukautiskierron häiriöitä kuin muilla urheilijoilla. Nuorempi ikä oli yhteydessä korkeampaan kuukautishäiriöiden esiintyvyyteen. Myöhäisempi kuukautisten alkamisikä yhdistyi alhaisempaan kehon rasvamassan määrään keski-ikässä, mutta myös alhaisempaan luuntiheyteen. Syömisen haasteet olivat yhteydessä suurempaan vammojen ilmaantuvuuteen ja lyhyempään urheilu-uraan. Kuukautisten poisjääminen urheilu-uran aikana oli yhteydessä suurempaan vammojen aiheuttamaan haittaan ja uran lopettamiseen vamman vuoksi.

Syömisen ja kuukautiskierron haasteiden yleisyys naisurheilijoilla tulisi tunnistaa. Urheilijoiden ja heidän kanssaan toimivien ammattilaisten tulisi olla tietoisia näiden tilojen epäedullisesta yhteydestä terveyteen ja urheilu-uraan.

Asiasanat: urheilijat, syömiskäyttäytyminen, kuukautishäiriöt, urheiluvammat

Author

Suvi Ravi, MSc
Faculty of Sport and Health Sciences
University of Jyväskylä
Jyväskylä, Finland
suvi.m.ravi@jyu.fi
ORCID: 0000-0001-9706-5449

Supervisors

Professor Jari Parkkari, MD, PhD
Faculty of Sport and Health Sciences
University of Jyväskylä
Jyväskylä, Finland

Medical Director Maarit Valtonen, MD, PhD
Finnish Institute of High Performance Sport KIHU
Jyväskylä, Finland

Adjunct Professor Benjamin Waller, PhD
Sports Science Department
Physical Activity, Physical Education, Health and Sport
Research Centre (PAPESH)
Reykjavik University
Reykjavik, Iceland

Professor emeritus Urho M. Kujala, MD, PhD
Faculty of Sport and Health Sciences
University of Jyväskylä
Jyväskylä, Finland

Reviewers

Specialist in Obstetrics and Gynecology and
Reproductive Medicine & Clinical Lecturer
Kaisu Luiro-Helve, MD, PhD
University of Helsinki & Helsinki University Hospital
Helsinki, Finland

Specialist in Pediatric Surgery and Adolescent
Psychiatry
Satu-Liisa Pauniahho, MD, PhD, Docent
Wellbeing Services County of Pirkanmaa
Tampere, Finland

Opponent

Chief Physician Arja Uusitalo, MD, PhD, Docent
Helsinki University Hospital Diagnostic Center &
Clinic for Sports and Exercise Medicine,
Foundation for Sports and Exercise
Helsinki, Finland

ACKNOWLEDGMENTS

This dissertation would not have been possible without the support and invaluable help of other people. First, I want to express my deepest gratitude to my supervisors, Professor Jari Parkkari, Medical Director Maarit Valtonen, Adjunct Professor Benjamin Waller, and Professor emeritus Urho Kujala. Thank you all for the guidance and support you have given me during my journey. Jari, thank you for always finding time to answer my questions, strengthening my trust in myself, and sharing your extensive expertise with me. It has been a pleasure to work under your encouraging supervision. Maarit, I appreciate your clinical experience working with female athletes who are facing challenges with issues pertaining to eating and menstrual function, and the passion you have toward the topic of my dissertation. Thank you for sharing thoughts with me and helping me to understand the clinical value of our findings. Ben, I admire your acuity and expertise in sports injury research. I am also grateful for the meticulous and sharp-eyed comments you have given me and for helping me with grammatical issues. Finally, Urho, I admire the extensive expertise and knowledge you have. Thank you for having faith in me from the day I expressed my desire to start this journey, for helping me at the beginning of my academic career, and for the possibilities you created and discovered that made this dissertation possible.

I also want to express my sincere thanks to Senior Lecturer Johanna Ihalainen for her unofficial supervision along the way. I truly appreciate all the help and support you have given me throughout this journey. Your constructive and encouraging comments and the time you have given me have eased this journey and helped me when facing challenges. I am also grateful for the privilege of using the Female Athlete 2.0 data for my dissertation.

I also wish to express my gratitude to Associate Professor Sami Kokko and Associate Professor Eija Laakkonen for offering me the opportunity to utilize the data from the Finnish Health Promoting Sports Club study and from the Estrogenic Regulation of Muscle Apoptosis study, respectively, in this dissertation and for their support when writing the manuscripts. I am also thankful to both Professor emeritus Ari Heinonen, a member of my steering group and a former Dean of the Faculty of Sport and Health Sciences, and to Professor Sarianna Sipilä, the Dean of the Faculty of Sport and Health Sciences, for the possibility of conducting my research in the faculty. Sarianna, thank you also for the constructive comments when writing my second article. I also want to thank all the other co-authors I have been privileged to work with. Your contributions have improved the quality of the studies included in this dissertation. My sincere thanks also go to all the study participants without whom this dissertation would not have been possible.

I am also thankful to the official reviewers of this dissertation, Specialist in Obstetrics and Gynecology and Reproductive Medicine, Clinical Lecturer Kaisu Luiro-Helve and Specialist in Pediatric Surgery and Adolescent Psychiatry Satu-Liisa Pauniahho, whose valuable comments significantly helped me to improve the dissertation. Sincerely thanks also to Chief Physician Arja Uusitalo for

agreeing to be the opponent in the public defense of my dissertation. Moreover, I want to express my gratitude to Senior Lecturer Anne Viljanen for the scientific editing of the dissertation, and Editor from Scribendi for the language editing.

I also had the honor of being granted a full-time doctoral researcher position by the Faculty of Sport and Health Sciences at the University of Jyväskylä, an opportunity for which I am deeply thankful.

Throughout this journey, I have been fortunate to have had the opportunity to share thoughts, engage in meaningful discussions, and get help and support from several co-workers. My sincere gratitude goes to Doctoral Researchers Anna Lee, Maija Puupponen, Katja Rinta-Antila, Tuula Aira, Hanna Juppi, Jari Karppinen and Heidi Pesonen, Postdoctoral Researchers Lotta Palmberg and Tiina Savikangas, and all other co-workers who have contributed to my dissertation journey. Your help and peer support have been a source of strength and inspiration, and this journey would not have been the same without you.

There is also life outside the academic world. I am always grateful to my family and friends for being in my life, supporting me, and bringing joy to my life. My heartfelt gratitude goes to my parents and sister for their trust and support before and during my dissertation journey. Maria, our longstanding friendship has been a source of comfort and support, and I am grateful for your presence in my life. I also want to thank Tuija for our regular morning walks and conversations that have provided me much-needed emotional support and time to breathe. Furthermore, I would like to extend my sincere appreciation to all my other friends for bringing joy and support to my life during these dissertation years. Finally, Jani, thank you for your presence in my life over these years. You have helped me view things from a different angle and reminded me that life does not always have to be taken so seriously. I am more than happy that I can share my life with you.

Jyväskylä 11.5.2023

Suvi Ravi

ORIGINAL PUBLICATIONS AND AUTHOR CONTRIBUTION

This thesis is based on the following original publications, which will be referred to by their Roman numbers.

- I Ravi, S., Waller, B., Valtonen, M., Villberg, J., Vasankari, T., Parkkari, J., Heinonen, O. J., Alanko, L., Savonen, K., Vanhala, M., Selänne, H., Kokko, S., & Kujala, U. M. (2021). Menstrual dysfunction and body weight dissatisfaction among Finnish young athletes and non-athletes. *Scandinavian Journal of Medicine & Science in Sports*, 31(2), 405–417. <https://doi.org/10.1111/sms.13838>
- II Ravi, S., Kujala, U. M., Tammelin, T. H., Hirvensalo, M., Kovanen, V., Valtonen, M., Waller, B., Aukee, P., Sipilä, S., & Laakkonen, E. K. (2020). Adolescent sport participation and age at menarche in relation to midlife body composition, bone mineral density, fitness, and physical activity. *Journal of Clinical Medicine*, 9(12), Article 3797. <https://doi.org/10.3390/jcm9123797>
- III Ravi, S., Ihalainen, J. K., Taipale-Mikkonen, R. S., Kujala, U. M., Waller, B., Mierlahti, L., Lehto, J., & Valtonen, M. (2021). Self-reported restrictive eating, eating disorders, menstrual dysfunction, and injuries in athletes competing at different levels and sports. *Nutrients*, 13(9), Article 3275. <https://doi.org/10.3390/nu13093275>
- IV Ravi, S., Valtonen, M., Ihalainen, J. K., Holopainen, E., Kosola, S., Heinonen, S., Waller, B., Kujala, U. M., & Parkkari, J. (2023). Eating behaviours, menstrual history, and the athletic career: A retrospective survey from adolescence to adulthood in female endurance athletes. *BMJ Open Sport & Exercise Medicine*, 9(1), Article e001489. <https://doi.org/10.1136/bmjsem-2022-001489>

As the first author of the original publications, considering the comments from the co-authors, I drafted the study questions, and performed the statistical analysis independently or with help from the statistician. I also took the main responsibility for data interpretation and writing the manuscripts. With the help of my supervisors, I prepared and conducted the data collection for the Menstrual Function, Eating Attitudes, Body Image, and a Sports Career (MEBS) study, the data used in Study IV. I also participated in developing the research questionnaire used in the Female Athlete 2.0 study, the data which was used in Study III. In Studies I and II, I was privileged to use pre-existing data.

FIGURES

FIGURE 1	The spectrums of energy availability, menstrual status, and bone health in the Female Athlete Triad model. Modified from Nattiv et al. (2007).	28
FIGURE 2	The health consequences in the Relative Energy Deficiency in Sport (RED-S) model. Note that psychological problems can either precede or be the result of RED-S. Modified from Mountjoy et al. (2014).	29
FIGURE 3	The potential performance consequences in the Relative Energy Deficiency in Sport (RED-S) model. Modified from Mountjoy et al. (2014).	30
FIGURE 4	Anatomy and function of the hypothalamus–pituitary–ovarian axis. Modified from Bulun (2020). GnRH = gonadotropin-releasing hormone; LH = luteinizing hormone; FSH = follicle-stimulating hormone.	37
FIGURE 5	An overview of selected factors affecting GnRH secretion from GnRH neurons in women within the fertility age. The main regulator of GnRH neurons is kisspeptin secreted from kisspeptin neurons, which stimulates (+) GnRH secretion. In addition, other factors stimulate or inhibit (-) GnRH secretion directly or indirectly via kisspeptin neurons. Estrogen can stimulate (via kisspeptin neurons located in the arcuate nucleus of the hypothalamus) or inhibit (via kisspeptin neurons located in the anteroventral periventricular nucleus of the hypothalamus) GnRH secretion. GnRH = gonadotropin-releasing hormone; NPY = neuropeptide Y; AgRP = agouti-related peptide; GABA = γ -aminobutyric acid; α -MSH = α -melanocyte-stimulating hormone.	38
FIGURE 6	Continuum of menstrual dysfunction. Modified from De Souza (2003).	40
FIGURE 7	The time points in the athletes' lifespan when each study was conducted. The time between puberty and menopause marks the reproductive period in a female's life. FHPSC = the Finnish Health Promoting Sports Club study; MEBS = the Menstrual Function, Eating Attitudes, Body Image, and a Sports Career study; ERMA = the Estrogenic Regulation of Muscle Apoptosis study.	54
FIGURE 8	Summary of selected associations of low energy availability-related conditions, age at menarche, and sports participation with health- and sports career-related factors found in this study. The two-way arrows indicate an association where no temporal precedence could not be shown, while in the associations marked with a one-way arrow, a predictive variable occurred before an outcome variable. BMD = bone mineral density; BMI = body mass index.	92

TABLES

TABLE 1	Summary of the datasets, study designs, and participants in Studies I-IV.....	54
TABLE 2	Summary of the study variables.....	58
TABLE 3	Descriptive characteristics and covariates used in this dissertation study.....	59
TABLE 4	Background characteristics of the participants from the different datasets used in this study.....	69
TABLE 5	Menstrual cycle-related characteristics of the participants from the different datasets used in this study.....	70
TABLE 6	Age, body mass index (BMI), and training hours of the athletes in Study III classified according to their competition level, age, and type of sport.....	72
TABLE 7	Low energy availability-related conditions in athletes and non-athletes in the Finnish Health Promoting Sports Club study baseline (FHPSC1), Finnish Health Promoting Sports Club study follow-up (FHPSC2), Estrogenic Regulation of Muscle Apoptosis study (ERMA), Female athlete 2.0 study (FA 2.0) and Menstrual Function, Eating Attitudes, Body Image, and a Sports Career study (MEBS).....	74
TABLE 8	Eating disorder examination questionnaire short form (EDE-QS) scores of the athletes and non-athletes in Study IV.....	75
TABLE 9	Proportion of athletes reporting current or past eating disorder (ED), restrictive eating (RE), and menstrual dysfunction (MD) by participation level, age, and type of sports in Study III.....	75
TABLE 10	Attitudes toward weight in adolescent athletes and non-athletes by different weight groups in Study I.....	76
TABLE 11	Attitudes toward weight in young adult athletes and non-athletes by different weight groups in Study I.....	77
TABLE 12	Midlife health-related physical fitness components and bone health of participants according to their age at menarche (AAM) in Study II.....	78
TABLE 13	Correlation coefficients and their p-values between the physical activity level at age 13-16 and other age categories in Study II.....	79
TABLE 14	Midlife characteristics of participants according to physical activity level at age 13-16 in Study II.....	80
TABLE 15	Frequencies and proportions of athletes with one or more injuries during the preceding year and athletes with ≥ 22 missed participation days due to injuries during the preceding year along with crude odds ratios by eating disorders (ED), restrictive eating (RE), and menstrual dysfunction (MD) in Study III.....	81

TABLE 16	Body mass index (BMI), training volume, and amount of moderate-to-vigorous physical activity (MVPA) among participants reporting and not reporting current or past eating disorder (ED), restrictive eating (RE), and menstrual dysfunction (MD) in Studies I and III.	82
TABLE 17	Associations of eating behaviors and menstrual history with sports career length, participation level, injury-related harms during the sports career, and injury-related sports career termination in Study IV.	83

ABBREVIATIONS

AAM	Age at menarche
ACL	Anterior cruciate ligament
ACOG	American College of Obstetricians and Gynecologists
ALMI	Appendicular lean mass index
BED	Binge-eating disorder
BMD	Bone mineral density
BMI	Body mass index
CI	Confidence interval
DE	Disordered eating
DSM	Diagnostic and Statistical Manual of Mental Disorders
DXA	Dual-energy X-ray absorptiometry
EA	Energy availability
EB	Energy balance
ED	Eating disorder
EDE-Q	Eating Disorder Examination Questionnaire
EDE-QS	Eating Disorder Examination Questionnaire short form
ERMA	Estrogenic Regulation of Muscle Apoptosis (study)
FFM	Fat-free mass
FHPSC	Finnish Health Promoting Sports Club (study)
FSH	Follicle-stimulating hormone
GEE	Generalized estimation equation
GnRH	Gonadotropin-releasing hormone
HPO	Hypothalamus–pituitary–ovarian
ICD	International Statistical Classification of Diseases and Related Health Problems
IGF-1	Insulin-like growth factor 1
IOC	International Olympic Committee
kcal	Kilocalorie
KNDy	Kisspeptin/neurokinin B/dynorphin
LBM	Lean body mass
LH	Luteinizing hormone
MAD	Mean amplitude deviation
MD	Menstrual dysfunction
MEBS	Menstrual Function, Eating Attitudes, Body Image, and a Sports Career (study)
MET	Metabolic equivalent of task
MVPA	Moderate-to-vigorous physical activity
OR	Odds ratio
OSFED	Other specified feeding or eating disorders
RED-S	Relative Energy Deficiency in Sport
Triad	Female Athlete Triad
WHO	World Health Organization

CONTENTS

ABSTRACT

TIIVISTELMÄ (ABSTRACT IN FINNISH)

ACKNOWLEDGMENTS

ORIGINAL PUBLICATIONS AND AUTHOR CONTRIBUTION

FIGURES

TABLES

ABBREVIATIONS

CONTENTS

1	INTRODUCTION	17
2	REVIEW OF THE LITERATURE	19
2.1	Energy needs of female athletes	19
2.1.1	Energy balance and energy availability	19
2.1.2	Reasons for low energy availability in female athletes	22
2.1.2.1	Eating disorders and disordered eating behaviors.....	22
2.1.2.2	Undereating without an eating disorder/disordered eating	25
2.1.3	Acute and chronic consequences of low energy availability ..	26
2.1.4	The Female Athlete Triad and Relative Energy Deficiency in Sport.....	27
2.2	Female endocrinology	31
2.2.1	Endocrine system and the hypothalamus-pituitary-ovarian axis.....	31
2.2.1.1	Female sex steroids.....	32
2.2.1.2	Ovaries and follicle development	33
2.2.1.3	The menstrual cycle	34
2.2.1.4	Regulation of the hypothalamus-pituitary-ovarian axis.....	36
2.2.2	Dysfunction in the hypothalamus-pituitary-ovarian axis caused by low energy availability	38
2.2.3	Endocrinological development in females during the lifespan.....	40
2.2.3.1	Fetal and neonatal periods and childhood	40
2.2.3.2	Puberty	41
2.2.3.3	Adulthood, menopause, and postmenopause	44
2.3	Female athletic participation and health.....	46
2.3.1	Definitions of physical activity, exercise, athlete, and sports competition	46
2.3.2	Competitive sports during the female lifespan	47

2.3.3	A role of physical activity and competitive sports in female health.....	47
2.3.4	Injuries among female athletes	48
2.3.4.1	Association of low energy availability and other lifestyle-related factors with injuries	49
2.3.4.2	Effects of injuries on a sports career and later life	50
3	AIMS OF THE STUDY	52
4	METHODS	53
4.1	Datasets and participants	53
4.1.1	Finnish Health Promoting Sports Club study (Study I).....	55
4.1.2	Estrogenic Regulation of Muscle Apoptosis study (Study II)	55
4.1.3	Female Athlete 2.0 study (Study III).....	56
4.1.4	Menstrual Function, Eating Attitudes, Body Image, and a Sports Career study (Study IV)	56
4.2	Ethics.....	57
4.3	Measurements	58
4.3.1	Eating problems and body perceptions.....	59
4.3.2	The menstrual cycle and puberty	60
4.3.3	Injuries	61
4.3.4	Sports career.....	62
4.3.5	Bone mineral density and physical fitness components	64
4.3.6	Descriptive measurements and covariates.....	64
4.4	Statistical analyses	66
5	OVERVIEW OF THE RESULTS.....	68
5.1	Participant characteristics.....	68
5.2	Prevalence of low energy availability-related conditions among athletes and non-athletes	72
5.3	Associations between age at menarche and midlife characteristics..	77
5.4	Associations between sports participation in adolescence and midlife characteristics.....	79
5.5	Factors associated with eating problems, body weight perceptions, and menstrual dysfunction.....	81
5.5.1	Cross-sectional differences and associations	81
5.5.2	Longitudinal associations	83
6	DISCUSSION	84
6.1	Prevalence of low energy availability-related conditions among athletes.....	84
6.1.1	Attitudes toward eating and body weight perceptions	84
6.1.2	Menstrual dysfunction	87
6.2	Between-group comparisons	88
6.2.1	Athletes and non-athletes	88
6.2.2	Lean sport and non-lean sport athletes.....	89

6.2.3	Age and level of participation.....	90
6.3	Associations	91
6.3.1	Age at menarche and midlife characteristics	92
6.3.2	Competitive sports participation and midlife characteristics.....	93
6.3.3	Factors associated with eating disorders, disordered eating, and body weight dissatisfaction	95
6.3.4	Factors associated with menstrual dysfunction	96
6.4	Methodological considerations.....	98
6.5	Implications	100
6.6	Future directions.....	101
7	MAIN FINDINGS AND CONCLUSIONS.....	103
	YHTEENVETO (SUMMARY IN FINNISH)	105
	REFERENCES.....	108
	ORIGINAL PAPERS	

1 INTRODUCTION

Female participation in sports has constantly increased over the last few decades. In 1976, 20% of the athletes participating in the Olympic Games were women, while the figure in 2020 was 48% (International Olympic Committee [IOC], 2021). In Finland, 32% of female upper secondary school students vs 45% of males participated in activities organized by sports clubs in 2020, while a higher proportion of females (36%) than males (19%) participated in activities organized by third companies (Kokko et al., 2021).

While it is known that regular physical activity and sports participation are beneficial for females' overall health (Booth et al., 2012; Ekelund et al., 2019; Poitras et al., 2016), some female athletes present with menstrual dysfunction (MD), such as absence of menstrual bleeding or long and irregular cycles (Mountjoy et al., 2014; Nattiv et al., 2007). This can be a result of low energy availability (EA), that is, inadequate energy intake in relation to exercise energy expenditure (Loucks et al., 2011). Low EA, in turn, can result from clinical eating disorder (ED), subclinical disordered eating (DE) behaviors, energy intake restriction without ED/DE, or inadvertent undereating (Williams et al., 2017).

Some factors that are specific to the sports context are considered risk factors for ED/DE. These include assessment of body mass and composition, injuries and other events leading to time away from training, early specialization, weight cycling, pressures to change body weight or composition, and performance pressures (Arthur-Cameselle et al., 2017; Bratland-Sanda & Sundgot-Borgen, 2013; Sundgot-Borgen, 1994; K. R. Wells et al., 2020). In addition to these sports-specific risk factors, the internalization of a thin and fit body shape as the current societal standard of attractiveness in Western countries is causing pressures on many girls and women (Neighbors & Sobal, 2007; Stice & Shaw, 2002; Thompson & Stice, 2001). This thin-ideal internalization and pressure to achieve or maintain certain body weight or shape can lead to body dissatisfaction, which is a well-known risk factor for ED/DE (Phelps et al., 1999; Rohde et al., 2015; Stice & Shaw, 2002).

In addition to MD, low EA can result in many other health and performance issues. The health problems related to low EA include, but are not limited to,

impairments in bone health, endothelial dysfunction, issues with growth and development in children and adolescents, and an increased risk of illnesses (Mountjoy et al., 2018). Additionally, low EA has been associated with an elevated injury risk (Mountjoy et al., 2018); however, the evidence regarding the association between low EA and non-bone injuries is somewhat conflicting. Moreover, the effects of problems in eating and menstrual function on a sports career have yet to be elucidated.

The aim of this dissertation research was to explore the prevalence of low EA-related conditions (ED/DE, MD, and body weight dissatisfaction) in Finnish female athletes. Furthermore, the aim was to investigate whether athletes and non-athletes and athletes of different ages and those competing at different levels and engaging in different sport types differ from each other regarding the prevalence of these low EA-related conditions. The final aim of this dissertation was to investigate the association of age at menarche (AAM) and sports participation during adolescence with health-related physical fitness components and bone health in middle age, as well as the associations of low EA-related conditions with injuries, health, and sports career.

2 REVIEW OF THE LITERATURE

2.1 Energy needs of female athletes

Adequate and appropriate nutrition is crucial for the health and performance of athletes (D. T. Thomas et al., 2016). The most important factor relating to nutrition in female athletes is sufficient energy intake and hydration (Holtzman & Ackerman, 2021). However, humans have no strong biological drive to compensate for the energy expended through exercise by eating. Therefore, athletes should plan their eating based on their nutritional needs rather than relying solely on appetite (Loucks, 2013). Achieving a body composition that is optimal for peak performance is challenging and needs to be carefully planned to prevent long-term health and performance issues (D. T. Thomas et al., 2016).

2.1.1 Energy balance and energy availability

When estimating the sufficiency of energy intake, the terms energy balance (EB) and energy availability (EA) are salient, with the latter being currently widely used in studies conducted on athletes. EB, used in the field of dietetics, is calculated as energy intake minus total energy expenditure, which consists of resting metabolic rate, dietary-induced thermogenesis, non-exercise activity thermogenesis, and exercise energy expenditure. In turn, EA, used in the field of exercise physiology, is calculated as energy intake minus exercise energy expenditure divided by fat-free mass (FFM; Loucks et al., 2011; Nattiv et al., 2007). When EB is negative, energy intake is lower than energy expenditure, leading to the use of the body's energy stores. Conversely, when EB is positive, energy is stored in the body's energy stores (Loucks et al., 2011). EA in turn refers to the dietary energy remaining after exercise training for other metabolic processes in the body and can be seen as an input for the body's physiological systems (Areta et al., 2021; Loucks et al., 2011).

In healthy young females, an EA of 45 kcal per kilogram of FFM or lean body mass (LBM) per day has been associated with EB (Loucks, 2013). However, EB provides no information on whether the body's physiological processes are functioning at a healthy level (Loucks et al., 2011). In energy-deficient conditions, resting and non-resting energy expenditure decreases, a phenomenon called adaptive thermogenesis (Müller & Bosy-Westphal, 2013). This may result in a zero EB and a stable weight, while the body's endocrine and metabolic systems function suboptimally (Areta et al., 2021). In contrast, the value of EA is unaffected by adaptive thermogenesis as it only takes into account energy intake and exercise energy expenditure. Thus, it is a more useful concept when assessing the sufficiency of energy intake in athletes (Loucks et al., 2011).

While the concept of EA may be simple in laboratory-based investigations, its assessment has some challenges in field-based settings. This is compounded by the lack of a gold standard method for measuring EA. Each component in the EA equation, i.e., energy intake, exercise energy expenditure, and FFM/LBM can be assessed in a number of ways and in different periods of assessments (Burke et al., 2018; Heikura et al., 2021). Moreover, measurements of these components of EA have substantial error rates (Burke et al., 2018). Briefly, energy intake is usually assessed via (weighted) dietary records (Burke et al., 2018), which are known to be prone to underreporting (and in some cases overreporting), as well as have difficulties in gaining a truthful picture of a long-term diet (Magkos & Yannakoulia, 2003). In studies calculating EA, exercise energy expenditure is usually assessed by heart rate monitors and training logs, accelerometers, or a combination of those (Burke et al., 2018); however, those methods can have substantial error rates (Murakami et al., 2016). Furthermore, there is no clear definition of what constitutes an exercise. As a result, different definitions have been used, ranging from purposeful exercise to all forms of physical activity (Burke et al., 2018). Finally, FFM/LBM can be assessed, for example, by dual-energy X-ray absorptiometry (DXA) or skinfold caliber (Heikura et al., 2021), but they require trained staff and may not be accessible to everyone. In addition, errors and differences exist between different techniques and machines (Burke et al., 2018).

It should also be emphasized that the definition of EA has evolved over time (Areta et al., 2021). At first, EA was defined as energy intake minus exercise energy expenditure relative to total body mass (Loucks & Callister, 1993). After that, it was noted that LBM was more relevant than fat mass with regard to EA, and thus, EA was reported relative to LBM (Loucks & Heath, 1994a, 1994b). Furthermore, the most current EA definition acknowledges that the value of exercise energy expenditure also includes the habitual energy expenditure (i.e., resting metabolic rate and non-exercise activity thermogenesis), which is subtracted from the exercise energy expenditure to assess the true energy cost of exercise (Areta et al., 2021; Loucks et al., 1998). These different formulas lead to different values of EA, thereby making the comparisons between studies challenging (Areta et al., 2021).

Studies focusing on EA have also used FFM and LBM interchangeably. LBM is different from FFM, as lipids in cellular membranes, in the central

nervous system, and in bone marrows are included in LBM, but not in FFM. However, the difference is small because the lipids included in LBM in females consist only about 3% of the total body weight (Janmahasatian et al., 2005). Thus, it can be assumed that studies using FFM in the EA equation are comparable to those using LBM.

Seminal work conducted by Dr. Loucks demonstrated that there is a specific threshold of EA (30 kcal/kg FFM/d) below which physiological functions are disturbed (Ihle & Loucks, 2004; Loucks & Heath, 1994a; Loucks & Thuma, 2003). Nevertheless, it has recently been suggested that no such strict threshold for EA exists (De Souza et al., 2019; Lieberman et al., 2018; Reed et al., 2015; Williams et al., 2015). In turn, it seems that there is individual variability in the level of EA at which the consequences of low EA exist, and no strict threshold that would apply to everyone exist (De Souza et al., 2019).

Indeed, variables such as gynecological age (the difference between chronological and menarcheal age), genetics, psychological factors, and within-day EB can explain individual variability in susceptibility to low EA (Fahrenholtz et al., 2018; Williams et al., 2017). However, none of these factors have been widely studied in the low EA literature. Some evidence regarding the association between susceptibility to low EA and gynecological age was found in a study conducted by Loucks (2006). This study indicated that luteinizing hormone (LH; discussed in section 2.2) pulse frequency was reduced and mean 24-h LH concentration was suppressed as a result of low EA among women with 5–8 years of gynecological age, but not those with 14–18 years of gynecological age. It should be noted, however, that this was a short-term study with low EA treatment lasting only five days. Moreover, it has been suggested that genetic factors may explain why some individuals are more vulnerable to low EA than others (Williams et al., 2017).

In addition to gynecological age and genetic factors, the role of psychological factors in susceptibility to low EA has yet to be elucidated. The question is challenging to study as it is difficult to differentiate psychological stress from metabolic stress (i.e., stress caused by low EA) (Pauli & Berga, 2010; Williams et al., 2017). Thus, more research is required to better understand this issue (Williams et al., 2017). Finally, it has been shown that in athletes with similar daily EA, more time spent in negative EB within the day was associated with disruptions in the menstrual cycle (Fahrenholtz et al., 2018). This indicates that within-day energy deficiency would be more harmful to athletes than high meal frequency regardless of similar daily EA. However, assessment of within-day EB is even more time consuming than assessment of daily EA.

In summary, EA is the amount of energy remaining for physiological processes after exercise training and is theoretically a rather simple concept for assessing the sufficiency of energy in athletes. However, measuring EA in free-living athletes requires resources and expertise and is susceptible to significant errors (Burke et al., 2018; Heikura et al., 2021). In addition, there is individual variation in the susceptibility to low EA. For these reasons, some scholars have suggested that assessing signs of low EA (e.g., menstrual dysfunction; MD) and

using different questionnaires assessing, for example, eating behaviors would give a better estimation of a true EA than direct measurements of EA (De Souza et al., 2019; Heikura et al., 2018).

2.1.2 Reasons for low energy availability in female athletes

Athletes can have low EA for a variety of reasons. Some athletes have a clinical eating disorder (ED), some have ED symptoms without fulfilling the criteria of an ED (i.e., disordered eating, DE), some restrict their caloric intake without ED/DE, and some undereat inadvertently (Williams et al., 2017). Indeed, eating attitudes and habits can be seen as a continuum ranging from adequate energy intake and healthy body image to a clinical ED (Shisslak et al., 1995; Sundgot-Borgen & Torstveit, 2010; K. R. Wells et al., 2020). In between, there are dieting behaviors ranging from healthy dieting (such as well-planned slow weight loss) to DE (Sundgot-Borgen & Torstveit, 2010). DE include pathogenic weight control methods, such as skipping meals, excessive exercise, laxative pill use, binge eating, and vomiting but do not meet the criteria of clinical EDs (Nattiv et al., 2007). An athlete can move back and forth on the continuum during the athletic career and between the seasons (K. R. Wells et al., 2020).

2.1.2.1 Eating disorders and disordered eating behaviors

EDs are serious mental disorders characterized by disrupted attitudes toward eating and body weight and shape (Treasure et al., 2020; World Health Organization [WHO], 2023). Two main international disease classifications have been used to classify EDs: the International Classification of Diseases and Related Health Problems (ICD) by the WHO and the Diagnostic and Statistical Manual of Mental Disorders (DSM) by the American Psychiatric Association. ED classification has evolved over time and has been different in the ICD and DSM classification systems. The ICD 10th revision (ICD-10), released in 1992 (Hirsch et al., 2016), has been criticized for being unsatisfactory as, for example, many patients do not fulfill the criteria of any specific ED and thus receive a diagnosis of “other” or “unspecified” ED (Uher & Rutter, 2012). Thus, many changes have been made to the diagnostic criteria of EDs in the ICD 11th revision (ICD-11). It is now better aligning with the DSM fifth edition (DSM-5) and seems to be more clinically useful than the ICD-10 (Claudino et al., 2019).

The most common EDs include anorexia nervosa, bulimia nervosa, and binge-eating disorder (BED) (Hay et al., 2023). The ICD-11 includes also feeding disorders (avoidant-restrictive food intake disorder (ARFID), pica (consumption of non-nutritive substances), and rumination-regurgitation disorder) and other specified feeding or eating disorders (OSFED; WHO, 2023). Anorexia nervosa is characterized by a fear of weight gain, a disturbed body image, and dietary restriction or other weight loss behaviors, such as purging or excessive physical activity (WHO, 2023; Zipfel et al., 2015). Bulimia nervosa is marked by episodes of binge eating and compensatory behaviors thereafter (T. D. Wade, 2019). BED

patients differ from bulimia nervosa patients with fewer compensatory behaviors, although they both share the purging tendency (Hilbert, 2019).

In addition to the above-mentioned EDs, recent literature has discussed whether orthorexia nervosa should be included within the ED spectrum (Bartel et al., 2020; Cena et al., 2019; Donini et al., 2022; Strahler & Stark, 2020). Orthorexia nervosa is characterized with a pathological obsession with high-quality food as well as rigid and inflexible rules pertaining to nutrition (Bartel et al., 2020; Donini et al., 2022). It differs from other EDs in that individuals with orthorexia nervosa typically do not have body image disturbances or fear of weight gain, and they focus on the quality rather than the quantity of food (Bhattacharya et al., 2022). However, as is the case in clinical EDs, also orthorexia nervosa is associated with impaired physical and mental health and decreased quality of life (Donini et al., 2022). Orthorexia nervosa may coexist with other EDs or lead to a development of a clinical ED (Donini et al., 2022).

EDs are associated with increased mortality and morbidity and decreased quality of life (Pohjolainen et al., 2016; van Eeden et al., 2021; van Hoeken & Hoek, 2020), although a recent investigation suggests that the mortality risk may not be higher than in other mental disorders (Tuohisto-Kokko et al., 2023). However, readmission rates seem to be higher among patients with ED treated in inpatient care than among those treated because of other mental disorders (Tuohisto-Kokko et al., 2023). EDs also have a high comorbidity burden. Over 70% of patients with ED report comorbidity, such as anxiety, mood and personality disorders, substance use, or posttraumatic stress disorder (Keski-Rahkonen & Mustelin, 2016; Udo & Grilo, 2019). In addition, EDs have been associated with somatic diseases. For example, anorexia nervosa, bulimia nervosa, and OSFED have been linked with autoimmune diseases (e.g., type 1 diabetes and celiac disease) with a bidirectional association, such that autoimmune disease increases the risk of subsequent ED, and ED in turn increases the risk of subsequent autoimmune disease (Hedman et al., 2019). Moreover, anorexia nervosa has been associated with fibromyalgia, cancer, anemia, and osteoporosis (Udo & Grilo, 2019), and BED with obesity, type 2 diabetes, hypertension, high cholesterol, and triglycerides (Keski-Rahkonen & Mustelin, 2016; Mustelin et al., 2015; Udo & Grilo, 2019).

The etiology behind ED is multifactorial. Genes play a role in the development of the disease (Bulik et al., 2016, 2022; Trace et al., 2013); however, genes alone do not predict who will suffer from an ED (Schaumberg et al., 2017). Instead, there is an interplay between genetic and environmental factors behind the development of an ED and it is probable that genetic factors may explain why some individuals will get affected and some do not when they experience the same environmental triggers (Bulik et al., 2016; Schaumberg et al., 2017; Trace et al., 2013). Risk factors of ED can be divided into predisposing factors, precipitating factors, and perpetuating factors (Bratland-Sanda & Sundgot-Borgen, 2013; Treasure et al., 2020) as well as into biological, psychological, psychosocial, and behavioral factors (Treasure et al., 2020). Biological factors include, for example, genetics and female gender along with obsessive-

compulsive or autistic spectrum traits in restrictive-type EDs. Examples of psychological factors include body dissatisfaction and alexithymia (difficulty to identify emotions experienced by oneself or others) as well as some personality traits such as perfectionism, rigidity, and attention to detail in restrictive-type EDs and attention-deficit hyperactivity disorder (ADHD) traits in bulimic spectrum EDs. In addition, psychosocial factors include bullying, trauma, and thin-ideal internalization, and behavioral factors, for example, social isolation, weight control behaviors, and coping by avoidance or perfectionism (Bratland-Sanda & Sundgot-Borgen, 2013; Treasure et al., 2020; K. R. Wells et al., 2020).

Some risk factors are specific to the sports context, and it has been suggested that risk factors may be partly different between athletes and their non-athletic peers (Arthur-Cameselle et al., 2017). Sports-specific risk factors include coaching behavior, assessment of body mass and composition, injuries, illnesses, and other events leading to time away from training, early specialization, weight cycling, pressures to change body weight or composition, rules and regulations, performance pressures, teammate modeling of ED behaviors, and loss of coach (Arthur-Cameselle et al., 2017; Bratland-Sanda & Sundgot-Borgen, 2013; K. R. Wells et al., 2020).

Typically, girls and women are regarded as being more at risk of ED than boys and men in the general population (Striegel-Moore & Bulik, 2007; Weissman, 2019) and also in athletes (Bratland-Sanda & Sundgot-Borgen, 2013; Giel et al., 2016). The typical age of onset of an ED is in adolescence or early adulthood, although ED can begin before puberty or not until late adulthood (Schaumberg et al., 2017). While research regarding ED outside the typical age range is limited, there is evidence that late-onset EDs might be associated with less severe symptoms than EDs with the typical age of onset (Bueno et al., 2014). It has been shown that divorce, health issues, and menopause may increase the susceptibility to recurrence or onset of ED in middle-aged and older women (Kally & Cumella, 2008; Peat et al., 2008).

While EDs are severe illnesses affecting many athletes (e.g., Bratland-Sanda & Sundgot-Borgen, 2013), DE behaviors are even more common among athletes than clinical EDs (Reardon et al., 2019; K. R. Wells et al., 2020). ED behaviors can be defined as disturbed eating patterns that do not fulfill the criteria of clinical EDs and can include, for example, restrictive diets and pathogenic behaviors used to control weight, such as purging or excessive exercise (Reardon et al., 2019; K. R. Wells et al., 2020). While the health risks are more severe with regard to ED, DE can also result in health and performance problems (K. R. Wells et al., 2020). Furthermore, dieting and DE behaviors can lead to a clinical ED (Nattiv et al., 2007; K. R. Wells et al., 2020).

One commonly identified risk factor for ED/DE is body dissatisfaction, that is, negative subjective judgments of one's physical body (Phelps et al., 1999; Rohde et al., 2015; Stice & Shaw, 2002). The prevalence of body dissatisfaction in U.S. women is reported to range from 11% to 72% (Fiske et al., 2014). Indeed, some scholars have even suggested that body dissatisfaction is so common that

there is a “normative discontent” of body image (Matthiasdottir et al., 2012; Tantleff-Dunn et al., 2011).

A specific form of body dissatisfaction is body shape and body weight dissatisfaction, which refer to the discrepancy between current and idealized body shape and weight, respectively (Neighbors & Sobal, 2007). In Western countries, a thin and fit body shape is internalized as current societal standards of attractiveness and pursuing this body shape is placing pressure on many girls and women (Neighbors & Sobal, 2007; Schaefer et al., 2019; Stice & Shaw, 2002; Thompson & Stice, 2001). It has also been reported that a remarkable proportion of normal-weight women misclassify themselves as overweight and are trying to lose weight, indicating problems with body weight satisfaction (Chang & Christakis, 2003; Lowry et al., 2000; Matthiasdottir et al., 2012; Neighbors & Sobal, 2007).

The lifetime prevalence of clinical EDs among females in the general population is reported to be about 3–8% (Hay et al., 2023). However, it is probable that a significant portion of ED cases remain undetected in healthcare (Silén et al., 2021). Among female athletes, the ED prevalence rates are somewhat elusive and differ depending on the population studied and the assessment methods used (Bratland-Sanda & Sundgot-Borgen, 2013). However, some evidence shows that eating pathologies, including clinical EDs and subclinical DE behaviors, are more common among athletes than among non-athletes (Byrne & McLean, 2002; Martinsen & Sundgot-Borgen, 2013; Sundgot-Borgen & Torstveit, 2004; Torstveit et al., 2008). Nevertheless, there are also contradictory findings showing that problems with eating are more common in non-athletes than in athletes (Martinsen et al., 2010; Rosendahl et al., 2009). The evidence seems more consistent with the ED/DE risk being higher in sports that emphasize leanness, i.e., in endurance, gravitational, aesthetic, and weight-class sports (Mountjoy et al., 2018; Nattiv et al., 2007). It has also been suggested that characteristics associated with the development of ED may lead an individual to choose an athletic career (Schaumberg et al., 2017). However, more research is needed to confirm this.

2.1.2.2 Undereating without an eating disorder/disordered eating

Athletes can also undereat without presenting ED/DE. One cause for low EA without ED/DE is purposeful weight loss without signs of DE (De Souza et al., 2014). An athlete can start dieting because of perceived performance improvements; however, it is possible that these weight loss habits will lead to ED/DE at least when these are conducted without proper supervision (Loucks, 2013; Sundgot-Borgen, 1994).

Another reason for undereating without ED/DE is inadvertent undereating (De Souza et al., 2014), which is characterized by the absence of conscious restriction of energy intake, but energy needs still do not meet energy requirements (Williams et al., 2017). The prevalence of this phenomenon and its contribution to low EA in athletes is currently unclear. It has been suggested that the practical and logistic challenges experienced by athletes can be one

explanation for inadvertent undereating (Williams et al., 2017). These may include, for example, lack of time, financial challenges, or access to appropriate meals during the day away from home or when traveling (Heaney et al., 2008). Another possibility is an emphasis on healthy eating, such as preferring low-energy-dense foods with high fiber and low fat content or avoiding carbohydrate-rich foods. These modifications in diet may lead to low EA, especially during intense training periods (A. Melin et al., 2016). Moreover, a significant proportion of athletes suffer from gastrointestinal complaints (Waterman & Kapur, 2012), which may change eating habits and contribute to reductions in EA.

2.1.3 Acute and chronic consequences of low energy availability

When EA is low, the body allocates the received energy into functions that are essential for survival, such as thermoregulation and cellular maintenance, and non-essential functions, such as growth and reproductive functions, are not prioritized (De Souza et al., 2019). In their study conducted in healthy, sedentary women, Loucks and Thuma (2003) found that a 5-day low EA with 30, 20, or 10 kcal/kg FFM per day reduced carbohydrate availability and plasma glucose concentrations, plasma insulin, triiodothyronine and leptin concentrations, and increased cortisol concentrations in a linear manner. Similarly, this intervention found that low EA affected bone markers, resulting in reduced plasma osteocalcin concentrations and serum type I procollagen carboxy-terminal propeptide, indicating suppressed bone formation (Ihle & Loucks, 2004). Low EA with 20 or 10 kcal/kg FFM per day was additionally found to be associated with suppressed LH pulse frequency and increased LH pulse amplitude, as well as decreased growth hormone and insulin-like growth factor 1 (IGF-1) concentrations (Loucks & Thuma, 2003). However, the above-mentioned studies were conducted in sedentary women, who are characterized by different bone characteristics, body composition, and training habits than their physically more active counterparts. Nevertheless, it has later been shown that the negative effects of short-term low EA on bone health are also observed in physically active women (Papageorgiou et al., 2017).

Consequently, chronic, long-term (months to years) low EA has been found to cause physiological malfunctions in numerous body systems, including skeletal, reproductive, cardiovascular, endocrine, gastrointestinal, renal, immunological, and central nervous systems (De Souza et al., 2014; Heikura et al., 2021; Mountjoy et al., 2018). The time needed to elicit these responses may depend at least on the magnitude of an energy deficit, as well as an individual's characteristics, as discussed in subsection 2.1.1, and remains to be elucidated (Areta et al., 2021). The effects of low EA on bone health are both direct and indirect, as both energy deficiency and estrogen deficiency caused by low EA (for estrogen deficiency see section 2.2) have been found to be associated with impaired bone health with the presence of both exacerbating the negative bone effects (De Souza et al., 2008; Southmayd et al., 2017). While the mechanisms behind the association between low EA and impaired bone health are not yet fully understood

(Papageorgiou, Dolan, et al., 2018), it has been suggested that low EA alters the concentrations of some hormones, e.g., cortisol, growth hormone, insulin, IGF-1, triiodothyronine, and leptin, which in turn directly affects bone health (Nattiv et al., 2007; Papageorgiou, Dolan, et al., 2018; Popp et al., 2022). Alterations in those hormone levels contribute to the suppression of the hypothalamic-pituitary-ovarian (HPO) axis (see subsections 2.2.1-2.2.2), resulting in decreased levels of estrogen and further indirect impairments in bone health (Nattiv et al., 2007; Papageorgiou, Dolan, et al., 2018). In addition to low EA itself, a deficiency of some macro- and micronutrients, such as protein, calcium, and vitamin D, can impair bone health (Sale & Elliott-Sale, 2019). While low EA is always harmful to bone health, it is possible that an athlete's type of sport may affect the association between low EA and bone health. Indeed, there is some evidence that high- and odd-impact sports, such as soccer, volleyball, or gymnastics, may provide a higher osteogenic stimulus and thus mitigate the harmful impact of low EA on bone, at least up to a certain point (Hutson et al., 2021; Papageorgiou, Dolan, et al., 2018; Tenforde & Fredericson, 2011).

Dysfunctions in the reproductive system caused by low EA in females are addressed in subsection 2.2.2 and the consequences of low EA to other salient body systems are briefly discussed in the next subsection. Moreover, injuries in athletes, an issue partly related to low EA, are addressed in subsection 2.3.4.

2.1.4 The Female Athlete Triad and Relative Energy Deficiency in Sport

Two concepts have been developed to demonstrate the consequences of low EA. The first, the Female Athlete Triad (Triad), was launched in 1992 by the American College of Sports Medicine (Yeager et al., 1993). In the original version of the Triad, the following three components were included: DE, amenorrhea (absence of menarche at age 15 or missing three consecutive menses after menarche), and osteoporosis. Later, it was noticed that these three components are only the extremes of the spectrums of EA, menstrual function, and bone health, respectively, and the Triad was re-defined in 2007 (Nattiv et al., 2007). The re-defined Triad consists of three interrelated components: low EA with or without an ED, MD, and low bone mineral density (BMD). Each of these components presents along a continuum from the optimal situation to the unhealthiest extreme. In the optimal situation, EA is adequate, the menstrual cycle is regular, and bone health is optimal, in contrast to the unhealthiest extreme where EA is low, and amenorrhea and osteoporosis are present. The spectrums of EA, menstrual status, and BMD are presented in Figure 1. The black arrows on the left-hand side of the figure explain the interrelationships of the Triad components: low EA leads to amenorrhea, and bone health is impaired directly due to low EA by suppressing hormones that promote bone formation and indirectly via amenorrhea and related low estrogen concentrations (Nattiv et al., 2007) as presented in the previous subsection. In contrast, on the right-hand side of the figure, black arrows show that optimal EA supports menstrual function, and both optimal EA and regular menstrual cycle support optimal bone health. The gray arrows indicate

the continuum of EA, menstrual function, and bone health, along which an athlete can move depending on her diet and exercise habits (Nattiv et al., 2007).

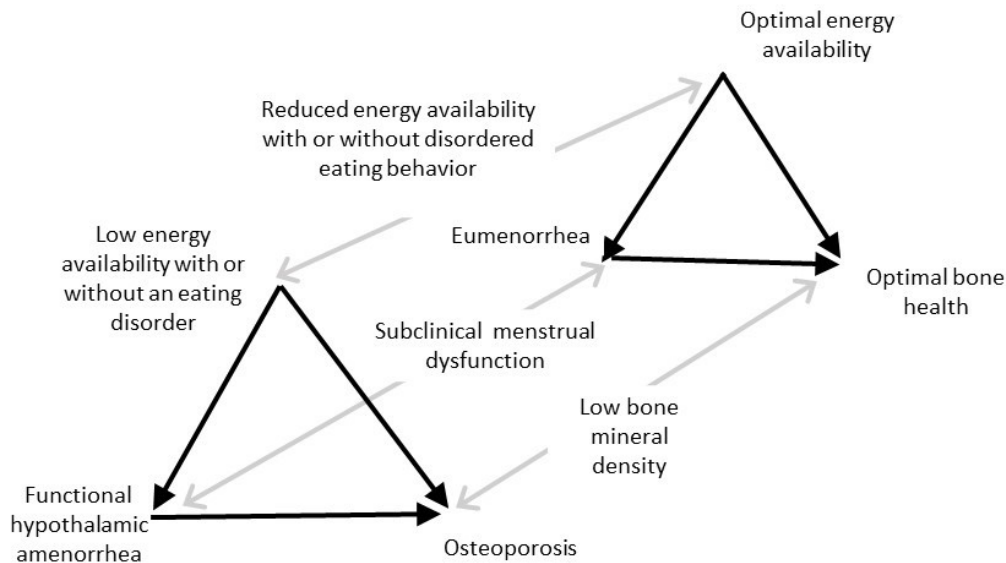


FIGURE 1 The spectrums of energy availability, menstrual status, and bone health in the Female Athlete Triad model. Modified from Nattiv et al. (2007).

In 2014, the International Olympic Committee (IOC) launched a new, more comprehensive and broader term for the syndrome: Relative Energy Deficiency in Sport (RED-S) (Mountjoy et al., 2014). In the RED-S model, similar to the Triad model, low EA is the underlying problem causing harmful effects on the body. However, the RED-S model emphasizes that in addition to the skeletal and reproductive systems, other body systems are affected by low EA. In addition, the RED-S model also includes male athletes and athletes with disabilities, and in addition to the health effects of low EA, it introduces the effects of low EA on performance (Mountjoy et al., 2014). The health and performance consequences of the RED-S model are presented in Figures 2 and 3, respectively. In addition to menstrual function and bone health presented already in the Triad model (Nattiv et al., 2007), according to the RED-S model, low EA affects i) the endocrine function by disrupting the function of hypothalamus-pituitary-gonadal axis, altering thyroid function, decreasing insulin and IGF-1 concentrations, decreasing growth hormone and cortisol concentrations, and altering the concentrations of appetite-regulating hormones, ii) the metabolic function by reducing resting metabolic rate, iii) the hematologic function by contributing iron deficiency, iv) growth and development in adolescents by increasing growth hormone resistance and increasing IGF-1/IGF binding protein-1 ratio, v) psychological health by lowering psychological well-being, vi) cardiovascular health by leading to endothelial dysfunction, unfavorable lipid profiles, and early atherosclerosis, vii) gastrointestinal health by resulting in, for example, delayed gastric emptying, increased intestinal transit time, and constipation, and viii)

immunological health by increasing the likelihood of illnesses (Mountjoy et al., 2018). Notably, in the RED-S model, psychological challenges can either precede or be the result of low EA. In addition to the health consequences in the RED-S model, low EA has been suggested to affect performance directly, for example, by impairing glycogen stores and protein synthesis or indirectly via injuries and illnesses, which make consistent and high-quality training difficult (Mountjoy et al., 2018).

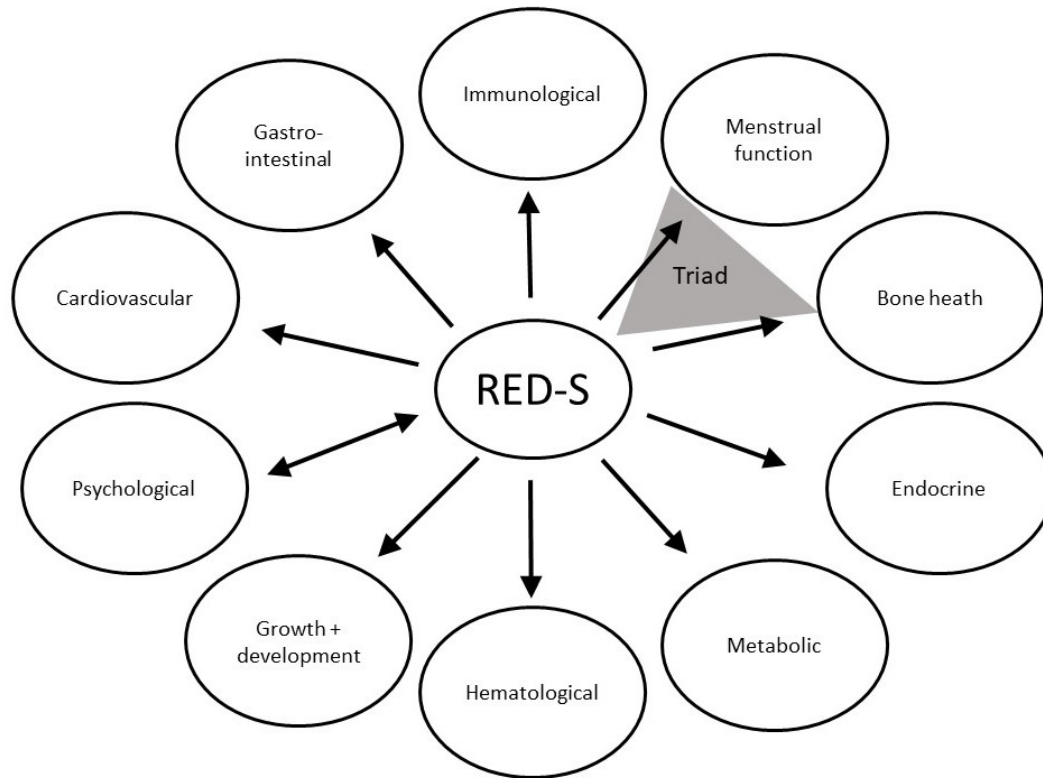


FIGURE 2 The health consequences in the Relative Energy Deficiency in Sport (RED-S) model. Note that psychological problems can either precede or be the result of RED-S. Modified from Mountjoy et al. (2014).

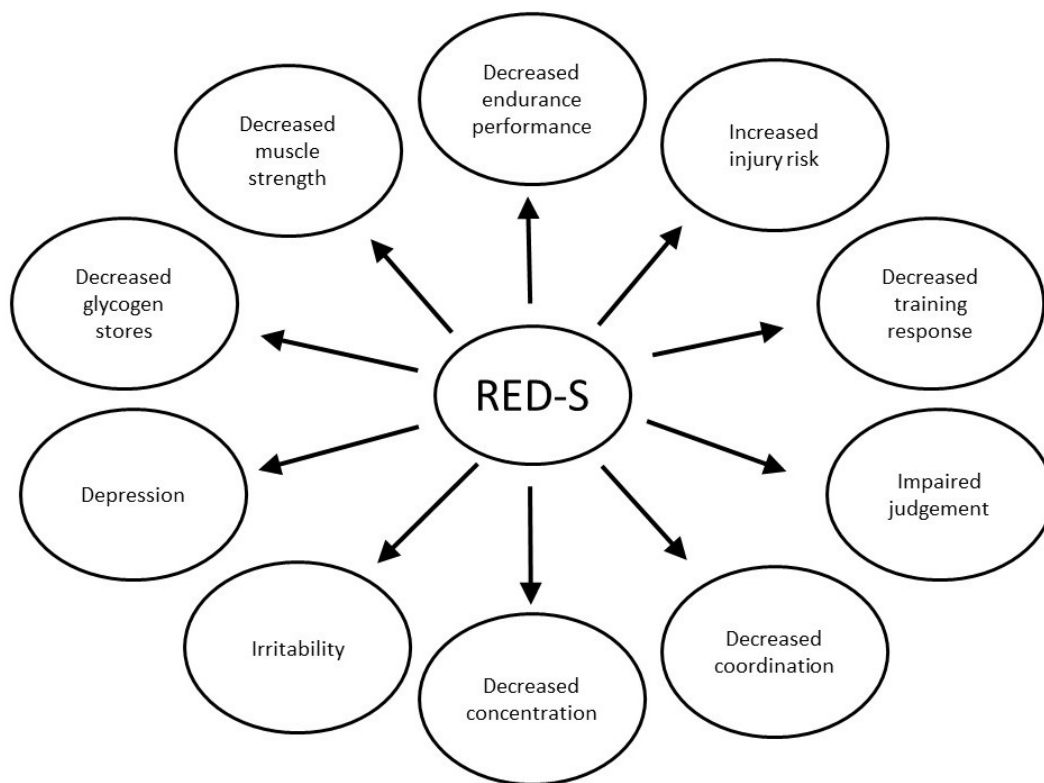


FIGURE 3 The potential performance consequences in the Relative Energy Deficiency in Sport (RED-S) model. Modified from Mountjoy et al. (2014).

The RED-S model was criticized by the Triad authors for not being as scientifically valid as the Triad and for oversimplification by presenting physiological systems, functions, and mechanisms independent of each other, while in fact they interact with each other (De Souza et al., 2014). Indeed, while there is strong evidence behind the Triad (De Souza et al., 2014; Warrick et al., 2020; Williams et al., 2019), some of the associations of the RED-S model still remain speculative, and more high-quality research conducted in different populations is needed (A. K. Melin et al., 2023; Mountjoy et al., 2018; Williams et al., 2019). However, the purpose of both the Triad model and the RED-S model is to call attention to the importance of adequate EA in order to prevent negative health outcomes associated with sports and exercise (Williams et al., 2019) and while the authors of the Triad and RED-S models disagree with each other to some extent (De Souza et al., 2014; Mountjoy, Sundgot-Borgen, et al., 2015; Williams et al., 2019), it has been stated that adversarial between the concepts should be avoided (Warrick et al., 2020).

2.2 Female endocrinology

2.2.1 Endocrine system and the hypothalamus–pituitary–ovarian axis

To maintain homeostasis in the organism, active and effective communication among the cells is needed (Deschenes & Keiichiro, 2005). The nervous and endocrine systems are mostly responsible for this communication (Deschenes & Keiichiro, 2005; Kovacs & Ojeda, 2011). The nervous system operates with electrochemical signals and functions by transmitting signals to peripheral organs and by receiving information from them (Kovacs & Ojeda, 2011). The signals of the nervous system are usually rapid and brief (Deschenes & Keiichiro, 2005). In contrast, the endocrine system functions by transporting chemical agents, i.e., hormones, in the bloodstream to their target organs (Kovacs & Ojeda, 2011). The responses of the endocrine system are typically slow and last longer than those of the nervous system (Deschenes & Keiichiro, 2005). However, the nervous and endocrine systems are closely connected. The nervous system innervates most of the endocrine organs but is also regulated by the endocrine system (Kovacs & Ojeda, 2011). Neuroendocrinology studies the interrelationships between nervous and endocrine systems, and its fundamental principle is that neurons secrete signal molecules into the circulation, and those signal molecules then regulate the secretion of hormones and expression of genes encoding these hormones (Lechan, 2020).

The core of the endocrine system is the hypothalamus–pituitary axis. The hypothalamic hormones are classified as stimulating and inhibiting hormones based on their actions in the pituitary. The pituitary is composed of two parts: anterior and posterior pituitary (i.e., adeno- and neurohypophysis, respectively) (Samson, 2011). The latter is responsible for the secretion of antidiuretic hormone and oxytocin. The anterior pituitary secretes hormones that are consequently affecting their target organs and further regulating, for instance, gonadal, thyroid, and adrenal function as well as growth and development (Samson, 2011).

The hypothalamus–pituitary–ovarian (HPO) axis is the main regulator of sex steroid production in females and involves the hypothalamus, pituitary, and ovaries. The signaling chain of the HPO axis begins from the hypothalamus, which secretes gonadotropin-releasing hormone (GnRH) into the portal circulation. GnRH, in turn, travels to the anterior pituitary and stimulates secretion of gonadotropins, i.e., follicle-stimulating hormone (FSH) and LH from the specialized cell types of the anterior pituitary (gonadotrophs) to the systemic circulation (Bulun, 2020; Hope & Hackney, 2017; Ruohonen et al., 2020). Gonadotrophs constitute approximately 7–15% of the anterior pituitary cells, and most of them are able to synthesize both LH and FSH (Bulun, 2020). GnRH is released from the hypothalamus in a pulsatile manner every 60–90 minutes, which in turn stimulates the secretion of LH and FSH from the pituitary approximately once every hour (Allaway et al., 2016). However, unlike LH, FSH secretion is not always pulsatile and when it is, the pulses are only partially concordant with LH pulses (Lechan, 2020). LH and FSH are, in turn, needed for

the secretion of sex steroid hormones, mainly estrogen and progesterone, from the ovaries (Ojeda, 2011).

The HPO axis is established in the first trimester in the uterus and becomes active in the second trimester (Bordini & Rosenfield, 2011). It is mostly responsible for the female's reproductive development and is active at three stages of development: fetal, neonatal, and adult (Bordini & Rosenfield, 2011; Colvin & Abdullatif, 2013). These developmental stages are discussed in more detail in subsection 2.2.3. Proper functioning of the HPO axis is vital for female's reproductive and overall health (Hope & Hackney, 2017).

2.2.1.1 Female sex steroids

As already mentioned, the two major biologically active sex steroid hormones in females are estrogen and progesterone (Hope & Hackney, 2017; Ojeda, 2011). Sex steroids in the body are initially synthesized from cholesterol derived from food or synthesized in the body in the mitochondria (Ojeda, 2011). The term estrogen refers to a group of hormones, which include estrone, estriol, and estradiol (Hope & Hackney, 2017) as well as estetrol (Fruzzetti et al., 2021). Estradiol, the most potent estrogen in women within the fertility age, is the major estrogen produced by the ovaries (Wierman, 2007); however, estrone is also secreted by the ovaries (Riggs et al., 2002). In the ovaries, both estradiol and estrone are produced from androgens by the action of the aromatase enzyme, which is stimulated by FSH (Hope & Hackney, 2017; Ojeda, 2011). In addition to ovarian production, estradiol is also produced in peripheral tissues, such as subcutaneous fat, skin, and brain (Bulun, 2020). Estrone is also produced peripherally, mainly from androstenedione (one of the androgens), which is secreted from the ovaries and the adrenal gland and is converted into estrone by the aromatase enzyme. Estrone is further converted to estradiol in peripheral tissues (Bulun, 2020; Ojeda, 2011). Estriol can be synthesized from both estradiol and estrone in the liver, and it is the main estrogen in pregnancy (Ali et al., 2017; Ding & Zhu, 2016). Estetrol, in turn, is synthesized from estradiol and estriol and produced only during pregnancy in the liver of the fetus (Fruzzetti et al., 2021). About 70% of estrogens in the bloodstream are bound to their transport proteins (albumin and sex hormone-binding globulin) (Ojeda, 2011; Riggs et al., 2002).

Another female sex steroid secreted by the ovaries is progesterone, a major progestogen. In addition to ovarian production, small amounts of progesterone are also secreted locally from peripheral tissues (Hope & Hackney, 2017; Ojeda, 2011; Wierman, 2007). LH stimulates the conversion of cholesterol via pregnenolone to progesterone in the ovary (Bulun, 2020; Hope & Hackney, 2017; Ojeda, 2011). Circulating progesterone is bound to corticosteroid-binding protein, also known as transcortin (Ojeda, 2011).

The female body also produces some amounts of androgens, such as androstenedione and testosterone, which can act on target tissues directly or after conversion to dihydrotestosterone. Androstenedione is not biologically active but is converted to testosterone and estrone in the ovary and peripheral tissues (Bulun, 2020). A significant portion of testosterone in the bloodstream is

synthesized to estradiol by the aromatase enzyme, but testosterone is also converted to dihydrotestosterone, the most potent androgen (Bulun, 2020; Ojeda, 2011). Unlike in the case of progesterone, only LH secreted from the pituitary stimulates testosterone secretion from the ovaries. However, FSH is needed to activate the aromatase enzyme complex. In females, almost all (97–99%) of the testosterone in the bloodstream is bound to sex hormone-binding globulin (Ojeda, 2011).

Female sex steroids are responsible for the development of primary and secondary sex characteristics (sexual characteristics that are present at birth and those appearing during puberty, respectively), but they also regulate a variety of other functions throughout the body, playing an important role in various physiological processes (Hope & Hackney, 2017). Estrogen and progesterone are needed to maintain the menstrual and uterine cycles (see subsection 2.2.1.3) (Ojeda, 2011). Estradiol is responsible for the proliferation of the uterine endometrium, while progesterone prepares the endometrium for embryo implantation (Bulun, 2020). Progesterone also plays an important role in the maintenance of pregnancy and preparation of milk secretion from the mammary gland (Graham & Clarke, 1997).

In addition to the above-mentioned fertility-related effects of sex steroids, estrogen affects many signaling pathways in the body and is thus important for female health (Hipólito Rodrigues & Carneiro, 2022; Rettberg et al., 2014). For instance, it affects the regulation of body fat and blood glucose levels, salt and water balance and oxygen levels in the cells (Kraemer et al., 2012). Moreover, estrogen prevents bone resorption and bone remodeling while maintaining bone formation (Cauley, 2015), thus, affecting bone health. Estrogen is also known to be associated with better vascular health (Usselman et al., 2016).

2.2.1.2 Ovaries and follicle development

Ovaries are responsible for sex steroid production as well as the production of germ cells (oocytes) (Ojeda, 2011). The ovary is composed of the outer cortex, the inner medulla, and the hilum. The outer cortex contains the follicles, the functional units of the ovary, and secretes sex steroids and is thus considered a functional component of the ovary (Hope & Hackney, 2017; Ojeda, 2011). The number of oocytes in the ovaries is fixed, being at highest during pregnancy and decreasing exponentially during the female's life. At week 20 of gestation, there are approximately seven million oocytes, and the number is decreasing to about two million at birth, further about 400 000 before puberty, and about 1000 at menopause (Coulombre & Russell, 1954; Faddy, 2000; Finch, 2014).

Two types of cells in the ovaries produce sex steroids, namely, granulosa and theca cells (Havelock et al., 2004). FSH receptors are located only on granulosa cells, which secrete estrogen. However, the formation of estrogen requires androgens as androgens are converted into androgens via aromatization (as discussed in subsection 2.2.1.1). Theca cells, stimulated by LH, synthesize androgens and are thus also needed in the formation of estrogen (Havelock et al., 2004; Ojeda, 2011).

The primordial follicles containing primary oocytes surrounded by a single layer of flattened epithelial (pre-granulosa) cells start to develop during the fetal stage of life (V. R. Araújo et al., 2014; Ojeda, 2011). The development of primary follicles from the primordial follicles occurs when the epithelial cells become cuboidal granulosa cells (Ojeda, 2011). During the reproductive age of a female's life, cohorts of primary follicles develop into the secondary (pre-antral) follicle, which has at least two layers of cuboidal granulosa cells surrounding a primary oocyte and a small number of theca cells separated from granulosa cells by a basal membrane and surrounding the granulosa cells (V. R. Araújo et al., 2014; Ojeda, 2011). The last two stages of follicle development are tertiary (antral) follicle and the Graafian (preovulatory) follicle. In the last stage, the follicle reaches its maximum size and contains a secondary oocyte (an oocyte after the first meiotic division) and is ready for ovulation (V. R. Araújo et al., 2014; Bulun, 2020; Ojeda, 2011).

The first stages of follicular development occur independent of gonadotropins and are suggested to be controlled by intraovarian mechanisms (Bulun, 2020). The development from antral to preovulatory follicle, however, requires the presence of FSH (Bulun, 2020; Hope & Hackney, 2017). The ovulatory follicle is one of a cohort recruited during the transition from the luteal phase to the follicular phase (Bulun, 2020; see the next subsection).

2.2.1.3 The menstrual cycle

Reproductive function in females is cyclic. It consists of fluctuating production of reproductive hormones (i.e., gonadotropins and sex steroids) over approximately 28 days and a series of events including rupture of an ovarian follicle and mature egg releasing from the ovary (i.e., ovulation). These interrelated series of events are called the menstrual cycle (Hope & Hackney, 2017; Ojeda, 2011). A normal menstrual cycle length varies between 21 and 35 days in healthy adult female (Elliott-Sale et al., 2021; Hope & Hackney, 2017). A regular menstrual cycle can be seen as a vital sign of a female's overall health and well-being (American College of Obstetricians and Gynecologists [ACOG], 2015). The menstrual cycle can be divided into two main phases, namely, the follicular and luteal phases, which are divided by ovulation (Allaway et al., 2016; Hope & Hackney, 2017).

The menstrual cycle begins from the first day of menstrual bleeding, the external sign of the menstrual cycle, which also starts the follicular phase of the cycle (Allaway et al., 2016; Hope & Hackney, 2017). Menstrual bleeding typically lasts 3–6 days, and the follicular phase is approximately 10–16 days (Allaway et al., 2016; Ojeda, 2011). The follicular phase ends with ovulation, which typically lasts typically about 36 hours (Ojeda, 2011). The last part of the menstrual cycle is the luteal phase, the length of which is usually 12–14 days but may range between 11–17 days (Practice Committees of the American Society for Reproductive Medicine and the Society for Reproductive Endocrinology and Infertility, 2021). While both follicular and luteal phases vary by length, most of

the variability in the cycle length results from the variability of the follicular phase (Ojeda, 2011; Waller et al., 1998).

The HPO axis is responsible for the regulation of the menstrual cycle, and the follicular and luteal phases are characterized by different hormonal milieus. During menstrual bleeding and the early follicular phase, serum FSH levels increase, while LH, estrogen, progesterone, and androgen levels are low (Ojeda, 2011). Because of the FSH secretion at the early follicular phase, a cohort of follicles (usually 3–11) starts to grow and secrete estrogen, thus increasing the estrogen levels (Allaway et al., 2016). The rise in FSH levels also increases the sensitivity of FSH receptors in the granulosa cells, thus increasing the effect of bioactive FSH (Allaway et al., 2016; Hope & Hackney, 2017). Typically, only one follicle per cycle will become dominant, while others undergo programmed apoptosis called atresia (Hope & Hackney, 2017; Ojeda, 2011). The selection of a dominant follicle occurs in the mid-part of the follicular phase, after which estrogen secretion of the dominant follicle becomes more rapid (Allaway et al., 2016; Mihm et al., 2011). The selection of the dominant follicle occurs based on a number of FSH receptors on its cell membrane, but the factors contributing to this selection have yet to be explored (Hope & Hackney, 2017; Ojeda, 2011). The highest FSH levels are observed at the time of dominant follicle emergence, after which it gradually starts to decline as a result of inhibin-B secretion (stimulated by FSH itself) at the same time that estrogen levels start to rise (Mihm et al., 2011). Plasma progesterone, androgen, and LH levels are low throughout the follicular phase (Ojeda, 2011).

Before ovulation, estrogen levels are at their highest, about nine-fold over the basal level (Ojeda, 2011). This stimulates gonadotropin, specifically LH secretion from the pituitary, typically 24–36 hours after peak estrogen secretion (Allaway et al., 2016; Ojeda, 2011). In response to this surge, the dominant follicle grows further, reaching its maximum diameter in 48 hours and is then called the Graafian (preovulatory) follicle (Ojeda, 2011). Ovulation is a complex series of events during which the follicle ruptures and the oocyte is released from the follicle (Allaway et al., 2016; Ojeda, 2011). LH is essential for stimulating the rupture of the mature follicle (Bulun, 2020). After the surge of gonadotropin secretion, estrogen levels fall, progesterone levels start to rise, and there is also a moderate rise in androgen levels (Ojeda, 2011).

After ovulation, the ruptured follicle develops into the corpus luteum, which marks the beginning of the luteal phase of the menstrual cycle (Allaway et al., 2016; Ojeda, 2011). The corpus luteum secretes large amounts of progesterone, and thus, the luteal phase is characterized by high levels of progesterone. The corpus luteum also secretes estrogens, androgens, and inhibin-A during the luteal phase of the cycle, while FSH and LH levels remain low until the late stage of the luteal phase (Ojeda, 2011). If no fertilization occurs, the corpus luteum remains functional for approximately 12–16 days (Bulun, 2020) after which it is degraded by proteolytic enzymes (Hope & Hackney, 2017; Ojeda, 2011). Without fertilization, progesterone levels start to decrease approximately 8–10 days after ovulation, reaching the lowest levels about 14 days after ovulation (Ojeda, 2011).

This decline in progesterone levels, as well as a decline in estrogen and inhibin levels, removes the negative feedback on the pituitary; thus, FSH levels start to rise approximately four days before menstrual bleeding, initiating follicular emergence for the follicular phase of the next menstrual cycle (Allaway et al., 2016; Bulun, 2020; Mihm et al., 2011).

During the menstrual cycle, the oocyte released from the ruptured follicle travels from the ovary to the uterus, and if it is not fertilized, it is shed during menstrual bleeding (Hope & Hackney, 2017). The uterus has its own cycle, which coincides with the ovarian cycle. The uterine cycle has a proliferative phase (during the follicular phase of the menstrual cycle) and secretory phase (during the luteal phase of the menstrual cycle) (Bulun, 2020). During the proliferative phase, circulating estrogen (produced mainly by the dominant follicle) causes thickening of the endometrium (Hope & Hackney, 2017) and by the end of the proliferative phase, the endometrium thickness has increased approximately three- to five-fold (Ojeda, 2011). After ovulation, increased levels of progesterone contribute to the secretion of glycogen from the endometrium and increase endometrium vascularization. These changes are necessary for embryonic implantation in the case of fertilization. In the absence of fertilization, the rapid decline in estrogen and progesterone levels at the latter part of the luteal phase causes constriction of the arteries supporting the endometrium, which further causes ischemia and necrotic epithelium of the endometrial tissue and thus leads to shrinking of the endometrium (Allaway et al., 2016; Hope & Hackney, 2017). This necrotic epithelium and blood are discharged during menstruation (Allaway et al., 2016).

2.2.1.4 Regulation of the hypothalamus–pituitary–ovarian axis

The function of the HPO axis is regulated by both negative and positive feedback. Estrogen, and in smaller amounts progesterone, secreted by the ovaries inhibits both GnRH secretion by the hypothalamus and gonadotropin secretion by the anterior pituitary. Negative feedback is also regulated by inhibin, which is secreted by the ovaries and controls the secretion of FSH (Ojeda, 2011). Inhibin-B secretion (in the early follicular phase of the menstrual cycle) is stimulated by FSH, and inhibin-A (in the late follicular phase and in the luteal phase) by LH (Bulun, 2020). Moreover, activin and follistatin from the pituitary control the secretion of gonadotropins: activin stimulates FSH production, while follistatin inhibits the action of activin (Bulun, 2020). High levels of estrogen and progesterone can also stimulate the secretion of GnRH and gonadotropins (positive feedback) (Bulun, 2020; Ojeda, 2011; Veldhuis & Weltman, 2005). A simplified scheme of the anatomy and function of the HPO axis is presented in Figure 4.

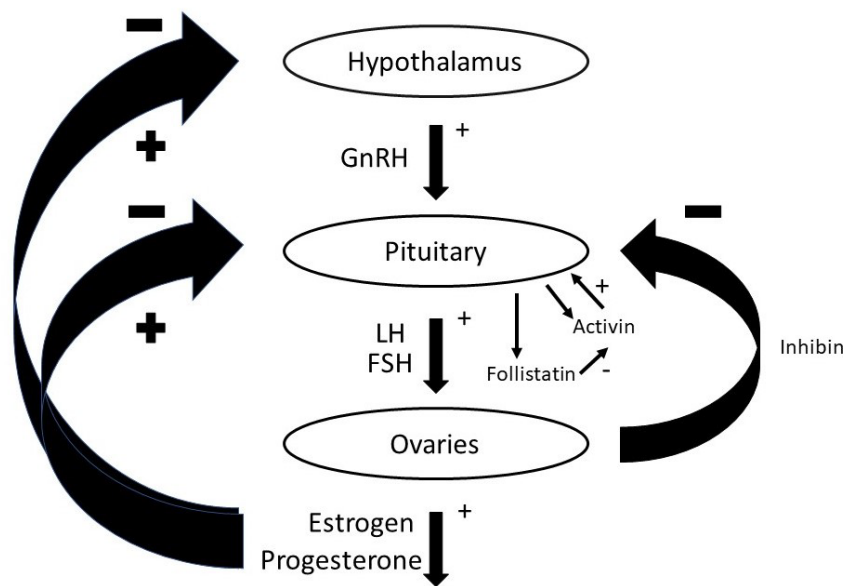


FIGURE 4 Anatomy and function of the hypothalamus–pituitary–ovarian axis. Modified from Bulun (2020). GnRH = gonadotropin-releasing hormone; LH = luteinizing hormone; FSH = follicle-stimulating hormone.

The regulation of the pulsatile GnRH secretion from GnRH neurons is complex, as many neurotransmitter systems convey information to GnRH neurons (Lechan, 2020). One of the most salient signaling systems combining metabolic and endocrine pathways is kisspeptin pathway, which provides information on the body’s metabolic status to GnRH neurons (Maeda et al., 2007; Pope et al., 2015; Tsukamura, 2022; Wahab et al., 2013). The synthesis and pulsatile secretion of GnRH is dependent on the secretion of hypothalamic peptide kisspeptin from kisspeptin neurons (Bulun, 2020; Iwasa et al., 2022). As kisspeptin neurons in the arcuate nucleus (one of the main hypothalamic nuclei that control food intake and GnRH secretion) also express two additional peptides, namely neurokinin B and dynorphin, these neurons are also called kisspeptin/neurokinin B/dynorphin (KNDy) neurons (Guzmán et al., 2019; Ikegami et al., 2022; Lechan, 2020). The actions of neurokinin B and dynorphin are opposite to each other, as the first-mentioned peptide stimulates, and the last-mentioned peptide inhibits kisspeptin secretion (Lechan, 2020). Kisspeptin is also suggested to contribute to other levels of the HPO axis, but the relevance of kisspeptin outside the hypothalamus is still undetermined (Ruohonen et al., 2020).

In addition to kisspeptin, other neuropeptides and neurotransmitters control GnRH secretion independently and via kisspeptin neurons. These include, for example, neuropeptide Y, α -melanocyte-stimulating hormone, γ -aminobutyric acid, glutamate, and noradrenaline (Allaway et al., 2016; Bulun, 2020; Goodman et al., 2022; Guzmán et al., 2019; Roa, 2013). Moreover, many peripheral signals modulate, that is, stimulate or inhibit, KNDy and other neurons affecting the function of the HPO axis (Guzmán et al., 2019; Iwasa et al., 2022). These include, but are not limited to, estrogen, progesterone, leptin, ghrelin, and adiponectin (Allaway et al., 2016; Budak et al., 2006; García-Galiano

et al., 2012; Gordon et al., 2017; Hill et al., 2008; Ikegami et al., 2022; Iwasa et al., 2022; Prashar et al., 2021; Wahab et al., 2013). For example, estrogen is known to inhibit kisspeptin release from KNDy neurons (i.e., negative feedback) (Ikegami et al., 2022; Uenoyama et al., 2021). It has been suggested, however, that estrogen can also act by stimulating kisspeptin release from the kisspeptin neurons located in the anteroventral periventricular nucleus (García-Galiano et al., 2012; Uenoyama et al., 2021). Leptin and ghrelin, in turn, the hormones secreted from the adipocytes and gastric cells, respectively, transform information on the metabolic status to the hypothalamus (e.g., Allaway et al., 2016; Wahab et al., 2013). Leptin concentrations are positively correlated with fat mass, and caloric deprivation has been linked to decreased leptin levels (Allaway et al., 2016; Boden et al., 1996; Chan & Mantzoros, 2005; Shimizu et al., 1997). While the exact mechanisms of the role of leptin in GnRH secretion stimulation are not well understood, recent evidence suggests that leptin modulates the secretion of kisspeptin and other neuropeptides and further stimulates GnRH secretion from GnRH neurons, which lack leptin receptors (Childs et al., 2021; Guzmán et al., 2019). Ghrelin levels, in contrast to leptin levels, are increased in the fasted state and stimulate appetite (Budak et al., 2006). Ghrelin plays an inhibiting role in GnRH secretion from the hypothalamus (Navarro & Kaiser, 2013). An overview of some selected factors contributing to the regulation of GnRH secretion is presented in Figure 5.

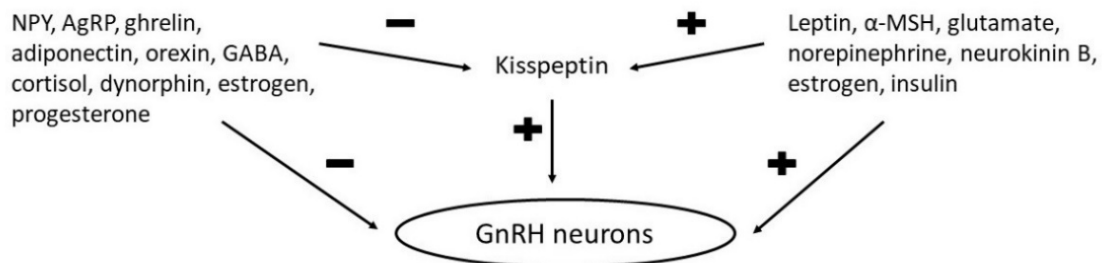


FIGURE 5 An overview of selected factors affecting GnRH secretion from GnRH neurons in women within the fertility age. The main regulator of GnRH neurons is kisspeptin secreted from kisspeptin neurons, which stimulates (+) GnRH secretion. In addition, other factors stimulate or inhibit (-) GnRH secretion directly or indirectly via kisspeptin neurons. Estrogen can stimulate (via kisspeptin neurons located in the arcuate nucleus of the hypothalamus) or inhibit (via kisspeptin neurons located in the anteroventral periventricular nucleus of the hypothalamus) GnRH secretion. GnRH = gonadotropin-releasing hormone; NPY = neuropeptide Y; AgRP = agouti-related peptide; GABA = γ -aminobutyric acid; α -MSH = α -melanocyte-stimulating hormone.

2.2.2 Dysfunction in the hypothalamus–pituitary–ovarian axis caused by low energy availability

The female's reproductive cycle, including ovulation, conception, pregnancy, and lactation, are highly energetically demanding activities (G. N. Wade et al.,

1996) and compared with males, the energy cost of reproduction in females is much higher (Caldwell & Hooper, 2017). While many environmental factors influence mammals' reproduction, food availability is the one of the most important factors (Bronson, 1985).

In the 1980s, it was noted that amenorrhea was associated with exercise training, with many female athletes being amenorrheic (Bullen et al., 1985; Loucks, 1990). Since then, much evidence has been accumulated on the mechanisms behind the disruption of the menstrual cycle. It is now known that when EA is low, the HPO axis is disturbed: the pulsatile secretion of GnRH from the hypothalamus is suppressed, leading to suppressed secretion of gonadotropins and especially reduced LH pulsatility from the pituitary. This consequently leads to the suppressed secretion of estrogen and progesterone from the ovaries (Gordon et al., 2017; Mallinson & De Souza, 2014). The suppression of GnRH pulsatility from the GnRH neurons in the hypothalamus is thought to be attributed to alterations in metabolism- and appetite-related factors and subsequent neuro-modulatory signals (Allaway et al., 2016; Gordon et al., 2017; Iwasa et al., 2018; Mallinson & De Souza, 2014; G. N. Wade et al., 1996). While the exact mechanisms behind this phenomenon are not fully understood, one of the most salient factors among others seems to be suppressed leptin levels and reduced kisspeptin expression (Childs et al., 2021; Iwasa et al., 2022).

Disorders in the menstrual cycle resulting from low EA range from clinical disorders, i.e., functional hypothalamic amenorrhea and oligomenorrhea, to sub-clinical disorders, i.e., anovulation and luteal phase deficiency (Figure 6) (De Souza & Williams, 2004). In the most severe form of disturbance, functional hypothalamic amenorrhea, the term "functional" indicates that amenorrhea is not caused by any organic reason but rather dysfunction in the HPO axis (Gordon et al., 2017). Amenorrhea can be classified as primary and secondary, with the first defined as a failure to attain menarche by the age of 15 years and the second as cessation of menses after menarche for at least 90 days (Elliott-Sale et al., 2021; Gordon et al., 2017; Nattiv et al., 2007). Oligomenorrhea is defined as long and inconsistent menstrual cycles occurring at intervals of 36–90 days, and the cycles can be ovulatory or anovulatory (Mallinson & De Souza, 2014; Nattiv et al., 2007). While clinical menstrual disorders are easily detectable, subclinical disorders require hormonal assessment as they do not cause any changes in menstrual bleeding (De Souza et al., 2010). In an anovulatory cycle, ovulation does not occur and progesterone is low during the luteal phase, while a luteal phase defect is characterized by a short luteal phase and/or insufficient progesterone concentration in the luteal phase (Mallinson & De Souza, 2014).

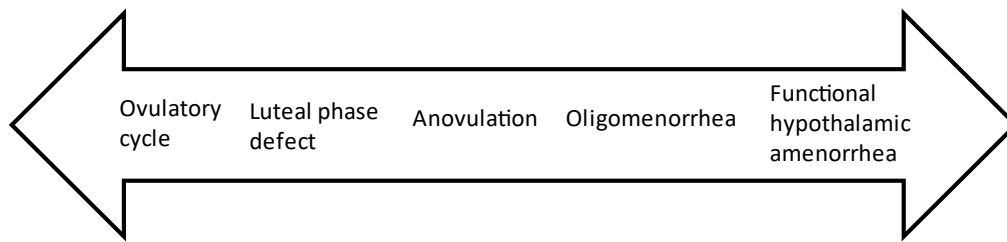


FIGURE 6 Continuum of menstrual dysfunction. Modified from De Souza (2003).

In addition to functional hypothalamic amenorrhea or other disturbances in the menstrual cycle due to low EA, absent or irregular menstrual cycles can result from other conditions. The most common differential diagnoses are pregnancy and polycystic ovary syndrome, while other conditions related to the hypothalamus, pituitary, ovaries, and uterus (e.g., tumors, irradiation, trauma, and surgery) or some diseases (e.g., hypo- or hyperthyroidism, Cushing syndrome, and Addison disease) can result in disturbances in the menstrual cycle (Gordon et al., 2017). Moreover, rare gene mutations may render some individuals more prone to functional hypothalamic amenorrhea (Caronia et al., 2011).

2.2.3 Endocrinological development in females during the lifespan

2.2.3.1 Fetal and neonatal periods and childhood

Differences between the sex hormone secretion of the HPO axis in females compared with the hypothalamus–pituitary–testicular axis in males during the fetal and neonatal periods of life contribute to sexual differentiation of behavior, metabolism, and neuroendocrine function in later life (Bordini & Rosenfield, 2011). Indeed, the HPO axis is active during the fetal stage and again during the first months of life, causing an elevation in LH, FSH, and estrogen secretion. These changes in hormone secretion do not lead to physical changes in sexual characteristics or reproductive function but are called endocrine puberties, with the latter, that is, activation of the HPO axis after birth, also known as minipuberty (Becker & Hesse, 2020; Bordini & Rosenfield, 2011). In girls, LH and FSH during the fetal phase are highest at midgestation and are progressively suppressed after that (Becker & Hesse, 2020). Meanwhile, estrogen secretion peaks in the third trimester and is predominantly secreted from the placenta (Becker & Hesse, 2020; Takagi et al., 1977). The suppression of LH and FSH is caused by the placental rise in estrogen secretion (Becker & Hesse, 2020).

Minipuberty was first described in 1973 (Forest et al., 1973). While it is still unknown if minipuberty is essential to female reproductive function (Bizzarri & Cappa, 2020), hormonal changes during minipuberty have been investigated since the 1970s. It is now known that the absence of placental estrogen secretion causes activation of the HPO axis approximately one week after birth (Becker & Hesse, 2020). In girls, FSH levels are higher and LH levels are lower than in boys

during the first four weeks of life (Bizzarri & Cappa, 2020; Schmidt & Schwarz, 2000). The rise of gonadotropins leads to ovarian stimulation, resulting in increased, although fluctuating, estradiol levels (Bizzarri & Cappa, 2020; Kuiri-Hänninen et al., 2011, 2013). The rise in estradiol levels during minipuberty is more prominent in girls than in boys (Becker & Hesse, 2020; Kuiri-Hänninen et al., 2013).

The activation of the HPO axis gradually decreases after a few months of life with mechanisms that are not yet understood (Becker & Hesse, 2020). In girls, FSH levels may remain high until the first 3–4 years of life, while LH levels drop after the first six months (Bizzarri & Cappa, 2020). After these changes, the HPO axis is nearly inactive until the late prepubertal period, about the eighth to tenth year of life (Bordini & Rosenfield, 2011; Hope & Hackney, 2017).

2.2.3.2 Puberty

Puberty is one of the most critical parts of a female's life cycle, a period leading from childhood to adulthood and from non-fertility to fertility (Colvin & Abdullatif, 2013; DiVall & Radovick, 2009). It is characterized by the maturation of gametogenesis and gonadal hormone secretion, along with the development of secondary sexual characteristics and reproductive functions (Bordini & Rosenfield, 2011). During puberty, the female's body achieves the ability to produce and deliver a gamete, a capacity to provide a suitable environment for gestation, and a source of infant nutrition (Colvin & Abdullatif, 2013).

The triggering factors for the reactivation of the HPO axis at the beginning of "true" puberty in adolescence are poorly understood, but include a complex interaction between a variety of neurotransmitters, hormones, and proteins (Colvin & Abdullatif, 2013; Herbison, 2016; Karapanou & Papadimitriou, 2010; Uenoyama et al., 2019). The accumulated evidence thus far suggests that KNDy neurons are crucial for pubertal onset, as they regulate kisspeptin synthesis and secretion and thus GnRH secretion (Uenoyama et al., 2019). In addition, sufficient energy stores and nutritional status are needed for pubertal onset and progression (Martos-Moreno et al., 2010). Indeed, leptin has been suggested to be important for the proceeding of pubertal development (Sanchez-Garrido & Tena-Sempere, 2013; Uenoyama et al., 2019).

As a result of the activation of the HPO axis at the beginning of puberty, the pulsatile release of LH and FSH secretion increase (Bordini & Rosenfield, 2011; Colvin & Abdullatif, 2013). The sensitivity and size of gonads increase as a result of gonadotropin release, leading to increased secretion of estradiol (Bordini & Rosenfield, 2011). More specifically, FSH induces estradiol production from androgen precursors by increasing ovarian granulosa cell aromatase activity and also promotes the growth and proliferation of granulosa cells, while LH, in turn, stimulates androgen production in ovarian theca cells (Colvin & Abdullatif, 2013). These hormones then promote pubertal changes throughout the body (Bordini & Rosenfield, 2011; Colvin & Abdullatif, 2013). Moreover, sex steroids stimulate the secretion of growth hormone from the pituitary gland (Colvin & Abdullatif, 2013). Sex steroids and growth hormone, as well as IGF-1, which secretion is stimulated

by growth hormone, have synergistic roles in pubertal growth spurt and reproductive organ development (Bordini & Rosenfield, 2011; Colvin & Abdullatif, 2013; Riggs et al., 2002).

Hormonal changes, especially the elevated levels of estrogen, occurring during puberty also affect uterine growth (Salardi et al., 1985) and the endometrium, which grows until it has reached a point where a decrease in estrogen levels leads to first menstruation, i.e., menarche (Hope & Hackney, 2017). In females, menarche is one of the landmarks of puberty in addition to breast tissue development and peak height velocity (Karapanou & Papadimitriou, 2010). It typically occurs 2–3 years after thelarche (i.e., breast building) and six months after peak height velocity, while in girls with early breast building, the interval between thelarche and age at menarche (AAM) can be longer (ACOG, 2015; Karapanou & Papadimitriou, 2010). In Europe, an average AAM has declined by 2–3 months per decade from the early 1800s to the mid-1950s (Wyshak & Frisch, 1982). While some evidence suggests a further decrease after that (Talma et al., 2013), other studies have reported that the decline in AAM has been slowed or even leveled off in developed countries during the last decades (Bau et al., 2009; Gottschalk et al., 2020; Lee et al., 2016; Mul et al., 2001; Papadimitriou, 2016; Rigon et al., 2010). In most recent studies, the mean AAM in developed countries has been reported as approximately 12–13 years (ACOG, 2015; Bau et al., 2009; Gottschalk et al., 2020; InterLACE Study Team, 2019; Lee et al., 2016; Rigon et al., 2010). However, AAM varies internationally, being higher in less developed countries (ACOG, 2015). The decrease in AAM during the last centuries in developed countries has been explained by improved living conditions, socioeconomic status, and general health (Gluckman & Hanson, 2006; Karapanou & Papadimitriou, 2010; F. Thomas et al., 2001). Improvements in nutrition have been suggested to be one of the major reasons for the decreased AAM (Gluckman & Hanson, 2006; F. Thomas et al., 2001).

Several factors can influence AAM, of which the most significant are presented here. Genetic factors play an important role in explaining about half of the phenotypic variation in AAM among girls living in developed countries (Towne et al., 2005). Indeed, maternal AAM is associated with daughter's AAM both in the general population (e.g., Pouta et al., 2005; Wohlfahrt-Veje et al., 2016) and in athletes (Malina et al., 1994). AAM may also differ between ethnicities and geographic locations, which may reflect genetic or environmental factors (Karapanou & Papadimitriou, 2010). Moreover, body mass index (BMI) and fat mass in infancy, prepuberty, and puberty are important factors for explaining AAM and are negatively associated with it (Druet et al., 2012; Juul et al., 2017; Karapanou & Papadimitriou, 2010; Mumby et al., 2011; Yermachenko & Dvornyk, 2014). The association between a higher BMI and fat mass and earlier AAM may be explained by the role of leptin. As discussed earlier, leptin is an adipocyte-derived hormone reflecting the body's fat mass and stimulating the pulsatile release of GnRH in the hypothalamus, thus serving as a signal for menarche (Mantzoros, 2000). Other factors that influence AAM include socioeconomic status, parental education, endocrine-disrupting chemicals, i.e., substances in the

environment, food, and consumer products that interfere with hormone function, and the absence of a father, with the first three of them being positively associated with earlier maturation in girls and the absence of a father being linked with earlier AAM (Diamanti-Kandarakis et al., 2009; Karapanou & Papadimitriou, 2010; Yermachenko & Dvornyk, 2014).

Moreover, premenarcheal physical activity is associated with later AAM (Calthorpe et al., 2019) and a sedentary lifestyle has been reported to be linked with earlier AAM (Ramraj et al., 2021). In line with these findings, athletes tend to attain menarche later than their non-athletic peers (Calthorpe et al., 2019; Torstveit & Sundgot-Borgen, 2005). However, it is not known if there is a causal relationship between sports participation and later AAM or if there are genetic, behavioral, or body type-related factors between athletes and non-athletes that explain that relationship (Calthorpe et al., 2019; Torstveit & Sundgot-Borgen, 2005).

Furthermore, there is an association between AAM and women's health in later life. In their systematic review and meta-analysis, Prentice and Viner (2013) found that earlier AAM was associated with a higher BMI in later life, the association being stronger in women under 40 years of age. An association, albeit weaker, was also found between AAM and adult cardiovascular disease, suggesting that earlier AAM is associated with a higher risk of cardiovascular disease (Prentice & Viner, 2013). This association was later confirmed in a systematic review by Luijken et al. (2017); however, one large cohort study also suggested a U-shaped association between AAM and cardiovascular disease, with both early and late AAM being associated with an increased risk of cardiovascular disease (Canoy et al., 2015). In further meta-analyses, early menarche has been associated with all-cause deaths (Charalampopoulos et al., 2014; Chen et al., 2018), ischemic heart disease mortality (Chen et al., 2018), hypertension (Bubach et al., 2018) and metabolic disease (Kim & Je, 2019) in adulthood as well as endometrial (Gong et al., 2015), breast (Collaborative Group on Hormonal Factors in Breast Cancer, 2012), and ovarian (Gong et al., 2013) cancers.

The mechanisms behind the above-mentioned associations have yet to be resolved. However, it has been suggested that higher BMI or lower height associated with earlier AAM may explain the association between earlier AAM and a higher risk of cardiovascular disease (Luijken et al., 2017) and that estrogen exposure and/or a number of ovulations could at least partly explain the association between early AAM and the risk of endometrial, breast, and ovarian cancers (Collaborative Group on Hormonal Factors in Breast Cancer, 2012; Gong et al., 2013, 2015). Early menarche has also been associated with a higher risk of depressive symptoms (Joinson et al., 2011; Kaltiala-Heino et al., 2003), less time spent in education (Gill et al., 2017), sexual risk-taking (Belsky et al., 2010), and ED risk (Klump, 2013). AAM also has implications for bone health, with later AAM predicting lower BMD in both premenopausal (Chevalley et al., 2008, 2009b; Ito et al., 1995; Rosenthal et al., 1989) and postmenopausal (Ito et al., 1995; Tuppurainen et al., 1995; Varena et al., 1999) women. In line with that, fracture

risk has been shown to be higher in women with late AAM in contrast to those with earlier AAM (G. S. Cooper & Sandler, 1997; Johnell et al., 1995; Roy et al., 2003; Silman, 2003).

Menstrual cycles in the early years after menarche are typically anovulatory and irregular (ACOG, 2015; Carlson & Shaw, 2019). However, variability between individuals is high, and in some studies, early menarche has been associated with the early onset of ovulatory cycles (Apter & Vihko, 1983; Vihko & Apter, 1984). Indeed, it has been reported that 50% of the cycles are ovulatory in the first year after menarche among girls with AAM lower than 12 years, but when AAM is about 13 years, 50% of girls had ovulatory cycles 4.5 years after menarche (Apter & Vihko, 1983). However, these studies were conducted a rather long time ago, and it is possible that girls are achieving ovulatory cycles more rapidly nowadays (Carlson & Shaw, 2019). However, there is limited evidence to support this hypothesis. Although cycle length varies among individuals, in the first few years after menarche most cycles range from 21 days to 45 days, and the trend is toward shorter and more regular cycles with increasing gynecologic age (ACOG, 2015). While it is typical that a normal-length menstrual cycle is observed after about three years of menarche, cycles longer than 90 days are not normal even in the first gynecologic years (ACOG, 2015; Nattiv et al., 2007).

2.2.3.3 Adulthood, menopause, and postmenopause

Studies conducted several decades ago suggest that individuals reach their normal cycle length at about the sixth gynecologic year (American Academy of Pediatrics Committee on Adolescence et al., 2006). After that, cycle length seems to be rather stable (Guo et al., 2006; Harlow et al., 2000) until one to five years before menopause when the variability in cycle length and the number of anovulatory cycles increase (Harlow et al., 2012; O'Connor et al., 2001).

Reproductive aging in women is a process rather than an event (Soules et al., 2001). It begins at birth and ends at menopause, which is defined as the loss of ovarian follicular activity leading to a permanent cessation of menstrual cycles and is confirmed after one year of absence of menses (Davis et al., 2015; Harlow et al., 2012; Soules et al., 2001). Menopause is characterized by a depletion of ovarian follicles, while other factors also contribute to this process (Davis et al., 2015; Hall, 2015; Wise, 1999). Indeed, natural menopause is a consequence of ovarian function loss, which occurs after changes in the function of the HPO axis (Burger et al., 2008; Davis et al., 2015). These changes occurring both in the brains and in the ovaries are driven by aging and genetic factors, along with environmental- and lifestyle-related factors and possible systemic diseases (Davis et al., 2015).

Some years before menopause, the function of the HPO axis begins to weaken due to hypothalamic aging. More specifically, pulsatile GnRH secretion becomes desynchronized, which leads to impaired timing of LH pulse secretion. This, combined with ovarian aging and follicles' decreased sensitivity to gonadotropins, leads to suppressed levels of estradiol, inhibin, and anti-

Müllerian hormone (a marker of ovarian reserve; Oh et al., 2019) secretion from the ovaries, as well as impairments of follicle maturation. As a result, the menstrual cycle becomes irregular and FSH levels begin to rise because of the lack of negative feedback of estradiol (Davis et al., 2015; Hall, 2015). The perimenopausal transition is a period that starts when there is a sustained difference of ≥ 7 days in the length of 10 consecutive menstrual cycles. The last menstrual period is the marker for the end of the menopausal transition and the beginning of postmenopause (Harlow et al., 2012). Thereafter, FSH levels continue to rise, and estradiol levels continue to decrease until approximately two years after menopause. After that, the levels of these hormones become more stable (Davis et al., 2015; Harlow et al., 2012). Thus, postmenopausal women are characterized by high FSH levels and low estradiol levels (Davis et al., 2015). The term perimenopause is defined as a period from the beginning of the menopausal transition until one year after the final menstrual period (Harlow et al., 2012).

The overall mean age at menopause is 48.8 years, with European women having a slightly higher menopausal age (50.5 years) (Schoenaker et al., 2014). In the U.S., age at menopause has been reported to increase by 1.5 years from 1959–1962 to 2015–2018 (from 48.4 years to 49.9 years) (Appiah et al., 2021). While the factors explaining the variation between individuals in age at menopause are still largely unknown (Davis et al., 2015), it has been reported that genes may explain approximately 50% of this variation (Murabito et al., 2005; van Asselt et al., 2004). In addition, smoking has been associated with earlier age at menopause in a dose-response manner, while higher socioeconomic status/education (Gold et al., 2013; InterLACE Study Team, 2019; Pelosi et al., 2015; Schoenaker et al., 2014), a higher number of parities (Pelosi et al., 2015), and a higher BMI (Tao et al., 2015) has been linked with a later menopausal age.

Studies have been inconsistent regarding the possible association between physical activity and age at menopause. Nagata and colleagues (2012) reported that among Japanese women with a baseline age of 35–56 years, physical activity during the 10-year follow-up was moderately associated with earlier menopausal age. In contrast, Dratva and research group (2009) reported that low physical activity was associated with an earlier age at menopause compared with high and medium physical activity among 30–60-year-old women from nine European countries. In line with this, Gudmundsdottir and colleagues (2012) reported that physical inactivity was associated with a lower menopausal age among Norwegian women with a baseline age of 40–49 years. However, among 50–59-year-old women from the same sample, no association between physical activity and menopausal age was found. The authors concluded that the association between physical activity and menopausal age may be age-dependent (Gudmundsdottir et al., 2012).

2.3 Female athletic participation and health

2.3.1 Definitions of physical activity, exercise, athlete, and sports competition

The most widely accepted definition for *physical activity* is “any bodily movement produced by skeletal muscles that results in energy expenditure,” while *exercise* is defined as “physical activity that is planned, structured, repetitive, and purposeful in the sense that improvement or maintenance of one or more components of physical fitness is an objective” (Caspersen et al., 1985). Thus, physical activity is a wider term consisting of any bodily movement from daily tasks to competitive sports, whereas exercise is a subset of physical activity. Instead, physical fitness is characterized as a set of attributes that people have or achieve and that are either health- or skill-related. Health-related attributes can be classified as i) cardiorespiratory endurance, ii) muscular endurance, iii) muscular strength, iv) body composition, and v) flexibility, while skill-related attributes are related to athletic ability (Caspersen et al., 1985).

The term *athlete* has been used in a variety of ways in the literature. The American Heart Association has defined an athlete as “one who participates in an organized team or individual sport that requires systematic training and regular competition against others and places a high premium on athletic excellence and achievement” (Maron et al., 2007). It has also been suggested that for being an athlete, a person must have exercise training and competitions as his/her major activity and spend several hours at least most of the days for these activities (C. G. S. Araújo & Scharhag, 2016), but this definition has been considered too strict as it only applies for professional athletes (MacMahon & Parrington, 2017; McKinney et al., 2019). Recently, MacMahon and Parrington (2017) suggested that an athlete differs from an exerciser in the motivation and goals of an exercise. While an exerciser trains because of, for instance, increasing fitness, promoting health, and improving physique, an athlete’s primary goal is to improve performance for competition (MacMahon & Parrington, 2017; McKinney et al., 2019). *Sports competitions*, in turn, are organized teams or individual sports events that place a high premium on athletic excellence and achievement (Pelliccia et al., 2005). A characteristic of competitive sports is that, regardless of the level of participation, participants seek to exert themselves physically until their limits and improve performance (Pelliccia et al., 2005).

The level of exercise and competitive sports range from sedentary individuals to world class athletes, and similarly to the term athlete, the level of participation has been defined in a variety of ways in the literature. Therefore, McKay and colleagues (2021) recently presented a 6-tiered Participant Classification Framework, which can be used when classifying athletes and non-athletic individuals. According to the Participant Classification Framework, an individual can be classified into one of the following tiers based on his/her training volume and performance: Tier 0 = sedentary; Tier 1 = recreationally active; Tier 2 =

trained/developmental; Tier 3 = highly trained/national level; Tier 4 = elite/international level; Tier 5 = world class (McKay et al., 2021).

2.3.2 Competitive sports during the female lifespan

In Finland, sports club participation among girls starts on average at the age of 6 years and dropout occurs on average at the age of 11 years (Blomqvist et al., 2019, 2023). In 2018, over half (58%) of 11-year-old girls and boys were actively engaged in sports clubs, while the proportion among 13-year-olds was 46% and among 15-year-olds 38% (Blomqvist et al., 2019). In 2022, after the COVID-19 pandemic, the figures were 53%, 42%, and 34% among 11-, 13-, and 15-year-olds, respectively (Blomqvist et al., 2023). In 2018, 23% of the 11–15-year-old girls participating in sports clubs competed at the national level (Tier 3; McKay et al., 2021), while the proportion in 2022 was 9% (Blomqvist et al., 2019, 2023). Further, regional/district-level sports participation was reported by 26% of the 15-year-old girls participating in sports clubs in 2018 and by 30% of the girls of similar ages in 2022 (Blomqvist et al., 2019, 2023). Among Finnish females from upper secondary schools (from 16- to 20-year-olds), 32% participate in sports clubs at least once a week (Kokko et al., 2021). Of these, 28% competed at the national level (Tier 3) and 24% at the regional/district level (Tier 2) (Mononen et al., 2021).

The timing of competitive sports participation during the lifespan varies between individuals being still emphasized to late childhood and adolescence. The most talented athletes are usually introduced to organized sports during their childhood (about the age of six or seven), become more dedicated at a mean age of 12–13 years, and reach their highest competition level in young adulthood (Wylleman & Reints, 2010). Nevertheless, there are differences between sports disciplines. For example, most elite gymnasts start intensive training at the age of eight or nine and reach their highest performance level already during late adolescence (G. Kerr & Dacyshyn, 2000). The discontinuation stage, where athletes transit out from competitive sports, typically occurs from age 28 onwards (Wylleman & Reints, 2010). However, some individuals continue their competitive sports participation into late adulthood, and some start their competitive sports careers after the age of 35 years (Kontro et al., 2022).

2.3.3 A role of physical activity and competitive sports in female health

Physical activity/exercise and health-related physical fitness can vary from low to high and have important implications for an individual's health (e.g., Caspersen et al., 1985; Haskell et al., 2007). For instance, physical activity in adulthood has been associated with reductions in the risk of type 2 diabetes, metabolic syndrome, cardiovascular diseases, dementia, and osteoporosis, along with colon, breast, and endometrial cancers (Booth et al., 2012) and mortality (Ekelund et al., 2019). In addition, physical activity in children and adolescents has been reported to be related to a decreased adiposity and cardiometabolic risk factors (such as high cholesterol, insulin resistance, and blood pressure) and to better physical fitness and bone health (Poitras et al., 2016). Physical activity

during childhood and adolescence also seems to have long-term health benefits, as it has been linked to better metabolic health in adulthood (Yang et al., 2009). A physically active lifestyle during childhood and adolescence also predicts physical activity later in life, and changing from a physically inactive lifestyle to a physically active lifestyle in adulthood may be challenging (Hirvensalo & Lintunen, 2011). In addition, in females, competitive sports participation during adolescence has been associated with better bone health in adulthood (Andreoli et al., 2012; Erlandson et al., 2012; N. K. Pollock et al., 2006) and better adulthood mental health, lower risk of obesity, and lower smoking rates (Callison & Lowen, 2022). However, not all studies have confirmed this finding regarding the association between competitive sports participation in adolescence and later bone health (Valdimarsson et al., 2005). It is also known that a type of physical activity plays an important role in bone health, as only exercises that include high or odd impact are considered beneficial (Tenforde & Fredericson, 2011).

In addition to physical activity, health-related physical fitness is also an important determinant of health. Several studies have demonstrated a link between cardiorespiratory fitness and cardiovascular diseases and morbidity (Blair et al., 2001; Kodama et al., 2009; Myers et al., 2015). Evidence regarding muscular fitness and health is less strong, but the available evidence suggests that muscular fitness is an independent protective factor for all-cause and cancer mortality as well as metabolic syndrome in adults (Artero et al., 2012). In children and adolescents, physical fitness has been associated with lower BMI and cardiovascular risk, better insulin resistance, and higher BMD later in life (Artero et al., 2012; García-Hermoso et al., 2019; Smith et al., 2014).

2.3.4 Injuries among female athletes

As discussed above, participation in sports provides many health benefits. However, it is also associated with injuries (Maffulli et al., 2011). The International Olympic Committee (IOC) has defined sports injury as “a tissue damage or other derangement of normal physical function due to participation in sports, resulting from rapid or repetitive transfer of kinetic energy” (Bahr et al., 2020). This is different from illness, which is defined as “a complaint or disorder experienced by an athlete, not related to injury” (Bahr et al., 2020). According to the IOC (Bahr et al., 2020), illnesses are problems in physical (e.g., influenza), mental (e.g., depression), or social well-being, or the removal or loss of vital elements (e.g., water).

Traditionally, injuries have been divided into acute and overuse injuries. An acute injury results from a specific, identifiable event, and an overuse injury is without a specific, identifiable event responsible for its occurrence (Clarsen et al., 2013; Fuller et al., 2006). Acute, sudden-onset injuries are most common in sports disciplines with high speed and falling risk, while overuse injuries occur more commonly in aerobic and technical sports, in which the same movement is repeated multiple times (Bahr, 2009). Indeed, overuse injuries have been reported to be more common in individual sports than in team sports (Franco et al., 2021).

Compared to their male counterparts, female athletes have been reported to be at a higher risk of specific injuries. These include noncontact anterior

cruciate ligament (ACL) injuries, patellofemoral pain syndrome, and bone stress injuries (de Borja et al., 2022; Edison et al., 2022; Hilibrand et al., 2015; Ireland, 2002; Wentz et al., 2011; Wright et al., 2015). It is thought that anatomical (e.g., increased Q angle, that is, the angle formed by the quadriceps muscle and patellar tendon), hormonal (higher risk of ACL injury during the follicular phase of the menstrual cycle compared with the luteal phase), and neuromuscular (e.g., imbalance of hamstring-quadriceps ratio or weakness in gluteal muscles) factors contribute to the higher susceptibility of knee injuries among females compared to males (American Academy of Family Physicians et al., 2018; Balachandar et al., 2017; de Borja et al., 2022; Edison et al., 2022; Renstrom et al., 2008). In addition, while bone stress injuries are multifactorial, females' higher risk of these injuries is thought to be attributed to, for instance, bone anatomy, lower aerobic capacity, and smaller muscles (Wentz et al., 2011).

2.3.4.1 Association of low energy availability and other lifestyle-related factors with injuries

As described earlier, low EA impairs bone health. Even short-term low EA has been linked with decreased bone formation and increased bone resorption in females (Ihle & Loucks, 2004; Papageorgiou et al., 2017). Interestingly, it was found that short-term (three days) low EA induced by dietary restriction was associated with decreased bone formation, while low EA induced by increased exercise energy expenditure did not affect bone formation, highlighting the importance of the dietary intake of athletes (Papageorgiou, Martin, et al., 2018). In field-based studies investigating bone stress injuries in athletes, direct assessments of EA have rarely been conducted; instead, proxies of low EA, for example, MD, later AAM, lower BMI, ED/DE, and dietary restraint, are linked with bone stress injuries (Ackerman et al., 2015; Barrow & Saha, 1988; Beals & Manore, 2002; Guest & Barr, 2005; Myburgh et al., 1990; Nattiv et al., 2013; Nose-Ogura et al., 2019; Tenforde et al., 2013). In addition, Barrack and colleagues (2014) showed that the risk of bone stress injuries increased with increasing numbers of Triad-related risk factors (e.g., low BMD, dietary restraint, and low BMI), so that 15–21% of females with only one risk factor developed a bone stress injury compared with 29–50% of females with three risk factors developing such an injury. Similar findings were found by Gibbs and colleagues (2014) who reported that the risk of low BMD increases with an increasing number of Triad risk factors.

Less evidence has been accumulated on the relationship between low EA and non-bone injuries, and the available evidence is conflicting. While some studies have reported that DE is associated with musculoskeletal injuries in athletes (Rauh et al., 2010; Thein-Nissenbaum et al., 2011; J. J. Thomas et al., 2011), other studies have failed to find an association between low EA and injuries (Gerlach et al., 2008; Heikura et al., 2018; Vanheest et al., 2014) or between ED/DE and injuries (Ackerman et al., 2019; Tenforde et al., 2011). In addition, MD has been associated with injuries in some studies (Rauh et al., 2010, 2014), but most of them have not found such an association (Beals & Manore, 2002; Bratland-Sanda et al., 2015; Tenforde et al., 2011; Thein-Nissenbaum et al., 2011).

In addition to low EA and its surrogates, other lifestyle-related factors may also play a role in the risk of injury. Early sports specialization, defined as “year-round (8 months or more per year) intensive training in a single sport at the exclusion of other sports,” is associated with overuse injuries in young athletes, especially in the lower extremities (Myer et al., 2015; Puzzitiello et al., 2021). Moreover, a rapid increase in training volume is associated with an increased risk of injury (Gabbett, 2016) as is earlier injury (Fulton et al., 2014). Additionally, sleeping for less than eight hours per night was found to be associated with injuries in elite adolescent athletes (von Rosen et al., 2017). In females, the risk of muscle, tendon, and ACL injuries may be highest in the late follicular/preovulatory phase of the menstrual cycle (Balachandar et al., 2017; Herzberg et al., 2017; Martin et al., 2021).

2.3.4.2 Effects of injuries on a sports career and later life

Most injuries heal well, allowing an athlete to return to full sports participation, although this does not necessarily mean that the injury will not cause any long-term consequences in later life (Garrick & Requa, 2003). Indeed, some injuries sustained by an athlete may compromise function in later life by causing pain and functional limitations (Garrick & Requa, 2003; Kujala et al., 2003; Palmer et al., 2021; Thornton et al., 2023). In addition, sports injuries have negative impacts on athletes’ psychological well-being causing, for example, depression, sadness, isolation, anger, and frustration (American College of Sports Medicine et al., 2006; Johnston & Carroll, 2000; Leddy et al., 1994; Putukian, 2016). Moreover, serious injuries can cause fear of re-injury along with issues in confidence and performance when returning to play (Podlog & Eklund, 2007). Some injuries can also force an athlete to terminate his/her sports career. For example, a recent study conducted on female soccer players in Poland (n=93) reported that 30% of athletes had terminated their athletic careers due to injury (Grygorowicz et al., 2019). Among the 52 retired Finnish male and female mixed-sport athletes who provided a reason for their retirement (n=50), over half (54%) reported that an injury resulted in their career termination (Ristolainen et al., 2012). A transition from athlete to non-athlete is usually difficult when it is caused by an unplanned incident, such as a career-ending injury (Alfermann et al., 2004; Stokowski et al., 2019).

A recent meta-analysis found that former athletes’ physical component scores assessing physical functioning, bodily pain, general health perceptions, and physical role limitations did not differ from those of the general population (Filbay et al., 2019). However, the findings of the studies included in the meta-analysis varied so that three studies reported worse physical component scores among athletes compared with the general population, while in three studies, the physical component scores among athletes were higher than among the general population (Filbay et al., 2019). Indeed, while the well-being of retired athletes seems, on average, to be similar or even higher than in the general population, there are subgroups of former athletes with lowered health-related quality of life linked, for example, with injuries and persistent pain (Z. Y. Kerr et al., 2014;

Mannes et al., 2019; Thornton et al., 2023). Moreover, osteoarthritis seems to be more prevalent in former athletes than in the general population (Gouttebauge et al., 2015). This is probably at least partly a result of joint injuries and consequent surgeries, which are linked with an increased risk of early osteoarthritis (Ajuied et al., 2014; Gelber et al., 2000; Kujala et al., 1995; L.-J. Wang et al., 2020; Wilder et al., 2002).

3 AIMS OF THE STUDY

The first aim of this study was to assess the prevalence rates of low energy availability (EA)-related conditions (i.e., eating disorders (ED), disordered eating (DE), body weight dissatisfaction, and menstrual dysfunction (MD)) in Finnish female athletes. Further, this study aimed to evaluate whether there are differences between athletes and non-athletes in terms of these conditions and whether athletes of different ages and those competing at different levels and sports differ from each other regarding these issues. The last aim of this dissertation was to investigate the association of age at menarche (AAM) and sports participation during adolescence with health-related physical fitness and bone health in middle age and the associations of low EA-related conditions with injuries, health, and sports career.

The specific research questions were as follows:

1. What are the prevalence rates of ED, DE, body weight dissatisfaction, and MD in Finnish female athletes? (Studies I and III)
2. Are there differences in low EA-related conditions between athletes and non-athletes or between different-aged athletes competing at different levels and sport types? (Studies I, III, and IV)
3. Is there an association of AAM and competitive sports participation with midlife health-related physical fitness components and bone health in women? (Study II)
4. What health- and sports career-related attributes are associated with low EA-related factors? (Studies I, III, and IV)

4 METHODS

4.1 Datasets and participants

Data from four different research projects were used in this dissertation: the Finnish Health Promoting Sports Club (FHPSC) study consisting of baseline and follow-up data (Study I), the Estrogenic Regulation of Muscle Apoptosis (ERMA) study (Study II), the Female Athlete 2.0 study (Study III), and the Menstrual Function, Eating Attitudes, Body Image, and a Sports Career (MEBS) study (Study IV). The datasets, study designs, and participants are summarized in Table 1. The time points in the athletes' lifespans when each study was conducted are presented in Figure 7.

TABLE 1 Summary of the datasets, study designs, and participants in Studies I-IV.

Study	Dataset	Design	n	Participants
I	FHPSC	Observational, cross-sectional/longitudinal	Baseline: 178 athletes + 105 non-athletes Follow-up: 52 athletes + 159 non-athletes	Baseline: 14- to 16-year-old female athletes and non-athletes Follow-up: 18- to 20-year-old female athletes and non-athletes
II	ERMA	Observational, retrospective	1098	47- to 55-year-old community-dwelling women
III	Female Athlete 2.0	Observational, cross-sectional	846	15- to 45-year-old female athletes
IV	MEBS	Observational, retrospective	100 athletes + 98 non-athletes	30- to 53-year-old females who had participated in endurance sports in their adolescence, and their age-, gender-, and municipality-matched peers

FHPSC = the Finnish Health Promoting Sports Club study; ERMA = the Estrogenic Regulation of Muscle Apoptosis study; MEBS = the Menstrual Function, Eating Attitudes, Body Image, and a Sports Career study.

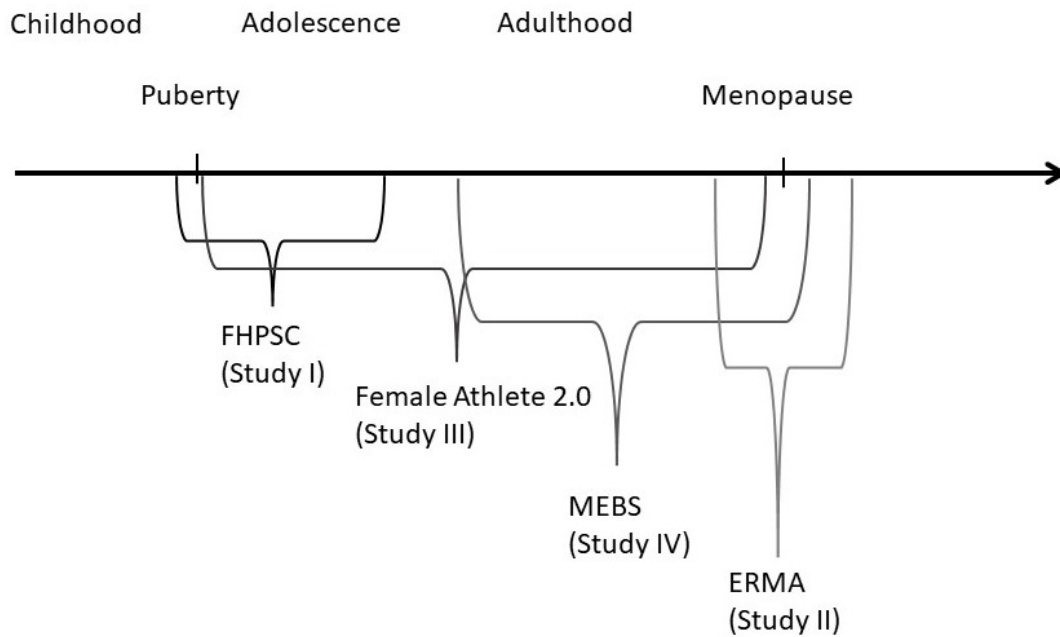


FIGURE 7 The time points in the athletes' lifespan when each study was conducted. The time between puberty and menopause marks the reproductive period in a female's life. FHPSC = the Finnish Health Promoting Sports Club study; MEBS = the Menstrual Function, Eating Attitudes, Body Image, and a Sports Career study; ERMA = the Estrogenic Regulation of Muscle Apoptosis study.

4.1.1 Finnish Health Promoting Sports Club study (Study I)

The Finnish Health Promoting Sports Club (FHPSC) study (Kokko et al., 2015) was a longitudinal cohort study investigating the health status and behaviors of adolescents and young adults participating in sports clubs, in addition to the health promotion orientation of those clubs. The baseline data were gathered in 2013 and the follow-up data in 2017–2018. The participants were 14–16 year-olds at baseline and 18–20 year-olds at follow-up. At baseline, 272 sports clubs from the 10 most popular sports disciplines in Finland were approached, and 156 clubs (85 winter disciplines and 71 summer disciplines) agreed to participate in the study. Study I used data from female sports club members who underwent the preparticipation screening of the FHPSC study, in which medical history, height, and weight were assessed. The inclusion criterion for the athlete in Study I was that she had to exercise at least four times a week during the training season.

At baseline (i.e., in adolescence), the number of total athletes invited to the preparticipation screening of the FHPSC study was 1123, from which 699 could not be contacted or refused to participate. Thus, 424 athletes underwent the preparticipation screening. Of those, 215 were girls, and of those, 37 exercised less than four times a week in the training season and were excluded from the data used in Study I. Thus, in Study I, there were 178 adolescent athletes.

At the same time, when collecting data from athletes, data from non-sports club members (i.e., non-athletes) were collected from schools. A total of 100 schools from 159 invited schools agreed to participate, with 512 non-athletes invited for the preparticipation screening. Of those, 353 could not be contacted or refused to participate, and thus 159 non-athletes underwent the preparticipation screening at baseline. Of these, 105 were girls and were included in Study I.

All participants from the baseline who had agreed to be recontacted were invited to participate in the follow-up of the FHPSC study. Of the 315 females invited, 88 could not be contacted or refused to participate, leaving 227 females in the study. Of those, 68 were members of a sports club. Of the sports club members, 16 females were excluded from the data used in Study I because they exercised less than four times a week during the training season. Thus, at follow-up (i.e., young adulthood), data from 52 athletes and 159 non-athletes were included in Study I.

4.1.2 Estrogenic Regulation of Muscle Apoptosis study (Study II)

The Estrogenic Regulation of Muscle Apoptosis (ERMA) study (Kovanen et al., 2018; Laakkonen et al., 2022) was a population-based study consisting of both cross-sectional and longitudinal parts. This study investigated the role of hormonal differences associated with menopause on physiological and psychological functioning in middle-aged women. In Study II, the cross-sectional part of the ERMA study, including data from participants who attended two laboratory visits and filled out a questionnaire survey was used. The baseline data were collected between January 2015 and November 2016.

In the ERMA project, a written invitation was sent to 6878 women representing a random sample of 47–55-year-old women living in Jyväskylä or neighboring municipalities in Central Finland. The response rate was 46.9% (n=3229). Of these, 1102 participated in two laboratory visits and provided a questionnaire survey, while 1259 women were excluded due to conditions that met the exclusion criteria (see below), and 868 women were unwilling to participate or continue their participation. Four questionnaires were lost due to technical errors; thus, the final sample size in Study II was 1098. Of those, 988 provided information on their physical activity in adolescence, and the question on AAM was answered by 1081 participants.

In the ERMA, the exclusion criteria were conditions and the use of medications that affect ovarian function. In addition, obesity (self-reported BMI >35kg/m²), along with conditions or medications affecting daily physical or mental functioning or systemic hormone or inflammatory status, were considered exclusion criteria.

4.1.3 Female Athlete 2.0 study (Study III)

The Female Athlete 2.0 study was a survey-based cross-sectional study aimed at investigating the menstrual cycle and low EA-related problems in Finnish female athletes. All female athletes who were at least 15 years old and competed at any level (Tier 2 upward; McKay et al., 2021) in any sport were eligible to participate in the study. A link to the survey was promoted by the Finnish Olympic Committee, along with national sports federations and sports academies, and the research team distributed it via social media.

Data were collected between May and August 2020. Initially, 926 athletes filled out the questionnaire. Of these, 32 questionnaires were excluded due to incomplete survey responses. In Study III, athletes over 45 years of age (n=23) or those who did not report their age (n=9) were excluded for the purpose of including only premenopausal women. Furthermore, those who did not report their participation level (n=9) or sports discipline (n=7) were excluded. Thus, the sample size in Study III was 846.

4.1.4 Menstrual Function, Eating Attitudes, Body Image, and a Sports Career study (Study IV)

The Menstrual Function, Eating Attitudes, Body Image, and a Sports Career (MEBS) study was a retrospective survey study that was conducted for this dissertation. Data for the MEBS study were collected between May and July 2022.

Athletes for the MEBS study were invited through the result lists, with the following inclusion criteria: 1) an athlete among the best 30 in junior national championships in cross-country running between 1990 and 2005 in the girls' division aged 16 years (from 1990 to 1994) or 17 years (from 1995 to 2005) or 2) an athlete among the best 10 in the national youth championships in adolescents' track and field between 1992 and 2003 in the 2000 meter running in the division for girls aged 14 years (from 1992 to 1994) or 15 years (from 1995 to 2003). These

criteria were chosen with the purpose of 1) having a sufficiently large sample size, 2) including participants who are well beyond their adolescence to ensure a long enough time span between adolescence and response time, and 3) including participants who had succeeded in sports during their adolescence, regardless of the length of their sports career.

A total of 295 athletes met the inclusion criteria. Their names and dates of birth were collected from the result lists and their postal addresses were collected from the population registry. Four athletes could not be identified, and from the 291 athletes, two were dead and 13 were living abroad. Of these 276 athletes, five had unknown addresses. Thus, invitation to participate in the study was sent to 271 athletes. One of the invited participants indicated that she was not the intended person; thus, the final number of invited athletes was 270.

Two controls, matched for age, municipality, and gender were randomly selected from the general population for each available athlete (n=552). One invitation was returned by the post office because the selected person had moved abroad.

The response rates for the athletes and controls were 37.0% (100/270) and 20.1% (111/551), respectively. Thirteen controls reported that they have participated in competitive sports and were excluded from the analysis. Thus, the final number of controls (i.e., non-athletes) in Study IV was 98.

4.2 Ethics

The FHPSC and ERMA studies were approved by the Ethics Committee of the Health Care District of Central Finland. The MEBS study was approved by the Ethical Committee of the University of Jyväskylä. The Female Athlete 2.0 study was considered not to require an ethical review as the Ethical Committee of the University of Jyväskylä determined that the study did not meet the criteria set by the Finnish National Board of Research Integrity TENK.

Good scientific practice was followed in every project and in all phases of the research. Participants were informed about the study protocols before providing their written informed consent. In the FHPSC, informed consent was also requested from a guardian when a participant was under 18 years of age. In every project, participants were informed that participation was voluntary and that they had the right to withdraw from the study at any stage.

In each project, the data were stored and managed confidentially. Data analyses were conducted using pseudonymized data, and data were reported in a way that no personal characteristics would be identifiable.

4.3 Measurements

The measurements used in this study are presented in Table 2. Descriptive characteristics and covariates are presented in Table 3.

TABLE 2 Summary of the study variables.

Variable	Study	Methods and references
Eating problems and body perceptions		
Restrictive eating	III	Single question (Mountjoy et al., 2015)
Current or past eating disorder	III & IV	Single question (Mountjoy et al., 2015)
Disordered eating behaviors	IV	Questionnaire (Gideon et al., 2016)
Body weight dissatisfaction	I	Single question
Desire to lose weight	I	Single question
Desire to gain weight	I	Single question
The menstrual cycle and puberty		
Menstrual dysfunction	I & III	Questionnaire (A. Melin et al., 2014)
Age at menarche/primary amenorrhea	I-IV	Single question
Gynecological age	I	Single question
Secondary amenorrhea	IV	Single question
Spontaneous menarche	IV	Single question (A. Melin et al., 2014)
Age of attaining regular menses	IV	Single question
Regular menses	IV	Single question (modified from Nicodemus et al., 2001)
Cycle length 21-35 days	IV	Single question (modified from Y.-X. Wang et al., 2020)
Delayed puberty	IV	Single question
Hormonal contraceptive use	I-III	Single questions
Injury-related variables		
Injury occurrence	III	Single question (A. Melin et al., 2014)
Missed participation days due to injury	III	Single question (A. Melin et al., 2014)
Injury-related harms	IV	Single question
Injury-related sports career termination	IV	Single question (modified from Ristolainen et al., 2012)
Type of career-ending injury	IV	Single questions (Bahr et al., 2020)
Sports career		
Training volume	I & III	Single question
Competitive sports participation	II & IV	Single question
Type of sports	I & III	Single question
Competition level	III & IV	Single questions (McKay et al., 2021)
Career length	IV	Single questions

continues

TABLE 2 continues

Variable	Study	Methods and references
Bone parameters and physical fitness components		
Bone mineral density	II	Dual-energy X-ray absorptiometry (DXA)
Fat percentage	II	Dual-energy X-ray absorptiometry (DXA)
Fat mass index	II	Dual-energy X-ray absorptiometry (DXA)
Lean mass index	II	Dual-energy X-ray absorptiometry (DXA)
Appendicular lean mass index	II	Dual-energy X-ray absorptiometry (DXA)
Hand grip force	II	Hand grip test
Isometric knee extension force	II	Isometric maximal knee extension using a custom-made dynamometer chair
Lower body muscle power	II	Countermovement jump
Aerobic capacity	II	6-minute walking test
Walking speed	II	10-meter walk

TABLE 3 Descriptive characteristics and covariates used in this dissertation study.

Age	I-IV	Single question
Body height	I-IV	Stadiometer (Studies I and II), self-report (Studies III and IV)
Body weight	I-IV	Beam scale (Studies I and II), self-report (Studies III and IV)
BMI	I-IV	
Menopausal status	II	Bleeding diary, serum FSH concentration
Education	II	Single question
Number of parities	II	Single question
MVPA	I & II	Hookie AM20 (Study I), GT3X+ and wGT3X+ accelerometers (Study II)
Daily step count	I & II	Hookie AM20 (Study I), GT3X+ and wGT3X+ accelerometers (Study II)
Self-reported LTPA	II	Questionnaire (Hyvärinen et al., 2020; Kujala et al., 1998)

BMI = body mass index; FSH = follicle-stimulating hormone; MVPA = moderate-to-vigorous physical activity; LTPA = leisure-time physical activity.

4.3.1 Eating problems and body perceptions

In the Female Athlete 2.0 study (Study III), restrictive eating was assessed by the following question from the female athlete triad screening questionnaire (Mountjoy, Hutchinson, et al., 2015): “Do you limit or carefully control the foods that you eat?” The response options were binary (yes or no). Current or previous EDs in the Female Athlete 2.0 study were measured by asking the participants whether they currently have or had ever had an ED (Mountjoy, Hutchinson, et al., 2015). In the MEBS study (Study IV), participants were asked whether they had ever been diagnosed with an ED by a doctor.

DE behaviors were assessed in the MEBS study using the Eating Disorder Examination Questionnaire (EDE-Q) short form (EDE-QS). EDE-QS is a 12-item version of the 28-item EDE-Q and assesses respondent's perceptions toward eating and body image on a 4-point scale (Gideon et al., 2016). EDE-QS has demonstrated high internal consistency (Cronbach's $\alpha=.913$), test-retest validity (ICC=.93; $p<.001$), and convergent validity with the EDE-Q both among people with ($r=.91$) and without ($r=.82$) an ED (Gideon et al., 2016). Since the MEBS study was retrospective, participants were asked to fill out the EDE-QS several times by recalling their thoughts at different age periods, i.e., at ages 13–15, 16–18, 19–21, and 22–25. In addition, the participants completed the EDE-QS by thinking about their current situation. EDE-QS scores were calculated for each age period, and a cut-off score of ≥ 15 was used for the presence of DE behaviors (Prnjak et al., 2020). The EDE-QS scores were also treated as a continuous variable.

In the FHPSC study (Study I), the following questions were used to inquire about participants' perceptions of their body weight: "Are you satisfied with your present weight?" "Do you think that you should lose weight?" and "Do you think that you should gain weight?" The response options were binary "yes" or "no."

4.3.2 The menstrual cycle and puberty

All menstrual cycle- and puberty-related variables except menopausal status in the ERMA (Study II) were assessed using a questionnaire. Because of the differences in the questionnaires used in the FHPSC and the Female Athlete 2.0 studies, there was a slight discrepancy between the criteria for MD used in Studies I and III. In Study I, a participant was assigned to the MD group if she reported ≤ 9 periods in a preceding year, if her menstrual cycle was over 35 days, if she had not reached menarche despite being 15 years of age or older, or if she had not had periods in the preceding three months, even if she had reached menarche (Cobb et al., 2003; Nattiv et al., 2007). In Study III, all other criteria were similar than in Study I except the first: in Study III, MD was noted if a participant reported having < 9 periods in a preceding year (Elliott-Sale et al., 2021). In addition, in Study III, MD was noted if a participant reported using hormonal contraceptives to restore the absent menstrual cycle (Koltun et al., 2020).

In Study I, participants who reported using hormonal contraceptives (oral contraceptives, implants, injections, transdermal patches, vaginal rings, or intrauterine systems, etc.) at all, and in Study III participants who reported using oral contraceptives for reasons other than to maintain the menstrual cycle (i.e., contraception, reduction in menstrual pains, reduction in bleeding, and regulation of the menstrual cycle in relation to performance) were excluded from the analyses regarding MD. In addition, in Study I, participants whose menstrual cycle was 36–89 days and whose gynecological age (chronological age minus AAM) was one year or less were excluded from the analysis regarding MD. This was because after menarche, it takes time to achieve a regular menstrual cycle. However, menstrual cycles exceeding 90 days are not normal, even when they occur in the first gynecological year (ACOG, 2015).

The AAM was assessed using an open-ended question in the FHPSC, ERMA, and MEBS studies (Studies I, II, and IV, respectively). In the Female Athlete 2.0 study (Study III), the response options were categorized as follows: ≤ 11 , 12–14, ≥ 15 , I do not remember, or I have never menstruated (A. Melin et al., 2014). Primary amenorrhea was defined as the absence of menarche by the age of 15 years in Studies I and III (Elliott-Sale et al., 2021; Nattiv et al., 2007). In Studies III and IV, the criterion for primary amenorrhea was an absence of menarche by the age of 16 years because the participants in the ERMA and MEBS studies were in their adolescence mostly at times when this criterion was effective (Nattiv et al., 2007; Practice Committee of the American Society for Reproductive Medicine, 2004). Secondary amenorrhea (Study IV) was defined as the absence of at least three menstrual bleedings for reasons other than pregnancy or the use of hormonal contraceptives (Elliott-Sale et al., 2021; Nattiv et al., 2007).

In the MEBS (Study IV), participants were asked whether their menarche occurred spontaneously (by itself) or whether they were treated for delayed menarche (A. Melin et al., 2014). They also reported the age at which they attained a regular menstrual cycle, defined as a 21–35 day long cycle in which menses are predictable within 10 days at a maximum. In addition, participants reported whether they had had regular menses during their sports career (athletes) or ever (non-athletes) at times when they did not use hormonal contraceptives. The response options were: “always,” “nearly always,” “sometimes,” “seldom,” “never or I have never had periods,” “I always used hormonal contraceptives,” and “I do not know” (modified from Nicodemus et al., 2001). In addition, participants were asked to report the length of their usual menstrual cycle at the times when they did not use hormonal contraceptives with the following response options: “under 21 days,” “21–35 days,” “36–90 days,” “over 90 days,” “my periods were too irregular for estimation of the length of the cycle,” “I always used hormonal contraceptives”, and “I do not know” (modified from Y.-X. Wang et al., 2020).

Participants in the MEBS study also reported whether they had been examined by a medical professional because of delayed puberty. Current hormonal contraceptive (e.g., oral contraceptives, transdermal patches, vaginal rings, intrauterine systems, or other hormonal contraceptives) use was assessed using a questionnaire in the FHPSC (Study I) and the Female Athlete 2.0 (Study III) studies. Furthermore, lifetime hormonal contraceptive use was investigated through a questionnaire in the ERMA study (Study II).

4.3.3 Injuries

In the Female Athlete 2.0 study (Study III), injury and missed participation days from training or competition due to injury were assessed using questions from the Low Energy Availability in Females Questionnaire (LEAF-Q) (A. Melin et al., 2014). First, the participants were divided into two groups based on their injury status during the preceding year as follows: injured participants, i.e., those who had had absences from training or competitions during the preceding year because of sports injury/injuries (either an acute or an overuse injury) and non-

injured participants, i.e., those who had not had absences from training or competitions during the preceding year because of sports injury/injuries. Second, the injured participants were classified into two groups based on the number of missed participation days as follows: those who had missed ≥ 22 participation days because of injuries during the preceding year and those who had missed < 22 participation days because of injuries during the preceding year.

Injury-related harms during the sports career were assessed in the MEBS study (Study IV) by asking the athletes whether injuries or musculoskeletal pain hindered their sports careers. The response options were: “not at all or very little: I usually did not have to skip competitions or pause normal training because of injury or musculoskeletal pain,” “somewhat: I sometimes had to skip competitions and/or pause normal training because of injury or musculoskeletal pain,” “quite a lot: I regularly had to skip competitions and/or pause normal training for a long time because of injury or musculoskeletal pain,” and “significant amount: I often had to skip competitions and pause training for a long time because of injury or musculoskeletal pain.” The two first and two last response options were combined for analyses.

In addition, in the MEBS, retired athletes were asked whether an injury or several injuries had contributed to their decision to terminate their sports career. The response options were: “not at all,” “some injury or several injuries contributed to sports career termination,” and “some injury or several injuries was/were the main cause for terminating the sports career.” The two last response options were combined for analyses.

Athletes in the MEBS study also reported whether there was an acute or overuse injury that impacted their decision to terminate their careers. An acute injury was defined as an injury that occurs suddenly or accidentally, interrupts training or ability to compete, or causes an identifiable trauma. An overuse injury was defined as an injury that causes worsening pain during or after exercise without any noticeable external cause of injury (Ristolainen et al., 2012). Athletes in the MEBS also provided information on the type of career-ending injury/injuries, which were further classified by tissue, as recommended by Bahr and colleagues (Bahr et al., 2020).

4.3.4 Sports career

In the FHPSC study (Study I), athletes were asked about their training hours per week for competition and training seasons. They were asked to include training hours from their primary discipline and any other training in which they were engaging. In the Female Athlete 2.0 study (Study III), participants were asked to estimate how many hours they had trained in the past year.

In the ERMA (Study II), competitive sports participation at ages 7-12, 13-16, 17-19, 20-29, 30-39, and 40-50 was asked using a questionnaire. The questionnaire included four options to choose from for each age category: 1) no exercise, 2) regular independent leisure-time physical activity, 3) regular other supervised physical activity in a sports club, etc., and 4) regular competitive sport and training related to that sport (Hirvensalo et al., 2000). A definition for regular

independent leisure-time physical activity was physical activity during a journey to or from school/work (>2 km/one way) or regular physical activity causing sweating, which was not organized by a school, sports club, fitness center, etc. Supervised physical activity was defined as regular, non-competitive physical activity organized by a fitness center, sports club, etc., while competitive sport and related training was defined as goal-oriented, regular competitive sport within a sports club etc., and competing and training in that discipline. Participants were assigned into three groups based on their responses regarding ages 13–16 years as follows: no-exercise group (respondents who chose the response option 1), regular physical activity group (respondents who chose the response option 2 or 3), and athletes (respondents who chose the response option 4).

In the MEBS study (Study IV), participants invited to the control group were asked if they had ever participated in competitive sports. Those who responded positively were excluded. Regarding the type of sports, sports disciplines were classified into endurance, aesthetic, technical, ball games, and power/anti-gravitation sports in Study I and lean and non-lean sports in Study III. In Study I, modified criteria by Sundgot-Borgen and Torstveit (2004) were used, and in Study III, sports disciplines were categorized into lean and non-lean sports according to Torstveit and Sundgot-Borgen's (2005) criteria.

Athletes' competition levels were assessed in both the Female Athlete 2.0 (Study III) and MEBS (Study IV) studies by asking athletes to choose their current (Female Athlete 2.0) or highest (MEBS) competition levels from the following response options: "recreational athlete" (only in Female Athlete 2.0), "regional/district level," "national level," and "international level." If an athlete in the MEBS study had specialized in more than one discipline, the highest competition level in the first and main disciplines was inquired about separately. In Study III, athletes were classified into elite (competing at the national or international level, i.e., Tiers 3 and ≥ 4 ; McKay et al. 2021) or non-elite (competing at regional/district level or being a recreational athlete, i.e., Tier 2) athletes. In Study IV, national- and international-level athletes were categorized into their own groups (Tiers 3 and ≥ 4 ; McKay et al., 2021). One athlete in the MEBS data reported that her highest competition level had been regional/district level and she was classified into the group of national-level athletes in the analysis.

Career length was assessed in the MEBS study (Study IV) by asking athletes at what age they started goal-oriented (at least two times a week) training in the first discipline they competed in and at what age they terminated their sports career. Sports career termination was defined as terminating a sports career at the level one had practiced and actively competed in (Ristolainen et al., 2012). Career length was calculated by subtracting the sports career starting age from the age at the end of the career. If an athlete had not ended her sports career, the length of the career was calculated by subtracting the starting age from the age at response.

4.3.5 Bone mineral density and physical fitness components

Femoral neck areal BMD and body composition in the ERMA study (Study II) were assessed by dual-energy X-ray absorptiometry (DXA; LUNAR; GE Healthcare, Chicago, IL, USA) after an overnight fast. Fat percentage, fat and lean mass indexes, and appendicular lean mass index (ALMI) were calculated from the DXA-derived parameters as follows: fat percentage = total fat mass (kg) ÷ total mass (kg) × 100; fat mass index = fat mass (kg) ÷ height squared (m²); lean mass index = lean mass (kg) ÷ height squared (m²); ALMI = (arms lean mass (kg) + legs lean mass (kg)) ÷ height squared (m²).

Cardiorespiratory and muscular fitness in middle age was measured in the ERMA using multiple performance tests measuring hand grip, maximal isometric knee extension strength, lower body muscle power, aerobic capacity, and walking speed. Hand grip force was measured on the dominant arm using an adjustable dynamometer chair (Good Strength, Metitur Oy, Palokka, Finland) with the elbow flexed at a 90° angle. Participants were instructed to squeeze the handle for 2–3 s as forcefully as possible.

Maximal isometric knee extension force was assessed from the side of the dominant hand using a custom-made dynamometer chair (Good Strength, Metitur Oy, Palokka, Finland). Participants were in a sitting position and were instructed to extend the knee toward a full extension to produce maximal force.

Lower body muscle power was measured with a countermovement jump performed on a contact mat. Vertical jumping height (cm) was calculated as follows: $(g \times t^2) / 8 \times 100$, where g is the acceleration of gravity (9.81 m/s²) and t is the recorded flight time in seconds. In all three above-mentioned muscle strength and power tests, 3–5 maximal attempts were performed, and the highest value was recorded.

Aerobic capacity was assessed with a 6-minute walking test on a 20-meter indoor track. Participants were encouraged to walk as many laps as possible in six minutes, and the distance walked in meters was recorded as a result.

Walking speed was assessed via a 10-meter walk in a laboratory corridor using photocells. Five meters were allowed for acceleration before the starting line. Two attempts were performed, and the fastest result was recorded.

4.3.6 Descriptive measurements and covariates

Age/date of birth (all studies), education (ERMA; Study II), and number of parities (ERMA) were asked by a questionnaire. In Study III, athletes were categorized into younger and older athletes as follows: younger = 15–24 years of age, older = 25–45 years of age. Weight and height were measured with a stadiometer and a beam scale, respectively, in the FHPSC (Study I) and ERMA (Study II) studies, and were self-reported in the Female Athlete 2.0 (Study III) and MEBS (Study IV) studies. BMI was calculated as body weight (kg) divided by height squared (m²). In Study I, participants were divided into three groups according to their BMI. In adolescence, BMI was age-adjusted using the participants' mean age (15 years) (Cole et al., 2000, 2007). The classification was

as follows: BMI <17.5kg/m²: underweight, BMI = 17.5-23.9kg/m²: normal weight, and BMI >23.9kg/m²: overweight. In young adulthood, the cut-offs were as follows: BMI <18.5kg/m²: underweight, BMI 18.5-24.9kg/m²: normal weight, and BMI ≥25.0kg/m²: overweight.

Menopausal status (premenopausal, early perimenopausal, late perimenopausal, and postmenopausal) in the ERMA (Study II) was assessed according to slightly modified Stages of Reproductive Aging Workshop + 10 guidelines (Harlow et al., 2012) by bleeding diaries and measuring serum FSH concentrations and are explained in detail in the protocol article of Kovanen et al. (2018).

Physical activity was measured using accelerometers in the FHPSC (Study I) and using self-reports and accelerometers in the ERMA (Study II). Accelerometer-measured physical activity was assessed using triaxial accelerometers (Hookie AM20, Traxmeet Ltd., Espoo, Finland, in the FHPSC and ActiGraph GT3X+ and wGT3X, Pensacola, FL, USA, in the ERMA). In both FHPSC and ERMA studies, participants were instructed to wear the accelerometer on their hips during waking hours (excluding activities in water) for seven consecutive days. In ERMA, participants were also provided with a diary in which they were instructed to report their wake-up time, working hours, and periods when the accelerometer was removed for over 30 minutes.

In the FHPSC (Study I), accelerometer-measured data analysis was based on algorithms that employed the mean amplitude deviation (MAD) of the resultant acceleration in 6-s epochs and the angle for posture estimation of the body. As the MAD had been validated through directly measured incident oxygen uptake from slow walking to fast running on an indoor track (Vähä-Ypyä et al., 2015), it was possible to convert the MAD values into metabolic equivalents of tasks (METs). MET values 3-6 were used as cut-offs for moderate activities, and over 6 MET for vigorous activities (Sievänen & Kujala, 2017).

In the ERMA (Study II), accelerometer-measured data were analyzed in 60-s epochs. Corresponding triaxial vector magnitude cut-off points for moderate and vigorous physical activity were >2690-6166 counts per minute and >6166 counts per minute, respectively (Laakkonen et al., 2017; Sasaki et al., 2011).

In both FHPSC and ERMA, the mean times spent in moderate and vigorous activity per day were summed to obtain the total mean time spent in moderate-to-vigorous physical activity (MVPA) per day. In addition, the mean step count per day during the monitoring week was recorded.

Self-reported physical activity in the ERMA was assessed using a series of modified, structured questions (Kovanen et al., 2018; Kujala et al., 1998). Participants reported the frequency, mean duration, and mean intensity of their physical activity sessions as well as physical activity during commuting. The METs of physical activity were calculated as a product of the frequency, duration, and intensity of physical activity during leisure time and commuting. The following MET values were used: 4 for exercise intensity corresponding to walking and for physical activity during commuting, 6 for vigorous walking to

jogging, 10 for jogging, and 13 for running. Self-reported physical activity during leisure time and commuting was expressed as MET-hours/day.

4.4 Statistical analyses

Continuous variables were tested for normality prior to the statistical analysis. Descriptive statistics were reported as means and standard deviations for continuous variables, and as counts and percentages for categorical variables. In Study III, medians and interquartile ranges for non-normally distributed data were presented.

Differences between groups for categorical data were tested using chi-square or Fisher's exact tests. For continuous data, differences between two groups were analyzed using a two-independent-sample t-test or Mann-Whitney U test (Studies I, III, and IV) and between three groups by one-way analysis of variance (ANOVA) with Bonferroni post-hoc pairwise comparisons or Kruskal-Wallis rank sum test with subsequent pairwise comparisons (Study II). Adjustments for potential confounding factors were made using analysis of covariance (ANCOVA) in Study II. The following confounders were included in all the adjusted models: age, menopausal status, number of parities, education, and hormonal contraceptive use during the 10 preceding years. Moreover, physical activity level at age 13–16, self-reported leisure-time physical activity in midlife, and AAM were considered confounders in the models in which they were not treated as independent variables.

In Study I, McNemar's test was used to analyze the differences in the proportions of participants with MD in adolescence and young adulthood. Binary logistic regression was used to investigate the associations of adolescents' BMI, MD, attitudes toward body weight, physical activity, and AAM on MD in young adulthood in Study I and to determining crude and adjusted odds ratios (OR) and their 95% confidence intervals (CIs) for the association of ED, restrictive eating, and MD with injury occurrence and missed participation days in Study III.

In Study IV, longitudinal analyses of the EDE-QS scores at different age stages were conducted using Friedman's non-parametric test for repeated measures with pairwise post-hoc comparisons. Friedman's test was used because of the non-normal distribution of the outcome variable. Associations of primary and secondary amenorrhea and eating behaviors at different age stages with sports career-related outcome variables were analyzed using generalized estimation equation (GEE) models with an exchangeable working correlation matrix. GEE does not require outcome variables to be normally distributed, and it can be used in estimating population-average effects in repeatedly measured data as it takes into account the within-subject correlation between the different measurements (Ballinger, 2004). In the GEE models, primary and secondary amenorrhea, together with EDE-QS scores, were used as independent variables. Models were adjusted for the age phase (i.e., time). Only EDE-QS scores at the

age of each athlete's sports career were considered and scores after the age of sports career termination were excluded. Ages 13–15, 16–18, and 19–21 years were included in the models, as most of the athletes had terminated their sports careers by the age of 23 years. Missing data were not imputed.

All statistical analyses in Studies I–III and descriptive analyses, as well as the Friedman's test in Study IV were conducted using the Statistical Package for Social Sciences (SPSS) Versions 24.0 and 26.0 (IBM, Armonk, NY, USA). The GEE models in Study IV were performed using R Project for Statistical Computing version 4.0.2 (R core team, Vienna, Austria). Statistical significance was set at $p < .05$, two-tailed.

5 OVERVIEW OF THE RESULTS

5.1 Participant characteristics

The background characteristics of the participants in the FHPSC (Study I), ERMA (Study II), Female Athlete 2.0 (Study III), and MEBS (Study IV) studies whose data were used in this dissertation study are presented in Table 4. Menstrual cycle-related characteristics from these participants are shown in Table 5.

The athletes had higher AAM compared with the non-athletes in Study I in adolescence and in Study IV, while no differences in AAM were found among young adult athletes and non-athletes in Study I or between former athletes and non-athletes in Study II (Table 5). However, when the participants of Study II were classified into groups of AAM ≤ 12 , 13, and ≥ 14 years of age, the athletes and the regular physical activity group differed from the no-exercise group in terms of AAM ($p=0.01$; data not shown).

TABLE 4 Background characteristics of the participants from the different datasets used in this study.

	FHPSC (Study I)		FHPSC2 (Study I)		ERMA (Study II)			FA 2.0 (Study III)	MEBS (Study IV)	
	Athletes (n=178)	Non-athletes (n=105)	Athletes (n=52)	Non-athletes (n=159)	Athletes (n=136)	RPA (n=689)	NE (n=163)	Athletes (n=846)	Athletes (n=100)	Non-athletes (n=98)
Age, years	14.9 ± 0.6	15.0 ± 0.4	18.8 ± 0.7	18.9 ± 0.7	50.9 ± 2.0*	51.4 ± 2.1*	51.6 ± 2.0*	24.3 ± 7.5	39.6 ± 4.5	39.4 ± 4.5
BMI, kg/m ²	20.9 ± 2.2*	21.8 ± 3.1*	22.2 ± 2.4	23.4 ± 3.9 ^a	25.4 ± 3.7 ^b	25.6 ± 3.8 ^c	25.2 ± 3.4 ^d	23.2 ± 3.6 ^e	22.0 ± 2.5^{f*}	26.2 ± 5.8*
Bachelor or higher education, n (%)	N/A	N/A	N/A	N/A	66 (48.5)*	286 (41.5)*	56 (34.4)*	N/A	N/A	N/A
Number of parities	N/A	N/A	N/A	N/A	2.0 ± 1.1*	2.1 ± 1.2^{g*}	1.9 ± 1.4*	N/A	N/A	N/A
Menopausal status, n (%)	N/A	N/A	N/A	N/A				N/A	N/A	N/A
Premenopausal					41 (30.1)	189 (27.4)	40 (24.5) ^h			
Early perimenopausal					26 (19.1)	128 (18.6)	32 (19.0) ^h			
Late perimenopausal					29 (21.3)	133 (19.3)	34 (20.9) ^h			
Postmenopausal					40 (29.4)	239 (34.7)	58 (35.6) ^h			
MVPA/day, hr:min	1:19 ± 0:28^{i*}	0:59 ± 0:23^{i*}	1:21 ± 0:38^{k*}	0:54 ± 0:30^{l*}	0:54 ± 0:31 ^m	0:50 ± 0:26 ⁿ	0:47 ± 0:23 ^o	N/A	N/A	N/A
Step count/day	9310 ± 2816^{i*}	7674 ± 2535^{j*}	10117 ± 4273^{k*}	7236 ± 3179^{l*}	8903 ± 2887 ^m	8698 ± 2872 ⁿ	8510 ± 2398 ^o	N/A	N/A	N/A
LTPA, MET-hr/day	N/A	N/A	N/A	N/A	4.9 ± 4.0*	4.2 ± 3.7*	3.6 ± 2.8*	N/A	N/A	N/A

Data are presented as means ± standard deviations unless otherwise indicated.

^an=158; ^bn=113; ^cn=609; ^dn=146 ^en=841; ^fn=99; ^gn=687; ^hn=161; ⁱn=164; ^jn=87; ^kn=39; ^ln=130; ^mn=88; ⁿn=516; ^on=126.

*Denotes statistically significant difference (p<.05) between the groups of the same dataset.

FHPSC1 = the Finnish Health Promoting Sports Club study baseline; FHPSC2 = the Finnish Health Promoting Sports Club study follow-up; ERMA = Estrogenic Regulation of Muscle Apoptosis study; FA = the Female Athlete 2.0 study; MEBS = the Menstrual Function, Eating Attitudes, Body Image, and a Sports Career study; RPA = regular physical activity group; NE = no-exercise group; BMI = body mass index; N/A = not applicable; MVPA = moderate-to-vigorous physical activity; LTPA = leisure-time physical activity (self-reported); MET = metabolic equivalent of task.

TABLE 5 Menstrual cycle-related characteristics of the participants from the different datasets used in this study.

	FHPSC (Study I)		FHPSC2 (Study I)		ERMA (Study II)			FA 2.0 (Study III)	MEBS (Study IV)	
	Athletes (n=178)	Non-athletes (n=105)	Athletes (n=52)	Non-athletes (n=159)	Athletes (n=136)	RPA (n=689)	NE (n=163)	Athletes (n=846)	Athletes (n=100)	Non-athletes (n=98)
AAM, years	12.7 ± 1.1^{a*}	12.2 ± 1.1^{b*}	12.9 ± 1.5 ^c	12.5 ± 1.3	13.0 ± 1.3 ^d	13.0 ± 1.4 ^e	12.8 ± 1.2 ^f		14.0 ± 2.0[*]	12.5 ± 1.2^{h*}
<12 years, n (%)								104 (12.7) ^g		
12-14 years, n (%)								599 (73.0) ^g		
≥15 years, n (%)								117 (14.3) ^g		
Gynecological age, years	2.4 ± 1.2^{a*}	2.8 ± 1.1^{b*}	5.9 ± 1.6 ^c	6.3 ± 1.4	N/A	N/A	N/A	N/A	N/A	N/A
Age at regular menses, years	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	16.0 ± 3.7^{i*}	14.7 ± 2.9^{i*}
Hormonal contraceptive use, n (%)	9 (5.1)[*]	18 (17.1)[*]	21 (40.4) ^j	79 (49.7) ^k	85 (62.5)^{§*}	350 (50.8)^{§*}	80 (49.1)^{§*}	352 (42.6)	N/A	N/A

Data are presented as means ± standard deviations unless otherwise indicated.

^an=165; ^bn=104; ^cn=50; ^dn=134; ^en=677; ^fn=162; ^gn=820; ^hn=97; ⁱn=82; ^jn=51; ^kn=157.

*Denotes statistically significant difference (p<.05) between the groups of the same dataset; §At some point of life.

FHPSC1 = the Finnish Health Promoting Sports Club study baseline; FHPSC2 = the Finnish Health Promoting Sports Club study follow-up; ERMA = the Estrogenic Regulation of Muscle Apoptosis study; FA = the Female Athlete 2.0 study; MEBS = the Menstrual Function, Eating Attitudes, Body Image, and Sports a Career study; RPA = regular physical activity group; NE = no-exercise group; AAM = age at menarche; N/A = not applicable.

The athletes in this study engaged in various sports disciplines. In Study I, there were 53 (29.8%) endurance athletes, 62 (34.8%) aesthetic athletes, 42 (23.6%) ball game athletes, 18 (10.1%) athletes in power or anti-gravitation sports, and 3 (1.7%) technical sports athletes in adolescence. In young adulthood, the number of athletes in each sports category was as follows: endurance, n=22 (42.3% of the athletes); aesthetic, n=12 (23.1%); ball games, n=10 (19.2%); and power/anti-gravitation: n=8 (15.4%). None of the athletes in young adulthood engaged in technical sports.

In Study II, the most common sports disciplines among the participants who participated in competitive sports at age 13–16 were track and field (n=39, 28.7%), volleyball (n=28, 20.6%), cross-country skiing (n=25, 18.4%), running (n=23, 16.9%), Finnish baseball (n=10, 7.4%), and gymnastics (n=10, 7.4%). Most of the participants who engaged in competitive sports at age 13–16 reported competing in two or more sports disciplines (n=98, 72.1%).

The athletes in Study III represented 67 different sports disciplines. A total of 545 (64.4%) of the athletes engaged in lean sports, while the remaining 301 (35.6%) participated in non-lean sports. The most common lean sports among the athletes were weightlifting (n=51, 6.0% of the whole sample), figure skating (n=51, 6.0%), cheerleading (n=43, 5.1%), and cross-country skiing (n=42, 5.0%). Among non-lean sport athletes, the most common sports were soccer (n=78, 9.2%), ice hockey (n=33, 3.9%), and American football (n=22, 2.6%). A total of 625 (73.9%) of the athletes were classified as elite athletes (national or international level), and 496 (58.6%) of the athletes were grouped into the younger athletes group.

In Study IV, 83 (83%) of the athletes had had only one main discipline, while 17 had specialized in at least two disciplines. The disciplines in which the athletes had started their sports careers were distance running (n=64, 64%), track and field (n=16, 16%), cross-country skiing (n=12, 12%), and other (orienteering, swimming, gymnastic, and Finnish baseball; n=8, 8%). Participants who had commenced their sports careers in gymnastics or Finnish baseball had later changed to endurance sports. All track and field athletes had competed in middle- and long-distance running, and one had also participated in jumping disciplines.

Table 6 shows the age, BMI, and training hours of the athletes classified according to their level of participation, age, and type of sports (lean/non-lean) in Study III. Elite athletes were younger and trained more than non-elite athletes, younger athletes had lower BMI and higher training hours than older athletes, and lean sport athletes had lower BMI than non-lean sport athletes (Table 6). Moreover, over half (55.3%) of the participants in Study III reported sustaining an injury during the preceding year, and 30.8% of those athletes reported that they had missed at least 22 participation days due to an injury (data not shown). In Study I, no statistically significant differences in MD were found between the athletes who engaged in different types of sports, but the number of athletes in each category was low.

TABLE 6 Age, body mass index (BMI), and training hours of the athletes in Study III classified according to their competition level, age, and type of sport.

	Age (years), mean \pm SD	BMI (kg/m ²), median (IQR)	Training hours ^a , median (IQR)
Non-elite athletes	27.0 (8.6)* (n=221)	22.8 (21.0–25.1) (n=219)	484 (250–574)* (n=189)
Elite athletes	23.3 (6.8)* (n=625)	22.5 (20.8–24.2) (n=622)	600 (450–786)* (n=537)
Younger athletes	19.0 (2.6)* (n=496)	22.5 (20.7–23.8)* (n=491)	624 (436–850)* (n=413)
Older athletes	31.8 (5.4)* (n=350)	23.2 (21.1–25.6)* (n=350)	467 (300–600)* (n=313)
Lean sport athletes	24.3 (5.8) (n=545)	22.3 (20.6–24.1)* (n=544)	540 (390–730) (n=484)
Non-lean sport athletes	24.4 (6.9) (n=301)	23.2 (21.6–25.4)* (n=297)	561 (380–728) (n=242)

^aDuring the preceding year.

*Statistically significant difference ($p < .05$) compared to the comparison group.

SD = standard deviation; IQR = interquartile range.

In Study IV, 94% (n=94) of the athletes reported that they had ended their sports careers. Among those who had already retired, the mean length of the career was 12.0 ± 5.8 years (n=92). Thirty-four athletes (34%) reported having competed at the international level (Tier ≥ 4 ; McKay et al. 2021), while 65 (65%) had competed at the national level (Tier 3), and one (1%) reported that the regional/district level (Tier 2) was her highest competition level. Sixteen athletes (n=16%) reported that injuries or musculoskeletal pain had affected their sports career quite a lot or a significant amount, and 44.7% (n=42) of the retired athletes reported that an injury had influenced their sports career termination.

Ten (23.8%) of the retired athletes in Study IV reported that an acute injury had contributed to their career termination, while 34 (81.0%) athletes reported that an overuse injury played a role in their career termination. Indeed, two athletes reported that acute and overuse injuries contributed to their career termination. Twenty (47.6%) athletes who had terminated their career because of injuries identified one single contributing injury, while 22 (52.4%) athletes reported several injuries that had impacted their decision to terminate their career. The most common acute injury reported was ligament/joint capsule injury (n=6, 14.3%), and the most common overuse injuries were muscle/tendon injuries and non-specific injuries, i.e., undiagnosed pains (n=13, 31.0% for both).

5.2 Prevalence of low energy availability-related conditions among athletes and non-athletes

Tables 7 and 8 summarize the prevalence rates of low EA-related conditions among study participants from different datasets and comparisons between

athletes and non-athletes. In general, athletes tended to have higher rates of MD and delayed puberty, while non-athletes tended to be more dissatisfied with their weight than athletes and wanted to lose weight more often (Table 7). No differences were found in lifetime ED prevalence (Table 7) or eating behaviors between athletes and non-athletes at any age (Table 8). However, athletes and non-athletes differed in terms of changes in the EDE-QS scores over time, with the trends in the EDE-QS scores of athletes fluctuating more than the scores of non-athletes. Among the athletes, significant differences were observed between ages 13–15 and 16–18 ($p < .001$), 13–15 and 19–21 ($p = .004$), 16–18 and 22–25 ($p = .038$), 16–18 and currently ($p = .001$), and between ages 19–21 and currently ($p = .008$). Among the non-athletes, significant differences were found only between ages 13–15 and 22–25 ($p = .013$) and between ages 22–25 and current EDE-QS scores ($p = .034$). However, after the Bonferroni correction for multiple tests, significant differences were observed only among the athletes between ages of 13–15 and 16–18 ($p = .002$), 13–15 and 19–21 ($p = .040$), and 16–18 and currently ($p = .005$), while no differences were found among the non-athletes between the respective age ranges (Table 8).

TABLE 7 Low energy availability-related conditions in athletes and non-athletes in the Finnish Health Promoting Sports Club study base-line (FHPSC1), Finnish Health Promoting Sports Club study follow-up (FHPSC2), Estrogenic Regulation of Muscle Apoptosis study (ERMA), Female athlete 2.0 study (FA 2.0) and Menstrual Function, Eating Attitudes, Body Image, and a Sports Career study (MEBS).

	FHPSC1 (Study I)		FHPSC2 (Study I)		ERMA (Study II)		FA 2.0 (Study III)	MEBS (Study IV)	
	Athletes	Non-athletes	Athletes	Non-athletes	Athletes	Non-athletes [§]	Athletes	Athletes	Non-athletes
Current or past eating disorder, n (%)	N/A	N/A	N/A	N/A	N/A	N/A	155/843 (18.4)	7/98 (7.1)	8/95 (8.4)
Restrictive eating, n (%)	N/A	N/A	N/A	N/A	N/A	N/A	207/841 (24.6)	N/A	N/A
Current menstrual dysfunction, n (%)	25/141 (17.7)	14/80 (17.5)	12/31 (38.7)	4/71 (5.6)	N/A	N/A	160/506 (31.6)	N/A	N/A
Secondary amenorrhea, n (%)*	N/A	N/A	N/A	N/A	N/A	N/A	N/A	31/91 (34.1)	20/98 (20.4)
Regular menses, n (%)*	N/A	N/A	N/A	N/A	N/A	N/A	N/A	50/92 (54.3)	72/92 (78.3)
Cycle length 21–35 days, n (%)*	N/A	N/A	N/A	N/A	N/A	N/A	N/A	55/85 (64.7)	81/91 (89.0)
Primary amenorrhea ^a , n (%)	14/172 (8.1)	0/102 (0.0)	6/50 (12.0)	12/159 (7.5)	5/134 (3.7)	33/839 (3.9)	129/832 (15.5)	20/100 (20.0)	2/98 (2.0)
Spontaneous menarche, n (%)	N/A	N/A	N/A	N/A	N/A	N/A	821/838 (98.0)	93/100 (93.0)	98/98 (100.0)
Delayed puberty, n (%)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	13/100 (13.0)	2/98 (2.0)
Dissatisfied with weight, n (%)	32/174 (18.4)	46/104 (44.2)	12/51 (23.5)	59/157 (37.6)	N/A	N/A	N/A	N/A	N/A
Desire to lose weight, n (%)	33/171 (19.3)	45/103 (43.7)	15/51 (29.4)	72/155 (46.5)	N/A	N/A	N/A	N/A	N/A
Desire to gain weight, n (%)	24/172 (14.0)	4/100 (4.0)	1/48 (2.1)	8/148 (5.4)	N/A	N/A	N/A	N/A	N/A

[§]Regular physical activity and no-exercise groups combined; *During the sports career (athletes) or ever (non-athletes). Statistically significant differences (p<.05) between athletes and non-athletes from the same dataset are indicated in bold. N/A = not applicable.

TABLE 8 Eating disorder examination questionnaire short form (EDE-QS) scores of the athletes and non-athletes in Study IV.

	Athletes (n=100)	Non-athletes (n=98)	P-value
EDE-QS scores, mean \pm SD			
At age 13–15	6.3 \pm 7.3	6.4 \pm 7.4	.943
At age 16–18	8.4 \pm 7.9*	7.3 \pm 7.7	.340
At age 19–21	8.0 \pm 8.0 ^{a,*}	7.4 \pm 7.8	.589
At age 22–25	7.2 \pm 7.4 ^b	7.5 \pm 7.8	.737
Currently	5.6 \pm 6.4 ^s	6.0 \pm 6.2	.697
EDE-QS \geq 15, n (%)			
At age 13–15	16 (16.0)	15 (15.3)	.893
At age 16–18	20 (20.0)	18 (18.4)	.771
At age 19–21	19 (19.8) ^a	15 (15.3)	.411
At age 22–25	17 (18.9) ^b	13 (13.3)	.293
Currently	12 (12.0)	10 (10.2)	.688

^an=96; ^bn=90.

*Differed statistically significantly ($p < .05$) from the EDE-QS scores at age 13–15 after the Bonferroni correction for multiple tests; ^sDiffered statistically significantly ($p < .05$) from the EDE-QS scores at age 16–18 after the Bonferroni correction for multiple tests. SD = standard deviation.

Table 9 presents the prevalence rates and differences in current or previous ED, restrictive eating, and MD by participation level, age, and type of sports (Study III). No differences were found between elite and non-elite athletes. Younger athletes reported higher rates of MD and lower rates of ED, while lean sport athletes exhibited higher rates of ED, restrictive eating, and MD.

TABLE 9 Proportion of athletes reporting current or past eating disorder (ED), restrictive eating (RE), and menstrual dysfunction (MD) by participation level, age, and type of sports in Study III.

	ED	RE	MD
Non-elite athletes, n (%)	40/221 (18.1)	54/219 (24.7)	33/122 (27.0)
Elite athletes, n (%)	115/622 (18.5)	153/622 (24.6)	127/384 (33.1)
OR (95% CI)	0.97 (0.66–1.45)	1.00 (0.70–1.43)	0.75 (0.48–1.18)
Younger athletes, n (%)	72/494 (14.6)	116/494 (23.5)	111/317 (35.0)
Older athletes, n (%)	83/349 (23.8)	91/347 (26.2)	49/189 (25.9)
OR (95% CI)	0.55 (0.39–0.78)	0.86 (0.63–1.19)	1.54 (1.03–2.29)
Lean sport athletes, n (%)	112/543 (20.6)	146/542 (26.9)	115/323 (35.6)
Non-lean sport athletes, n (%)	43/300 (14.3)	61/299 (20.4)	45/183 (24.6)
OR (95% CI)	1.55 (1.06–2.28)	1.44 (1.03–2.02)	1.70 (1.13–2.55)

Statistically significant differences ($p < .05$) are indicated in bold.

OR = odds ratio; CI = confidence interval.

Differences in attitudes toward weight between athletes and non-athletes of different weight categories in adolescence (Study I) are presented in Table 10. Athletes in the normal-weight category were less dissatisfied with their weight ($p = .006$) than non-athletes in that category. Normal-weight athletes had a lower

desire to lose weight ($p=.01$) and a higher desire to gain weight ($p=.04$) than non-athletes in the same weight category. Similarly, athletes in the overweight category were less dissatisfied with their weight than non-athletes in the same category ($p=.004$). Athletes in the overweight category also wished to lose weight less often than non-athletes in the overweight category ($p=.004$).

TABLE 10 Attitudes toward weight in adolescent athletes and non-athletes by different weight groups in Study I.

	Athletes			Non-athletes		
	Under-weight (n=7)	Normal weight (n=150)	Over-weight (n=18)	Under-weight (n=7)	Normal weight (n=70)	Overweight (n=27)
Dissatisfied with weight	1 (14.3)	23 (15.3)^N	8 (44.4)^O	1 (14.3)	22 (31.4)^N	23 (85.2)^O
Desire to lose weight	0 (0.0)	27 (18.0)^N	8 (44.4)^O	0 (0.0)	23 (32.9)^N	23 (85.2)^O
Desire to gain weight	2 (28.6)	20 (13.3)^N	1 (5.6)	1 (14.3)	3 (4.3)^N	0 (0.0)

Data are presented as counts (percentages). N and O denote statistically significant difference ($p<.05$) between athletes and non-athletes in the normal and overweight categories, respectively.

In young adulthood (Study I), athletes in the overweight category less dissatisfied with their weight ($p=.005$) and had less desire to lose weight ($p=.001$) than non-athletes in that category. No differences in attitudes toward weight were found between athletes and non-athletes in the normal and underweight categories (Table 11).

TABLE 11 Attitudes toward weight in young adult athletes and non-athletes by different weight groups in Study I.

	Athletes			Non-athletes		
	Under-weight (n=1)	Normal weight (n=43)	Overweight (n=8)	Under-weight (n=10)	Normal weight (n=107)	Overweight (n=40)
Dissatisfied with weight	0 (0.0)	10 (23.3)	2 (25.0)^o	1 (10.0)	27 (25.2)	32 (80.0)^o
Desire to lose weight	0 (0.0)	13 (30.2)	2 (25.0)^o	0 (0.0)	37 (34.6)	35 (87.5)^o
Desire to gain weight	0 (0.0)	1 (2.3)	0 (0.0)	4 (40.0)	4 (3.7)	0 (0.0)

Data are presented as counts (percentages). O denotes statistically significant difference ($p < .05$) between athletes and non-athletes in the overweight category.

5.3 Associations between age at menarche and midlife characteristics

Midlife health-related physical fitness components and bone health of the participants according to their menarcheal age in Study II are presented in Table 12. There was a linear downward trend in middle-aged BMI, fat percentage, and fat mass index across the menarcheal age groups, with later AAM being associated with a lower BMI, fat percentage, and fat mass index in middle age. All pairwise comparisons were statistically significant except for fat percentage between the group of AAM ≤ 12 years and the group of AAM = 13 years. Femoral neck BMD was higher in the group reporting AAM of ≤ 12 years compared with the group with AAM of ≥ 14 years. The only difference in cardiorespiratory and muscular fitness tests between the AAM groups was found in jumping height, where the group of AAM ≥ 14 years performed better than the group of AAM ≤ 12 years.

The differences in BMI, fat percentage, fat mass index, and jumping height remained significant after adjusting for confounding factors. No differences between the groups were found in BMD after the adjustment, but an exclusion of postmenopausal women from this analysis demonstrated an inverse association between AAM and BMD even after adjusting for confounding factors ($p = .041$).

TABLE 12 Midlife health-related physical fitness components and bone health of participants according to their age at menarche (AAM) in Study II.

	AAM ≤ 12	AAM = 13	AAM ≥ 14	P-value	Adj. p-value ^a
BMI, kg/m ²	26.2 ± 3.8 ^{M,L} (n=335)	25.4 ± 3.5 ^L (n=341)	24.6 ± 3.5 (n=266)	<.001	<.001
Fat percentage	36.4 ± 6.9 ^L (n=315)	35.1 ± 7.4 ^L (n=319)	33.1 ± 8.0 (n=247)	<.001	<.001
FMI, kg/m ²	9.7 ± 3.0 ^{M,L} (n=315)	9.1 ± 3.0 ^L (n=319)	8.4 ± 3.1 (n=247)	<.001	<.001
LMI, kg/m ²	15.5 ± 1.3 (n=315)	15.3 ± 1.3 (n=319)	15.3 ± 1.2 (n=247)	.135	.047
ALMI, kg/m ²	6.7 ± 0.7 (n=315)	6.6 ± 0.7 (n=319)	6.6 ± 0.6 (n=247)	.237	.124
FN BMD, g/cm ²	0.97 ± 0.1 ^L (n=315)	0.96 ± 0.1 (n=319)	0.94 ± 0.1 (n=247)	.034	.056
Hand grip, N	314 ± 62 (n=321)	315 ± 59 (n=324)	311 ± 57 (n=254)	.715	.195
Knee extension force, N	469 ± 95 (n=282)	458 ± 90 (n=271)	460 ± 100 (n=230)	.344	.442
Jumping height, cm	18.7 ± 4.1 ^L (n=300)	19.2 ± 4.1 (n=302)	19.8 ± 4.5 (n=243)	.013	.013
Walking speed, m/s	2.6 ± 0.5 (n=319)	2.7 ± 0.4 (n=324)	2.7 ± 0.5 (n=255)	.443	.507
6-min walking dist., m	666 ± 61 (n=300)	670 ± 57 (n=293)	674 ± 63 (n=240)	.259	.093

The data are presented as means ± standard deviations.

^aAdjusted for physical activity level at ages 13–16, self-reported leisure-time physical activity in midlife, age, menopausal status, number of parities, education, and hormonal contraceptive use during the 10 preceding years.

M and L indicate statistically significant differences ($p < .05$) between the middle and the latest menarcheal groups, respectively.

Statistically significant p-values are indicated in bold.

BMI = body mass index; FMI = fat mass index; LMI = lean mass index; ALMI = appendicular lean mass index; FN BMD = femoral neck bone mineral density.

5.4 Associations between sports participation in adolescence and midlife characteristics

The correlation coefficients between the physical activity level at age 13–16 and the physical activity levels at ages 7–12, 17–19, 20–29, 30–39, and 40–50 are presented in Table 13 (Study II). The correlations with the physical activity level at age 13–16 were at their highest at ages 7–12 and 17–20 and gradually decreased thenceforth. All the other correlation coefficients were statistically significant except for the correlation between physical activity levels at ages 13–16 and 40–50.

TABLE 13 Correlation coefficients and their p-values between the physical activity level at age 13–16 and other age categories in Study II.

Age category	Correlation coefficient	P-value
7-12	.54	<.001
17-19	.60	<.001
20-29	.29	<.001
30-39	.19	<.001
40-50	-.02	.463

Statistically significant correlations ($p < .05$) are indicated in bold.

Table 14 presents the midlife health-related physical fitness components and bone health of the participants according to their physical activity level at age 13–16 in Study II. The athletes had higher lean mass index, ALMI, and femoral neck BMD than the participants in the other two groups. They also performed better in cardiorespiratory and muscular fitness tests. Differences in lean mass index, ALMI, femoral neck BMD, and physical fitness test results between the athletes and the other two groups persisted after adjusting for potential confounders.

TABLE 14 Midlife characteristics of participants according to physical activity level at age 13–16 in Study II.

	Athletes	Regular PA	No exercise	P-value	Adj. p-value ^a
Fat percentage	33.3 ± 7.3 ^R (n=101)	35.4 ± 7.5 (n=584)	35.0 ± 7.2 (n=138)	.028	.026
FMI, kg/m ²	8.7 ± 2.9 (n=101)	9.3 ± 3.1 (n=584)	9.0 ± 2.8 (n=138)	.143	.062
LMI, kg/m ²	15.8 ± 1.3 ^{R,N} (n=101)	15.3 ± 1.3 (n=584)	15.2 ± 1.3 (n=138)	.002	.001
ALMI, kg/m ²	6.9 ± 0.7 ^{R,N} (n=101)	6.6 ± 0.6 (n=584)	6.6 ± 0.7 (n=138)	<.001	.001
FN BMD, g/cm ²	1.00 ± 0.13 ^{R,N} (n=101)	0.96 ± 0.12 (n=584)	0.95 ± 0.12 (n=138)	<.001	.002
Hand grip force, N	334 ± 57 ^{R,N} (n=106)	314 ± 60 (n=590)	306 ± 54 (n=142)	.001	.002
Knee extension force, N	509 ± 100 ^{R,N} (n=92)	461 ± 90 (n=508)	442 ± 91 (n=118)	<.001	<.001
Jumping height, cm	21.3 ± 4.1 ^{R,N} (n=101)	19.0 ± 4.1 (n=547)	18.7 ± 4.1 (n=136)	<.001	<.001
Walking speed, m/s	2.9 ± 0.5 ^{R,N} (n=106)	2.6 ± 0.4 (n=591)	2.6 ± 0.4 (n=140)	<.001	<.001
6-min walking dist., m	697 ± 58 ^{R,N} (n=95)	668 ± 61 (n=543)	661 ± 55 (n=131)	<.001	.001

The data are presented as means ± standard deviations.

^aAdjusted for self-reported leisure-time physical activity in midlife, age, menarcheal age, menopausal status, number of parities, education, and hormonal contraceptive use during the 10 preceding years.

R and N indicate significant difference ($p < .05$) from the regular physical activity and no-exercise groups, respectively.

Statistically significant p-values are indicated in bold.

PA = physical activity; FMI = fat mass index; LMI = lean mass index; ALMI = appendicular lean mass index; FN BMD = femoral neck bone mineral density.

5.5 Factors associated with eating problems, body weight perceptions, and menstrual dysfunction

5.5.1 Cross-sectional differences and associations

In Study III, athletes who reported current or previous ED or restrictive eating were more likely to have suffered an injury during the previous year than those who did not report these eating problems. No differences were found in injury occurrence by menstrual status; however, athletes who reported MD were more likely to report ≥ 22 days of injury-related absence from training or competitions in the preceding year than those who reported menstruating regularly (Table 15).

TABLE 15 Frequencies and proportions of athletes with one or more injuries during the preceding year and athletes with ≥ 22 missed participation days due to injuries during the preceding year along with crude odds ratios by eating disorders (ED), restrictive eating (RE), and menstrual dysfunction (MD) in Study III.

One or more injuries				≥ 22 missed participation days due to injuries ^a			
Variable	n	Proportion (%)	OR (95% CI)	Variable	n	Proportion (%)	OR (95% CI)
No ED	362	52.6	Ref	No ED	117	32.6	Ref
ED	105	67.7	1.89 (1.31–2.73)	ED	26	25.0	0.69 (0.42–1.13)
No RE	339	53.5	Ref	No RE	104	30.8	Ref
RE	128	61.8	1.41 (1.02–1.94)	RE	39	31.2	1.02 (0.66–1.59)
No MD	187	54.0	Ref	No MD	50	27.3	Ref
MD	87	54.4	1.01 (0.70–1.48)	MD	35	40.2	1.79 (1.05–3.07)

^a Only injured participants were included in this analysis.

Statistically significant odds ratios ($p < .05$) are indicated in bold.

OR = odds ratio; CI = confidence interval.

The association of ED (OR 2.29, 95% CI 1.52–3.44) and restrictive eating (OR 1.50, 95% CI 1.05–2.14) with the occurrence of injury during the previous year in all participants persisted after adjusting for BMI, training hours in the preceding year, and age. However, in the adjusted model, no association was found between MD and missed participation days (OR 1.63, 95% CI 0.91–2.91).

When investigating these associations in different groups of athletes, the relationship between ED and injuries was found among elite (OR 1.91, 95% CI 1.24–2.93) and non-elite athletes (OR 2.41, 95% CI 1.11–5.23), among older (OR 2.46, 95% CI 1.44–4.19), lean (OR 1.91, 95% CI 1.24–2.93), and non-lean athletes (OR 2.41, 95% CI 1.11–5.23). The association between restrictive eating and injury occurrence was observed among non-elite athletes (OR 1.90, 95% CI 1.01–3.57) and lean sport athletes (OR 1.68, 95% CI 1.14–2.47), but not among elite, non-lean, younger, or older athletes. The relationship between MD and ≥ 22 missed participation days was observed among elite (OR 2.37, 95% CI 1.28–4.36) and lean sport athletes (OR 2.07, 95% CI 1.05–4.11).

Table 16 shows differences in BMI, training volume, and MVPA in athletes with and without MD (Studies I and III), ED, and restrictive eating (Study III). Athletes who reported MD had lower BMI (Study I, adolescence, and Study III), trained more (Study III), and had higher rates of MVPA (Study I, young adulthood) than those who reported regular menstrual cycles. However, athletes who reported MD in adolescence in Study I reported fewer weekly training hours than those who did not report MD. No differences in BMI or training volume were found among those with and without ED or restrictive eating.

TABLE 16 Body mass index (BMI), training volume, and amount of moderate-to-vigorous physical activity (MVPA) among participants reporting and not reporting current or past eating disorder (ED), restrictive eating (RE), and menstrual dysfunction (MD) in Studies I and III.

	BMI, kg/m ²	Training volume, hr*	MVPA per day, hr:min
Study I, adolescence	mean ± SD	mean ± SD	mean ± SD
MD [§] (n=31)	20.1 ± 2.0	8.9 ± 3.5^a	1:17 ± 0:30 ^c
No MD (n=116)	21.5 ± 2.2	10.5 ± 4.3^b	1:18 ± 0:26 ^d
P-value	0.002	0.042	0.907
Study I, young adulthood	mean ± SD	mean ± SD	mean ± SD
MD (n=12)	22.6 ± 3.3	12.7 ± 4.6	1:37 ± 0:22^e
No MD (n=19)	22.6 ± 2.5	10.9 ± 4.3	0:57 ± 0:26^f
P-value	0.985	0.289	0.001
Study III	median (IQR)	median (IQR)	
ED (n=155)	22.3 (20.6–24.1)	555 (366–726) ^h	N/A
No ED (n=688)	22.6 (21.0–24.4) ^g	540 (400–730) ⁱ	N/A
P-value	.198	.983	
RE (n=207)	22.5 (20.6–24.8) ^j	600 (364–800) ^l	N/A
No RE (n=634)	22.6 (21.0–24.3) ^k	520 (390–720) ^m	N/A
P-value	.741	.092	
MD (n=160)	21.6 (20.1–23.6)ⁿ	627 (414–832)^p	N/A
No MD (n=346)	22.7 (21.0–24.3)^o	550 (400–720)^q	N/A
P-value	<.001	.041	

^an=27; ^bn=113; ^cn=26; ^dn=107; ^en=9; ^fn=14; ^gn=683; ^hn=136; ⁱn=588; ^jn=205; ^kn=631; ^ln=179; ^mn=543; ⁿn=158; ^on=344; ^pn=134; ^qn=294.

*During a week in the training season in Study I and during the preceding year in Study III; [§]Current MD (n=25) or history of primary amenorrhea within the preceding year (n=6). Statistically significant differences (p<.05) between the groups are indicated in bold. SD = standard deviation; IQR = interquartile range; N/A = not applicable.

In Study I in adolescence, higher weight satisfaction was associated with lower BMI among the athletes (p=.01), while a higher desire to lose weight was linked with higher BMI among the athletes (p=.02). No relationship was found between a desire to gain weight and BMI in the adolescent athletes (p=.28). In the young adult athletes, no differences were found in attitudes toward weight between the three weight groups.

5.5.2 Longitudinal associations

In the longitudinal analysis in Study I, MD in adolescence was the only variable that predicted MD in young adulthood in those who had been athletes in adolescence (OR 18.00, 95% CI 3.69–87.70, $p=.001$) and also in all participants (OR 11.33, 95% CI 3.26–39.34, $p<.001$). BMI, body weight perceptions, physical activity, or AAM assessed in adolescence were not found to be associated with MD in young adulthood ($p>.05$ for all).

In Study IV, 19 (19%) of the athletes reported DE during their sports career and 17 of them had retired from sports. Thirty-five percent ($n=6$) of the retired athletes with DE behaviors during their sports career also reported current DE compared with 8% ($n=6$) of the retired athletes reporting current DE without DE during their sports career ($p=.001$). A similar association was observed among the non-athletes: current DE was more likely among those who reported DE at least at some point during ages 13–21 than among those without DE at that age (43% vs 1%, $p<.001$).

The GEE model in Study IV showed an inverse relationship between EDE-QS scores and sports career length, with one point in the EDE-QS score associated with a 0.15-year shorter sports career. In addition, secondary amenorrhea was associated with a lower participation level, injury-related harms during the career, and injury-related sports career termination (Table 17).

TABLE 17 Associations of eating behaviors and menstrual history with sports career length, participation level, injury-related harms during the sports career, and injury-related sports career termination in Study IV.

	Career length ($n=100$)		Participation level ($n=100$)		Injury-related harms ($n=100$)		Injury-related career termination ($n=94$)	
	B	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
PA	2.28	-0.55, 5.11	1.09	0.54–2.21	1.22	0.53–2.81	0.61	0.30–1.23
SA	-0.16	-2.05, 1.72	0.51	0.27–0.95	4.00	1.88–8.48	1.89	1.02–3.51
EDE-QS	-0.15	-0.26, -0.05	0.99	0.95–1.03	0.97	0.93–1.01	1.01	0.97–1.05

Models are adjusted for age (categories 13–15, 16–18, and 19–21).

Statistically significant differences ($p<.05$) are indicated in bold.

B = unstandardized regression coefficient; CI = confidence interval; OR = odds ratio; PA = primary amenorrhea; SA = secondary amenorrhea; EDE-QS = Eating Disorder Examination Questionnaire short form scores.

6 DISCUSSION

This dissertation study showed that low EA-related conditions, such as ED, DE behaviors, body weight dissatisfaction, and MD are not uncommon problems among Finnish female athletes. However, while MD was more common in athletes, athletes did not differ from their non-athletic peers in terms of ED or DE behaviors. In addition, athletes showed less body weight dissatisfaction and a desire to lose weight than did non-athletes. Athletes who competed in lean sports were at a higher risk for ED, DE, and MD than athletes who participated in non-lean sports, and younger athletes exhibited higher rates of MD than older athletes. No association was found between DE behaviors and athletes' levels of competition. Later AAM was associated with lower BMI and fat mass at midlife, but also lower BMD in pre- and perimenopausal women regardless of adjustments for potential confounding factors. Sports participation in adolescence was associated with higher midlife LBM and BMD and better midlife cardiorespiratory and muscular fitness. ED and restrictive eating were associated with a higher injury risk. Higher EDE-QS scores, indicating more unhealthy attitudes toward eating and body image, were associated with a shorter sports career. Secondary amenorrhea was associated with higher rates of injury-related harms during the sports career and career termination due to injury. DE during the sports career predicted DE after the career, and MD in adolescence predicted MD in young adulthood.

6.1 Prevalence of low energy availability-related conditions among athletes

6.1.1 Attitudes toward eating and body weight perceptions

In the present study, the prevalence of ED among athletes varied from 7.1% (Study IV) to 18.4% (Study III), depending on the data. The prevalence of DE behaviors among athletes, in turn, varied from 16.0% (EDE-QS scores ≥ 15 at age

13–16 in Study IV) to 24.6% (restrictive eating in Study III). However, even higher prevalence rates were observed when investigating subsamples of athletes. For example, 23.8% of the older athletes in Study III reported past or current ED, and 26.9% of the athletes who participated in lean sports reported DE (i.e., restrictive eating). All these figures lie in between the prevalence rates found by other studies (5–47%) (Bratland-Sanda & Sundgot-Borgen, 2013; Dervish et al., 2022; Kampouri et al., 2019; Rousselet et al., 2017; Shriver et al., 2016; Whitehead et al., 2020).

Body weight dissatisfaction was reported by 18.4% of the adolescent athletes and 23.5% of the young adult athletes in this study. However, almost half (44.4%) of the adolescent athletes in the overweight group were dissatisfied with their weight. About one-fifth of the adolescent athletes (19.3%) and almost one-third (29.4%) of the young adult athletes desired to lose weight, even when some of them were already classified as normal weight. However, these figures are lower than those observed among female U.S. Olympic marathon trial participants, among which body weight dissatisfaction was reported to be 44.5% (Sophia et al., 2022). In a recent meta-analysis, the prevalence of body weight dissatisfaction among non-athletic adolescents ranged from 18–57% (Martini et al., 2022). The findings of this study are at the lower end of this range. In addition, some studies conducted on athletes have found higher rates of desire to lose weight than were found in the current study. For example, in a study conducted on female collegiate gymnasts, swimmers, and divers with a mean age of 19.1 years, a desire to lose weight was reported by 55% of the participants (Tackett et al., 2016). In addition, a study conducted on Portuguese mixed-sports female and male athletes with a mean age of 17.8 years and competing at regional, national, and top levels of their sports reported that 51.4% of the participants set their ideal weight lower than their present weight (Gomes et al., 2011).

The differences in findings regarding the prevalence of ED/DE and body dissatisfaction are likely related to the sample and/or assessment method (de Bruin, 2017). Some sports have a higher risk of ED/DE than others, which will be discussed further in subsection 6.2.2. In addition, there are plenty of different self-report methods regarding DE/ED used in the studies, but few of them have been validated in the athletic population (Pope et al., 2015). Methods for assessing body image/weight satisfaction, in turn, vary considerably from study to study, making the comparison between studies challenging. Moreover, many athletes have been found to underreport the use of pathogenic weight control methods in self-reports compared with clinical interviews (Martinsen & Sundgot-Borgen, 2013). Selection bias may also play a role, as athletes with eating and/or body image problems may be more or less prone to participating in these studies than athletes without such issues. Finally, COVID-19 pandemic has been reported to lead to worsening of DE behaviors and body image issues (Buckley et al. 2021). Thus, it is possible that data collected after the beginning of COVID-19 pandemic, as was the case in Study III, are influenced by the COVID-19 pandemic.

The rather high prevalence of eating problems, body dissatisfaction, and desire to lose weight found among both athletes and non-athletes is concerning given that both body dissatisfaction (S. B. Wang et al., 2019) and also DE behaviors (Micali et al., 2017; Neumark-Sztainer et al., 2011; Pearson et al., 2017) have been reported to be relatively stable from adolescence to adulthood. Pressure to be thin, thin-ideal internalization, and body dissatisfaction at the age of 14 years have been associated with future ED in girls (Rohde et al., 2015). Moreover, body dissatisfaction has been reported to be associated with higher levels of dieting and unhealthy weight control methods, as well as lower levels of physical activity among girls, regardless of BMI (Neumark-Sztainer et al., 2006). Body dissatisfaction and weight concerns also predict overweight (Haines et al., 2007), and body satisfaction, not body dissatisfaction, appears to be beneficial for weight management in overweight adolescent girls (Loth et al., 2015; van den Berg & Neumark-Sztainer, 2007). Moreover, body image problems have been associated with low self-esteem and depressive symptoms (Holsen et al., 2001; Paxton et al., 2006). Thus, to prevent further health issues, attention should be paid to the prevention and early identification of body dissatisfaction and the promotion of body satisfaction in adolescent girls.

It has been stated that sports environment can support and hinder the development of body satisfaction and that every stakeholder, including coaches, parents, referees, and sports administrators, in addition to athletes themselves, can contribute to this environment (Koulanova et al., 2021). For example, coaches' communication about body image and the value they place on body weight and composition, rather than performance, play an important role in female athletes' body image (Beckner & Record, 2016; Koulanova et al., 2021). In addition, coach-related weight pressure and critical comments about weight from coaches, family members, peers, etc. are associated with higher rates of DE behaviors (Gomes et al., 2011; G. Kerr et al., 2006; Muscat & Long, 2008). All stakeholders of the athlete can reduce the risk of athlete's body dissatisfaction and eating problems by acting as body-positive role models, making support resources accessible, discussing negative body image with athletes, and valuing skills over appearance. In addition, it is important to present images of female athletes that represent a variety of body shapes and sizes, use uniforms that are comfortable and fit for every type of body, and educate coaches on athletes' body image concerns (Koulanova et al., 2021). Furthermore, coaches and other people working with athletes, and parents of adolescent athletes should be able to identify early signs of eating and body image issues. These include behavioral changes, such as avoidance of eating-related social activities and secretive behavior relating to eating and/or exercise, physical changes, such as rapid weight changes, and physiological changes, such as declining mental health (K. R. Wells et al., 2020). Thus, athletes' stakeholders play important roles in preventing body image- and eating-related issues and identifying their early signs.

6.1.2 Menstrual dysfunction

In this study, the prevalence of MD among athletes ranged from 17.7% (Study I, adolescence) to 38.7% (Study I, young adulthood). While previous studies have shown that MD is prevalent among athletes (e.g., Hoch et al., 2009; Nichols et al., 2006, 2007; Tenforde et al., 2017; Thein-Nissenbaum et al., 2011; Torstveit & Sundgot-Borgen, 2005), few studies have addressed this issue among Finnish athletes (Fogelholm & Hiilloskorpi, 1999; Heikura et al., 2018). The present study showed that MD prevalence in Finnish athletes seems to be in line with studies conducted in other Western countries and with a study conducted in Finnish athletes over two decades ago (Fogelholm & Hiilloskorpi, 1999). For example, in a large study conducted among Norwegian athletes from a variety of sports with a mean age of 21.3 years (n=938), the prevalence of current MD was 16.5% (Torstveit & Sundgot-Borgen, 2005). In addition, in a study conducted on mixed-sports athletes (n=323) from the U.S. with a mean age of 20.0 years, the prevalence of MD (oligomenorrhea or secondary amenorrhea) was 26.8% (Tenforde et al., 2017). Moreover, in a study conducted by Fogelholm and Hiilloskorpi (1999), MD was found among 32–37% of 14–40 year-olds athletes participating in aesthetic, endurance, and weight-class sports, whereas the prevalence rates of similar-aged speed and ball game athletes were lower. In another study, however, 50% of Finnish world-class female middle- and long-distance runners and race walkers (n=12) were amenorrheic (Heikura et al., 2018). Studies conducted on adolescent mixed-sport athletes have shown prevalence rates between 19% and 24% (Nichols et al., 2006, 2007; Thein-Nissenbaum et al., 2011, 2012), which is slightly higher than our finding on MD prevalence in adolescent athletes in Study I. Some of these studies (Nichols et al., 2006, 2007; Thein-Nissenbaum et al., 2011), however, did not report excluding females with hormonal contraceptives from the analyses. Thus, the findings may not be fully comparable with the findings of the current study.

The prevalence of primary amenorrhea among athletes in this study ranged from 3.7% (Study II) to 20.0% (Study IV). In Study I, the prevalence among adolescent athletes was 8.1%, which is fairly consistent with the prevalence rates of 8.9%, 7.3%, and 6.0% found in other studies (Beals & Hill, 2006; Hoch et al., 2009; Torstveit & Sundgot-Borgen, 2005). However, in some studies, the prevalence of primary amenorrhea among athletes was found to be low, at about 1% (Martinsen et al., 2010; Nichols et al., 2006, 2007; Thein-Nissenbaum et al., 2012). The prevalence of primary amenorrhea in Study IV is even higher than the 11.8% found in a study by Tenforde and colleagues (2017) and nearly as high as the 21.9% found by Torstveit and Sundgot-Borgen (2005) among aesthetic athletes. As the athletes in Study IV were going through puberty in the 1990s and early 2000s, recall bias may play a role. However, in Study II, the participants were older than in those in Study IV, yet less than 4% of those with a competitive sports background reported primary amenorrhea. One possible explanation for the variation in findings may be the differences between lean and non-lean sport athletes. These differences are discussed in subsection 6.2.2.

6.2 Between-group comparisons

6.2.1 Athletes and non-athletes

Studies comparing the prevalence of ED or DE in athletes and non-athletes have yielded mixed findings. In their meta-analysis, Smolak and colleagues (2000) found that eating problems (ED or DE) were more common in athletes than in non-athletes, albeit with a small effect size. In contrast, consistent with the present study, a recent meta-analysis by Chapa et al. (2022) found no differences in ED/DE between athletes and non-athletes.

Although this study found no differences in eating problems between athletes and non-athletes, the trends in eating behaviors over time were different between the groups. The scores of the EDE-QS among the athletes increased significantly after the age of 13–15 and were lower in adulthood than in adolescence, while among the non-athletes, the EDE-QS scores were more stable over time. This may be related to sports-specific pressures of controlling body weight or composition, which can further result in restrictive eating and other disturbances in eating behaviors (K. R. Wells et al., 2020).

The findings of this study regarding the trends in eating behaviors are somewhat different from those reported by Slane and colleagues (2014). They found that DE behaviors in general increased almost linearly between the ages of 11 and 25 in females participating in the Minnesota Twin Family Study. In contrast, bulimic behaviors increased from 11 until only 18 years of age and then leveled off (Slane et al., 2014). The differences in findings may be related to different trajectories between individuals. While eating problems may increase in some individuals during adolescence, they may decline or remain stable in others (Fairweather-Schmidt & Wade, 2016; Pearson et al., 2017). Further studies would be beneficial in exploring the trajectories of DE behaviors among athletes and non-athletes and in finding factors that are associated with continued DE.

The findings of the current study align with the findings of the meta-analyses showing that athletes have more positive body images than their non-athletic peers (Hausenblas & Symons Downs, 2001) and that athletes have lower levels of body dissatisfaction than non-athletes (Chapa et al., 2022). It can be speculated that a higher BMI in non-athletes might have played a role in this difference. However, the present study found that athletes and non-athletes in the same weight category differed from each other in terms of body weight satisfaction and desire to lose weight, with athletes being more satisfied with their weight and desiring to lose weight less often than non-athletes. Indeed, the differences in body satisfaction and desire to lose weight between athletes and non-athletes may be related to body composition. In Western societies, a fit body type is idealized for females (Homan, 2010), and athletes' bodies may more often meet the form of this body type. In addition, body weight dissatisfaction has been linked to lower odds of meeting physical activity recommendations in adolescents with a high BMI (Sampasa-Kanyinga et al., 2017). Thus, it remains

unknown whether physical activity itself affects body image positively or whether individuals with a positive body image are more likely to engage in sports than those with a more negative body image (Hausenblas & Symons Downs, 2001).

While some previous studies have found higher MD prevalence rates among athletes compared with non-athletes (Hoch et al., 2009; Muia et al., 2016; Raymond-Barker et al., 2007; Rouveix et al., 2007), others have failed to find this association (e.g., Reinking & Alexander, 2005; Torstveit & Sundgot-Borgen, 2005). In line with these inconsistent findings, the present study found similar MD prevalence rates among adolescent athletes and non-athletes in Study I, but higher rates of MD among young adult athletes compared with non-athletes in Study I and among athletes in Study IV. Differences between the studied populations, particularly in terms of age and type of sports, may at least partly explain these inconsistent findings. The role of sports type is discussed in the next subsection. In sum, it can be stated that the findings of this study show that MD is more common in athletes than in non-athletes, at least in females above adolescent age.

The present study is also in line with other studies showing that athletes have later AAM than non-athletes (Constantini & Warren, 1995; Hoch et al., 2009; Torstveit & Sundgot-Borgen, 2005) along with higher rates of primary amenorrhea and delayed puberty (Torstveit & Sundgot-Borgen, 2005; Warren, 1980). The mean AAM among athletes in the present study varied between 12.7 years (Study I, adolescence) and 14.0 years (Study IV). The former AAM is in line with other studies that have reported that, among mixed-sports athletes, AAM varied between 12.4 and 13.4 years (Beals & Hill, 2006; Nichols et al., 2007; Rauh et al., 2010; Thein-Nissenbaum et al., 2012; Torstveit & Sundgot-Borgen, 2005). Moreover, the AAM of 14.0 years found among the athletes in Study IV is similar to the AAM found among aesthetic athletes from Norway (14.0 years) (Torstveit & Sundgot-Borgen, 2005) and among runners from the United Kingdom (14.1 years) (Hulley et al., 2007).

The mechanism behind later AAM in athletes compared with non-athletes may be intensive exercise and/or low EA among athletes, which can consequently result in alterations in hormone concentrations. More specifically, it has been suggested that decreased EA may lead to decreased leptin and increased ghrelin concentrations, as well as alterations in other peptides, which further suppress the function of the HPO axis, especially via KNDy neurons, and decreased kisspeptin release from these neurons (Ikegami et al., 2022; Martos-Moreno et al., 2010; Muñoz-Calvo & Argente, 2016; Uenoyama et al., 2019).

6.2.2 Lean sport and non-lean sport athletes

The results of the present study support the findings of other studies showing that athletes who participate in lean sports have higher rates of ED/DE compared with athletes who participate in non-lean sports (Mancine et al., 2020; McDonald et al., 2020; E. K. Wells et al., 2015). The findings of this study are also in line with previous studies showing that athletes competing in lean sports have higher rates

of MD (Gibbs et al., 2013; Nichols et al., 2007; Torstveit & Sundgot-Borgen, 2005) and primary amenorrhea (Torstveit & Sundgot-Borgen, 2005). In the current study, lean sports athletes had a lower BMI compared to non-lean sports athletes, but no differences were found in training hours. The finding regarding lower BMI among lean sports athletes is in agreement with previous studies (Beals & Hill, 2006; Nichols et al., 2007; Torstveit & Sundgot-Borgen, 2005) and may be an indicator of lower EA compared with non-lean sports athletes. Indeed, lean sports athletes have been found to have higher rates of body dissatisfaction (Reinking & Alexander, 2005) as well as the desire and external social pressure to be thin (E. K. Wells et al., 2015) than non-lean sports athletes, probably resulting in restrictive eating and reductions in EA. This may render athletes competing in lean sports more susceptible to MD compared to athletes competing in non-lean sports. It should be noted, however, that both ED/DE and MD still exist among non-lean sports athletes, although to a lesser extent.

6.2.3 Age and level of participation

The present study found a higher lifetime ED prevalence among older athletes compared with younger athletes. However, while current and past ED diagnoses were not differentiated, the prevalence of current ED cannot be interpreted from the results. In general, ED incidence peaks in adolescence and young adulthood (Jacobi et al., 2004). This observation is supported by the findings of the present study, which show that the EDE-QS scores among athletes increased after the age of 15 and were lower in adulthood than in adolescence.

In this study, younger athletes had higher rates of current MD compared with older athletes. This contrasts with the findings of previous studies (Prather et al., 2016; Torstveit & Sundgot-Borgen, 2005), which found no differences in MD prevalence between age groups. However, in their study conducted on high-level runners and race walkers, Heikura and colleagues (2018) found that athletes with secondary amenorrhea were younger than those with regular menstrual cycles (23.8 years vs 26.7 years, $p < .01$). The findings of the current study regarding higher rates of MD in younger athletes compared with older athletes is plausible, as it has been suggested that the risk of MD decreases with advanced gynecological age (Williams et al., 2017).

This study found no differences in ED/DE prevalence between athletes competing at different participation levels. Previous studies have provided inconsistent findings on this issue. Kong and Harris (2015) found that athletes competing at the international or national level and training at least 12 h per week had higher levels of DE than non-competitive athletes or those competing at the local to national level and training less than 12 h per week. In addition, Joubert and research group (2020) reported that elite-level climbers had higher levels of DE than those competing at lower levels. In contrast, Hopkinson and Lock (2004) found that recreational runners had higher levels of DE compared with varsity athletes, while Gomes et al. (2011) did not find any differences in DE behaviors between athletes who had won national titles and those who had not. The

findings of the current and previous studies indicate that, in addition to high-level athletes, athletes competing at lower levels are also at risk for DE.

This study provided conflicting findings regarding the association between the level of participation and MD. In Study III, no differences in MD were found between elite and non-elite athletes, while in Study IV, secondary amenorrhea predicted a lower participation level. The differences in the type of sports (mixed-sports athletes in Study III vs endurance athletes in Study IV), competition level (Tier 2 vs Tier ≥ 3 athletes in Study III vs Tier 3 vs Tier ≥ 4 athletes in Study IV), or statistical method used (chi-square test in Study III vs GEE model in Study IV) may explain the inconsistencies in the findings. The findings of the study by Adam and colleagues (2022) are in line with the results of Study III, as they found no differences in MD rates between athletes competing at the local, regional, or provincial levels compared to those competing at the national or international level. Further research is needed to investigate whether high-level athletes differ from those competing at lower levels in terms of MD prevalence.

6.3 Associations

A summary of selected associations of low EA-related conditions, AAM, and sports participation with health- and sports career-related factors found in this dissertation and their occurrence during the lifespan are presented in Figure 8.

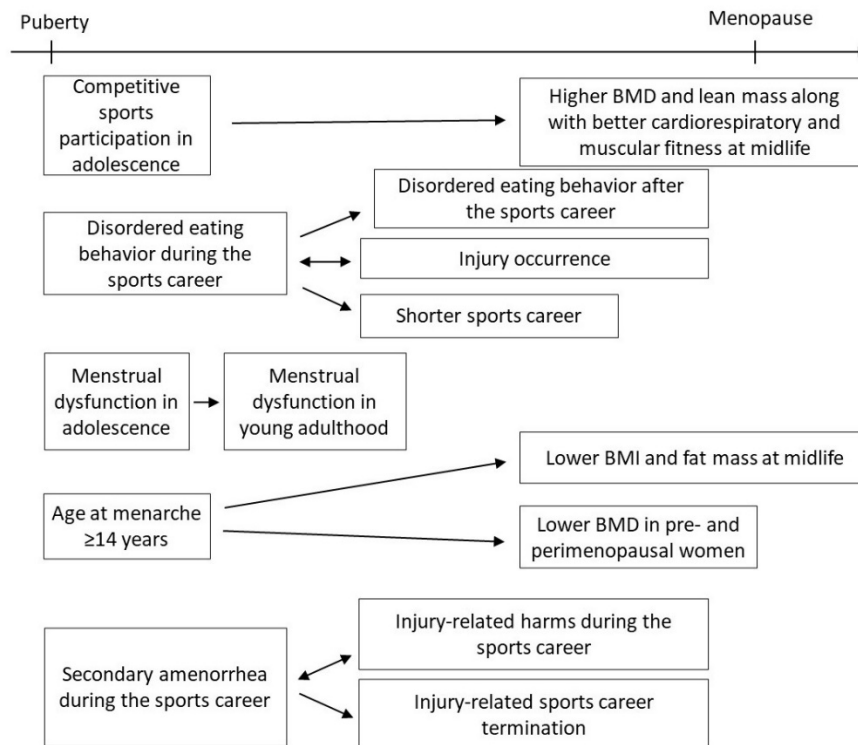


FIGURE 8 Summary of selected associations of low energy availability-related conditions, age at menarche, and sports participation with health- and sports career-related factors found in this study. The two-way arrows indicate an association where no temporal precedence could not be shown, while in the associations marked with a one-way arrow, a predictive variable occurred before an outcome variable. BMD = bone mineral density; BMI = body mass index.

6.3.1 Age at menarche and midlife characteristics

The results of this study are in line with previous observational studies (Farahmand et al., 2020; Magnus et al., 2018; Prentice & Viner, 2013; Trikudanathan et al., 2013) showing that AAM is inversely associated with BMI, fat mass, and fat percentage in middle age. However, Mendelian randomization studies have found a causal association between childhood adiposity and early AAM (Bell et al., 2018; Mumby et al., 2011) suggesting that early AAM is not causally linked to high adiposity in middle age. Rather, it is a marker of childhood adiposity, which can subsequently lead to obesity in adulthood (Bell et al., 2018).

Fewer studies have investigated the association between AAM and LBM in adulthood. In line with the findings of the present study, Kirchengast and colleagues (1998) found no differences in LBM among pre- or postmenopausal women grouped according to their AAM. It has also been suggested that AAM and LBM may share common genetic components (Hai et al., 2012).

Consistent with previous studies conducted among women with a mean age above 40 years (Chevalley et al., 2009b; Guo et al., 2005) and postmenopausal women (Mohammadi et al., 2019; Parker et al., 2014; Tuppurainen et al., 1995;

Varenna et al., 1999), the current study shows that AAM is inversely associated with BMD in middle-aged pre-, peri-, and postmenopausal women. However, after adjusting for potential confounding factors, no association was found between AAM and BMD in the entire group. In line with this, other studies have failed to find an association between AAM and BMD among pre- and postmenopausal women (Blum et al., 2001; Hagemans et al., 2004; Hassa et al., 2005; Nakaoka et al., 2001; Ozdemir et al., 2005; Sioka et al., 2010). It has been suggested that the number of reproductive years may be a better estimator of postmenopausal BMD than AAM (Grainge et al., 2001; Hagemans et al., 2004; Parker et al., 2014; Sioka et al., 2010). Other factors that have been found to be linked with a low adulthood BMD are low BMI, MD, a high number of parities, long lactation periods, low levels of physical activity, low calcium intake, and strong family histories of osteoporosis (Fujita et al., 1999; Hyassat et al., 2017; Khoo et al., 2011; Ozdemir et al., 2005; Varenna et al., 1999; Y.-X. Wang et al., 2020; Y.-Y. Zhang et al., 2003).

Common genetic factors have been shown to influence both AAM and BMD (Chevalley et al., 2009a; Cousminer et al., 2018; Guo et al., 2005; Z.-X. Zhang et al., 2009); however, menopause may interfere with this relationship (Z.-X. Zhang et al., 2009). Supporting this hypothesis, the present study found an association between AAM and BMD after excluding postmenopausal women from the analysis, regardless of adjusting for confounding factors. Thus, the results of this study indicate that late AAM is a predictor for low BMD until the age of menopause. Thereafter, other factors play a more important role in determining BMD. It is important to note that the relationship between AAM and middle-aged BMD in pre- and perimenopausal women was observed regardless of physical activity levels in adolescence or middle age. Therefore, special attention should be paid to women with late menarcheal age, as they may be at risk for low BMD in adulthood.

It should be noted, however, that this study did not investigate the influence of BMI on the relationship between AAM and BMD. It has been reported that BMI and body weight in adolescence (Blum et al., 2001) and adulthood (Edelstein & Barrett-Connor, 1993; Liel et al., 1988) are positively associated with adulthood BMD. Moreover, it has been suggested that ALMI may mediate the association between AAM and later BMD (Ning et al., 2021). Therefore, it is possible that BMI/ALMI may have affected the relationship between AAM and BMD observed in this study. Participants with higher AAM had lower middle-aged BMI; however, no differences in ALMI were found between the AAM groups. Mendelian randomization studies have suggested a causal link between AAM and BMD in adolescence (Cousminer et al., 2018) and adulthood (Q. Zhang et al., 2018); however, further studies considering the mediating role of other factors are required.

6.3.2 Competitive sports participation and midlife characteristics

In contrast with the findings of studies conducted on former swimmers, runners, and gymnasts (Andreoli et al., 2012; N. K. Pollock et al., 2006), the present study

found no association between physical activity status in adolescence and fat mass in middle age. Furthermore, contrary to a study conducted on gymnasts (N. K. Pollock et al., 2006) but in line with a study conducted on swimmers and runners (Andreoli et al., 2012), this study found an association between adolescent sports participation and higher LBM in middle age. The inconsistencies between the studies could be related to different study populations. As found in this study (Study III) and by others (Torstveit & Sundgot-Borgen, 2005) lean sport athletes tend to have lower BMI than athletes in non-lean sports. That may explain why swimmers, runners, and gymnastics (all of whom are lean athletes) also had lower fat mass in adulthood, in contrast with the mixed-sports athletes in the ERMA (Study II). It is also possible that this study lacked statistical power to show the difference in fat percentage between the athletes and the no-exercise group. Furthermore, gymnasts have been reported to have lower levels of lean mass than swimmers and non-athletic controls (Peltenburg et al., 1984), which may explain the inconsistencies in previous studies regarding lean mass.

In line with previous studies conducted on women (Andreoli et al., 2012; N. K. Pollock et al., 2006), the present study found an association between competitive sports in adolescence and higher midlife BMD. In addition, Valdimarsson and colleagues (2005) found that despite an increased loss of BMD related to reduced training in retired middle-aged female soccer players, retired athletes still had higher BMD levels in their legs compared with those of controls.

The findings of this study regarding an association between adolescent competitive sports participation and better cardiorespiratory and muscular fitness in midlife are in contrast with Simon and Docherty's (2017) findings that showed that middle-aged male and female former Division I athletes performed worse in cardiorespiratory and muscular fitness tests than their similar-aged peers who were recreationally active in college. They speculated that the difference in physical fitness between the groups may be attributed to pain, injuries, or motivation to exercise (Simon & Docherty, 2017). In Study II, however, physical activity level at adolescence correlated with physical activity level until the age of 39 years, and those with competitive sports backgrounds also reported higher rates of leisure-time physical activity than other groups at midlife. Thus, it can be assumed that those engaging in competitive sports during adolescence continued their sports career until adulthood and also maintained at least moderate activity in midlife. This can explain the findings of the current study regarding better midlife cardiorespiratory and muscular fitness among those who participated in competitive sports in adolescence compared to their non-athletic peers. Previous studies have shown that while cardiorespiratory and muscular fitness decline with age, the rate of this decline is lower in individuals with higher physical activity than others (M. L. Pollock et al., 1997; Sandler et al., 1991; Trappe et al., 1996).

It has been suggested that physical activity, combined with sufficient calcium intake in adolescence, during the years of peak bone mass acquisition, may be particularly important in terms of BMD in later life (Ondrak & Morgan, 2007; Välimäki et al., 1994). Rideout and research group (2006) reported that

physical activity at ages 12–18 was positively associated with current BMD among postmenopausal women, while no association between physical activity and other (later) age stages was found. However, studies conducted in males have reported that impact loading during young adulthood is more important for adulthood BMD than impact loading in adolescence (Rogers & Hinton, 2010; Van Langendonck et al., 2003). The difference between studies may be due to gender differences, as the rapid increase in bone mineral mass in adolescence occurs about two years earlier in females compared with males (Rizzoli & Bonjour, 1999). However, it was not within the scope of the present study to evaluate the most important age period for middle-aged BMD. The models investigating the association between sports participation in adolescence and midlife BMD were adjusted for physical activity at midlife, but as physical activity levels between ages 13–16 and all other age stages except the last (40–50) were correlated, it cannot be interpreted whether competitive physical activity during adolescence was an independent predictor of middle-aged BMD or whether physical activity at other ages also played a role. However, the results of this and other studies suggest that physical activity during adolescence and young adulthood is important for BMD at older ages.

While the present study did not investigate differences in sports disciplines in terms of adulthood BMD, it is known that not all disciplines are beneficial in terms of bone health. In males, it has been shown that the type of sport is a more important predictor of adulthood BMD than the time spent in physical activity (Van Langendonck et al., 2003). Sports that involve high- or odd-impact loading for bones (e.g., jumping sports, gymnastics, soccer) enhance BMD in contrast to non-impact sports (e.g., cycling and swimming), which do not seem to be associated with improved bone health (Tenforde & Fredericson, 2011). Thus, sports that include impact loading for bones are recommended in terms of bone health.

6.3.3 Factors associated with eating disorders, disordered eating, and body weight dissatisfaction

The finding of this study regarding an association of both restrictive eating and ED with injury occurrence is in line with other studies conducted on adolescent and young adult athletes (Gusfa et al., 2022; Prus et al., 2022; Rauh et al., 2014; Scheid & Stefanik, 2019; Sweeney et al., 2021; Thein-Nissenbaum et al., 2011; J. J. Thomas et al., 2011). The findings of the present study extend the findings of previous studies by indicating that increasing age does not protect athletes from the relationship between ED and injuries, as it was found that the association was also observed in older athletes in Study III. As it is not possible to infer causality from the findings of the present or other studies, prospective studies are needed to establish whether there is a causal relationship between ED/DE and injury incidence. In the case of a causal association, the direction of causality needs to be investigated. While it may be tempting to assume that those with ED/DE would have a higher risk of getting injured, the possibility of reverse causality

(i.e., the possibility that injured athletes are more prone to restricting their eating or engaging in other unhealthy eating behaviors) cannot be ruled out.

In agreement with the finding of Sweeney et al., (2021) this study did not find an association between DE behaviors and sports career termination due to injury. However, the present study found a negative association between DE behaviors and sports career length, indicating that problems with eating and body image are linked with a shorter career among athletes. To the best of the author's knowledge, no other studies have focused on an association between DE and sports career length. Hence, prospective studies are needed to confirm the finding of the current study and to evaluate the mechanisms behind this association. One possible explanation for this association is an increased injury risk and/or decreased BMD associated with DE (Mountjoy et al., 2018; Nattiv et al., 2007). However, while this study did not find an association of DE behaviors with injury-related harms during the sports career or with career termination due to injury, a possible explanation for the relationship between higher EDE-QS scores and a shorter career may be related to factors other than injuries.

This study found that DE behaviors during the sports career were also associated with DE behaviors after the career. A similar association was observed in non-athletes; thus, this relationship may not be related to sports participation. These findings highlight the need to find efficient interventions to prevent and treat ED behavior in athletes and in non-athletes. As discussed in subsection 6.1.1, it is important that parents and people working with athletes are able to identify early signs of DE and have the courage to intervene when observing these signs.

The present study found that in adolescent athletes, a higher BMI was associated with higher body weight dissatisfaction, which is in line with other studies (Karr et al., 2013; Swami et al., 2009). Regarding young adult athletes, the number of athletes in the under- and overweight categories were small; thus, analyses regarding the association between BMI and body weight dissatisfaction in these athletes might have lacked statistical power. This study did not investigate the role of factors other than BMI in body weight dissatisfaction in athletes. However, other studies have found that participation in lean sports and the internalization of athletic media messages are associated with higher body dissatisfaction among athletes (Swami et al., 2009). In turn, athletic self-efficacy has been found to protect against body dissatisfaction in athletes (Karr et al., 2013).

6.3.4 Factors associated with menstrual dysfunction

Studies investigating the association between MD and injuries have provided mixed and inconsistent findings. While Rauh and research group (2014) found an association between MD and injury incidence in their logistic regression model, which was adjusted for DE and BMD less than -2.0 standard deviations, other studies have found no association between MD and injuries (Beals & Manore, 2002; Beckvid Henriksson et al., 2000; Bratland-Sanda et al., 2015; Tenforde et al., 2011; Thein-Nissenbaum et al., 2011). This was also the case in Study III, in which no association between MD and the occurrence of injury was

found. However, Study IV provides interesting findings by showing that MD, and more specifically secondary amenorrhea, was associated with both injury-related harms during the career and career termination due to injury. The results of Study III, in which MD was associated with higher rates of missed participation days among athletes, also provide support for the findings of previous studies (Beckvid Henriksson et al., 2000; Ihalainen et al., 2021). Because performance enhancement among high-level athletes requires systematic training and high training volumes (Saavedra et al., 2018; Wasserfurth et al., 2020), injuries and missed training days may compromise athletic performance and consequently impair the success of the athletic career.

It is likely that chronic low EA explains the association between MD and missed participation days due to injury. As was speculated by Rauh and colleagues (2014), it is possible that the anti-anabolic state caused by the altered hormone profile resulting from chronic low EA might impair the health of tissues other than bone. These alterations in hormonal concentrations include, but are not limited to, elevated cortisol and suppressed IGF-1, thyroid hormones, and leptin levels (De Souza & Williams, 2004; Dipla et al., 2021; Elliott-Sale et al., 2018). A low BMI and high training volume can be related to low EA, which in turn may contribute to both MD and injuries. This was supported by the results from Study III, which showed that athletes with MD trained more and had a lower BMI than those with a regular menstrual cycle.

However, this study found inconsistent findings regarding the association between MD and training volume/physical activity. Young adult athletes with MD had higher accelerometer-measured physical activity than those with a regular menstrual cycle in Study I, and athletes with MD in Study III reported higher training hours compared with those without MD. These findings parallel those of some previous studies, which found a higher training volume in athletes with MD compared with those without (Beckvid Henriksson et al., 2000; Cobb et al., 2003). However, no differences in accelerometer-measured physical activity were found among adolescent athletes with and without MD in Study I. Instead, athletes without MD reported higher training hours in the training season. The finding regarding no differences in physical activity between athletes with and without MD is in accordance with other studies that found no differences in training volume or estimated energy expenditure between athletes with MD and eumenorrheic athletes (Ackerman et al., 2013, 2015; Tornberg et al., 2017; Torstveit & Sundgot-Borgen, 2005; Zanker & Swaine, 1998).

Both the adolescent athletes in Study I and the participants in Study III who reported MD had lower BMIs compared with their peers without MD. This finding is consistent with previous studies conducted on adult athletes (Ackerman et al., 2015; Tornberg et al., 2017; Zanker & Swaine, 1998). However, this is not always the case, as other studies conducted in adolescent, young adult (Ackerman et al., 2013; Armento et al., 2021; Cobb et al., 2003), and adult athletes (Micklesfield et al., 2007) have found no differences in BMI between athletes with MD and those without. In line with this, young adult athletes with MD in Study I had a similar mean BMI to their peers without MD.

Overall, the findings of this study and other investigations suggest that although athletes with MD may have a lower BMI and higher training volume than their regularly menstruating peers, this is not always the case. The present study did not investigate DE behaviors between athletes with and without MD, but other studies have found that DE behaviors are reported more frequently among athletes with MD than among naturally menstruating athletes (Cobb et al., 2003). Indeed, low BMI and/or high training hours among athletes with MD may be indicators of low EA, which can consequently increase the risk of MD (De Souza et al., 2014; Mountjoy et al., 2018).

In this study, MD in adolescence was the only significant predictor of MD in young adulthood, whereas BMI, attitudes toward body weight, physical activity, or AAM in adolescence did not predict MD in young adulthood. The finding regarding no association between BMI and future MD is in line with the results reported by To and research group (2000), who found that baseline weight did not predict further MD in a 12-month follow-up among Chinese dancers. In addition, the findings of this study align with the results of a previous study, which found that menstrual cycles over 35 days during the first gynecologic year predicted persistent menstrual irregularity (Rigon et al., 2010). These findings emphasize the need to prevent MD and identify it as early as possible. This is particularly important because it is known that (clinical) MD results not from an acute low EA but from a long-term, chronic low EA (Heikura et al., 2021; Nattiv et al., 2007).

6.4 Methodological considerations

Data for this dissertation were drawn from four different research projects: the FHPSC (Study I), ERMA (Study II), Female Athlete 2.0 (Study III), and MEBS (Study IV) studies. Of those, only the MEBS study was specifically designed for the purposes of this dissertation. Three other projects were planned for other purposes; thus, the analyses included in this dissertation were not planned before the data collections. Consequently, some assessment methods were not optimal for the purposes of this dissertation. For instance, no data in the ERMA were collected on participants' menstrual cycle regularity during adolescence and adulthood. In addition, in Study I, it would have been beneficial to study participants' body image more closely rather than their body weight (dis)satisfaction. Moreover, no data in the FHPSC or ERMA studies were collected on participants' attitudes toward eating. Regarding Study III, a closer look at the types of injuries (acute or stress injury, bone or soft tissue injury) would have been interesting if data were available for this. Further, it may have been fruitful to investigate the relationship between AAM and lumbar spine BMD, in addition to femoral neck BMD, in the ERMA study. Finally, because of the differences in the assessment methods, the definition of MD was marginally different in Studies I and III.

Even though most of the data used in the present study were not collected for the purposes of this dissertation, both the FHPSC and ERMA studies are large, well-designed studies that used laboratory-based measurements in addition to survey data. The FHPSC is a multi-centered study aimed to obtain a nationally representative sample of the most popular sports among adolescents in Finland and a school-based sample (non-athletes) stratified depending on school location, size, and type of school (rural/urban) (Kokko et al., 2015). In the ERMA, the target was to get a nationally representative sample, and to achieve this goal, no information for the exclusion criteria was included in the invitation letter. Instead, a stepwise exclusion protocol was applied with the aim of reaching as large a sample size as possible but taking into account all the potential confounding factors (Kovanen et al., 2018).

The strengths of this study include moderately large sample sizes in Studies II and III, the use of control groups in Studies I, II, and IV, and laboratory-based measurements (e.g., height, weight, BMD, physical activity, and physical fitness components) in Studies I and II. In addition, previously validated questionnaires were used when collecting survey data in the Female Athlete 2.0 and MEBS studies. The recruitment process of the MEBS study was designed in such a way that athletes who had terminated their sports careers at a young age were also included. This was done because of a desire to reach those female athletes who had succeeded at a young age but whose sports careers had been short. Another strength of this study is that in all four studies, females using hormonal contraceptives for reasons other than to maintain their menstrual bleeding were excluded from the analyses regarding MD. This was done because hormonal contraceptive use may mask the presence of MD (Cheng et al., 2021; De Souza et al., 2014; DiVasta & Gordon, 2008).

A further strength of this study is the fact that it includes both retrospective and cross-sectional designs, and a longitudinal analysis. While the cross-sectional design may be less prone to recall bias, the retrospective design made it possible to collect data from longer time periods in a time-consuming manner.

The major limitation of the present study is the possibility of recall bias, as participants were asked to provide responses that were dependent on their memories. For example, the AAM was based on the participants' memory in every research project included in this dissertation. While some studies have reported that long-term recall of AAM is fairly accurate (Casey et al., 1991; Must et al., 2002), some have recommended using self-reported AAM with caution and acknowledging the possibility of bias when using it (R. Cooper et al., 2006; Zarów & Cichocka, 2008). Furthermore, a recall of menstrual cycle regularity seems to be even less accurate than a recall of AAM (Must et al., 2002). Moreover, eating behaviors and attitudes at different age periods inquired about in Study IV are dependent on the respondent's memory and are thus susceptible to recall bias. Indeed, the possibility of recall bias should be kept in mind when interpreting the results of the present study.

Another limitation of this study is that menstrual cycle-related assessments were based on questionnaires, not laboratory-based assessments or clinical

evaluations. Thus, it was not possible to detect subclinical MD, such as anovulation, or exclude reasons other than low EA for MD. While the possibility of pregnancy was asked in the MEBS study, it was not evaluated in the FHPSC and Female Athlete 2.0 studies. In addition, other reasons, such as polycystic ovary syndrome or some other diseases may have been the reason for MD (Gordon et al., 2017). Thus, it is possible that some participants classified as having MD had it for some reason other than because of low EA.

In addition, ED was self-reported in this study, and the results most likely underestimate the actual prevalence rates. This is because only a third of EDs are detected in healthcare in Finland (Silén et al., 2021). Moreover, in the Female Athlete 2.0 study, restrictive eating was assessed using a simple yes/no question, which may have affected the findings.

An additional limitation of this study is the possibility of selection bias. In particular, the Female Athlete 2.0 and MEBS studies, which were designed to investigate menstrual cycle- and eating-related issues, were prone to selection bias. It is likely that those who were interested in these topics were more willing to reply and that those who replied may differ in terms of eating behaviors and/or menstrual function from those who did not. The response rate in MEBS among athletes was 37.0%, and the characteristics of the remaining 63.0% of the invited athletes cannot be evaluated. However, the main purpose of the FHPSC and ERMA studies was not to investigate the topics discussed in this dissertation. Thus, it may be less likely that selection bias influenced the results regarding these parameters. However, in the ERMA, those who participated were relatively healthy and well-functioning due to exclusion criteria. Thus, the results may not be generalizable to all middle-aged women.

Finally, the study designs used do not enable causal inferences. Although it could be shown that there was a temporal precedence of some predictive variables before outcome variables (e.g., adolescent characteristics and MD in young adulthood in Study I, AAM and adolescent sports participation and midlife characteristics in Study II, and MD during the career and career terminations due to injury in Study IV; Figure 7), these investigations were observational and thus unable to exclude all possible confounding factors. Although the models were adjusted for a variety of confounders, it is possible that some external confounding factors influenced the results of this study.

6.5 Implications

Previous studies have shown that low EA-related conditions (ED/DE, body dissatisfaction, and MD) are commonly observed in female athletes (e.g., Mountjoy et al., 2018; Torstveit et al., 2008; Torstveit & Sundgot-Borgen, 2005; K. R. Wells et al., 2020). This study shows that, regardless of age, type of sports, or level of participation, Finnish female athletes also have relatively high prevalence rates of these issues. Based on the findings of the present study, it is important

that parents and those working with female athletes are aware of these issues, are able to identify early indicators of eating and body image issues, and understand how to deal with athletes with eating- and/or menstrual cycle-related issues to prevent further health and performance problems. In addition, this study's finding that ED/DE and MD are associated with injuries emphasizes the importance of recognizing the possibility of these issues in the case of an athlete experiencing repetitive or long-term injuries.

The results of this study can help to reduce shame and stigma around eating-/body image- and menstrual cycle-related issues and to increase openness regarding these topics to make it easier for athletes to disclose their problems and seek help. The findings of this study, showing that problems with eating and menstrual function are common, may help athletes suffering from these issues understand that they are not alone in their challenges. The findings can also motivate athletes to seek help, as it was found that problems with eating and menstrual function were associated with injuries and sports career-related factors in a negative way.

In sum, this study showed that, although competitive sports participation in adolescence appears to have long-term health benefits, low EA-related factors are common among Finnish female athletes and are associated with negative aspects of both health and a sports career. By ignoring the importance of sufficient EA, there is a risk that an athlete will not achieve her full potential. It would be important to educate athletes, parents, and those working with athletes on the importance of adequate EA and healthy menstrual function. Moreover, the prevention of ED/DE and body image problems is important to prevent future health problems. In the sports context, the focus should be shifted from body weight and composition to health and performance, and it should be acknowledged that optimizing body weight includes the risk of harmful effects, such as injuries.

6.6 Future directions

The present study raises at least as many questions as it answers. What are the mechanisms behind the found association between higher EDE-QS scores and shorter career length, and can other studies replicate this association? Is there a causal relationship between restrictive eating and injuries, and if yes, what is the direction of this relationship? What mechanisms explain the link between MD and more missed participation days, injury-related harms during the career, and career termination due to injury? Is late AAM causally linked to lower midlife BMD, regardless of long-term physical activity status or body mass? Is the higher MD prevalence found among athletes compared with non-athletes a result of competitive sports itself, or does selection bias play a role? How could the EA-related conditions investigated in this study be prevented in the most effective

way? Are athletes themselves worried about these conditions, and what are their views on preventing and treating them?

To gain a deeper understanding of the conditions investigated in this dissertation, future studies should strive to answer the above-mentioned questions. In addition to well-designed randomized controlled trials and prospective cohort studies, which help to gain an understanding of causal relationships and mechanisms behind the associations, qualitative studies are also needed to increase our understanding of athletes' own experiences. Moreover, genomic studies would be beneficial for understanding the genetic components of these issues and associations found in this study.

7 MAIN FINDINGS AND CONCLUSIONS

The main findings and conclusions of this dissertation study can be summarized as follows:

1. Low EA-related conditions are common in Finnish female athletes. The prevalence rates for ED, DE, MD, and body weight dissatisfaction varied between 7–18%, 16–25%, 18–39%, and 18–24%, respectively, depending on the data used. Even higher rates for ED, DE, and body weight dissatisfaction were observed when examining subsamples of athletes (i.e., ED prevalence of 24% among athletes over 24 years of age, DE (restrictive eating) prevalence of 27% among lean sport athletes, and body weight dissatisfaction prevalence of 44% among overweight adolescent athletes).
2. Female athletes appeared to have higher rates of MD and delayed puberty compared with non-athletes, whereas no differences in ED/DE were found between the groups. Athletes were more satisfied with their weight and wanted to lose weight less often than non-athletes. Lean sport athletes were at a higher risk for low EA-related conditions than non-lean sport athletes, and MD was more common in young athletes compared with their older peers. The level of participation was not found to be associated with eating problems. The findings regarding the association between MD and a participation level are conflicting.
3. Later AAM was associated with lower BMI and fat mass in midlife but also with lower BMD in pre- and perimenopausal women, regardless of physical activity level in adolescence or middle age. Participation in competitive sports in adolescence was associated with a higher LBM and BMD in midlife and better midlife cardiorespiratory and muscular fitness compared with regular physical activity or no exercise in adolescence.
4. Problems with eating patterns were associated with an increased injury risk and a shorter sports career, and DE during the sports career was associated with DE after the career. MD was associated with higher MVPA in young adult athletes and with injury-related harms during the sports

career and career termination due to injury. MD in adolescence predicted MD in young adulthood.

YHTEENVETO (SUMMARY IN FINNISH)

Alhaiseen energiansaatavuuteen liittyvät tilat naisurheilijoilla: Esiintyvyys ja yhteydet terveyteen, vammoihin ja urheilu-uraan

Runsas fyysinen aktiivisuus lisää energiankulutusta, ja riittävä energiansaanti tukee urheilijan terveyttä sekä suorituskykyä. Syöminen voi jäädä kulutukseen nähden liian niukaksi kliinisen syömishäiriön tai häiriintyneen syömiskäyttäytymisen, mutta myös tahallisen, terveellä tavalla toteutetun ja lyhytaikaisen energianrajoituksen tai tahattoman alisyömisestä vuoksi. Yleisiä riskitekijöitä syömisestä ja kehonkuvan haasteille ovat muun muassa yhteiskunnassa vallitsevan laihuuksihanteen sisäistäminen, tyytymättömyys kehon painoon, perfektionismi, traumaattiset tapahtumat ja kehoon kohdistuvat kommentit. Lisäksi urheiluun liittyy tekijöitä, jotka voivat osaltaan altistaa syömisestä ja kehonkuvan häiriöille. Näitä tekijöitä ovat muun muassa kehon painon tai koostumuksen tarkka seuranta, painon vaihtelut, valmentajan kehon painoon tai koostumukseen liittyvät kommentit, vammat ja paineet parantaa suorituskykyä tai muuttaa kehon painoa/koostumusta.

Jos keho ei saa riittävästi energiaa, se alkaa säästää elämälle ei-välttämättömistä toiminnoista, kuten kasvusta ja lisääntymiselimistön toiminnasta. Naisurheilijoilla matalan energiansaatavuuden seurauksia voivat olla muun muassa häiriöt kuukautiskierrossa sekä luun mineraalitiheyden aleneminen, joka voi johtaa luun rasitusvammoihin, mm. rasitusmurtumiin. On myös näyttöä siitä, että matala energiansaatavuus voi lisätä riskiä muille kuin luuvammoille.

Vaikka muualla maailmassa toteutetut tutkimukset ovat osoittaneet, että syömisestä, kehonkuvan ja kuukautiskierron häiriöt ovat kohtalaisen yleisiä naisurheilijoilla, suomalaisilla urheilijoilla asiaa oli tutkittu varsin vähän. Lisäksi on tarvittu tietoa siitä, miten nuoruuden aikaiset syömisestä, kehonkuvan ja kuukautiskierron haasteet ovat yhteydessä urheilu-uraan ja terveyteen myöhemmällä iällä. Tämän tutkimuksen tarkoituksena oli selvittää alhaiseen energiansaatavuuteen liittyvien tilojen (syömisestä haasteet, tyytymättömyys kehon painoon, kuukautiskierron häiriöt) esiintyvyyttä suomalaisilla naisurheilijoilla ja sitä, eroavatko urheilijat ja ei-urheilijat tai eri-ikäiset, eri tasoilla ja eri lajityypeissä kilpailevat urheilijat toisistaan näiden tilojen esiintyvyydessä. Lisäksi tarkoituksena oli tutkia alhaiseen energiansaatavuuteen liittyvien tilojen yhteyttä vammoihin, urheilu-uraan ja terveyteen sekä kuukautisten alkamisiensa ja nuoruuden kilpaurheilun yhteyttä myöhemmän iän terveyteen liittyviin tekijöihin.

Tässä väitöskirjatutkimuksen hyödynnettiin neljää eri aineistoa. Ensimmäisessä osatyössä käytettiin Terveyttä edistävä liikuntaseura (TELS) -tutkimuksen ensimmäisen ja toisen mittauskerran aineistoa (n = 283 ja n = 211, tässä järjestyksessä), joissa oli mukana sekä urheiluseurassa harjoittelevia että urheiluseuraan kuulumattomia nuoria. TELS-tutkimuksen ensimmäisessä vaiheessa tutkittavat olivat iältään 14–16-vuotiaita ja toisessa vaiheessa 18–20-vuotiaita. Tutkimuksen toisessa osatyössä hyödynnettiin Estrogeeni, vaihdevuodet ja toimintakyky (ERMA) -tutkimuksen poikkileikkausaineistoa (n = 1098), jossa tutkittavat olivat

kotona asuvia, 47–55-vuotiaita naisia. Väitöskirjan kolmannessa osatyössä käytettiin Naisurheilija 2.0 -aineistoa (n = 846). Naisurheilija 2.0 -tutkimus on kyselylomakkeella toteutettu poikkileikkaustutkimus, jossa tutkittiin 15–45-vuotiaita kilpaurheilua millä tahansa tasolla harrastavia naisia. Tutkimuksen neljännessä osatyössä käytettiin Kuukautiskierron häiriöiden sekä syömiseen ja kehonkuvaan liittyvien asenteiden yhteys urheilu-uraan (MEBS) -aineistoa (n = 198). MEBS on retrospektiivinen tutkimus, jossa tutkittavat olivat 30–53-vuotiaita nuoruudessaan kansallisella ja kansainvälisellä tasolla kestävyyslajeissa kilpailleita naisia. Väitöskirjassa hyödynnetyissä aineistoissa kuukautiskierron, syömiseen, vammoihin ja urheilu-uraan liittyvät tiedot kerättiin kyselylomakkeilla. Keski-ikäisen kehon koostumusta ja luun mineraalitiheyttä mitattiin kaksiennergiaisella röntgenabsorptiometrialla (DXA) ja fyysistä kuntoa keski-ikässä useilla eri lihas- ja kestävyyskuntoa mittaavilla testeillä.

Tulokset osoittivat, että alhaiseen energiansaataavuuteen liittyvät tilat ovat kohtalaisen yleisiä suomalaisilla naisurheilijoilla. Syömishäiriöiden esiintyvyys urheilijoilla oli tutkittavasta aineistosta riippuen 7–18 % ja häiriintyneen syömiskäyttäytymisen esiintyvyys 16–25 %. Kuukautiskierron häiriöistä raportoi 18–39 % urheilijoista, ja tyytymättömyyttä kehon painoon esiintyi 18–24 %:lla. Kuukautiskierron häiriöt olivat urheilijoilla yleisempiä kuin ei-urheilijoilla, mutta syömisen haasteissa ei nähty eroja urheilijoiden ja ei-urheilijoiden välillä. Sen sijaan tyytymättömyys kehon painoon sekä halu pudottaa painoa oli yleisempää ei-urheilijoilla kuin urheilijoilla. Kestävyys-, painoluokka- ja hyppylajien sekä esteettisten lajien urheilijat raportoivat enemmän syömisen haasteita sekä kuukautiskierron häiriöitä kuin palloilulajien, teknisten ja taitolajien sekä teho-/nopeuslajien harrastajat. Nuoremmilla, 15–24-vuotiailla urheilijoilla esiintyi enemmän häiriöitä kuukautiskierrossa kuin 25–45-vuotiailla urheilijoilla. Syömisen haasteissa ei nähty eroja korkeammalla ja matalammalla tasolla kilpailevilla urheilijoilla, mutta kuukautiskierron häiriöiden suhteen tulokset olivat ristiriitaisia. Myöhäisempi kuukautisten alkamisikä (≥ 14 vuotta) oli yhteydessä matalampaan kehon painoaindeksiin ja rasvamassan määrään keski-ikässä, mutta myös alhaisempaan luun mineraalitiheyteen. Nuoruuden kilpaurheilu yhdistyi suurempaan kehon rasvattoman massan määrään, korkeampaan luuntiheyteen ja parempaan fyysiseen kuntoon keski-ikässä. Syömisen haasteet olivat yhteydessä suurempaan vammaan ja lyhyempään urheilu-uraan. Kuukautisten poisjääminen urheilu-uran aikana yli kolmen kuukauden ajaksi ilman hormonivalmisteiden käyttöä tai raskautta (sekundaarinen amenorrea) oli yhteydessä suurempaan vammojen aiheuttamaan haittaan urheilu-uran aikana sekä uran lopettamiseen vamman vuoksi. Häiriintynyt syömiskäyttäytyminen urheilu-uran aikana ennusti häiriintynyttä syömiskäyttäytymistä myös uran jälkeen, ja kuukautiskierron häiriö nuoruudessa ennusti kuukautiskierron häiriötä nuorena aikuisuudessa.

Tämän väitöskirjatutkimuksen tulokset tukevat kliinistä käsitystä siitä, että myös suomalaisilla naisurheilijoilla esiintyy melko yleisesti matalaan energiansaataavuuteen liittyviä tiloja, kuten syömisen haasteita, tyytymättömyyttä kehon painoa kohtaan sekä kuukautiskierron häiriöitä. Tulokset tukevat myös

aikaisempaa näyttöä siitä, että ns. painoherkkien lajien urheilijoilla (kestävyys-, painoluokka- ja hyppylajit sekä esteettiset lajit) esiintyy kyseisiä haasteita yleisemmin kuin muiden lajien harrastajilla. Syömisen ja kuukautiskierron haasteita esiintyy kuitenkin suomalaisilla naisurheilijoilla lajista, iästä tai tasosta riippumatta.

Tietoisuuden lisääminen syömisen ja kehonkuvan sekä kuukautiskierron haasteiden yleisyydestä sekä niiden negatiivisesta yhteydestä terveyteen ja urheilu-uraan on tärkeää aiheen ympärillä olevan stigman vähentämiseksi, avoimuuden kasvattamiseksi, ongelmien välttämiseksi sekä varhaisen puuttumisen vuoksi. On tärkeää, että niin valmentajat ja muut urheilijoiden kanssa työskentelevät henkilöt kuin nuorten urheilijoiden vanhemmatkin tiedostavat tutkimuksessa havaitut yhteydet matalaan energiansaataavuuteen liittyvien tekijöiden ja vammojen sekä urheilu-uraan liittyvien tekijöiden välillä. Painon ja/tai kehon koostumuksen optimointiin tähtäävä valmennus voi pahimmassa tapauksessa kääntyä itseään vastaan ja aiheuttaa ongelmia syömiskäyttäytymisen häiriintymisen tai vammojen muodossa. Sekä yksilö- että joukkuevalmennuksessa urheilijan terveys ja hyvinvointi tulisikin asettaa aina etusijalle, sillä pitkässä juoksussa vain terve, täysipainoiseen harjoitteluun kykenevä urheilija menestyy.

REFERENCES

- Ackerman, K. E., Cano Sokoloff, N., DE Nardo Maffazioli, G., Clarke, H. M., Lee, H., & Misra, M. (2015). Fractures in relation to menstrual status and bone parameters in young athletes. *Medicine & Science in Sports & Exercise*, 47(8), 1577–1586. <https://doi.org/10.1249/MSS.0000000000000574>
- Ackerman, K. E., Holtzman, B., Cooper, K. M., Flynn, E. F., Bruinvels, G., Tenforde, A. S., Popp, K. L., Simpkin, A. J., & Parziale, A. L. (2019). Low energy availability surrogates correlate with health and performance consequences of Relative Energy Deficiency in Sport. *British Journal of Sports Medicine*, 53(10), 628–633. <https://doi.org/10.1136/bjsports-2017-098958>
- Ackerman, K. E., Patel, K. T., Guereca, G., Pierce, L., Herzog, D.B., & Misra, M. (2013). Cortisol secretory parameters in young exercisers in relation to LH secretion and bone parameters. *Clinical Endocrinology*, 78(1), 114–119. <https://doi.org/10.1111/j.1365-2265.2012.04458.x>
- Adam, M. E. K., Bristow, A., Neely, K. C., & Erlandson, M. C. (2022). Do women athletes' experiences of menstrual function and dysfunction vary across competition levels? A mixed methods exploration. *Psychology of Sport and Exercise*, 63, Article 102270. <https://doi.org/10.1016/j.psychsport.2022.102270>
- Ajuied, A., Wong, F., Smith, C., Norris, M., Earnshaw, P., Back, D., & Davies, A. (2014). Anterior cruciate ligament injury and radiologic progression of knee osteoarthritis: A systematic review and meta-analysis. *The American Journal of Sports Medicine*, 42(9), 2242–2252. <https://doi.org/10.1177/0363546513508376>
- Alfermann, D., Stambulova, N., & Zemaityte, A. (2004). Reactions to sport career termination: A cross-national comparison of German, Lithuanian, and Russian athletes. *Psychology of Sport and Exercise*, 5(1), 61–75. [https://doi.org/10.1016/S1469-0292\(02\)00050-X](https://doi.org/10.1016/S1469-0292(02)00050-X)
- Ali, E. S., Mangold, C., & Peiris, A. N. (2017). Estriol: Emerging clinical benefits. *Menopause*, 24(9), Article 1081. <https://doi.org/10.1097/GME.0000000000000855>
- Allaway, H. C. M., Southmayd, E. A., & De Souza, M. J. (2016). The physiology of functional hypothalamic amenorrhea associated with energy deficiency in exercising women and in women with anorexia nervosa. *Hormone Molecular Biology and Clinical Investigation*, 25(2), 91–119. <https://doi.org/10.1515/hmbci-2015-0053>
- American Academy of Family Physicians, American Academy of Orthopaedic Surgeons, American College of Sports Medicine, American Medical Society for Sports Medicine, American Orthopaedic Society for Sports Medicine, & the American Osteopathic Academy of Sports Medicine. (2018). Female athlete issues for the team physician: A consensus statement–2017 update. *Current Sports Medicine Reports*, 17(5), 163–171. <https://doi.org/10.1249/JSR.0000000000000482>

- American Academy of Pediatrics Committee on Adolescence, American College of Obstetricians and Gynecologists Committee on Adolescent Health Care, Diaz, A., Laufer, M. R., & Breech, L. L. (2006). Menstruation in girls and adolescents: Using the menstrual cycle as a vital sign. *Pediatrics*, 118(5), 2245–2250. <https://doi.org/10.1542/peds.2006-2481>
- American College of Obstetricians and Gynecologists (2015). Committee Opinion No. 651: Menstruation in girls and adolescents: Using the menstrual cycle as a vital sign. *Obstetrics & Gynecology*, 126(6), e143–e146. <https://doi.org/10.1097/aog.0000000000001215>
- American College of Sports Medicine, American Academy of Family Physicians, American Academy of Orthopaedic Surgeons, American Medical Society for Sports Medicine, American Orthopaedic Society for Sports Medicine, & American Osteopathic Academy of Sports Medicine. (2006). Psychological issues related to injury in athletes and the team physician: A consensus statement. *Medicine & Science in Sports & Exercise*, 38(11), 2030–2034. <https://doi.org/10.1249/mss.0b013e31802b37a6>
- Andreoli, A., Celi, M., Volpe, S. L., Sorge, R., & Tarantino, U. (2012). Long-term effect of exercise on bone mineral density and body composition in post-menopausal ex-elite athletes: A retrospective study. *European Journal of Clinical Nutrition*, 66(1), 69–74. <https://doi.org/10.1038/ejcn.2011.104>
- Appiah, D., Nwabuo, C. C., Ebong, I. A., Wellons, M. F., & Winters, S. J. (2021). Trends in age at natural menopause and reproductive life span among US women, 1959–2018. *JAMA*, 325(13), 1328–1330. <https://doi.org/10.1001/jama.2021.0278>
- Apter, D., & Vihko, R. (1983). Early menarche, a risk factor for breast cancer, indicates early onset of ovulatory cycles. *The Journal of Clinical Endocrinology & Metabolism*, 57(1), 82–86. <https://doi.org/10.1210/jcem-57-1-82>
- Araújo, C. G. S., & Scharhag, J. (2016). Athlete: A working definition for medical and health sciences research. *Scandinavian Journal of Medicine & Science in Sports*, 26(1), 4–7. <https://doi.org/10.1111/sms.12632>
- Araújo, V. R., Gastal, M. O., Figueiredo, J. R., & Gastal, E. L. (2014). In vitro culture of bovine preantral follicles: A review. *Reproductive Biology and Endocrinology*, 12, Article 78. <https://doi.org/10.1186/1477-7827-12-78>
- Areta, J. L., Taylor, H. L., & Koehler, K. (2021). Low energy availability: History, definition and evidence of its endocrine, metabolic and physiological effects in prospective studies in females and males. *European Journal of Applied Physiology*, 121(1), 1–21. <https://doi.org/10.1007/s00421-020-04516-0>
- Armento, A., VanBaak, K., Seehusen, C. N., Sweeney, E. A., Wilson, J. C., & Howell, D. R. (2021). Presence and perceptions of menstrual dysfunction and associated quality of life measures among high school female athletes. *Journal of Athletic Training*, 56(10), 1094–1099. <https://doi.org/10.4085/624-20>

- Artero, E. G., Lee, D., Lavie, C. J., España-Romero, V., Sui, X., Church, T. S., & Blair, S. N. (2012). Effects of muscular strength on cardiovascular risk factors and prognosis. *Journal of Cardiopulmonary Rehabilitation and Prevention*, 32(6), 351–358.
<https://doi.org/10.1097/HCR.0b013e3182642688>
- Arthur-Cameselle, J., Sossin, K., & Quatromoni, P. (2017). A qualitative analysis of factors related to eating disorder onset in female collegiate athletes and non-athletes. *Eating Disorders*, 25(3), 199–215.
<https://doi.org/10.1080/10640266.2016.1258940>
- Bahr, R. (2009). No injuries, but plenty of pain? On the methodology for recording overuse symptoms in sports. *British Journal of Sports Medicine*, 43(13), 966–972. <https://doi.org/10.1136/bjism.2009.066936>
- Bahr, R., Clarsen, B., Derman, W., Dvorak, J., Emery, C. A., Finch, C. F., Hägglund, M., Junge, A., Kemp, S., Khan, K. M., Marshall, S. W., Meeuwisse, W., Mountjoy, M., Orchard, J. W., Pluim, B., Quarrie, K. L., Reider, B., Schweltnus, M., Soligard, T., ... Chamari, K. (2020). International Olympic Committee consensus statement: Methods for recording and reporting of epidemiological data on injury and illness in sports 2020 (including the STROBE Extension for Sports Injury and Illness Surveillance (STROBE-SIIS)). *British Journal of Sports Medicine*, 54(7), 372–389. <https://doi.org/10.1136/bjsports-2019-101969>
- Balachandar, V., Marciniak, J.-L., Wall, O., & Balachandar, C. (2017). Effects of the menstrual cycle on lower-limb biomechanics, neuromuscular control, and anterior cruciate ligament injury risk: A systematic review. *Muscles, Ligaments and Tendons Journal*, 7(1), 136–146.
<https://doi.org/10.11138/mltj/2017.7.1.136>
- Ballinger, G. A. (2004). Using generalized estimating equations for longitudinal data analysis. *Organizational Research Methods*, 7(2), 127–150.
<https://doi.org/10.1177/1094428104263672>
- Barrack, M. T., Gibbs, J. C., De Souza, M. J., Williams, N. I., Nichols, J. F., Rauh, M. J., & Nattiv, A. (2014). Higher incidence of bone stress injuries with increasing Female Athlete Triad-related risk factors: A prospective multisite study of exercising girls and women. *The American Journal of Sports Medicine*, 42(4), 949–958.
<https://doi.org/10.1177/0363546513520295>
- Barrow, G. W., & Saha, S. (1988). Menstrual irregularity and stress fractures in collegiate female distance runners. *The American Journal of Sports Medicine*, 16(3), 209–216. <https://doi.org/10.1177/036354658801600302>
- Bartel, S. J., Sherry, S. B., Farthing, G. R., & Stewart, S. H. (2020). Classification of orthorexia nervosa: Further evidence for placement within the eating disorders spectrum. *Eating Behaviors*, 38, Article 101406.
<https://doi.org/10.1016/j.eatbeh.2020.101406>
- Bau, A. M., Ernert, A., Schenk, L., Wiegand, S., Martus, P., Grüters, A., & Krude, H. (2009). Is there a further acceleration in the age at onset of menarche? A cross-sectional study in 1840 school children focusing on age and

- bodyweight at the onset of menarche. *European Journal of Endocrinology*, 160(1), 107–113. <https://doi.org/10.1530/EJE-08-0594>
- Beals, K. A., & Hill, A. K. (2006). The prevalence of disordered eating, menstrual dysfunction, and low bone mineral density among US collegiate athletes. *International Journal of Sport Nutrition and Exercise Metabolism*, 16(1), 1–23. <https://doi.org/10.1123/ijsnem.16.1.1>
- Beals, K. A., & Manore, M. M. (2002). Disorders of the female athlete triad among collegiate athletes. *International Journal of Sport Nutrition and Exercise Metabolism*, 12(3), 281–293. <https://doi.org/10.1123/ijsnem.12.3.281>
- Becker, M., & Hesse, V. (2020). Minipuberty: Why does it happen? *Hormone Research in Paediatrics*, 93(2), 76–84. <https://doi.org/10.1159/000508329>
- Beckner, B. N., & Record, R. A. (2016). Navigating the thin-ideal in an athletic world: Influence of coach communication on female athletes' body image and health choices. *Health Communication*, 31(3), 364–373. <https://doi.org/10.1080/10410236.2014.957998>
- Beckvid Henriksson, G., Schnell, C., & Lindén Hirschberg, A. (2000). Women endurance runners with menstrual dysfunction have prolonged interruption of training due to injury. *Gynecologic and Obstetric Investigation*, 49(1), 41–46. <https://doi.org/10.1159/000010211>
- Bell, J. A., Carslake, D., Wade, K. H., Richmond, R. C., Langdon, R. J., Vincent, E. E., Holmes, M. V., Timpson, N. J., & Davey Smith, G. (2018). Influence of puberty timing on adiposity and cardiometabolic traits: A Mendelian randomisation study. *PLoS Medicine*, 15(8), Article e1002641. <https://doi.org/10.1371/journal.pmed.1002641>
- Belsky, J., Steinberg, L., Houts, R. M., Halpern-Felsher, B. L., & NICHD Early Child Care Research Network. (2010). The development of reproductive strategy in females: Early maternal harshness --> earlier menarche --> increased sexual risk taking. *Developmental Psychology*, 46(1), 120–128. <https://doi.org/10.1037/a0015549>
- Bhattacharya, A., Cooper, M., McAdams, C., Peebles, R., & Timko, C. A. (2022). Cultural shifts in the symptoms of anorexia nervosa: The case of orthorexia nervosa. *Appetite*, 170, Article 105869. <https://doi.org/10.1016/j.appet.2021.105869>
- Bizzarri, C., & Cappa, M. (2020). Ontogeny of hypothalamus-pituitary gonadal axis and minipuberty: An ongoing debate? *Frontiers in Endocrinology*, 11, Article 187. <https://doi.org/10.3389/fendo.2020.00187>
- Blair, S. N., Cheng, Y., & Scott Holder, J. (2001). Is physical activity or physical fitness more important in defining health benefits? *Medicine & Science in Sports & Exercise*, 33(Suppl. 6), S379–S420. <https://doi.org/10.1097/00005768-200106001-00007>
- Blomqvist, M., Mononen, K., Koski, P., & Kokko, S. (2019). Urheilu ja seuraharrastaminen [Sports and sports club participation]. In S. Kokko & L. Martin (Eds.), *Lasten ja nuorten liikuntakäyttäytyminen Suomessa. LIITU-tutkimuksen tuloksia 2018* (pp. 47–54). Valtion liikuntaneuvoston julkaisuja

- 2019:1. <https://www.liikuntaneuvosto.fi/lausunnot-ja-julkaisut/lasten-ja-nuorten-liikuntakayttaytyminen-suomessa-liitu-tutkimuksen-tuloksia-2018/>
- Blomqvist, M., Mononen, K., Koski, P., & Kokko, S. (2023). Urheilu, seuraharrastaminen ja epäasiallinen kohtelu [Sports, sports club participation, and inappropriate treatment]. In S. Kokko & L. Martin (Eds.), *Lasten ja nuorten liikuntakäyttäytyminen Suomessa: LIITU-tutkimuksen tuloksia 2022* (pp. 83–98). Valtion liikuntaneuvoston julkaisuja 2023:1. <https://www.liikuntaneuvosto.fi/lausunnot-ja-julkaisut/liitu-2022/>
- Blum, M., Harris, S. S., Must, A., Phillips, S. M., Rand, W. M., & Dawson-Hughes, B. (2001). Weight and body mass index at menarche are associated with premenopausal bone mass. *Osteoporosis International*, 12(7), 588–594. <https://doi.org/10.1007/s001980170082>
- Boden, G., Chen, X., Mozzoli, M., & Ryan, I. (1996). Effect of fasting on serum leptin in normal human subjects. *The Journal of Clinical Endocrinology & Metabolism*, 81(9), 3419–3423. <https://doi.org/10.1210/jcem.81.9.8784108>
- Booth, F. W., Roberts, C. K., & Laye, M. J. (2012). Lack of exercise is a major cause of chronic diseases. *Comprehensive Physiology*, 2(2), 1143–1211. <https://doi.org/10.1002/cphy.c110025>
- Bordini, B., & Rosenfield, R. L. (2011). Normal pubertal development: Part I: The endocrine basis of puberty. *Pediatrics in Review*, 32(6), 223–229. <https://doi.org/10.1542/pir.32-6-223>
- Bratland-Sanda, S., & Sundgot-Borgen, J. (2013). Eating disorders in athletes: Overview of prevalence, risk factors and recommendations for prevention and treatment. *European Journal of Sport Science*, 13(5), 499–508. <https://doi.org/10.1080/17461391.2012.740504>
- Bratland-Sanda, S., Sundgot-Borgen, J., & Myklebust, G. (2015). Injuries and musculoskeletal pain among Norwegian group fitness instructors. *European Journal of Sport Science*, 15(8), 784–792. <https://doi.org/10.1080/17461391.2015.1062564>
- Bubach, S., De Mola, C. L., Hardy, R., Dreyfus, J., Santos, A. C., & Horta, B. L. (2018). Early menarche and blood pressure in adulthood: Systematic review and meta-analysis. *Journal of Public Health*, 40(3), 476–484. <https://doi.org/10.1093/pubmed/idx118>
- Buckley, G. L., Hall, L. E., Lassemillante, A.-C. M., & Belski, R. (2021). Disordered eating & body image of current and former athletes in a pandemic; A convergent mixed methods study – What can we learn from COVID-19 to support athletes through transitions? *Journal of Eating Disorders*, 9(1), 73. <https://doi.org/10.1186/s40337-021-00427-3>
- Budak, E., Fernández Sánchez, M., Bellver, J., Cerveró, A., Simón, C., & Pellicer, A. (2006). Interactions of the hormones leptin, ghrelin, adiponectin, resistin, and PYY3-36 with the reproductive system. *Fertility and Sterility*, 85(6), 1563–1581. <https://doi.org/10.1016/j.fertnstert.2005.09.065>
- Bueno, B., Krug, I., Bulik, C. m., Jiménez-Murcia, S., Granero, R., Thornton, L., Penelo, E., Menchón, J. m., Sánchez, I., Tinahones, F. j., & Fernández-

- Aranda, F. (2014). Late onset eating disorders in Spain: Clinical characteristics and therapeutic implications. *Journal of Clinical Psychology, 70*(1), 1–17. <https://doi.org/10.1002/jclp.22006>
- Bulik, C. M., Coleman, J. R. I., Hardaway, J. A., Breithaupt, L., Watson, H. J., Bryant, C. D., & Breen, G. (2022). Genetics and neurobiology of eating disorders. *Nature Neuroscience, 25*(5), Article 5. <https://doi.org/10.1038/s41593-022-01071-z>
- Bulik, C. M., Kleiman, S. C., & Yilmaz, Z. (2016). Genetic epidemiology of eating disorders. *Current Opinion in Psychiatry, 29*(6), 383–388. <https://doi.org/10.1097/YCO.0000000000000275>
- Bullen, B. A., Skrinar, G. S., Beitins, I. Z., von Mering, G., Turnbull, B. A., & McArthur, J. W. (1985). Induction of menstrual disorders by strenuous exercise in untrained women. *The New England Journal of Medicine, 312*(21), 1349–1353. <https://doi.org/10.1056/NEJM198505233122103>
- Bulun, S. E. (2020). Physiology and pathology of the female reproductive axis. In S. Melmed, R. J. Auchus, A. B. Goldfine, R. J. Koenig, & C. J. Rosen (Eds.), *Williams textbook of endocrinology* (14th ed., pp. 574–641). Elsevier.
- Burger, H. G., Hale, G. E., Dennerstein, L., & Robertson, D. M. (2008). Cycle and hormone changes during perimenopause: The key role of ovarian function. *Menopause, 15*(4), 603–612. <https://doi.org/10.1097/gme.0b013e318174ea4d>
- Burke, L. M., Lundy, B., Fahrenholtz, I. L., & Melin, A. K. (2018). Pitfalls of conducting and interpreting estimates of energy availability in free-living athletes. *International Journal of Sport Nutrition and Exercise Metabolism, 28*(4), 350–363. <https://doi.org/10.1123/ijsnem.2018-0142>
- Byrne, S., & McLean, N. (2002). Elite athletes: Effects of the pressure to be thin. *Journal of Science and Medicine in Sport, 5*(2), 80–94. [https://doi.org/10.1016/S1440-2440\(02\)80029-9](https://doi.org/10.1016/S1440-2440(02)80029-9)
- Caldwell, A. E., & Hooper, P. L. (2017). Sex hormones and physical activity in women: An evolutionary framework. In A. C. Hackney (Ed.), *Sex hormones, exercise and women: Scientific and clinical aspects* (pp. 139–149). Springer International Publishing AG. https://doi.org/10.1007/978-3-319-44558-8_9
- Callison, K., & Lowen, A. (2022). The long-run effects of adolescent athletic participation on women’s health. *Economics & Human Biology, 44*, Article 101087. <https://doi.org/10.1016/j.ehb.2021.101087>
- Calthorpe, L., Brage, S., & Ong, K. K. (2019). Systematic review and meta-analysis of the association between childhood physical activity and age at menarche. *Acta Paediatrica, 108*(6), 1008–1015. <https://doi.org/10.1111/apa.14711>
- Canoy, D., Beral, V., Balkwill, A., Wright, F. L., Kroll, M. E., Reeves, G. K., Green, J., & Cairns, B. J. (2015). Age at menarche and risks of coronary heart and other vascular diseases in a large UK cohort. *Circulation, 131*(3), 237–244. <https://doi.org/10.1161/CIRCULATIONAHA.114.010070>

- Carlson, L. J., & Shaw, N. D. (2019). Development of ovulatory menstrual cycles in adolescent girls. *Journal of Pediatric and Adolescent Gynecology*, 32(3), 249–253. <https://doi.org/10.1016/j.jpag.2019.02.119>
- Caronia, L. M., Martin, C., Welt, C. K., Sykiotis, G. P., Quinton, R., Thambundit, A., Avbelj, M., Dhruvakumar, S., Plummer, L., Hughes, V. A., Seminara, S. B., Boepple, P. A., Sidis, Y., Crowley, W. F., Martin, K. A., Hall, J. E., & Pitteloud, N. (2011). A genetic basis for functional hypothalamic amenorrhea. *The New England Journal of Medicine*, 364(3), 215–225. <https://doi.org/10.1056/NEJMoa0911064>
- Casey, V. A., Dwyer, J. T., Coleman, K. A., Krall, E. A., Gardner, J., & Valadian, I. (1991). Accuracy of recall by middle-aged participants in a longitudinal study of their body size and indices of maturation earlier in life. *Annals of Human Biology*, 18(2), 155–166. <https://doi.org/10.1080/03014469100001492>
- Caspersen, C. J., Powell, K. E., & Christenson, G. M. (1985). Physical activity, exercise, and physical fitness: Definitions and distinctions for health-related research. *Public Health Reports*, 100(2), 126–131.
- Cauley, J. A. (2015). Estrogen and bone health in men and women. *Steroids*, 99(Part A), 11–15. <https://doi.org/10.1016/j.steroids.2014.12.010>
- Cena, H., Barthels, F., Cuzzolaro, M., Bratman, S., Brytek-Matera, A., Dunn, T., Varga, M., Missbach, B., & Donini, L. M. (2019). Definition and diagnostic criteria for orthorexia nervosa: A narrative review of the literature. *Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity*, 24(2), 209–246. <https://doi.org/10.1007/s40519-018-0606-y>
- Chan, J. L., & Mantzoros, C. S. (2005). Role of leptin in energy-deprivation states: Normal human physiology and clinical implications for hypothalamic amenorrhoea and anorexia nervosa. *The Lancet*, 366(9479), 74–85. [https://doi.org/10.1016/S0140-6736\(05\)66830-4](https://doi.org/10.1016/S0140-6736(05)66830-4)
- Chang, V. W., & Christakis, N. A. (2003). Self-perception of weight appropriateness in the United States. *American Journal of Preventive Medicine*, 24(4), 332–339. [https://doi.org/10.1016/s0749-3797\(03\)00020-5](https://doi.org/10.1016/s0749-3797(03)00020-5)
- Chapa, D. A. N., Johnson, S. N., Richson, B. N., Bjorlie, K., Won, Y. Q., Nelson, S. V., Ayres, J., Jun, D., Forbush, K. T., Christensen, K. A., & Perko, V. L. (2022). Eating-disorder psychopathology in female athletes and non-athletes: A meta-analysis. *International Journal of Eating Disorders*, 55(7), 861–885. <https://doi.org/10.1002/eat.23748>
- Charalampopoulos, D., McLoughlin, A., Elks, C. E., & Ong, K. K. (2014). Age at menarche and risks of all-cause and cardiovascular death: A systematic review and meta-analysis. *American Journal of Epidemiology*, 180(1), 29–40. <https://doi.org/10.1093/aje/kwu113>
- Chen, X., Liu, Y., Sun, X., Yin, Z., Li, H., Liu, X., Zhang, D., Cheng, C., Liu, L., Liu, F., Zhou, Q., Wang, C., Li, L., Wang, B., Zhao, Y., Liu, D., Zhang, M., & Hu, D. (2018). Age at menarche and risk of all-cause and cardiovascular mortality: A systematic review and dose-response meta-analysis.

Menopause, 26(6), 670–676.

<https://doi.org/10.1097/GME.0000000000001289>

- Cheng, J., Santiago, K. A., Abutalib, Z., Temme, K. E., Hulme, A., Goolsby, M. A., Esopenko, C. L., & Casey, E. K. (2021). Menstrual irregularity, hormonal contraceptive use, and bone stress injuries in collegiate female athletes in the united states. *PM & R*, 13(11), 1207–1215.
<https://doi.org/10.1002/pmrj.12539>
- Chevalley, T., Bonjour, J.-P., Ferrari, S., & Rizzoli, R. (2008). Influence of age at menarche on forearm bone microstructure in healthy young women. *The Journal of Clinical Endocrinology & Metabolism*, 93(7), 2594–2601.
<https://doi.org/10.1210/jc.2007-2644>
- Chevalley, T., Bonjour, J. P., Ferrari, S., & Rizzoli, R. (2009a). The influence of pubertal timing on bone mass acquisition: A predetermined trajectory detectable five years before menarche. *The Journal of Clinical Endocrinology & Metabolism*, 94(9), 3424–3431. <https://doi.org/10.1210/jc.2009-0241>
- Chevalley, T., Bonjour, J.-P., Ferrari, S., & Rizzoli, R. (2009b). Deleterious effect of late menarche on distal tibia microstructure in healthy 20-year-old and premenopausal middle-aged women. *Journal of Bone and Mineral Research*, 24(1), 144–152. <https://doi.org/10.1359/jbmr.080815>
- Childs, G. V., Odle, A. K., MacNicol, M. C., & MacNicol, A. M. (2021). The importance of leptin to reproduction. *Endocrinology*, 162(2), Article bqaa204. <https://doi.org/10.1210/endo/bqaa204>
- Clarsen, B., Myklebust, G., & Bahr, R. (2013). Development and validation of a new method for the registration of overuse injuries in sports injury epidemiology: The Oslo Sports Trauma Research Centre (OSTRC) overuse injury questionnaire. *British Journal of Sports Medicine*, 47(8), 495–502.
<https://doi.org/10.1136/bjsports-2012-091524>
- Claudino, A. M., Pike, K. M., Hay, P., Keeley, J. W., Evans, S. C., Rebello, T. J., Bryant-Waugh, R., Dai, Y., Zhao, M., Matsumoto, C., Herscovici, C. R., Mellor-Marsá, B., Stona, A.-C., Kogan, C. S., Andrews, H. F., Monteleone, P., Pilon, D. J., Thiels, C., Sharan, P., ... Reed, G. M. (2019). The classification of feeding and eating disorders in the ICD-11: Results of a field study comparing proposed ICD-11 guidelines with existing ICD-10 guidelines. *BMC Medicine*, 17(1), Article 93.
<https://doi.org/10.1186/s12916-019-1327-4>
- Cobb, K. L., Bachrach, L. K., Greendale, G., Marcus, R., Neer, R. M., Nieves, J., Sowers, M. F., Brown, B. W., Gopalakrishnan, G., Luetters, C., Tanner, H. K., Ward, B., & Kelsey, J. L. (2003). Disordered eating, menstrual irregularity, and bone mineral density in female runners. *Medicine & Science in Sports & Exercise*, 35(5), 711–719.
<https://doi.org/10.1249/01.MSS.0000064935.68277.E7>
- Cole, T. J., Bellizzi, M. C., Flegal, K. M., & Dietz, W. H. (2000). Establishing a standard definition for child overweight and obesity worldwide: International survey. *The BMJ*, 320(7244), 1240–1243.
<https://doi.org/10.1136/bmj.320.7244.1240>

- Cole, T. J., Flegal, K. M., Nicholls, D., & Jackson, A. A. (2007). Body mass index cut offs to define thinness in children and adolescents: International survey. *The BMJ*, 335(7612), Article 194.
<https://doi.org/10.1136/bmj.39238.399444.55>
- Collaborative Group on Hormonal Factors in Breast Cancer. (2012). Menarche, menopause, and breast cancer risk: Individual participant meta-analysis, including 118 964 women with breast cancer from 117 epidemiological studies. *The Lancet Oncology*, 13(11), 1141–1151.
[https://doi.org/10.1016/S1470-2045\(12\)70425-4](https://doi.org/10.1016/S1470-2045(12)70425-4)
- Colvin, C. W., & Abdullatif, H. (2013). Anatomy of female puberty: The clinical relevance of developmental changes in the reproductive system. *Clinical Anatomy*, 26(1), 115–129. <https://doi.org/10.1002/ca.22164>
- Constantini, N. W., & Warren, M. P. (1995). Menstrual dysfunction in swimmers: A distinct entity. *The Journal of Clinical Endocrinology & Metabolism*, 80(9), 2740–2744. <https://doi.org/10.1210/jcem.80.9.7673417>
- Cooper, G. S., & Sandler, D. P. (1997). Long-term effects of reproductive-age menstrual cycle patterns on peri- and postmenopausal fracture risk. *American Journal of Epidemiology*, 145(9), 804–809.
<https://doi.org/10.1093/oxfordjournals.aje.a009173>
- Cooper, R., Blell, M., Hardy, R., Black, S., Pollard, T. M., Wadsworth, M. E. J., Pearce, M. S., & Kuh, D. (2006). Validity of age at menarche self-reported in adulthood. *Journal of Epidemiology & Community Health*, 60(11), 993–997.
<https://doi.org/10.1136/jech.2005.043182>
- Coulombre, J. L., & Russell, E. S. (1954). Analysis of the pleiotropism at the W-locus in the mouse. The effects of W and W^v substitution upon postnatal development of germ cells. *Journal of Experimental Zoology*, 126(2), 277–295.
<https://doi.org/10.1002/jez.1401260207>
- Cousminer, D. L., Mitchell, J. A., Chesi, A., Roy, S. M., Kalkwarf, H. J., Lappe, J. M., Gilsanz, V., Oberfield, S. E., Shepherd, J. A., & Kelly, A. (2018). Genetically determined later puberty impacts lowered bone mineral density in childhood and adulthood. *Journal of Bone and Mineral Research*, 33(3), 430–436. <https://doi.org/10.1002/jbmr.3320>
- Davis, S. R., Lambrinoudaki, I., Lumsden, M., Mishra, G. D., Pal, L., Rees, M., Santoro, N., & Simoncini, T. (2015). Menopause. *Nature Reviews Disease Primers*, 1, Article 150004. <https://doi.org/10.1038/nrdp.2015.4>
- de Borja, C., Chang, C. J., Watkins, R., & Senter, C. (2022). Optimizing health and athletic performance for women. *Current Reviews in Musculoskeletal Medicine*, 15(1), 10–20. <https://doi.org/10.1007/s12178-021-09735-2>
- de Bruin, A. P. K. (2017). Athletes with eating disorder symptomatology, a specific population with specific needs. *Current Opinion in Psychology*, 16, 148–153. <https://doi.org/10.1016/j.copsyc.2017.05.009>
- De Souza, M. J. (2003). Menstrual disturbances in athletes: A focus on luteal phase defects. *Medicine & Science in Sports & Exercise*, 35(9), 1553–1563.
<https://doi.org/10.1249/01.MSS.0000084530.31478.DF>

- De Souza, M. J., Koltun, K. J., Strock, N. C., & Williams, N. I. (2019). Rethinking the concept of an energy availability threshold and its role in the Female Athlete Triad. *Current Opinion in Physiology*, *10*, 35–42.
<https://doi.org/10.1016/j.cophys.2019.04.001>
- De Souza, M. J., Nattiv, A., Joy, E., Misra, M., Williams, N. I., Mallinson, R. J., Gibbs, J. C., Olmsted, M., Goolsby, M., Matheson, G., & Expert Panel. (2014). 2014 Female Athlete Triad coalition consensus statement on treatment and return to play of the Female Athlete Triad: 1st international conference held in San Francisco, California, May 2012 and 2nd international conference held in Indianapolis, Indiana, May 2013. *British Journal of Sports Medicine*, *48*(4), 289–289.
<https://doi.org/10.1136/bjsports-2013-093218>
- De Souza, M. J., Toombs, R. J., Scheid, J. L., O'Donnell, E., West, S. L., & Williams, N. I. (2010). High prevalence of subtle and severe menstrual disturbances in exercising women: Confirmation using daily hormone measures. *Human Reproduction*, *25*(2), 491–503.
<https://doi.org/10.1093/humrep/dep411>
- De Souza, M. J., West, S. L., Jamal, S. A., Hawker, G. A., Gundberg, C. M., & Williams, N. I. (2008). The presence of both an energy deficiency and estrogen deficiency exacerbate alterations of bone metabolism in exercising women. *Bone*, *43*(1), 140–148.
<https://doi.org/10.1016/j.bone.2008.03.013>
- De Souza, M. J., & Williams, N. I. (2004). Physiological aspects and clinical sequelae of energy deficiency and hypoestrogenism in exercising women. *Human Reproduction Update*, *10*(5), 433–448.
<https://doi.org/10.1093/humupd/dmh033>
- De Souza, M. J., Williams, N. I., Nattiv, A., Joy, E., Misra, M., Loucks, A. B., Matheson, G., Olmsted, M. P., Barrack, M., Mallinson, R. J., Gibbs, J. C., Goolsby, M., Nichols, J. F., Drinkwater, B., Sanborn, C. (Barney), Agostini, R., Otis, C. L., Johnson, M. D., Hoch, A. Z., ... McComb, J. (2014). Misunderstanding the Female Athlete Triad: Refuting the IOC consensus statement on Relative Energy Deficiency in Sport (RED-S). *British Journal of Sports Medicine*, *48*(20), 1461–1465. <https://doi.org/10.1136/bjsports-2014-093958>
- Dervish, R. A., Wilson, L. J., & Curtis, C. (2022). Investigating the prevalence of low energy availability, disordered eating and eating disorders in competitive and recreational female endurance runners. *European Journal of Sport Science*. Advance online publication.
<https://doi.org/10.1080/17461391.2022.2079423>
- Deschenes, M. R., & Keiichiro, D. (2005). Basic principles and mechanisms of endocrinology. In W. J. Kraemer & A. D. Rogol (Eds.), *The endocrine system in sports and exercise: An IOC Medical Commission publication, endocrine system in sports and exercise* (pp. 8–24). Blackwell Publishing.
<https://doi.org/10.1002/9780470757826>

- Diamanti-Kandarakis, E., Bourguignon, J.-P., Giudice, L. C., Hauser, R., Prins, G. S., Soto, A. M., Zoeller, R. T., & Gore, A. C. (2009). Endocrine-disrupting chemicals: An Endocrine Society scientific statement. *Endocrine Reviews*, 30(4), 293–342. <https://doi.org/10.1210/er.2009-0002>
- Ding, J., & Zhu, B.-T. (2016). Unique dose-dependent effects of the human pregnancy hormone estriol on the ratio of blood IgM to IgG in female mice. *Molecular Medicine Reports*, 13(1), 447–452. <https://doi.org/10.3892/mmr.2015.4491>
- Dipla, K., Kraemer, R. R., Constantini, N. W., & Hackney, A. C. (2021). Relative energy deficiency in sports (RED-S): Elucidation of endocrine changes affecting the health of males and females. *Hormones*, 20(1), 35–47. <https://doi.org/10.1007/s42000-020-00214-w>
- DiVall, S. A., & Radovick, S. (2009). Endocrinology of female puberty. *Current Opinion in Endocrinology, Diabetes and Obesity*, 16(1), 1–4. <https://doi.org/10.1097/MED.0b013e3283207937>
- DiVasta, A. D., & Gordon, C. M. (2008). Hormone replacement therapy for the adolescent patient. *Annals of the New York Academy of Sciences*, 1135(1), 204–211. <https://doi.org/10.1196/annals.1429.012>
- Donini, L. M., Barrada, J. R., Barthels, F., Dunn, T. M., Babeau, C., Brytek-Matera, A., Cena, H., Cerolini, S., Cho, H., Coimbra, M., Cuzzolaro, M., Ferreira, C., Galfano, V., Grammatikopoulou, M. G., Hallit, S., Håman, L., Hay, P., Jimbo, M., Lasson, C., ... Lombardo, C. (2022). A consensus document on definition and diagnostic criteria for orthorexia nervosa. *Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity*, 27(8), 3695–3711. <https://doi.org/10.1007/s40519-022-01512-5>
- Dratva, J., Gómez Real, F., Schindler, C., Ackermann-Liebrich, U., Gerbase, M. W., Probst-Hensch, N. M., Svanes, C., Omenaas, E. R., Neukirch, F., Wjst, M., Morabia, A., Jarvis, D., Leynaert, B., & Zemp, E. (2009). Is age at menopause increasing across Europe? Results on age at menopause and determinants from two population-based studies. *Menopause*, 16(2), 385–394. <https://doi.org/10.1097/gme.0b013e31818aefef>
- Druet, C., Stettler, N., Sharp, S., Simmons, R. K., Cooper, C., Smith, G. D., Ekelund, U., Lévy-Marchal, C., Jarvelin, M.-R., Kuh, D., & Ong, K. K. (2012). Prediction of childhood obesity by infancy weight gain: An individual-level meta-analysis. *Paediatric and Perinatal Epidemiology*, 26(1), 19–26. <https://doi.org/10.1111/j.1365-3016.2011.01213.x>
- Edelstein, S. L., & Barrett-Connor, E. (1993). Relation between body size and bone mineral density in elderly men and women. *American Journal of Epidemiology*, 138(3), 160–169. <https://doi.org/10.1093/oxfordjournals.aje.a116842>
- Edison, B. R., Pandya, N., Patel, N. M., & Carter, C. W. (2022). Sex and gender differences in pediatric knee injuries. *Clinics in Sports Medicine*, 41(4), 769–787. <https://doi.org/10.1016/j.csm.2022.06.002>
- Ekelund, U., Tarp, J., Steene-Johannessen, J., Hansen, B. H., Jefferis, B., Fagerland, M. W., Whincup, P., Diaz, K. M., Hooker, S. P., Chernofsky, A.,

- Larson, M. G., Spartano, N., Vasani, R. S., Dohrn, I.-M., Hagströmer, M., Edwardson, C., Yates, T., Shiroma, E., Anderssen, S. A., & Lee, I.-M. (2019). Dose-response associations between accelerometry measured physical activity and sedentary time and all cause mortality: Systematic review and harmonised meta-analysis. *The BMJ*, 366, Article l4570. <https://doi.org/10.1136/bmj.l4570>
- Elliott-Sale, K. J., Minahan, C. L., de Jonge, X. A. K. J., Ackerman, K. E., Sipilä, S., Constantini, N. W., Lebrun, C. M., & Hackney, A. C. (2021). Methodological considerations for studies in sport and exercise science with women as participants: A working guide for standards of practice for research on women. *Sports Medicine*, 51(5), 843–861. <https://doi.org/10.1007/s40279-021-01435-8>
- Elliott-Sale, K. J., Tenforde, A. S., Parziale, A. L., Holtzman, B., & Ackerman, K. E. (2018). Endocrine effects of Relative Energy Deficiency in Sport. *International Journal of Sport Nutrition and Exercise Metabolism*, 28(4), 335–349. <https://doi.org/10.1123/ijsnem.2018-0127>
- Erlandson, M. D., Kontulainen, S. A., Chilibeck, P. D., Arnold, C. M., Faulkner, R. A., & Baxter-Jones, A. D. G. (2012). Higher premenarcheal bone mass in elite gymnasts is maintained into young adulthood after long-term retirement from sport: A 14-year follow-up. *Journal of Bone and Mineral Research*, 27(1), 104–110. <https://doi.org/10.1002/jbmr.514>
- Faddy, M. J. (2000). Follicle dynamics during ovarian ageing. *Molecular and Cellular Endocrinology*, 163(1), 43–48. [https://doi.org/10.1016/S0303-7207\(99\)00238-5](https://doi.org/10.1016/S0303-7207(99)00238-5)
- Fahrenholtz, I. L., Sjödin, A., Benardot, D., Tornberg, Å. B., Skouby, S., Faber, J., Sundgot-Borgen, J. K., & Melin, A. K. (2018). Within-day energy deficiency and reproductive function in female endurance athletes. *Scandinavian Journal of Medicine & Science in Sports*, 28(3), 1139–1146. <https://doi.org/10.1111/sms.13030>
- Fairweather-Schmidt, A. K., & Wade, T. D. (2016). Characterizing and predicting trajectories of disordered eating over adolescence. *Journal of Abnormal Psychology*, 125(3), 369–380. <https://doi.org/10.1037/abn0000146>
- Farahmand, M., Ramezani Tehrani, F., Khalili, D., Cheraghi, L., & Azizi, F. (2020). Is there any association between age at menarche and anthropometric indices? A 15-year follow-up population-based cohort study. *European Journal of Pediatrics*, 179(9), 1379–1388. <https://doi.org/10.1007/s00431-020-03575-x>
- Filbay, S., Pandya, T., Thomas, B., McKay, C., Adams, J., & Arden, N. (2019). Quality of life and life satisfaction in former athletes: A systematic review and meta-analysis. *Sports Medicine*, 49(11), 1723–1738. <https://doi.org/10.1007/s40279-019-01163-0>
- Finch, C. E. (2014). The menopause and aging, a comparative perspective. *The Journal of Steroid Biochemistry and Molecular Biology*, 142, 132–141. <https://doi.org/10.1016/j.jsbmb.2013.03.010>

- Fiske, L., Fallon, E. A., Blissmer, B., & Redding, C. A. (2014). Prevalence of body dissatisfaction among United States adults: Review and recommendations for future research. *Eating Behaviors, 15*(3), 357–365. <https://doi.org/10.1016/j.eatbeh.2014.04.010>
- Fogelholm, M., & Hiilloskorpi, H. (1999). Weight and diet concerns in Finnish female and male athletes. *Medicine & Science in Sports & Exercise, 31*(2), 229–235. <https://doi.org/10.1097/00005768-199902000-00005>
- Forest, M. G., Cathiard, A. M., & Bertrand, J. A. (1973). Evidence of testicular activity in early infancy. *The Journal of Clinical Endocrinology & Metabolism, 37*(1), 148–151. <https://doi.org/10.1210/jcem-37-1-148>
- Franco, M. F., Madaleno, F. O., de Paula, T. M. N., Ferreira, T. V., Pinto, R. Z., & Resende, R. A. (2021). Prevalence of overuse injuries in athletes from individual and team sports: A systematic review with meta-analysis and GRADE recommendations. *Brazilian Journal of Physical Therapy, 25*(5), 500–513. <https://doi.org/10.1016/j.bjpt.2021.04.013>
- Fruzzetti, F., Fidicicchi, T., Montt Guevara, M. M., & Simoncini, T. (2021). Estetrol: A new choice for contraception. *Journal of Clinical Medicine, 10*(23), Article 23. <https://doi.org/10.3390/jcm10235625>
- Fujita, Y., Katsumata, K., Unno, A., Tawa, T., & Tokita, A. (1999). Factors affecting peak bone density in Japanese women. *Calcified Tissue International, 64*(2), 107–111. <https://doi.org/10.1007/s002239900587>
- Fuller, C. W., Ekstrand, J., Junge, A., Andersen, T. E., Bahr, R., Dvorak, J., Hägglund, M., McCrory, P., & Meeuwisse, W. H. (2006). Consensus statement on injury definitions and data collection procedures in studies of football (soccer) injuries. *British Journal of Sports Medicine, 40*(3), 193–201. <https://doi.org/10.1136/bjism.2005.025270>
- Fulton, J., Wright, K., Kelly, M., Zebrosky, B., Zanis, M., Drvol, C., & Butler, R. (2014). Injury risk is altered by previous injury: A systematic review of the literature and presentation of causative neuromuscular factors. *International Journal of Sports Physical Therapy, 9*(5), 583–595.
- Gabbett, T. J. (2016). The training – injury prevention paradox: Should athletes be training smarter and harder? *British Journal of Sports Medicine, 50*(5), 273–280. <https://doi.org/10.1136/bjsports-2015-095788>
- García-Galiano, D., Pinilla, L., & Tena-Sempere, M. (2012). Sex steroids and the control of the Kiss1 system: Developmental roles and major regulatory actions. *Journal of Neuroendocrinology, 24*(1), 22–33. <https://doi.org/10.1111/j.1365-2826.2011.02230.x>
- García-Hermoso, A., Ramírez-Campillo, R., & Izquierdo, M. (2019). Is muscular fitness associated with future health benefits in children and adolescents? A systematic review and meta-analysis of longitudinal studies. *Sports Medicine, 49*(7), 1079–1094. <https://doi.org/10.1007/s40279-019-01098-6>
- Garrick, J. G., & Requa, R. K. (2003). Sports and fitness activities: The negative consequences. *The Journal of the American Academy of Orthopaedic Surgeons, 11*(6), 439–443. <https://doi.org/10.5435/00124635-200311000-00008>

- Gelber, A. C., Hochberg, M. C., Mead, L. A., Wang, N.-Y., Wigley, F. M., & Klag, M. J. (2000). Joint injury in young adults and risk for subsequent knee and hip osteoarthritis. *Annals of Internal Medicine*, 133(5), 321–328.
<https://doi.org/10.7326/0003-4819-133-5-200009050-00007>
- Gerlach, K. E., Burton, H. W., Dorn, J. M., Leddy, J. J., & Horvath, P. J. (2008). Fat intake and injury in female runners. *Journal of the International Society of Sports Nutrition*, 5(1), Article 1. <https://doi.org/10.1186/1550-2783-5-1>
- Gibbs, J. C., Nattiv, A., Barrack, M. T., Williams, N. I., Rauh, M. J., Nichols, J. F., & De Souza, M. J. (2014). Low bone density risk is higher in exercising women with multiple triad risk factors. *Medicine & Science in Sports & Exercise*, 46(1), 167–176. <https://doi.org/10.1249/MSS.0b013e3182a03b8b>
- Gibbs, J. C., Williams, N. I., & De Souza, M. J. (2013). Prevalence of individual and combined components of the Female Athlete Triad. *Medicine & Science in Sports & Exercise*, 45(5), 985–996.
<https://doi.org/10.1249/MSS.0b013e31827e1bdc>
- Gideon, N., Hawkes, N., Mond, J., Saunders, R., Tchanturia, K., & Serpell, L. (2016). Development and psychometric validation of the EDE-QS, a 12 item short form of the Eating Disorder Examination Questionnaire (EDE-Q). *PLoS One*, 11(5), Article e0152744.
<http://dx.doi.org/10.1371/journal.pone.0152744>
- Giel, K. E., Hermann-Werner, A., Mayer, J., Diehl, K., Schneider, S., Thiel, A., Zipfel, S., & for the GOAL study group. (2016). Eating disorder pathology in elite adolescent athletes. *International Journal of Eating Disorders*, 49(6), 553–562. <https://doi.org/10.1002/eat.22511>
- Gill, D., Del Greco M, F., Rawson, T. M., Sivakumaran, P., Brown, A., Sheehan, N. A., & Minelli, C. (2017). Fifteen-year prevalence, trajectories, and predictors of body dissatisfaction from adolescence to middle adulthood. *Behavior Genetics*, 47(5), 480–485. <https://doi.org/10.1007/s10519-017-9862-2>
- Gluckman, P. D., & Hanson, M. A. (2006). Changing times: The evolution of puberty. *Molecular and Cellular Endocrinology*, 254–255, 26–31.
<https://doi.org/10.1016/j.mce.2006.04.005>
- Gold, E. B., Crawford, S. L., Avis, N. E., Crandall, C. J., Matthews, K. A., Waetjen, L. E., Lee, J. S., Thurston, R., Vuga, M., & Harlow, S. D. (2013). Factors related to age at natural menopause: Longitudinal analyses from SWAN. *American Journal of Epidemiology*, 178(1), 70–83.
<https://doi.org/10.1093/aje/kws421>
- Gomes, A. R., Martins, C., & Silva, L. (2011). Eating disordered behaviours in Portuguese athletes: The influence of personal, sport, and psychological variables. *European Eating Disorders Review*, 19(3), 190–200.
<https://doi.org/10.1002/erv.1113>
- Gong, T.-T., Wang, Y.-L., & Ma, X.-X. (2015). Age at menarche and endometrial cancer risk: A dose-response meta-analysis of prospective studies. *Scientific Reports*, 5, Article 14051. <https://doi.org/10.1038/srep14051>

- Gong, T.-T., Wu, Q.-J., Vogtmann, E., Lin, B., & Wang, Y.-L. (2013). Age at menarche and risk of ovarian cancer: A meta-analysis of epidemiological studies. *International Journal of Cancer*, 132(12), 2894–2900.
<https://doi.org/10.1002/ijc.27952>
- Goodman, R. L., Herbison, A. E., Lehman, M. N., & Navarro, V. M. (2022). Neuroendocrine control of gonadotropin-releasing hormone: Pulsatile and surge modes of secretion. *Journal of Neuroendocrinology*, 34(5), Article e13094. <https://doi.org/10.1111/jne.13094>
- Gordon, C. M., Ackerman, K. E., Berga, S. L., Kaplan, J. R., Mastorakos, G., Misra, M., Murad, M. H., Santoro, N. F., & Warren, M. P. (2017). Functional hypothalamic amenorrhea: An endocrine society clinical practice guideline. *The Journal of Clinical Endocrinology & Metabolism*, 102(5), 1413–1439. <https://doi.org/10.1210/jc.2017-00131>
- Gottschalk, M. S., Eskild, A., Hofvind, S., Gran, J. M., & Bjelland, E. K. (2020). Temporal trends in age at menarche and age at menopause: A population study of 312 656 women in Norway. *Human Reproduction*, 35(2), 464–471.
<https://doi.org/10.1093/humrep/dez288>
- Gouttebauge, V., Inklaar, H., Backx, F., & Kerckhoffs, G. (2015). Prevalence of osteoarthritis in former elite athletes: A systematic overview of the recent literature. *Rheumatology International*, 35(3), 405–418.
<https://doi.org/10.1007/s00296-014-3093-0>
- Graham, J. D., & Clarke, C. L. (1997). Physiological action of progesterone in target tissues. *Endocrine Reviews*, 18(4), 502–519.
<https://doi.org/10.1210/edrv.18.4.0308>
- Grainge, M. J., Coupland, C. A., Cliffe, S. J., Chilvers, C. E., & Hosking, D. J. (2001). Reproductive, menstrual and menopausal factors: Which are associated with bone mineral density in early postmenopausal women? *Osteoporosis International*, 12(9), 777–787.
<https://doi.org/10.1007/s001980170055>
- Grygorowicz, M., Michałowska, M., Jurga, P., Piontek, T., Jakubowska, H., & Kotwicki, T. (2019). Thirty percent of female footballers terminate their careers due to injury: A retrospective study among Polish former players. *Journal of Sport Rehabilitation*, 28(2), 109–114.
<https://doi.org/10.1123/jsr.2017-0190>
- Gudmundsdottir, S. L., Flanders, W. D., & Augestad, L. B. (2012). Physical activity and age at menopause: The Nord-Trøndelag population-based health study. *Climacteric*, 16(1), 78–87.
<https://doi.org/10.3109/13697137.2011.646344>
- Guest, N. S., & Barr, S. I. (2005). Cognitive dietary restraint is associated with stress fractures in women runners. *International Journal of Sport Nutrition and Exercise Metabolism*, 15(2), 147–159.
<https://doi.org/10.1123/ijsem.15.2.147>
- Guo, Y., Manatunga, A. K., Chen, S., & Marcus, M. (2006). Modeling menstrual cycle length using a mixture distribution. *Biostatistics*, 7(1), 100–114.
<https://doi.org/10.1093/biostatistics/kxi043>

- Guo, Y., Zhao, L.-J., Shen, H., Guo, Y., & Deng, H.-W. (2005). Genetic and environmental correlations between age at menarche and bone mineral density at different skeletal sites. *Calcified Tissue International*, 77(6), 356–360. <https://doi.org/10.1007/s00223-005-0181-x>
- Gusfa, D., Mancine, R., Kennedy, S., Bashir, D. A., & Saffarian, M. (2022). The relationship between disordered eating behaviors and injury rates in adolescent athletes. *International Journal of Eating Disorders*, 55(1), 131–134. <https://doi.org/10.1002/eat.23642>
- Guzmán, A., Hernández-Coronado, C. G., Rosales-Torres, A. M., & Hernández-Medrano, J. H. (2019). Leptin regulates neuropeptides associated with food intake and GnRH secretion. *Annales d'Endocrinologie*, 80(1), 38–46. <https://doi.org/10.1016/j.ando.2018.07.012>
- Hagemans, M. L. C., van der Schouw, Y. T., de Kleijn, M. J. J., van Staveren, W. A., Pop, V. J. M., Leusink, G. L., & Grobbee, D. E. (2004). Indicators for the total duration of premenopausal endogenous estrogen exposure in relation to BMD. *Human Reproduction*, 19(9), 2163–2169. <https://doi.org/10.1093/humrep/deh381>
- Hai, R., Zhang, L., Pei, Y., Zhao, L., Ran, S., Han, Y., Zhu, X., Shen, H., Tian, Q., & Deng, H. (2012). Bivariate genome-wide association study suggests that the DARC gene influences lean body mass and age at menarche. *Science China Life Sciences*, 55(6), 516–520. <https://doi.org/10.1007/s11427-012-4327-6>
- Haines, J., Neumark-Sztainer, D., Wall, M., & Story, M. (2007). Personal, behavioral, and environmental risk and protective factors for adolescent overweight. *Obesity*, 15(11), 2748–2760. <https://doi.org/10.1038/oby.2007.327>
- Hall, J. E. (2015). Endocrinology of the menopause. *Endocrinology and Metabolism Clinics of North America*, 44(3), 485–496. <https://doi.org/10.1016/j.ecl.2015.05.010>
- Harlow, S. D., Gass, M., Hall, J. E., Lobo, R., Maki, P., Rebar, R. W., Sherman, S., Sluss, P. M., de Villiers, T. J., & STRAW + 10 Collaborative Group. (2012). Executive summary of the Stages of Reproductive Aging Workshop + 10: Addressing the unfinished agenda of staging reproductive aging. *The Journal of Clinical Endocrinology & Metabolism*, 97(4), 1159–1168. <https://doi.org/10.1210/jc.2011-3362>
- Harlow, S. D., Lin, X., & Ho, M. J. (2000). Analysis of menstrual diary data across the reproductive life span applicability of the bipartite model approach and the importance of within-woman variance. *Journal of Clinical Epidemiology*, 53(7), 722–733. [https://doi.org/10.1016/S0895-4356\(99\)00202-4](https://doi.org/10.1016/S0895-4356(99)00202-4)
- Haskell, W. L., Lee, I.-M., Pate, R. R., Powell, K. E., Blair, S. N., Franklin, B. A., Macera, C. A., Heath, G. W., Thompson, P. D., & Bauman, A. (2007). Physical activity and public health: Updated recommendation for adults from the American College of Sports Medicine and the American Heart

- Association. *Circulation*, 116(8), 1081–1093.
<https://doi.org/10.1249/mss.0b013e3180616b27>
- Hassa, H., Tanir, H. M., Senses, T., Oge, T., & Sahin-Mutlu, F. (2005). Related factors in bone mineral density of lumbal and femur in natural postmenopausal women. *Archives of Gynecology and Obstetrics*, 273(2), 86–89. <https://doi.org/10.1007/s00404-005-0015-0>
- Hausenblas, H. A., & Symons Downs, D. (2001). Comparison of body image between athletes and nonathletes: A meta-analytic review. *Journal of Applied Sport Psychology*, 13, 323–339.
<https://doi.org/10.1080/104132001753144437>
- Havelock, J. C., Rainey, W. E., & Carr, B. R. (2004). Ovarian granulosa cell lines. *Molecular and Cellular Endocrinology*, 228(1), 67–78.
<https://doi.org/10.1016/j.mce.2004.04.018>
- Hay, P., Aouad, P., Le, A., Marks, P., Maloney, D., Barakat, S., Boakes, R., Brennan, L., Bryant, E., Byrne, S., Caldwell, B., Calvert, S., Carroll, B., Castle, D., Caterson, I., Chelius, B., Chiem, L., Clarke, S., Conti, J., ... National Eating Disorder Research Consortium. (2023). Epidemiology of eating disorders: Population, prevalence, disease burden and quality of life informing public policy in Australia – A rapid review. *Journal of Eating Disorders*, 11(1), Article 23. <https://doi.org/10.1186/s40337-023-00738-7>
- Heaney, S., O'Connor, H., Naughton, G., & Gifford, J. (2008). Towards an understanding of the barriers to good nutrition for elite athletes. *International Journal of Sports Science & Coaching*, 3(3), 391–401.
<https://doi.org/10.1260/174795408786238542>
- Hedman, A., Breithaupt, L., Hübel, C., Thornton, L. M., Tillander, A., Norring, C., Birgegård, A., Larsson, H., Ludvigsson, J. F., Sävendahl, L., Almqvist, C., & Bulik, C. M. (2019). Bidirectional relationship between eating disorders and autoimmune diseases. *Journal of Child Psychology and Psychiatry*, 60(7), 803–812. <https://doi.org/10.1111/jcpp.12958>
- Heikura, I. A., Stellingwerff, T., & Areta, J. L. (2021). Low energy availability in female athletes: From the lab to the field. *European Journal of Sport Science*, 22(5), 707–719. <https://doi.org/10.1080/17461391.2021.1915391>
- Heikura, I. A., Uusitalo, A. L. T., Stellingwerff, T., Bergland, D., Mero, A. A., & Burke, L. M. (2018). Low energy availability is difficult to assess but outcomes have large impact on bone injury rates in elite distance athletes. *International Journal of Sport Nutrition and Exercise Metabolism*, 28(4), 403–411. <https://doi.org/10.1123/ijsnem.2017-0313>
- Herbison, A. E. (2016). Control of puberty onset and fertility by gonadotropin-releasing hormone neurons. *Nature Reviews. Endocrinology*, 12(8), 452–466.
<https://doi.org/10.1038/nrendo.2016.70>
- Herzberg, S. D., Motu'apuaka, M. L., Lambert, W., Fu, R., Brady, J., & Guise, J.-M. (2017). The effect of menstrual cycle and contraceptives on ACL injuries and laxity: A systematic review and meta-analysis. *Orthopaedic Journal of Sports Medicine*, 5(7), Article 2325967117718781.
<https://doi.org/10.1177/2325967117718781>

- Hilbert, A. (2019). Binge-eating disorder. *Psychiatric Clinics of North America*, 42(1), 33–43. <https://doi.org/10.1016/j.psc.2018.10.011>
- Hilibrand, M. J., Hammoud, S., Bishop, M., Woods, D., Fredrick, R. W., & Dodson, C. C. (2015). Common injuries and ailments of the female athlete; Pathophysiology, treatment and prevention. *The Physician and Sportsmedicine*, 43(4), 403–411. <https://doi.org/10.1080/00913847.2015.1092856>
- Hill, J. W., Elmquist, J. K., & Elias, C. F. (2008). Hypothalamic pathways linking energy balance and reproduction. *American Journal of Physiology-Endocrinology and Metabolism*, 294(5), E827–E832. <https://doi.org/10.1152/ajpendo.00670.2007>
- Hipólito Rodrigues, M. A., & Carneiro, M. M. (2022). Estrogen in female health: Friend or foe? *Women & Health*, 62(2), 95–97. <https://doi.org/10.1080/03630242.2022.2035484>
- Hirsch, J. A., Nicola, G., McGinty, G., Liu, R. W., Barr, R. M., Chittle, M. D., & Manchikanti, L. (2016). ICD-10: History and context. *American Journal of Neuroradiology*, 37(4), 596–599. <https://doi.org/10.3174/ajnr.A4696>
- Hirvensalo, M., & Lintunen, T. (2011). Life-course perspective for physical activity and sports participation. *European Review of Aging and Physical Activity*, 8(1), 13–22. <https://doi.org/10.1007/s11556-010-0076-3>
- Hirvensalo, M., Lintunen, T., & Rantanen, T. (2000). The continuity of physical activity—A retrospective and prospective study among older people. *Scandinavian Journal of Medicine & Science in Sports*, 10(1), 37–41. <https://doi.org/10.1111/sms.13205>
- Hoch, A. Z., Pajewski, N. M., Moraski, L., Carrera, G. F., Wilson, C. R., Hoffmann, R. G., Schimke, J. E., & Gutterman, D. D. (2009). Prevalence of the Female Athlete Triad in high school athletes and sedentary students. *Clinical Journal of Sport Medicine*, 19(5), 421–428. <https://doi.org/10.1097/JSM.0b013e3181b8c136>
- Holsen, I., Kraft, P., & Røysamb, E. (2001). The relationship between body image and depressed mood in adolescence: A 5-year longitudinal panel study. *Journal of Health Psychology*, 6(6), 613–627. <https://doi.org/10.1177/135910530100600601>
- Holtzman, B., & Ackerman, K. E. (2021). Recommendations and nutritional considerations for female athletes: Health and performance. *Sports Medicine*, 51(Suppl. 1), 43–57. <https://doi.org/10.1007/s40279-021-01508-8>
- Homan, K. (2010). Athletic-ideal and thin-ideal internalization as prospective predictors of body dissatisfaction, dieting, and compulsive exercise. *Body Image*, 7(3), 240–245. <https://doi.org/10.1016/j.bodyim.2010.02.004>
- Hope, D. C., & Hackney, A. C. (2017). The hypothalamic–pituitary–ovarian axis and oral contraceptives: Regulation and function. In A. C. Hackney (Ed.), *Sex hormones, exercise and women: Scientific and clinical aspects* (pp. 1–17). Springer International Publishing AG. https://doi.org/10.1007/978-3-319-44558-8_9

- Hopkinson, R. A., & Lock, J. (2004). Athletics, perfectionism, and disordered eating. *Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity*, 9(2), 99–106. <https://doi.org/10.1007/BF03325052>
- Hulley, A., Currie, A., Njenga, F., & Hill, A. (2007). Eating disorders in elite female distance runners: Effects of nationality and running environment. *Psychology of Sport and Exercise*, 8(4), 521–533. <https://doi.org/10.1016/j.psychsport.2006.07.001>
- Hutson, M. J., O'Donnell, E., Brooke-Wavell, K., Sale, C., & Blagrove, R. C. (2021). Effects of low energy availability on bone health in endurance athletes and high-impact exercise as a potential countermeasure: A narrative review. *Sports Medicine*, 51(3), 391–403. <https://doi.org/10.1007/s40279-020-01396-4>
- Hyassat, D., Alyan, T., Jaddou, H., & Ajlouni, K. M. (2017). Prevalence and risk factors of osteoporosis among jordanian postmenopausal women attending the National Center for Diabetes, Endocrinology and Genetics in Jordan. *BioResearch Open Access*, 6(1), 85–93. <https://doi.org/10.1089/biores.2016.0045>
- Hyvärinen, M., Sipilä, S., Kulmala, J., Hakonen, H., Tammelin, T. H., Kujala, U. M., Kovanen, V., & Laakkonen, E. K. (2020). Validity and reliability of a single question for leisure-time physical activity assessment in middle-aged women. *Journal of Aging and Physical Activity*, 28(2), 231–241. <https://doi.org/10.1123/japa.2019-0093>
- Ihalainen, J. K., Kettunen, O., McGawley, K., Solli, G. S., Hackney, A. C., Mero, A. A., & Kyröläinen, H. (2021). Body composition, energy availability, training, and menstrual status in female runners. *International Journal of Sports Physiology and Performance*, 16(7), 1043–1048. <https://doi.org/10.1123/ijsp.2020-0276>
- Ihle, R., & Loucks, A. B. (2004). Dose-response relationships between energy availability and bone turnover in young exercising women. *Journal of Bone and Mineral Research*, 19(8), 1231–1240. <https://doi.org/10.1359/JBMR.040410>
- Ikegami, K., Watanabe, Y., Nakamura, S., Goto, T., Inoue, N., Uenoyama, Y., & Tsukamura, H. (2022). Cellular and molecular mechanisms regulating the KNDy neuronal activities to generate and modulate GnRH pulse in mammals. *Frontiers in Neuroendocrinology*, 64, Article 100968. <https://doi.org/10.1016/j.yfrne.2021.100968>
- InterLACE Study Team. (2019). Variations in reproductive events across life: A pooled analysis of data from 505 147 women across 10 countries. *Human Reproduction*, 34(5), 881–893. <https://doi.org/10.1093/humrep/dez015>
- International Olympic Committee. (2021). *Gender equality & inclusion report 2021*. https://stillmed.olympics.com/media/Documents/Beyond-the-Games/Gender-Equality-in-Sport/2021-IOC-Gender-Equality-Inclusion-Report.pdf?_ga=2.12945397.332830447.1678374803-562651686.1677842258

- Ireland, M. L. (2002). The female ACL: Why is it more prone to injury? *The Orthopedic Clinics of North America*, 33(4), 637–651.
[https://doi.org/10.1016/s0030-5898\(02\)00028-7](https://doi.org/10.1016/s0030-5898(02)00028-7)
- Ito, M., Yamada, M., Hayashi, K., Ohki, M., Uetani, M., & Nakamura, T. (1995). Relation of early menarche to high bone mineral density. *Calcified Tissue International*, 57(1), 11–14. <https://doi.org/10.1007/BF00298989>
- Iwasa, T., Matsuzaki, T., Yano, K., Mayila, Y., Yanagihara, R., Yamamoto, Y., Kuwahara, A., & Irahara, M. (2018). Effects of low energy availability on reproductive functions and their underlying neuroendocrine mechanisms. *Journal of Clinical Medicine*, 7(7), Article 166.
<https://doi.org/10.3390/jcm7070166>
- Iwasa, T., Minato, S., Imaizumi, J., Yoshida, A., Kawakita, T., Yoshida, K., & Yamamoto, Y. (2022). Effects of low energy availability on female reproductive function. *Reproductive Medicine and Biology*, 21(1), Article e12414. <https://doi.org/10.1002/rmb2.12414>
- Jacobi, C., Hayward, C., de Zwaan, M., Kraemer, H. C., & Agras, W. S. (2004). Coming to terms with risk factors for eating disorders: Application of risk terminology and suggestions for a general taxonomy. *Psychological Bulletin*, 130(1), 19–65. <https://doi.org/10.1037/0033-2909.130.1.19>
- Janmahasatian, S., Duffull, S. B., Ash, S., Ward, L. C., Byrne, N. M., & Green, B. (2005). Quantification of lean bodyweight. *Clinical Pharmacokinetics*, 44(10), 1051–1065. <https://doi.org/10.2165/00003088-200544100-00004>
- Johnell, O., Gullberg, B., Kanis, J. A., Allander, E., Elffors, L., Dequeker, J., Dilsen, G., Gennari, C., Lopes Vaz, A., & Lyritis, G. (1995). Risk factors for hip fracture in European women: The MEDOS Study. *Journal of Bone and Mineral Research*, 10(11), 1802–1815.
<https://doi.org/10.1002/jbmr.5650101125>
- Johnston, L. H., & Carroll, D. (2000). The psychological impact of injury: Effects of prior sport and exercise involvement. *British Journal of Sports Medicine*, 34(6), 436–439. <https://doi.org/10.1136/bjism.34.6.436>
- Joinson, C., Heron, J., Lewis, G., Croudace, T., & Araya, R. (2011). Timing of menarche and depressive symptoms in adolescent girls from a UK cohort. *The British Journal of Psychiatry*, 198(1), 17–23.
<https://doi.org/10.1192/bjp.bp.110.080861>
- Joubert, L. M., Gonzalez, G. B., & Larson, A. J. (2020). Prevalence of disordered eating among international sport lead rock climbers. *Frontiers in Sports and Active Living*, 2, Article 86. <https://doi.org/10.3389/fspor.2020.00086>
- Juul, F., Chang, V. W., Brar, P., & Parekh, N. (2017). Birth weight, early life weight gain and age at menarche: A systematic review of longitudinal studies. *Obesity Reviews*, 18(11), 1272–1288.
<https://doi.org/10.1111/obr.12587>
- Kally, Z., & Cumella, E. J. (2008). 100 midlife women with eating disorders: A phenomenological analysis of etiology. *The Journal of General Psychology*, 135(4), 359–378. <https://doi.org/10.3200/GENP.135.4.359-378>

- Kaltiala-Heino, R., Kosunen, E., & Rimpelä, M. (2003). Pubertal timing, sexual behaviour and self-reported depression in middle adolescence. *Journal of Adolescence*, 26(5), 531–545. [https://doi.org/10.1016/s0140-1971\(03\)00053-8](https://doi.org/10.1016/s0140-1971(03)00053-8)
- Kampouri, D., Kotopoulea-Nikolaidi, M., Daskou, S., & Giannopoulou, I. (2019). Prevalence of disordered eating in elite female athletes in team sports in Greece. *European Journal of Sport Science*, 19(9), 1267–1275. <https://doi.org/10.1080/17461391.2019.1587520>
- Karapanou, O., & Papadimitriou, A. (2010). Determinants of menarche. *Reproductive Biology and Endocrinology*, 8(1), Article 115. <https://doi.org/10.1186/1477-7827-8-115>
- Karr, T. M., Davidson, D., Bryant, F. B., Balague, G., & Bohnert, A. M. (2013). Sport type and interpersonal and intrapersonal predictors of body dissatisfaction in high school female sport participants. *Body Image*, 10(2), 210–219. <https://doi.org/10.1016/j.bodyim.2012.11.001>
- Kerr, G., Berman, E., & De Souza, M. J. (2006). Disordered eating in women's gymnastics: Perspectives of athletes, coaches, parents, and judges. *Journal of Applied Sport Psychology*, 18(1), 28–43. <https://doi.org/10.1080/10413200500471301>
- Kerr, G., & Dacyshyn, A. (2000). The retirement experiences of elite, female gymnasts. *Journal of Applied Sport Psychology*, 12(2), 115–133. <https://doi.org/10.1080/10413200008404218>
- Kerr, Z. Y., DeFreese, J. D., & Marshall, S. W. (2014). Current physical and mental health of former collegiate athletes. *Orthopaedic Journal of Sports Medicine*, 2(8), Article 2325967114544107. <https://doi.org/10.1177/2325967114544107>
- Keski-Rahkonen, A., & Mustelin, L. (2016). Epidemiology of eating disorders in Europe: Prevalence, incidence, comorbidity, course, consequences, and risk factors. *Current Opinion in Psychiatry*, 29(6), 340–345. <https://doi.org/10.1097/YCO.0000000000000278>
- Khoo, C. C., Woo, J., Leung, P. C., Kwok, A., & Kwok, T. (2011). Determinants of bone mineral density in older postmenopausal Chinese women. *Climacteric*, 14(3), 378–383. <https://doi.org/10.3109/13697137.2010.548566>
- Kim, Y., & Je, Y. (2019). Early menarche and risk of metabolic syndrome: A systematic review and meta-analysis. *Journal of Women's Health*, 28(1), 77–86. <https://doi.org/10.1089/jwh.2018.6998>
- Kirchengast, S., Gruber, D., Sator, M., & Huber, J. (1998). Impact of the age at menarche on adult body composition in healthy pre- and postmenopausal women. *American Journal of Physical Anthropology*, 105(1), 9–20. [https://doi.org/10.1002/\(SICI\)1096-8644\(199801\)105:1<9::AID-AJPA2>3.0.CO;2-T](https://doi.org/10.1002/(SICI)1096-8644(199801)105:1<9::AID-AJPA2>3.0.CO;2-T)
- Klump, K. L. (2013). Puberty as a critical risk period for eating disorders: A review of human and animal studies. *Hormones and Behavior*, 64(2), 399–410. <https://doi.org/10.1016/j.yhbeh.2013.02.019>

- Kodama, S., Saito, K., Tanaka, S., Maki, M., Yachi, Y., Asumi, M., Sugawara, A., Totsuka, K., Shimano, H., Ohashi, Y., Yamada, N., & Sone, H. (2009). Cardiorespiratory fitness as a quantitative predictor of all-cause mortality and cardiovascular events in healthy men and women: A meta-analysis. *JAMA*, 301(19), 2024–2035. <https://doi.org/10.1001/jama.2009.681>
- Kokko, S., Martin, L., Hämylä, R., Kwok, N., Villberg, J., & Suomi, K. (2021). Itsearvioitu liikunta-aktiivisuus, liikuntamuodot, -tilaisuudet ja -paikat, sekä liikkumisen seurantalaitteet ja -sovellukset [Self-reported physical activity, types of exercise, exercise opportunities and locations, as well as physical activity tracking devices and applications]. In S. Kokko, R. Hämylä, & L. Martin (Eds.), *Nuorten liikuntakäyttäytyminen Suomessa – LIITU-tutkimuksen tuloksia 2020* (pp. 16–21). Valtion liikuntaneuvoston julkaisuja 2021:1. <https://www.liikuntaneuvosto.fi/lausunnot-ja-julkaisut/liitu2020/>
- Kokko, S., Selänne, H., Alanko, L., Heinonen, O. J., Korpelainen, R., Savonen, K., Vasankari, T., Kannas, L., Kujala, U. M., Aira, T., Villberg, J., & Parkkari, J. (2015). Health promotion activities of sports clubs and coaches, and health and health behaviours in youth participating in sports clubs: The Health Promoting Sports Club study. *BMJ Open Sport & Exercise Medicine*, 1(1), Article e000034. <https://doi.org/10.1136/bmjsem-2015-000034>
- Koltun, K. J., Williams, N. I., & De Souza, M. J. (2020). Female Athlete Triad Coalition cumulative risk assessment tool: Proposed alternative scoring strategies. *Applied Physiology, Nutrition, and Metabolism*, 45(12), 1324–1331. <https://doi.org/10.1139/apnm-2020-0131>
- Kong, P., & Harris, L. M. (2015). The sporting body: Body image and eating disorder symptomatology among female athletes from leanness focused and nonleanness focused sports. *The Journal of Psychology*, 149(2), 141–160. <https://doi.org/10.1080/00223980.2013.846291>
- Kontro, T. K., Bondarev, D., Pyykönen, K.-M., Bonsdorff, M. von, Laakso, L., Suominen, H., & Korhonen, M. T. (2022). Motives for competitive sports participation in masters track and field athletes: Impact of sociodemographic factors and competitive background. *PloS One*, 17(11), Article e0275900. <https://doi.org/10.1371/journal.pone.0275900>
- Koulanova, A., Sabiston, C. M., Pila, E., Brunet, J., Sylvester, B., Sandmeyer-Graves, A., & Maginn, D. (2021). Ideas for action: Exploring strategies to address body image concerns for adolescent girls involved in sport. *Psychology of Sport and Exercise*, 56, Article 102017. <https://doi.org/10.1016/j.psychsport.2021.102017>
- Kovacs, W. J., & Ojeda, S. R. (2011). Organization of the endocrine system. In W. J. Kovacs & S. R. Ojeda (Eds.), *Textbook of endocrine physiology* (6th ed., pp. 3–20). Oxford University Press. <https://doi.org/10.1093/oso/9780199744121.001.0001>
- Kovanen, V., Aukee, P., Kokko, K., Finni, T., Tarkka, I. M., Tammelin, T., Kujala, U. M., Sipilä, S., & Laakkonen, E. K. (2018). Design and protocol of

- Estrogenic Regulation of Muscle Apoptosis (ERMA) study with 47 to 55-year-old women's cohort: Novel results show menopause-related differences in blood count. *Menopause*, 25(9), 1020–1032.
<https://doi.org/10.1097/GME.0000000000001117>
- Kraemer, R. R., Francois, M., & Castracane, V. D. (2012). Estrogen mediation of hormone responses to exercise. *Metabolism*, 61(10), 1337–1346.
<https://doi.org/10.1016/j.metabol.2012.03.009>
- Kuiri-Hänninen, T., Haanpää, M., Turpeinen, U., Hämäläinen, E., Seuri, R., Tyrväinen, E., Sankilampi, U., & Dunkel, L. (2013). Postnatal ovarian activation has effects in estrogen target tissues in infant girls. *The Journal of Clinical Endocrinology & Metabolism*, 98(12), 4709–4716.
<https://doi.org/10.1210/jc.2013-1677>
- Kuiri-Hänninen, T., Kallio, S., Seuri, R., Tyrväinen, E., Liakka, A., Tapanainen, J., Sankilampi, U., & Dunkel, L. (2011). Postnatal developmental changes in the pituitary-ovarian axis in preterm and term infant girls. *The Journal of Clinical Endocrinology & Metabolism*, 96(11), 3432–3439.
<https://doi.org/10.1210/jc.2011-1502>
- Kujala, U. M., Kaprio, J., Sarna, S., & Koskenvuo, M. (1998). Relationship of leisure-time physical activity and mortality: The Finnish twin cohort. *JAMA*, 279(6), 440–444. <https://doi.org/10.1001/jama.279.6.440>
- Kujala, U. M., Kettunen, J., Paananen, H., Aalto, T., Battié, M. C., Impivaara, O., Videman, T., & Sarna, S. (1995). Knee osteoarthritis in former runners, soccer players, weight lifters, and shooters. *Arthritis & Rheumatism*, 38(4), 539–546. <https://doi.org/10.1002/art.1780380413>
- Kujala, U. M., Orava, S., Parkkari, J., Kaprio, J., & Sarna, S. (2003). Sports career-related musculoskeletal injuries. *Sports Medicine*, 33(12), 869–875.
<https://doi.org/10.2165/00007256-200333120-00001>
- Laakkonen, E., Kovanen, V., & Sipilä, S. (2022). *Data from Estrogenic Regulation of Muscle Apoptosis (ERMA) study* [Data set].
<https://doi.org/10.17011/jyx/dataset/83491>
- Laakkonen, E. K., Kulmala, J., Aukee, P., Hakonen, H., Kujala, U. M., Lowe, D. A., Kovanen, V., Tammelin, T., & Sipilä, S. (2017). Female reproductive factors are associated with objectively measured physical activity in middle-aged women. *PloS One*, 12(2), Article e0172054.
<https://doi.org/10.1371/journal.pone.0172054>
- Lechan, R., M. (2020). Neuroendocrinology. In S. Melmed, R. J. Auchus, A. B. Goldfine, R. J. Koenig, & C. J. Rosen (Eds.), *Williams textbook of endocrinology* (14th ed., pp. 114–183). Elsevier.
- Leddy, M. H., Lambert, M. J., & Ogles, B. M. (1994). Psychological consequences of athletic injury among high-level competitors. *Research Quarterly for Exercise and Sport*, 65(4), 347–354.
<https://doi.org/10.1080/02701367.1994.10607639>
- Lee, M.-H., Kim, S. H., Oh, M., Lee, K.-W., & Park, M.-J. (2016). Age at menarche in Korean adolescents: Trends and influencing factors.

- Reproductive Health*, 13(1), Article 121. <https://doi.org/10.1186/s12978-016-0240-y>
- Lieberman, J. L., De Souza, M. J., Wagstaff, D. A., & Williams, N. I. (2018). Menstrual disruption with exercise is not linked to an energy availability threshold. *Medicine & Science in Sports & Exercise*, 50(3), 551–561. <https://doi.org/10.1249/MSS.0000000000001451>
- Liel, Y., Edwards, J., Shary, J., Spicer, K. M., Gordon, L., & Bell, N. H. (1988). The effects of race and body habitus on bone mineral density of the radius, hip, and spine in premenopausal women. *The Journal of Clinical Endocrinology & Metabolism*, 66(6), 1247–1250. <https://doi.org/10.1210/jcem-66-6-1247>
- Loth, K. A., Watts, A. W., van den Berg, P., & Neumark-Sztainer, D. (2015). Does body satisfaction help or harm overweight teens? A 10-year longitudinal study of the relationship between body satisfaction and body mass index. *The Journal of Adolescent Health*, 57(5), 559–561. <https://doi.org/10.1016/j.jadohealth.2015.07.008>
- Loucks, A. B. (1990). Effects of exercise training on the menstrual cycle: Existence and mechanisms. *Medicine & Science in Sports & Exercise*, 22(3), 275–280.
- Loucks, A. B. (2006). The response of luteinizing hormone pulsatility to 5 days of low energy availability disappears by 14 years of gynecological age. *The Journal of Clinical Endocrinology & Metabolism*, 91(8), 3158–3164. <https://doi.org/10.1210/jc.2006-0570>
- Loucks, A. B. (2013). Energy balance and energy availability. In R. J. Maughan (Ed.), *Sports nutrition: An IOC Medical Commission publication, sports nutrition* (pp. 72–87). John Wiley & Sons.
- Loucks, A. B., & Callister, R. (1993). Induction and prevention of low-T3 syndrome in exercising women. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, 264(5), R924–R930. <https://doi.org/10.1152/ajpregu.1993.264.5.R924>
- Loucks, A. B., & Heath, E. M. (1994a). Induction of low-T3 syndrome in exercising women occurs at a threshold of energy availability. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, 266(3), R817–R823. <https://doi.org/10.1152/ajpregu.1994.266.3.R817>
- Loucks, A. B., & Heath, E. M. (1994b). Dietary restriction reduces luteinizing hormone (LH) pulse frequency during waking hours and increases LH pulse amplitude during sleep in young menstruating women. *The Journal of Clinical Endocrinology & Metabolism*, 78(4), 910–915. <https://doi.org/10.1210/jcem.78.4.8157720>
- Loucks, A. B., Kiens, B., & Wright, H. H. (2011). Energy availability in athletes. *Journal of Sports Sciences*, 29(Suppl. 1), S7–S15. <https://doi.org/10.1080/02640414.2011.588958>
- Loucks, A. B., & Thuma, J. R. (2003). Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating

- women. *The Journal of Clinical Endocrinology & Metabolism*, 88(1), 297–311. <https://doi.org/10.1210/jc.2002-020369>
- Loucks, A. B., Verdun, M., & Heath, E. M. (1998). Low energy availability, not stress of exercise, alters LH pulsatility in exercising women. *Journal of Applied Physiology*, 84(1), 37–46. <https://doi.org/10.1152/jappl.1998.84.1.37>
- Lowry, R., Galuska, D. A., Fulton, J. E., Wechsler, H., Kann, L., & Collins, J. L. (2000). Physical activity, food choice, and weight management goals and practices among US college students. *American Journal of Preventive Medicine*, 18(1), 18–27. [https://doi.org/10.1016/s0749-3797\(99\)00107-5](https://doi.org/10.1016/s0749-3797(99)00107-5)
- Luijken, J., van der Schouw, Y. T., Mensink, D., & Onland-Moret, N. C. (2017). Association between age at menarche and cardiovascular disease: A systematic review on risk and potential mechanisms. *Maturitas*, 104, 96–116. <https://doi.org/10.1016/j.maturitas.2017.07.009>
- MacMahon, C., & Parrington, L. (2017). Not all athletes are equal, but don't call me an exerciser: Response to Araujo and Scharhag. *Scandinavian Journal of Medicine & Science in Sports*, 27(8), 904–906. <https://doi.org/10.1111/sms.12864>
- Maeda, K.-I., Adachi, S., Inoue, K., Ohkura, S., & Tsukamura, H. (2007). Metastin/kisspeptin and control of estrous cycle in rats. *Reviews in Endocrine and Metabolic Disorders*, 8(1), 21–29. <https://doi.org/10.1007/s11154-007-9032-6>
- Maffulli, N., Longo, U. G., Gougoulas, N., Caine, D., & Denaro, V. (2011). Sport injuries: A review of outcomes. *British Medical Bulletin*, 97(1), 47–80. <https://doi.org/10.1093/bmb/ldq026>
- Magkos, F., & Yannakoulia, M. (2003). Methodology of dietary assessment in athletes: Concepts and pitfalls. *Current Opinion in Clinical Nutrition and Metabolic Care*, 6(5), 539–549. <https://doi.org/10.1097/00075197-200309000-00007>
- Magnus, M. C., Lawlor, D. A., Iliodromiti, S., Padmanabhan, S., Nelson, S. M., & Fraser, A. (2018). Age at menarche and cardiometabolic health: A sibling analysis in the Scottish Family Health Study. *Journal of the American Heart Association*, 7(4), Article e007780. <https://doi.org/10.1161/JAHA.117.007780>
- Malina, R. M., Ryan, R. C., & Bonci, C. M. (1994). Age at menarche in athletes and their mothers and sisters. *Annals of Human Biology*, 21(5), 417–422. <https://doi.org/10.1080/03014469400003432>
- Mallinson, R. J., & De Souza, M. J. (2014). Current perspectives on the etiology and manifestation of the “silent” component of the Female Athlete Triad. *International Journal of Women's Health*, 6, 451–467. <https://doi.org/10.2147/IJWH.S38603>
- Mancine, R. P., Gusfa, D. W., Moshrefi, A., & Kennedy, S. F. (2020). Prevalence of disordered eating in athletes categorized by emphasis on leanness and activity type – A systematic review. *Journal of Eating Disorders*, 8(1), Article 47. <https://doi.org/10.1186/s40337-020-00323-2>

- Mannes, Z. L., Waxenberg, L. B., Cottler, L. B., Perlstein, W. M., Burrell, L. E., Ferguson, E. G., Edwards, M. E., & Ennis, N. (2019). Prevalence and correlates of psychological distress among retired elite athletes: A systematic review. *International Review of Sport and Exercise Psychology*, 12(1), 265–294. <https://doi.org/10.1080/1750984X.2018.1469162>
- Mantzoros, C. S. (2000). Role of leptin in reproduction. *Annals of the New York Academy of Sciences*, 900(1), 174–183. <https://doi.org/10.1111/j.1749-6632.2000.tb06228.x>
- Maron, B. J., Thompson, P. D., Ackerman, M. J., Balady, G., Berger, S., Cohen, D., Dimeff, R., Douglas, P. S., Glover, D. W., Hutter, A. M., Krauss, M. D., Maron, M. S., Mitten, M. J., Roberts, W. O., & Puffer, J. C. (2007). Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update. *Circulation*, 115(12), 1643–1655. <https://doi.org/10.1161/CIRCULATIONAHA.107.181423>
- Martin, D., Timmins, K., Cowie, C., Alty, J., Mehta, R., Tang, A., & Varley, I. (2021). Injury incidence across the menstrual cycle in international footballers. *Frontiers in Sports and Active Living*, 3, Article 616999. <https://doi.org/10.3389/fspor.2021.616999>
- Martini, M. C. S., Assumpção, D. de, Barros, M. B. de A., Mattei, J., & Barros Filho, A. de A. (2022). Prevalence of body weight dissatisfaction among adolescents: A systematic review. *Revista Paulista de Pediatria*, 41, Article e2021204. <https://doi.org/10.1590/1984-0462/2023/41/2021204>
- Martinsen, M., Bratland-Sanda, S., Eriksson, A. K., & Sundgot-Borgen, J. (2010). Dieting to win or to be thin? A study of dieting and disordered eating among adolescent elite athletes and non-athlete controls. *British Journal of Sports Medicine*, 44(1), 70–76. <https://doi.org/10.1136/bjism.2009.068668>
- Martinsen, M., & Sundgot-Borgen, J. (2013). Higher prevalence of eating disorders among adolescent elite athletes than controls. *Medicine & Science in Sports & Exercise*, 45(6), 1188–1197. <https://doi.org/10.1249/MSS.0b013e318281a939>
- Martos-Moreno, G. Á., Chowen, J. A., & Argente, J. (2010). Metabolic signals in human puberty: Effects of over and undernutrition. *Molecular and Cellular Endocrinology*, 324(1–2), 70–81. <https://doi.org/10.1016/j.mce.2009.12.017>
- Matthiasdottir, E., Jonsson, S. H., & Kristjansson, A. L. (2012). Body weight dissatisfaction in the Icelandic adult population: A normative discontent? *European Journal of Public Health*, 22(1), 116–121. <https://doi.org/10.1093/eurpub/ckq178>
- McDonald, A. H., Pritchard, M., & McGuire, M. K. (2020). Self-reported eating disorder risk in lean and non-lean NCAA collegiate athletes. *Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity*, 25(3), 745–750. <https://doi.org/10.1007/s40519-019-00681-0>
- McKay, A. K. A., Stellingwerff, T., Smith, E. S., Martin, D. T., Mujika, I., Goosey-Tolfrey, V. L., Sheppard, J., & Burke, L. M. (2021). Defining training and performance caliber: A Participant Classification Framework. *International*

- Journal of Sports Physiology and Performance*, 17(2), 317–331.
<https://doi.org/10.1123/ijsp.2021-0451>
- McKinney, J., Velghe, J., Fee, J., Isserow, S., & Drezner, J. A. (2019). Defining athletes and exercisers. *The American Journal of Cardiology*, 123(3), 532–535.
<https://doi.org/10.1016/j.amjcard.2018.11.001>
- Melin, A., Tornberg, Å. B., Skouby, S., Faber, J., Ritz, C., Sjödin, A., & Sundgot-Borgen, J. (2014). The LEAF questionnaire: A screening tool for the identification of female athletes at risk for the female athlete triad. *British Journal of Sports Medicine*, 48(7), 540–545.
<https://doi.org/10.1136/bjsports-2013-093240>
- Melin, A., Tornberg, Å. B., Skouby, S., Møller, S. S., Faber, J., Sundgot-Borgen, J., & Sjödin, A. (2016). Low-energy density and high fiber intake are dietary concerns in female endurance athletes. *Scandinavian Journal of Medicine & Science in Sports*, 26(9), 1060–1071. <https://doi.org/10.1111/sms.12516>
- Melin, A. K., Areta, J. L., Heikura, I. A., Stellingwerff, T., Torstveit, M. K., & Hackney, A. C. (2023). Direct and indirect impact of low energy availability on sports performance. *Scandinavian Journal of Medicine & Science in Sports*. Advance online publication.
<https://doi.org/10.1111/sms.14327>
- Micali, N., Horton, N. J., Crosby, R. D., Swanson, S. A., Sonnevile, K. R., Solmi, F., Calzo, J. P., Eddy, K. T., & Field, A. E. (2017). Eating disorder behaviours amongst adolescents: Investigating classification, persistence and prospective associations with adverse outcomes using latent class models. *European Child & Adolescent Psychiatry*, 26(2), 231–240.
<https://doi.org/10.1007/s00787-016-0877-7>
- Micklesfield, L. K., Hugo, J., Johnson, C., Noakes, T. D., & Lambert, E. V. (2007). Factors associated with menstrual dysfunction and self-reported bone stress injuries in female runners in the ultra- and half-marathons of the Two Oceans. *British Journal of Sports Medicine*, 41(10), 679–683.
<https://doi.org/10.1136/bjsem.2007.037077>
- Mihm, M., Gangooly, S., & Muttukrishna, S. (2011). The normal menstrual cycle in women. *Animal Reproduction Science*, 124(3), 229–236.
<https://doi.org/10.1016/j.anireprosci.2010.08.030>
- Mohammadi, F., Amirzadeh Iranagh, J., Motalebi, S. A., & Hamid, T. A. (2019). Reproductive factors influencing bone mineral density in postmenopausal women. *Women & Health*, 59(2), 145–154.
<https://doi.org/10.1080/03630242.2018.1434592>
- Mononen, K., Blomqvist, M., Koski, P., & Kokko, S. (2021). Urheilu ja seuraharrastaminen [Sports and sports club participation]. In S. Kokko, R. Hämylä, & L. Martin (Eds.), *Nuorten liikuntakäyttäytyminen Suomessa – LIITU-tutkimuksen tuloksia 2020* (pp. 16–21). Valtion liikuntaneuvoston julkaisuja 2021:1. <https://www.liikuntaneuvosto.fi/lausunnot-ja-julkaisut/liitu2020/>
- Mountjoy, M., Hutchinson, M., Cruz, L., & Lebrun, C. (2015). *Female athlete triad screening questionnaire*. <https://health4performance.co.uk/wp->

[content/uploads/2018/09/Female-Athlete-Triad-Pre-participation-Evaluation.pdf](#)

- Mountjoy, M., Sundgot-Borgen, J., Burke, L., Carter, S., Constantini, N., Lebrun, C., Meyer, N., Sherman, R., Steffen, K., Budgett, R., & Ljungqvist, A. (2014). The IOC consensus statement: Beyond the Female Athlete Triad – Relative Energy Deficiency in Sport (RED-S). *British Journal of Sports Medicine*, 48(7), 491–497. <https://doi.org/10.1136/bjsports-2014-093502>
- Mountjoy, M., Sundgot-Borgen, J., Burke, L., Carter, S., Constantini, N., Lebrun, C., Meyer, N., Sherman, R., Steffen, K., Budgett, R., & Ljungqvist, A. (2015). Authors' 2015 additions to the IOC consensus statement: Relative Energy Deficiency in Sport (RED-S). *British Journal of Sports Medicine*, 49(7), 417–420. <https://doi.org/10.1136/bjsports-2014-094371>
- Mountjoy, M., Sundgot-Borgen, J. K., Burke, L. M., Ackerman, K. E., Blauwet, C., Constantini, N., Lebrun, C., Lundy, B., Melin, A. K., Meyer, N. L., Sherman, R. T., Tenforde, A. S., Klungland Torstveit, M., & Budgett, R. (2018). IOC consensus statement on relative energy deficiency in sport (RED-S): 2018 update. *British Journal of Sports Medicine*, 52(11), 687–697. <https://doi.org/10.1136/bjsports-2018-099193>
- Muia, E. N., Wright, H. H., Onywera, V. O., & Kuria, E. N. (2016). Adolescent elite Kenyan runners are at risk for energy deficiency, menstrual dysfunction and disordered eating. *Journal of Sports Sciences*, 34(7), 598–606. <https://doi.org/10.1080/02640414.2015.1065340>
- Mul, D., Fredriks, A. M., van Buuren, S., Oostdijk, W., Verloove-Vanhorick, S. P., & Wit, J. M. (2001). Pubertal development in the Netherlands 1965–1997. *Pediatric Research*, 50(4), 479–486. <https://doi.org/10.1203/00006450-200110000-00010>
- Müller, M. J., & Bosy-Westphal, A. (2013). Adaptive thermogenesis with weight loss in humans. *Obesity*, 21(2), 218–228. <https://doi.org/10.1002/oby.20027>
- Mumby, H. S., Elks, C. E., Li, S., Sharp, S. J., Khaw, K.-T., Luben, R. N., Wareham, N. J., Loos, R. J. F., & Ong, K. K. (2011). Mendelian randomisation study of childhood BMI and early menarche. *Journal of Obesity*, 2011, Article 180729. <https://doi.org/10.1155/2011/180729>
- Muñoz-Calvo, M. T., & Argente, J. (2016). Nutritional and pubertal disorders. *Endocrine Development*, 29, 153–173. <https://doi.org/10.1159/000438884>
- Murabito, J. M., Yang, Q., Fox, C., Wilson, P. W. F., & Cupples, L. A. (2005). Heritability of age at natural menopause in the Framingham Heart Study. *The Journal of Clinical Endocrinology & Metabolism*, 90(6), 3427–3430. <https://doi.org/10.1210/jc.2005-0181>
- Murakami, H., Kawakami, R., Nakae, S., Nakata, Y., Ishikawa-Takata, K., Tanaka, S., & Miyachi, M. (2016). Accuracy of wearable devices for estimating total energy expenditure: Comparison with metabolic chamber and doubly labeled water method. *JAMA Internal Medicine*, 176(5), 702–703. <https://doi.org/10.1001/jamainternmed.2016.0152>

- Muscat, A. C., & Long, B. C. (2008). Critical comments about body shape and weight: Disordered eating of female athletes and sport participants. *Journal of Applied Sport Psychology, 20*(1), 1–24.
<https://doi.org/10.1080/10413200701784833>
- Must, A., Phillips, S. M., Naumova, E. N., Blum, M., Harris, S., Dawson-Hughes, B., & Rand, W. M. (2002). Recall of early menstrual history and menarcheal body size: After 30 years, how well do women remember? *American Journal of Epidemiology, 155*(7), 672–679.
<https://doi.org/10.1093/aje/155.7.672>
- Mustelin, L., Raevuori, A., Hoek, H. W., Kaprio, J., & Keski-Rahkonen, A. (2015). Incidence and weight trajectories of binge eating disorder among young women in the community. *International Journal of Eating Disorders, 48*(8), 1106–1112. <https://doi.org/10.1002/eat.22409>
- Myburgh, K. H., Hutchins, J., Fataar, A. B., Hough, S. F., & Noakes, T. D. (1990). Low bone density is an etiologic factor for stress fractures in athletes. *Annals of Internal Medicine, 113*(10), 754–759.
<https://doi.org/10.7326/0003-4819-113-10-754>
- Myer, G. D., Jayanthi, N., Difiori, J. P., Faigenbaum, A. D., Kiefer, A. W., Logerstedt, D., & Micheli, L. J. (2015). Sport specialization, part I: Does early sports specialization increase negative outcomes and reduce the opportunity for success in young athletes? *Sports Health, 7*(5), 437–442.
<https://doi.org/10.1177/1941738115598747>
- Myers, J., McAuley, P., Lavie, C. J., Despres, J.-P., Arena, R., & Kokkinos, P. (2015). Physical activity and cardiorespiratory fitness as major markers of cardiovascular risk: Their independent and interwoven importance to health status. *Progress in Cardiovascular Diseases, 57*(4), 306–314.
<https://doi.org/10.1016/j.pcad.2014.09.011>
- Nagata, C., Wada, K., Nakamura, K., Tamai, Y., Tsuji, M., & Shimizu, H. (2012). Associations of physical activity and diet with the onset of menopause in Japanese women. *Menopause, 19*(1), 75–81.
<https://doi.org/10.1097/gme.0b013e3182243737>
- Nakaoka, D., Sugimoto, T., Kaji, H., Kanzawa, M., Yano, S., Yamauchi, M., Sugishita, T., & Chihara, K. (2001). Determinants of bone mineral density and spinal fracture risk in postmenopausal Japanese women. *Osteoporosis International, 12*(7), 548–554. <https://doi.org/10.1007/s001980170075>
- Nattiv, A., Kennedy, G., Barrack, M. T., Abdelkerim, A., Goolsby, M. A., Arends, J. C., & Seeger, L. L. (2013). Correlation of MRI grading of bone stress injuries with clinical risk factors and return to play: A 5-year prospective study in collegiate track and field athletes. *The American Journal of Sports Medicine, 41*(8), 1930–1941.
<https://doi.org/10.1177/0363546513490645>
- Nattiv, A., Loucks, A. B., Manore, M. M., Sanborn, C. F., Sundgot-Borgen, J., Warren, M. P., & American College of Sports Medicine. (2007). American College of Sports Medicine position stand. The Female Athlete Triad.

- Medicine & Science in Sports & Exercise*, 39(10), 1867–1882.
<https://doi.org/10.1249/mss.0b013e318149f111>
- Navarro, V. M., & Kaiser, U. B. (2013). Metabolic influences on neuroendocrine regulation of reproduction. *Current Opinion in Endocrinology, Diabetes, and Obesity*, 20(4), 335–341. <https://doi.org/10.1097/MED.0b013e32836318ce>
- Neighbors, L. A., & Sobal, J. (2007). Prevalence and magnitude of body weight and shape dissatisfaction among university students. *Eating Behaviors*, 8(4), 429–439. <https://doi.org/10.1016/j.eatbeh.2007.03.003>
- Neumark-Sztainer, D., Paxton, S. J., Hannan, P. J., Haines, J., & Story, M. (2006). Does body satisfaction matter? Five-year longitudinal associations between body satisfaction and health behaviors in adolescent females and males. *Journal of Adolescent Health*, 39(2), 244–251.
<https://doi.org/10.1016/j.jadohealth.2005.12.001>
- Neumark-Sztainer, D., Wall, M., Larson, N. I., Eisenberg, M. E., & Loth, K. (2011). Dieting and disordered eating behaviors from adolescence to young adulthood: Findings from a 10-year longitudinal study. *Journal of the American Dietetic Association*, 111(7), 1004–1011.
<https://doi.org/10.1016/j.jada.2011.04.012>
- Nichols, J. F., Rauh, M. J., Barrack, M. T., Barkai, H.-S., & Pernick, Y. (2007). Disordered eating and menstrual irregularity in high school athletes in lean-build and nonlean-build sports. *International Journal of Sport Nutrition and Exercise Metabolism*, 17(4), 364–377.
<https://doi.org/10.1123/ijsnem.17.4.364>
- Nichols, J. F., Rauh, M. J., Lawson, M. J., Ji, M., & Barkai, H.-S. (2006). Prevalence of the Female Athlete Triad syndrome among high school athletes. *Archives of Pediatrics & Adolescent Medicine*, 160(2), 137–142.
<https://doi.org/10.1001/archpedi.160.2.137>
- Nicodemus, K. K., Folsom, A. R., & Anderson, K. E. (2001). Menstrual history and risk of hip fractures in postmenopausal women. The Iowa Women's Health Study. *American Journal of Epidemiology*, 153(3), 251–255.
<https://doi.org/10.1093/aje/153.3.251>
- Ning, H., Du, Y., Zhao, L.-J., Tian, Q., Feng, H., & Deng, H.-W. (2021). The mediating effect of skeletal muscle index on the relationship between menarcheal age and bone mineral density in premenopausal women by race/ethnicity. *Menopause*, 28(10), 1143–1149.
<https://doi.org/10.1097/GME.0000000000001814>
- Nose-Ogura, S., Yoshino, O., Dohi, M., Kigawa, M., Harada, M., Hiraike, O., Onda, T., Osuga, Y., Fujii, T., & Saito, S. (2019). Risk factors of stress fractures due to the female athlete triad: Differences in teens and twenties. *Scandinavian Journal of Medicine & Science in Sports*, 29(10), 1501–1510.
<https://doi.org/10.1111/sms.13464>
- O'Connor, K. A., Holman, D. J., & Wood, J. W. (2001). Menstrual cycle variability and the perimenopause. *American Journal of Human Biology*, 13(4), 465–478. <https://doi.org/10.1002/ajhb.1078>

- Oh, S. R., Choe, S. Y., & Cho, Y. J. (2019). Clinical application of serum anti-Müllerian hormone in women. *Clinical and Experimental Reproductive Medicine*, 46(2), 50–59. <https://doi.org/10.5653/cerm.2019.46.2.50>
- Ojeda, S. R. (2011). Female reproductive function. In W. J. Kovacs & S. R. Ojeda (Eds.), *Textbook of endocrine physiology* (6th ed., pp. 194–238). Oxford University Press. <https://doi.org/10.1093/oso/9780199744121.001.0001>
- Ondrak, K. S., & Morgan, D. W. (2007). Physical activity, calcium intake and bone health in children and adolescents. *Sports Medicine*, 37(7), 587–600. <https://doi.org/10.2165/00007256-200737070-00003>
- Ozdemir, F., Demirbag, D., & Rodoplu, M. (2005). Reproductive factors affecting the bone mineral density in postmenopausal women. *The Tohoku Journal of Experimental Medicine*, 205(3), 277–285. <https://doi.org/10.1620/tjem.205.277>
- Palmer, D., Cooper, D. J., Emery, C., Batt, M. E., Engebretsen, L., Scammell, B. E., Schamasch, P., Shroff, M., Soligard, T., Steffen, K., Whittaker, J. L., & Budgett, R. (2021). Self-reported sports injuries and later-life health status in 3357 retired Olympians from 131 countries: A cross-sectional survey among those competing in the games between London 1948 and PyeongChang 2018. *British Journal of Sports Medicine*, 55(1), 46–53. <https://doi.org/10.1136/bjsports-2019-101772>
- Papadimitriou, A. (2016). The evolution of the age at menarche from prehistorical to modern times. *Journal of Pediatric and Adolescent Gynecology*, 29(6), 527–530. <https://doi.org/10.1016/j.jpjag.2015.12.002>
- Papageorgiou, M., Dolan, E., Elliott-Sale, K. J., & Sale, C. (2018). Reduced energy availability: Implications for bone health in physically active populations. *European Journal of Nutrition*, 57(3), 847–859. <https://doi.org/10.1007/s00394-017-1498-8>
- Papageorgiou, M., Elliott-Sale, K. J., Parsons, A., Tang, J. C. Y., Greeves, J. P., Fraser, W. D., & Sale, C. (2017). Effects of reduced energy availability on bone metabolism in women and men. *Bone*, 105, 191–199. <https://doi.org/10.1016/j.bone.2017.08.019>
- Papageorgiou, M., Martin, D., Colgan, H., Cooper, S., Greeves, J. P., Tang, J. C. Y., Fraser, W. D., Elliott-Sale, K. J., & Sale, C. (2018). Bone metabolic responses to low energy availability achieved by diet or exercise in active eumenorrhic women. *Bone*, 114, 181–188. <https://doi.org/10.1016/j.bone.2018.06.016>
- Parker, S. E., Troisi, R., Wise, L. A., Palmer, J. R., Titus-Ernstoff, L., Strohsnitter, W. C., & Hatch, E. E. (2014). Menarche, menopause, years of menstruation, and the incidence of osteoporosis: The influence of prenatal exposure to diethylstilbestrol. *The Journal of Clinical Endocrinology & Metabolism*, 99(2), 594–601. <https://doi.org/10.1210/jc.2013-2954>
- Pauli, S. A., & Berga, S. L. (2010). Athletic amenorrhea: Energy deficit or psychogenic challenge? *Annals of the New York Academy of Sciences*, 1205(1), 33–38. <https://doi.org/10.1111/j.1749-6632.2010.05663.x>

- Paxton, S. J., Neumark-Sztainer, D., Hannan, P. J., & Eisenberg, M. E. (2006). Body dissatisfaction prospectively predicts depressive mood and low self-esteem in adolescent girls and boys. *Journal of Clinical Child & Adolescent Psychology*, 35(4), 539–549. https://doi.org/10.1207/s15374424jccp3504_5
- Pearson, C. M., Miller, J., Ackard, D. M., Loth, K. A., Wall, M. M., Haynos, A. F., & Neumark-Sztainer, D. (2017). Stability and change in patterns of eating disorder symptoms from adolescence to young adulthood. *International Journal of Eating Disorders*, 50(7), 748–757. <https://doi.org/10.1002/eat.22692>
- Peat, C. M., Peyerl, N. L., & Muehlenkamp, J. J. (2008). Body image and eating disorders in older adults: A review. *The Journal of General Psychology*, 135(4), 343–358. <https://doi.org/10.3200/GENP.135.4.343-358>
- Pelliccia, A., Fagard, R., Bjørnstad, H. H., Anastassakis, A., Arbustini, E., Assanelli, D., Biffi, A., Borjesson, M., Carrè, F., Corrado, D., Delise, P., Dorwarth, U., Hirth, A., Heidbuchel, H., Hoffmann, E., Mellwig, K. P., Panhuyzen-Goedkoop, N., Pisani, A., Solberg, E. E., ... Thiene, G. (2005). Recommendations for competitive sports participation in athletes with cardiovascular disease: A consensus document from the Study Group of Sports Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *European Heart Journal*, 26(14), 1422–1445. <https://doi.org/10.1093/eurheartj/ehi325>
- Pelosi, E., Simonsick, E., Forabosco, A., Garcia-Ortiz, J. E., & Schlessinger, D. (2015). Dynamics of the ovarian reserve and impact of genetic and epidemiological factors on age of menopause. *Biology of Reproduction*, 92(5), Article 130. <https://doi.org/10.1095/biolreprod.114.127381>
- Peltenburg, A. L., Erich, W. B. M., Bernink, M. J. E., Zonderland, M. L., & Huisveld, I. A. (1984). Biological maturation, body composition, and growth of female gymnasts and control groups of schoolgirls and girl swimmers, aged 8 to 14 years: A cross-sectional survey of 1064 girls. *International Journal of Sports Medicine*, 5(1), 36–42. <https://doi.org/10.1055/s-2008-1025878>
- Phelps, L., Johnston, L. S., & Augustyniak, K. (1999). Prevention of eating disorders: identification of predictor variables. *Eating Disorders*, 7(2), 99–108. <https://doi.org/10.1080/10640269908251189>
- Podlog, L., & Eklund, R. C. (2007). The psychosocial aspects of a return to sport following serious injury: A review of the literature from a self-determination perspective. *Psychology of Sport and Exercise*, 8(4), 535–566. <https://doi.org/10.1016/j.psychsport.2006.07.008>
- Pohjolainen, V., Koponen, S., Räsänen, P., Roine, R. P., Sintonen, H., & Karlsson, H. (2016). Long-term health-related quality of life in eating disorders. *Quality of Life Research*, 25(9), 2341–2346. <https://doi.org/10.1007/s11136-016-1250-5>
- Poitras, V. J., Gray, C. E., Borghese, M. M., Carson, V., Chaput, J.-P., Janssen, I., Katzmarzyk, P. T., Pate, R. R., Connor Gorber, S., Kho, M. E., Sampson,

- M., & Tremblay, M. S. (2016). Systematic review of the relationships between objectively measured physical activity and health indicators in school-aged children and youth. *Applied Physiology, Nutrition, and Metabolism*, 41(6 Suppl. 3), S197-S239. <https://doi.org/10.1139/apnm-2015-0663>
- Pollock, M. L., Mengelkoch, L. J., Graves, J. E., Lowenthal, D. T., Limacher, M. C., Foster, C., & Wilmore, J. H. (1997). Twenty-year follow-up of aerobic power and body composition of older track athletes. *Journal of Applied Physiology*, 82(5), 1508-1516. <https://doi.org/10.1152/jappl.1997.82.5.1508>
- Pollock, N. K., Laing, E. M., Modlesky, C. M., O'Connor, P. J., & Lewis, R. D. (2006). Former college artistic gymnasts maintain higher BMD: A nine-year follow-up. *Osteoporosis International*, 17(11), 1691-1697. <https://doi.org/10.1007/s00198-006-0181-3>
- Pope, Z., Gao, Y., Bolter, N., & Pritchard, M. (2015). Validity and reliability of eating disorder assessments used with athletes: A review. *Journal of Sport and Health Science*, 4(3), 211-221. <http://dx.doi.org/10.1016/j.jshs.2014.05.001>
- Popp, K. L., Cooke, L. M., Bouxsein, M. L., & Hughes, J. M. (2022). Impact of low energy availability on skeletal health in physically active adults. *Calcified Tissue International*, 110(5), 605-614. <https://doi.org/10.1007/s00223-022-00957-1>
- Pouta, A., Järvelin, M.-R., Hemminki, E., Sovio, U., & Hartikainen, A.-L. (2005). Mothers and daughters: Intergenerational patterns of reproduction. *European Journal of Public Health*, 15(2), 195-199. <https://doi.org/10.1093/eurpub/cki078>
- Practice Committee of the American Society for Reproductive Medicine. (2004). Current evaluation of amenorrhea. *Fertility and Sterility*, 82(1), 266-272. <https://doi.org/10.1016/j.fertnstert.2004.02.098>
- Practice Committees of the American Society for Reproductive Medicine and the Society for Reproductive Endocrinology and Infertility. (2021). Diagnosis and treatment of luteal phase deficiency: A committee opinion. *Fertility and Sterility*, 115(6), 1416-1423. <https://doi.org/10.1016/j.fertnstert.2021.02.010>
- Prashar, V., Arora, T., Singh, R., Sharma, A., & Parkash, J. (2021). Interplay of KNDy and nNOS neurons: A new possible mechanism of GnRH secretion in the adult brain. *Reproductive Biology*, 21(4), Article 100558. <https://doi.org/10.1016/j.repbio.2021.100558>
- Prather, H., Hunt, D., McKeon, K., Simpson, S., Meyer, E. B., Yemm, T., & Brophy, R. (2016). Are elite female soccer athletes at risk for disordered eating attitudes, menstrual dysfunction, and stress fractures? *PM & R*, 8(3), 208-213. <https://doi.org/10.1016/j.pmrj.2015.07.003>
- Prentice, P., & Viner, R. M. (2013). Pubertal timing and adult obesity and cardiometabolic risk in women and men: A systematic review and meta-analysis. *International Journal of Obesity*, 37(8), 1036-1043. <https://doi.org/10.1038/ijo.2012.177>

- Prnjak, K., Mitchison, D., Griffiths, S., Mond, J., Gideon, N., Serpell, L., & Hay, P. (2020). Further development of the 12-item EDE-QS: Identifying a cut-off for screening purposes. *BMC Psychiatry*, 20(1), Article 146. <https://doi.org/10.1186/s12888-020-02565-5>
- Prus, D., Mijatovic, D., Hadzic, V., Ostojic, D., Versic, S., Zenic, N., Jezdimirovic, T., Drid, P., & Zaletel, P. (2022). (Low) energy availability and its association with injury occurrence in competitive dance: Cross-sectional analysis in female dancers. *Medicina*, 58(7), Article 853. <https://doi.org/10.3390/medicina58070853>
- Putukian, M. (2016). The psychological response to injury in student athletes: A narrative review with a focus on mental health. *British Journal of Sports Medicine*, 50(3), 145–148. <https://doi.org/10.1136/bjsports-2015-095586>
- Puzzitiello, R. N., Rizzo, C. F., Garvey, K. D., Matzkin, E. G., & Salzler, M. J. (2021). Early sports specialisation and the incidence of lower extremity injuries in youth athletes: Current concepts. *Journal of ISAKOS*, 6(6), 339–343. <https://doi.org/10.1136/jisakos-2019-000288>
- Ramraj, B., Subramanian, V. M., & G, V. (2021). Study on age of menarche between generations and the factors associated with it. *Clinical Epidemiology and Global Health*, 11, Article 100758. <https://doi.org/10.1016/j.cegh.2021.100758>
- Rauh, M. J., Barrack, M., & Nichols, J. F. (2014). Associations between the female athlete triad and injury among high school runners. *International Journal of Sports Physical Therapy*, 9(7), 948–958.
- Rauh, M. J., Nichols, J. F., & Barrack, M. T. (2010). Relationships among injury and disordered eating, menstrual dysfunction, and low bone mineral density in high school athletes: A prospective study. *Journal of Athletic Training*, 45(3), 243–252. <https://doi.org/10.4085/1062-6050-45.3.243>
- Raymond-Barker, P., Petroczi, A., & Quested, E. (2007). Assessment of nutritional knowledge in female athletes susceptible to the Female Athlete Triad syndrome. *Journal of Occupational Medicine and Toxicology*, 2, Article 10. <https://doi.org/10.1186/1745-6673-2-10>
- Reardon, C. L., Hainline, B., Aron, C. M., Baron, D., Baum, A. L., Bindra, A., Budgett, R., Campriani, N., Castaldelli-Maia, J. M., Currie, A., Derevensky, J. L., Glick, I. D., Gorczynski, P., Gouttebauge, V., Grandner, M. A., Han, D. H., McDuff, D., Mountjoy, M., Polat, A., ... Engebretsen, L. (2019). Mental health in elite athletes: International Olympic Committee consensus statement (2019). *British Journal of Sports Medicine*, 53(11), 667–699. <https://doi.org/10.1136/bjsports-2019-100715>
- Reed, J. L., De Souza, M. J., Mallinson, R. J., Scheid, J. L., & Williams, N. I. (2015). Energy availability discriminates clinical menstrual status in exercising women. *Journal of the International Society of Sports Nutrition*, 12(1), Article 11. <https://doi.org/10.1186/s12970-015-0072-0>
- Reinking, M. F., & Alexander, L. E. (2005). Prevalence of disordered-eating behaviors in undergraduate female collegiate athletes and nonathletes. *Journal of Athletic Training*, 40(1), 47–51.

- Renstrom, P., Ljungqvist, A., Arendt, E., Beynon, B., Fukubayashi, T., Garrett, W., Georgoulis, T., Hewett, T. E., Johnson, R., Krosshaug, T., Mandelbaum, B., Micheli, L., Myklebust, G., Roos, E., Roos, H., Schamasch, P., Shultz, S., Werner, S., Wojtys, E., & Engebretsen, L. (2008). Non-contact ACL injuries in female athletes: An International Olympic Committee current concepts statement. *British Journal of Sports Medicine*, 42(6), 394–412. <https://doi.org/10.1136/bjism.2008.048934>
- Rettberg, J. R., Yao, J., & Brinton, R. D. (2014). Estrogen: A master regulator of bioenergetic systems in the brain and body. *Frontiers in Neuroendocrinology*, 35(1), 8–30. <https://doi.org/10.1016/j.yfrne.2013.08.001>
- Rideout, C. A., McKay, H. A., & Barr, S. I. (2006). Self-reported lifetime physical activity and areal bone mineral density in healthy postmenopausal women: The importance of teenage activity. *Calcified Tissue International*, 79(4), 214–222. <https://doi.org/10.1007/s00223-006-0058-7>
- Riggs, B. L., Khosla, S., & Melton, L. J. (2002). Sex steroids and the construction and conservation of the adult skeleton. *Endocrine Reviews*, 23(3), 279–302. <https://doi.org/10.1210/edrv.23.3.0465>
- Rigon, F., Bianchin, L., Bernasconi, S., Bona, G., Bozzola, M., Buzi, F., Cicognani, A., De Sanctis, C., De Sanctis, V., Radetti, G., Tatò, L., Tonini, G., & Perissinotto, E. (2010). Update on age at menarche in Italy: Toward the leveling off of the secular trend. *Journal of Adolescent Health*, 46(3), 238–244. <https://doi.org/10.1016/j.jadohealth.2009.07.009>
- Ristolainen, L., Kettunen, J. A., Kujala, U. M., & Heinonen, A. (2012). Sport injuries as the main cause of sport career termination among Finnish top-level athletes. *European Journal of Sport Science*, 12(3), 274–282. <https://doi.org/10.1080/17461391.2011.566365>
- Rizzoli, R., & Bonjour, J. P. (1999). Determinants of peak bone mass and mechanisms of bone loss. *Osteoporosis International*, 9(Suppl. 2), S17–S23. <https://doi.org/10.1007/pl00004155>
- Roa, J. (2013). Role of GnRH neurons and their neuronal afferents as key integrators between food intake regulatory signals and the control of reproduction. *International Journal of Endocrinology*, 2013, Article e518046. <https://doi.org/10.1155/2013/518046>
- Rogers, R. S., & Hinton, P. S. (2010). Bone loading during young adulthood predicts bone mineral density in physically active, middle-aged men. *The Physician and Sportsmedicine*, 38(2), 146–155. <https://doi.org/10.3810/psm.2010.06.1793>
- Rohde, P., Stice, E., & Marti, C. N. (2015). Development and predictive effects of eating disorder risk factors during adolescence: Implications for prevention efforts. *International Journal of Eating Disorders*, 48(2), 187–198. <https://doi.org/10.1002/eat.22270>
- Rosendahl, J., Bormann, B., Aschenbrenner, K., Aschenbrenner, F., & Strauss, B. (2009). Dieting and disordered eating in German high school athletes and non-athletes. *Scandinavian Journal of Medicine & Science in Sports*, 19(5), 731–739. <https://doi.org/10.1111/j.1600-0838.2008.00821.x>

- Rosenthal, D. I., Mayo-Smith, W., Hayes, C. W., Khurana, J. S., Biller, B. M., Neer, R. M., & Klibanski, A. (1989). Age and bone mass in premenopausal women. *Journal of Bone and Mineral Research*, 4(4), 533–538. <https://doi.org/10.1002/jbmr.5650040412>
- Rousselet, M., Guérineau, B., Paruit, M. C., Guinot, M., Lise, S., Destrupe, B., Ruffio-Thery, S., Dominguez, N., Brisseau-Gimenez, S., Dubois, V., Mora, C., Trolonge, S., Lambert, S., Grall-Bronnec, M., & Prétagut, S. (2017). Disordered eating in French high-level athletes: Association with type of sport, doping behavior, and psychological features. *Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity*, 22(1), 61–68. <https://doi.org/10.1007/s40519-016-0342-0>
- Rouveix, M., Bouget, M., Pannafieux, C., Champely, S., & Filaire, E. (2007). Eating attitudes, body esteem, perfectionism and anxiety of judo athletes and nonathletes. *International Journal of Sports Medicine*, 28(4), 340–345. <https://doi.org/10.1055/s-2006-924334>
- Roy, D. K., O'Neill, T. W., Finn, J. D., Lunt, M., Silman, A. J., Felsenberg, D., Armbrecht, G., Banzer, D., Benevolenskaya, L. I., Bhalla, A., Bruges Armas, J., Cannata, J. B., Cooper, C., Dequeker, J., Diaz, M. N., Eastell, R., Yershova, O. B., Felsch, B., Gowin, W., ... Reeve, J. (2003). Determinants of incident vertebral fracture in men and women: Results from the European Prospective Osteoporosis Study (EPOS). *Osteoporosis International*, 14(1), 19–26. <https://doi.org/10.1007/s00198-002-1317-8>
- Ruohonen, S. T., Poutanen, M., & Tena-Sempere, M. (2020). Role of kisspeptins in the control of the hypothalamic-pituitary-ovarian axis: Old dogmas and new challenges. *Fertility and Sterility*, 114(3), 465–474. <https://doi.org/10.1016/j.fertnstert.2020.06.038>
- Saavedra, J. M., Þorgeirsson, S., Kristjansdóttir, H., Halldórsson, K., Guðmundsdóttir, M. L., & Einarsson, I. Þ. (2018). Comparison of training volumes in different elite sportspersons according to sex, age, and sport practised. *Montenegrin Journal of Sports Science and Medicine*, 7(2), 37–42. <https://doi.org/10.26773/mjssm.180906>
- Salardi, S., Orsini, L. F., Cacciari, E., Bovicelli, L., Tassoni, P., & Reggiani, A. (1985). Pelvic ultrasonography in premenarcheal girls: Relation to puberty and sex hormone concentrations. *Archives of Disease in Childhood*, 60(2), 120–125. <https://doi.org/10.1136/adc.60.2.120>
- Sale, C., & Elliott-Sale, K. J. (2019). Nutrition and athlete bone health. *Sports Medicine*, 49(2), 139–151. <https://doi.org/10.1007/s40279-019-01161-2>
- Sampasa-Kanyinga, H., Hamilton, H. A., Willmore, J., & Chaput, J.-P. (2017). Perceptions and attitudes about body weight and adherence to the physical activity recommendation among adolescents: The moderating role of body mass index. *Public Health*, 146, 75–83. <https://doi.org/10.1016/j.puhe.2017.01.002>
- Samson, W. K. (2011). The posterior pituitary and water metabolism. In W. J. Kovacs & S. R. Ojeda (Eds.), *Textbook of endocrine physiology* (6th ed., pp.

- 148–171). Oxford University Press.
<https://doi.org/10.1093/oso/9780199744121.001.0001>
- Sanchez-Garrido, M. A., & Tena-Sempere, M. (2013). Metabolic control of puberty: Roles of leptin and kisspeptins. *Hormones and Behavior*, 64(2), 187–194. <https://doi.org/10.1016/j.yhbeh.2013.01.014>
- Sandler, R. B., Burdett, R., Zaleskiewicz, M., Sprowls-Repcheck, C., & Harwell, M. (1991). Muscle strength as an indicator of the habitual level of physical activity. *Medicine & Science in Sports & Exercise*, 23(12), 1375–1381.
- Sasaki, J. E., John, D., & Freedson, P. S. (2011). Validation and comparison of ActiGraph activity monitors. *Journal of Science and Medicine in Sport*, 14(5), 411–416. <https://doi.org/10.1016/j.jsams.2011.04.003>
- Schaefer, L. M., Burke, N. L., & Thompson, J. K. (2019). Thin-ideal internalization: How much is too much? *Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity*, 24(5), 933–937. <https://doi.org/10.1007/s40519-018-0498-x>
- Schaumberg, K., Welch, E., Breithaupt, L., Hübel, C., Baker, J. H., Munn-Chernoff, M. A., Yilmaz, Z., Ehrlich, S., Mustelin, L., Ghaderi, A., Hardaway, A. J., Bulik-Sullivan, E. C., Hedman, A. M., Jangmo, A., Nilsson, I. A. K., Wiklund, C., Yao, S., Seidel, M., & Bulik, C. M. (2017). The science behind the Academy for Eating Disorders' Nine Truths About Eating Disorders. *European Eating Disorders Review*, 25(6), 432–450. <https://doi.org/10.1002/erv.2553>
- Scheid, J. L., & Stefanik, M. E. (2019). Drive for thinness predicts musculoskeletal injuries in division II NCAA female athletes. *Journal of Functional Morphology and Kinesiology*, 4(3), Article 52. <https://doi.org/10.3390/jfmk4030052>
- Schmidt, H., & Schwarz, H. P. (2000). Serum concentrations of LH and FSH in the healthy newborn. *European Journal of Endocrinology*, 143(2), 213–215. <https://doi.org/10.1530/eje.0.1430213>
- Schoenaker, D. A. J. M., Jackson, C. A., Rowlands, J. V., & Mishra, G. D. (2014). Socioeconomic position, lifestyle factors and age at natural menopause: A systematic review and meta-analyses of studies across six continents. *International Journal of Epidemiology*, 43(5), 1542–1562. <https://doi.org/10.1093/ije/dyu094>
- Shimizu, H., Shimomura, Y., Hayashi, R., Ohtani, K., Sato, N., Futawatari, T., & Mori, M. (1997). Serum leptin concentration is associated with total body fat mass, but not abdominal fat distribution. *International Journal of Obesity*, 21(7), 536–541. <https://doi.org/10.1038/sj.ijo.0800437>
- Shisslak, C. M., Crago, M., & Estes, L. S. (1995). The spectrum of eating disturbances. *International Journal of Eating Disorders*, 18(3), 209–219. [https://doi.org/10.1002/1098-108x\(199511\)18:3<209::aid-eat2260180303>3.0.co;2-e](https://doi.org/10.1002/1098-108x(199511)18:3<209::aid-eat2260180303>3.0.co;2-e)
- Shriver, L. H., Wollenberg, G., & Gates, G. E. (2016). Prevalence of disordered eating and its association with emotion regulation in female college

- athletes. *International Journal of Sport Nutrition and Exercise Metabolism*, 26(3), 240–248. <https://doi.org/10.1123/ijsnem.2015-0166>
- Sievänen, H., & Kujala, U. M. (2017). Accelerometry-Simple, but challenging. *Scandinavian Journal of Medicine & Science in Sports*, 27(6), 574–578. <https://doi.org/10.1111/sms.12887>
- Silén, Y., Sipilä, P. N., Raevuori, A., Mustelin, L., Marttunen, M., Kaprio, J., & Keski-Rahkonen, A. (2021). Detection, treatment, and course of eating disorders in Finland: A population-based study of adolescent and young adult females and males. *European Eating Disorders Review*, 29(5), 720–732. <https://doi.org/10.1002/erv.2838>
- Silman, A. J. (2003). Risk factors for Colles' fracture in men and women: Results from the European Prospective Osteoporosis Study. *Osteoporosis International*, 14(3), 213–218. <https://doi.org/10.1007/s00198-002-1364-1>
- Simon, J. E., & Docherty, C. L. (2017). The impact of previous athletic experience on current physical fitness in former collegiate athletes and noncollegiate athletes. *Sports Health*, 9(5), 462–468. <https://doi.org/10.1177/1941738117705311>
- Sioka, C., Fotopoulos, A., Georgiou, A., Xourgia, X., Papadopoulos, A., & Kalef-Ezra, J. A. (2010). Age at menarche, age at menopause and duration of fertility as risk factors for osteoporosis. *Climacteric*, 13(1), 63–71. <https://doi.org/10.3109/13697130903075337>
- Slane, J. D., Klump, K. L., McGue, M., & Iacono, W. G. (2014). Developmental trajectories of disordered eating from early adolescence to young adulthood: A longitudinal study. *International Journal of Eating Disorders*, 47(7), 793–801. <https://doi.org/10.1002/eat.22329>
- Smith, J. J., Eather, N., Morgan, P. J., Plotnikoff, R. C., Faigenbaum, A. D., & Lubans, D. R. (2014). The health benefits of muscular fitness for children and adolescents: A systematic review and meta-analysis. *Sports Medicine*, 44(9), 1209–1223. <https://doi.org/10.1007/s40279-014-0196-4>
- Smolak, L., Murnen, S. K., & Ruble, A. E. (2000). Female athletes and eating problems: A meta-analysis. *International Journal of Eating Disorders*, 27(4), 371–380. [https://doi.org/10.1002/\(sici\)1098-108x\(200005\)27:4<371::aid-eat1>3.0.co;2-y](https://doi.org/10.1002/(sici)1098-108x(200005)27:4<371::aid-eat1>3.0.co;2-y)
- Sophia, B., Kelly, P., Ogan, D., & Larson, A. (2022). Self reported history of eating disorders, training, weight control methods, and body satisfaction in elite female runners competing at the 2020 U.S. Olympic marathon trials. *International Journal of Exercise Science*, 15(2), 721–732.
- Soules, M. R., Sherman, S., Parrott, E., Rebar, R., Santoro, N., Utian, W., & Woods, N. (2001). Executive summary: Stages of Reproductive Aging Workshop (STRAW). *Climacteric*, 4(4), 267–272. <https://doi.org/10.1080/cmt.4.4.267.272>
- Southmayd, E. A., Mallinson, R. J., Williams, N. I., Mallinson, D. J., & De Souza, M. J. (2017). Unique effects of energy versus estrogen deficiency on multiple components of bone strength in exercising women. *Osteoporosis International*, 28(4), 1365–1376. <https://doi.org/10.1007/s00198-016-3887-x>

- Stice, E., & Shaw, H. E. (2002). Role of body dissatisfaction in the onset and maintenance of eating pathology: A synthesis of research findings. *Journal of Psychosomatic Research*, 53(5), 985–993. [https://doi.org/10.1016/s0022-3999\(02\)00488-9](https://doi.org/10.1016/s0022-3999(02)00488-9)
- Stokowski, S., Paule-Koba, A., L., & Kaunert, C. (2019). Former college athletes' perceptions of adapting to transition. *Journal of Issues in Intercollegiate Athletics*, 12, 403–426.
- Strahler, J., & Stark, R. (2020). Perspective: Classifying orthorexia nervosa as a new mental illness - Much discussion, little evidence. *Advances in Nutrition*, 11(4), 784–789. <https://doi.org/10.1093/advances/nmaa012>
- Striegel-Moore, R. H., & Bulik, C. M. (2007). Risk factors for eating disorders. *American Psychologist*, 62(3), 181–198. <https://doi.org/10.1037/0003-066X.62.3.181>
- Sundgot-Borgen, J. (1994). Risk and trigger factors for the development of eating disorders in female elite athletes. *Medicine & Science in Sports & Exercise*, 26(4), 414–419.
- Sundgot-Borgen, J., & Torstveit, M. K. (2004). Prevalence of eating disorders in elite athletes is higher than in the general population. *Clinical Journal of Sport Medicine*, 14(1), 25–32. <https://doi.org/10.1097/00042752-200401000-00005>
- Sundgot-Borgen, J., & Torstveit, M. K. (2010). Aspects of disordered eating continuum in elite high-intensity sports. *Scandinavian Journal of Medicine & Science in Sports*, 20(Suppl. 2), 112–121. <https://doi.org/10.1111/j.1600-0838.2010.01190.x>
- Swami, V., Steadman, L., & Tovée, M. J. (2009). A comparison of body size ideals, body dissatisfaction, and media influence between female track athletes, martial artists, and non-athletes. *Psychology of Sport and Exercise*, 10(6), 609–614. <https://doi.org/10.1016/j.psychsport.2009.03.003>
- Sweeney, E., Howell, D. R., Seehusen, C. N., Tilley, D., & Casey, E. (2021). Health outcomes among former female collegiate gymnasts: The influence of sport specialization, concussion, and disordered eating. *The Physician and Sportsmedicine*, 49(4), 438–444. <https://doi.org/10.1080/00913847.2020.1850150>
- Tackett, B. P., Petrie, T. A., & Anderson, C. M. (2016). The frequency of weigh-ins, weight intentionality and management, and eating among female collegiate athletes. *Eating Behaviors*, 23, 82–85. <https://doi.org/10.1016/j.eatbeh.2016.08.007>
- Takagi, S., Yoshida, T., Tsubata, K., Ozaki, H., Fujii, T. K., Nomura, Y., & Sawada, M. (1977). Sex differences in fetal gonadotropins and androgens. *Journal of Steroid Biochemistry*, 8(5), 609–620. [https://doi.org/10.1016/0022-4731\(77\)90270-9](https://doi.org/10.1016/0022-4731(77)90270-9)
- Talma, H., Schönbeck, Y., van Dommelen, P., Bakker, B., van Buuren, S., & HiraSing, R. A. (2013). Trends in menarcheal age between 1955 and 2009 in the Netherlands. *PloS One*, 8(4), Article e60056. <https://doi.org/10.1371/journal.pone.0060056>

- Tantleff-Dunn, S., Barnes, R. D., & Larose, J. G. (2011). It's not just a "woman thing:" The current state of normative discontent. *Eating Disorders*, 19(5), 392-402. <https://doi.org/10.1080/10640266.2011.609088>
- Tao, X., Jiang, A., Yin, L., Li, Y., Tao, F., & Hu, H. (2015). Body mass index and age at natural menopause: A meta-analysis. *Menopause*, 22(4), 469-474. <https://doi.org/10.1097/GME.0000000000000324>
- Tenforde, A. S., Carlson, J. L., Chang, A., Sainani, K. L., Shultz, R., Kim, J. H., Cutti, P., Golden, N. H., & Fredericson, M. (2017). Association of the Female Athlete Triad risk assessment stratification to the development of bone stress injuries in collegiate athletes. *The American Journal of Sports Medicine*, 45(2), 302-310. <https://doi.org/10.1177/0363546516676262>
- Tenforde, A. S., & Fredericson, M. (2011). Influence of sports participation on bone health in the young athlete: A review of the literature. *PM&R*, 3(9), 861-867. <https://doi.org/10.1016/j.pmrj.2011.05.019>
- Tenforde, A. S., Sayres, L. C., McCurdy, M. L., Collado, H., Sainani, K. L., & Fredericson, M. (2011). Overuse injuries in high school runners: Lifetime prevalence and prevention strategies. *PM & R*, 3(2), 125-131. <https://doi.org/10.1016/j.pmrj.2010.09.009>
- Tenforde, A. S., Sayres, L. C., McCurdy, M. L., Sainani, K. L., & Fredericson, M. (2013). Identifying sex-specific risk factors for stress fractures in adolescent runners. *Medicine & Science in Sports & Exercise*, 45(10), 1843-1851. <https://doi.org/10.1249/MSS.0b013e3182963d75>
- Thein-Nissenbaum, J. M., Rauh, M. J., Carr, K. E., Loud, K. J., & McGuine, T. A. (2011). Associations between disordered eating, menstrual dysfunction, and musculoskeletal injury among high school athletes. *The Journal of Orthopaedic and Sports Physical Therapy*, 41(2), 60-69. <https://doi.org/10.2519/jospt.2011.3312>
- Thein-Nissenbaum, J. M., Rauh, M. J., Carr, K. E., Loud, K. J., & McGuine, T. A. (2012). Menstrual irregularity and musculoskeletal injury in female high school athletes. *Journal of Athletic Training*, 47(1), 74-82. <https://doi.org/10.4085/1062-6050-47.1.74>
- Thomas, D. T., Erdman, K. A., & Burke, L. M. (2016). American College of Sports Medicine joint position statement. Nutrition and athletic performance. *Medicine & Science in Sports & Exercise*, 48(3), 543-568. <https://doi.org/10.1249/MSS.0000000000000852>
- Thomas, F., Renaud, F., Benefice, E., de Meeüs, T., & Guegan, J. F. (2001). International variability of ages at menarche and menopause: Patterns and main determinants. *Human Biology*, 73(2), 271-290. <https://doi.org/10.1353/hub.2001.0029>
- Thomas, J. J., Keel, P. K., & Heatherton, T. F. (2011). Disordered eating and injuries among adolescent ballet dancers. *Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity*, 16(3), e216-e222. <https://doi.org/10.1007/BF03325136>
- Thompson, J. K., & Stice, E. (2001). Thin-ideal internalization: Mounting evidence for a new risk factor for body-image disturbance and eating

- pathology. *Current Directions in Psychological Science*, 10(5), 181–183.
<https://doi.org/10.1111/1467-8721.00144>
- Thornton, J., Rosen, C., Davenport, M., Mountjoy, M. L., Dorian, P., Gouttebauge, V., Breau, B., Pila, E., Reilly, K., Yuan, J., Mok, K., Ciacca, S. D., Speechley, M., & Crossley, K. (2023). Beyond the medals: A cross-sectional study exploring retired elite female athletes' health. *BMJ Open Sport & Exercise Medicine*, 9(1), Article e001479.
<https://doi.org/10.1136/bmjsem-2022-001479>
- To, W. W., Wong, M. W., & Lam, I. Y. (2000). Hormonal predisposition to menstrual dysfunction in collegiate dance students. *Acta Obstetrica Et Gynecologica Scandinavica*, 79(12), 1117–1123.
<https://doi.org/10.1034/j.1600-0412.2000.0790121117.x>
- Tornberg, Å. B., Melin, A., Koivula, F. M., Johansson, A., Skouby, S., Faber, J., & Sjödin, A. (2017). Reduced neuromuscular performance in amenorrheic elite endurance athletes. *Medicine & Science in Sports & Exercise*, 49(12), 2478–2485. <https://doi.org/10.1249/MSS.0000000000001383>
- Torstveit, M. K., Rosenvinge, J. H., & Sundgot-Borgen, J. (2008). Prevalence of eating disorders and the predictive power of risk models in female elite athletes: A controlled study. *Scandinavian Journal of Medicine & Science in Sports*, 18(1), 108–118. <https://doi.org/10.1111/j.1600-0838.2007.00657.x>
- Torstveit, M. K., & Sundgot-Borgen, J. (2005). Participation in leanness sports but not training volume is associated with menstrual dysfunction: A national survey of 1276 elite athletes and controls. *British Journal of Sports Medicine*, 39(3), 141–147. <https://doi.org/10.1136/bjsem.2003.011338>
- Towne, B., Czerwinski, S. A., Demerath, E. W., Blangero, J., Roche, A. F., & Siervogel, R. M. (2005). Heritability of age at menarche in girls from the Fels Longitudinal Study. *American Journal of Physical Anthropology*, 128(1), 210–219. <https://doi.org/10.1002/ajpa.20106>
- Trace, S. E., Baker, J. H., Peñas-Lledó, E., & Bulik, C. M. (2013). The genetics of eating disorders. *Annual Review of Clinical Psychology*, 9, 589–620.
<https://doi.org/10.1146/annurev-clinpsy-050212-185546>
- Trappe, S. W., Costill, D. L., Vukovich, M. D., Jones, J., & Melham, T. (1996). Aging among elite distance runners: A 22-yr longitudinal study. *Journal of Applied Physiology*, 80(1), 285–290.
<https://doi.org/10.1152/jappl.1996.80.1.285>
- Treasure, J., Duarte, T. A., & Schmidt, U. (2020). Eating disorders. *The Lancet*, 395(10227), 899–911. [https://doi.org/10.1016/S0140-6736\(20\)30059-3](https://doi.org/10.1016/S0140-6736(20)30059-3)
- Trikudanathan, S., Pedley, A., Massaro, J. M., Hoffmann, U., Seely, E. W., Murabito, J. M., & Fox, C. S. (2013). Association of female reproductive factors with body composition: The Framingham Heart Study. *The Journal of Clinical Endocrinology & Metabolism*, 98(1), 236–244.
<https://doi.org/10.1210/jc.2012-1785>
- Tsukamura, H. (2022). Kobayashi Award 2019: The neuroendocrine regulation of the mammalian reproduction. *General and Comparative Endocrinology*, 315, Article 113755. <https://doi.org/10.1016/j.ygcen.2021.113755>

- Tuohisto-Kokko, S., Holttinen, T., & Kaltiala, R. (2023). Morbidity and mortality after adolescent eating disorders requiring inpatient care. *Nordic Journal of Psychiatry*. Advance online publication.
<https://doi.org/10.1080/08039488.2023.2191007>
- Tuppurainen, M., Kröger, H., Saarikoski, S., Honkanen, R., & Alhava, E. (1995). The effect of gynecological risk factors on lumbar and femoral bone mineral density in peri- and postmenopausal women. *Maturitas*, 21(2), 137–145. [https://doi.org/10.1016/0378-5122\(94\)00878-b](https://doi.org/10.1016/0378-5122(94)00878-b)
- Udo, T., & Grilo, C. M. (2019). Psychiatric and medical correlates of DSM-5 eating disorders in a nationally representative sample of adults in the United States. *The International Journal of Eating Disorders*, 52(1), 42–50. <https://doi.org/10.1002/eat.23004>
- Uenoyama, Y., Inoue, N., Nakamura, S., & Tsukamura, H. (2019). Central mechanism controlling pubertal onset in mammals: A triggering role of kisspeptin. *Frontiers in Endocrinology*, 10, Article 312. <https://doi.org/10.3389/fendo.2019.00312>
- Uenoyama, Y., Inoue, N., Nakamura, S., & Tsukamura, H. (2021). Kisspeptin neurons and estrogen–estrogen receptor α signaling: Unraveling the mystery of steroid feedback system regulating mammalian reproduction. *International Journal of Molecular Sciences*, 22(17), Article 9229. <https://doi.org/10.3390/ijms22179229>
- Uher, R., & Rutter, M. (2012). Classification of feeding and eating disorders: Review of evidence and proposals for ICD-11. *World Psychiatry*, 11(2), 80–92. <https://doi.org/10.1016/j.wpsyc.2012.05.005>
- Usselman, C. W., Stachenfeld, N. S., & Bender, J. R. (2016). The molecular actions of estrogen in the regulation of vascular health. *Experimental Physiology*, 101(3), 356–361. <https://doi.org/10.1113/EP085148>
- Vähä-Ypyä, H., Vasankari, T., Husu, P., Mänttari, A., Vuorimaa, T., Suni, J., & Sievänen, H. (2015). Validation of cut-points for evaluating the intensity of physical activity with accelerometry-based mean amplitude deviation (MAD). *PloS One*, 10(8), Article e0134813. <https://doi.org/10.1371/journal.pone.0134813>
- Valdimarsson, O., Alborg, H. G., Düppe, H., Nyquist, F., & Karlsson, M. (2005). Reduced training is associated with increased loss of BMD. *Journal of Bone and Mineral Research*, 20(6), 906–912. <https://doi.org/10.1359/JBMR.050107>
- Välimäki, M. J., Kärkkäinen, M., Lamberg-Allardt, C., Laitinen, K., Alhava, E., Heikkinen, J., Impivaara, O., Mäkelä, P., Palmgren, J., Seppänen, R., Vuori, I., & the Cardiovascular Risk in Young Finns Study Group (1994). Exercise, smoking, and calcium intake during adolescence and early adulthood as determinants of peak bone mass. *The BMJ*, 309(6949), 230–235. <https://doi.org/10.1136/bmj.309.6949.230>
- van Asselt, K. M., Kok, H. S., Pearson, P. L., Dubas, J. S., Peeters, P. H. M., Te Velde, E. R., & van Noord, P. A. H. (2004). Heritability of menopausal age

- in mothers and daughters. *Fertility and Sterility*, 82(5), 1348–1351.
<https://doi.org/10.1016/j.fertnstert.2004.04.047>
- van den Berg, P., & Neumark-Sztainer, D. (2007). Fat 'n happy 5 years later: Is it bad for overweight girls to like their bodies? *The Journal of Adolescent Health*, 41(4), 415–417. <https://doi.org/10.1016/j.jadohealth.2007.06.001>
- van Eeden, A. E., van Hoeken, D., & Hoek, H. W. (2021). Incidence, prevalence and mortality of anorexia nervosa and bulimia nervosa. *Current Opinion in Psychiatry*, 34(6), 515–524.
<https://doi.org/10.1097/YCO.0000000000000739>
- van Hoeken, D., & Hoek, H. W. (2020). Review of the burden of eating disorders: Mortality, disability, costs, quality of life, and family burden. *Current Opinion in Psychiatry*, 33(6), 521–527.
<https://doi.org/10.1097/YCO.0000000000000641>
- Van Langendonck, L., Lefevre, J., Claessens, A. L., Thomis, M., Philippaerts, R., Delvaux, K., Lysens, R., Renson, R., Vanreusel, B., Vanden Eynde, B., Dequeker, J., & Beunen, G. (2003). Influence of participation in high-impact sports during adolescence and adulthood on bone mineral density in middle-aged men: A 27-year follow-up study. *American Journal of Epidemiology*, 158(6), 525–533. <https://doi.org/10.1093/aje/kwg170>
- VanHeest, J. L., Rodgers, C. D., Mahoney, C. E., & De Souza, M. J. (2014). Ovarian suppression impairs sport performance in junior elite female swimmers. *Medicine & Science in Sports & Exercise*, 46(1), 156–166.
<https://doi.org/10.1249/MSS.0b013e3182a32b72>
- Varena, M., Binelli, L., Zucchi, F., Ghiringhelli, D., Gallazzi, M., & Sinigaglia, L. (1999). Prevalence of osteoporosis by educational level in a cohort of postmenopausal women. *Osteoporosis International*, 9(3), 236–241.
<https://doi.org/10.1007/s001980050143>
- Veldhuis, J. D., & Weltman, A. L. (2005). The reproductive axis. In W. J. Kraemer & A. D. Rogol (Eds.), *The endocrine system in sports and exercise: An IOC Medical Commission publication, endocrine system in sports and exercise* (pp. 69–76). Blackwell Publishing. <https://doi.org/10.1002/9780470757826>
- Vihko, R., & Apter, D. (1984). Endocrine characteristics of adolescent menstrual cycles: Impact of early menarche. *Journal of Steroid Biochemistry*, 20(1), 231–236. [https://doi.org/10.1016/0022-4731\(84\)90209-7](https://doi.org/10.1016/0022-4731(84)90209-7)
- von Rosen, P., Frohm, A., Kottorp, A., Fridén, C., & Heijne, A. (2017). Too little sleep and an unhealthy diet could increase the risk of sustaining a new injury in adolescent elite athletes. *Scandinavian Journal of Medicine & Science in Sports*, 27(11), 1364–1371. <https://doi.org/10.1111/sms.12735>
- Wade, G. N., Schneider, J. E., & Li, H. Y. (1996). Control of fertility by metabolic cues. *American Journal of Physiology-Endocrinology and Metabolism*, 270(1), E1–E19. <https://doi.org/10.1152/ajpendo.1996.270.1.E1>
- Wade, T. D. (2019). Recent research on bulimia nervosa. *Psychiatric Clinics of North America*, 42(1), 21–32. <https://doi.org/10.1016/j.psc.2018.10.002>
- Wahab, F., Atika, B., & Shahab, M. (2013). Kisspeptin as a link between metabolism and reproduction: Evidences from rodent and primate

- studies. *Metabolism*, 62(7), 898–910.
<https://doi.org/10.1016/j.metabol.2013.01.015>
- Waller, K., Swan, S. H., Windham, G. C., Fenster, L., Elkin, E. P., & Lasley, B. L. (1998). Use of urine biomarkers to evaluate menstrual function in healthy premenopausal women. *American Journal of Epidemiology*, 147(11), 1071–1080. <https://doi.org/10.1093/oxfordjournals.aje.a009401>
- Wang, L.-J., Zeng, N., Yan, Z.-P., Li, J.-T., & Ni, G.-X. (2020). Post-traumatic osteoarthritis following ACL injury. *Arthritis Research & Therapy*, 22(1), Article 57. <https://doi.org/10.1186/s13075-020-02156-5>
- Wang, S. B., Haynos, A. F., Wall, M. M., Chen, C., Eisenberg, M. E., & Neumark-Sztainer, D. (2019). Fifteen-year prevalence, trajectories, and predictors of body dissatisfaction from adolescence to middle adulthood. *Clinical Psychological Science*, 7(6), 1403–1415.
<https://doi.org/10.1177/2167702619859331>
- Wang, Y.-X., Arvizu, M., Rich-Edwards, J. W., Stuart, J. J., Manson, J. E., Missmer, S. A., Pan, A., & Chavarro, J. E. (2020). Menstrual cycle regularity and length across the reproductive lifespan and risk of premature mortality: Prospective cohort study. *The BMJ*, 371, Article m3464. <https://doi.org/10.1136/bmj.m3464>
- Warren, M. P. (1980). The effects of exercise on pubertal progression and reproductive function in girls. *The Journal of Clinical Endocrinology & Metabolism*, 51(5), 1150–1157. <https://doi.org/10.1210/jcem-51-5-1150>
- Warrick, A., Faustin, M., & Waite, B. (2020). Comparison of Female Athlete Triad (Triad) and Relative Energy Deficiency in Sport (RED-S): A review of low energy availability, multidisciplinary awareness, screening tools and education. *Current Physical Medicine and Rehabilitation Reports*, 8(4), 373–384. <https://doi.org/10.1007/s40141-020-00296-y>
- Wasserfurth, P., Palmowski, J., Hahn, A., & Krüger, K. (2020). Reasons for and consequences of low energy availability in female and male athletes: Social environment, adaptations, and prevention. *Sports Medicine - Open*, 6(1), Article 44. <https://doi.org/10.1186/s40798-020-00275-6>
- Waterman, J. J., & Kapur, R. (2012). Upper gastrointestinal issues in athletes. *Current Sports Medicine Reports*, 11(2), 99–104.
<https://doi.org/10.1249/JSR.0b013e318249c311>
- Weissman, R. S. (2019). The role of sociocultural factors in the etiology of eating disorders. *Psychiatric Clinics of North America*, 42(1), 121–144.
<https://doi.org/10.1016/j.psc.2018.10.009>
- Wells, E. K., Chin, A. D., Tacke, J. A., & Bunn, J. A. (2015). Risk of disordered eating among division I female college athletes. *International Journal of Exercise Science*, 8(3), 256–264.
- Wells, K. R., Jeacocke, N. A., Appaneal, R., Smith, H. D., Vlahovich, N., Burke, L. M., & Hughes, D. (2020). The Australian Institute of Sport (AIS) and National Eating Disorders Collaboration (NEDC) position statement on disordered eating in high performance sport. *British Journal of Sports Medicine*, 54(21), 1247–1258. <https://doi.org/10.1136/bjsports-2019-101813>

- Wentz, L., Liu, P.-Y., Haymes, E., & Ilich, J. Z. (2011). Females have a greater incidence of stress fractures than males in both military and athletic populations: A systemic review. *Military Medicine*, 176(4), 420–430. <https://doi.org/10.7205/milmed-d-10-00322>
- Whitehead, J., Slater, G., Wright, H., Martin, L., O'Connor, H., & Mitchell, L. (2020). Disordered eating behaviours in female physique athletes. *European Journal of Sport Science*, 20(9), 1206–1214. <https://doi.org/10.1080/17461391.2019.1698659>
- Wierman, M. E. (2007). Sex steroid effects at target tissues: Mechanisms of action. *Advances in Physiology Education*, 31(1), 26–33. <https://doi.org/10.1152/advan.00086.2006>
- Wilder, F. V., Hall, B. J., Barrett, J. P., & Lemrow, N. B. (2002). History of acute knee injury and osteoarthritis of the knee: A prospective epidemiological assessment: The Clearwater Osteoarthritis Study. *Osteoarthritis and Cartilage*, 10(8), 611–616. <https://doi.org/10.1053/joca.2002.0795>
- Williams, N. I., Koltun, K. J., Strock, N. C. A., & De Souza, M. J. (2019). Female Athlete Triad and Relative Energy Deficiency in Sport: A focus on scientific rigor. *Exercise and Sport Sciences Reviews*, 47(4), 197–205. <https://doi.org/10.1249/JES.0000000000000200>
- Williams, N. I., Leidy, H. J., Hill, B. R., Lieberman, J. L., Legro, R. S., & De Souza, M. J. (2015). Magnitude of daily energy deficit predicts frequency but not severity of menstrual disturbances associated with exercise and caloric restriction. *American Journal of Physiology-Endocrinology and Metabolism*, 308(1), E29–E39. <https://doi.org/10.1152/ajpendo.00386.2013>
- Williams, N. I., Statuta, S. M., & Austin, A. (2017). Female Athlete Triad: Future directions for energy availability and eating disorder research and practice. *Clinics in Sports Medicine*, 36(4), 671–686. <https://doi.org/10.1016/j.csm.2017.05.003>
- Wise, P. M. (1999). Neuroendocrine modulation of the “menopause”: Insights into the aging brain. *American Journal of Physiology-Endocrinology and Metabolism*, 277(6), E965–970. <https://doi.org/10.1152/ajpendo.1999.277.6.E965>
- Wohlfahrt-Veje, C., Mouritsen, A., Hagen, C. P., Tinggaard, J., Mieritz, M. G., Boas, M., Petersen, J. H., Skakkebaek, N. E., & Main, K. M. (2016). Pubertal onset in boys and girls is influenced by pubertal timing of both parents. *The Journal of Clinical Endocrinology & Metabolism*, 101(7), 2667–2674. <https://doi.org/10.1210/jc.2016-1073>
- World Health Organization. (2023). *International Classification of Diseases, 11th Revision (ICD-11)*. <https://icd.who.int/en>
- Wright, A. A., Taylor, J. B., Ford, K. R., Siska, L., & Smoliga, J. M. (2015). Risk factors associated with lower extremity stress fractures in runners: A systematic review with meta-analysis. *British Journal of Sports Medicine*, 49(23), 1517–1523. <https://doi.org/10.1136/bjsports-2015-094828>
- Wylleman, P., & Reints, A. (2010). A lifespan perspective on the career of talented and elite athletes: Perspectives on high-intensity sports.

- Scandinavian Journal of Medicine & Science in Sports*, 20(Suppl. 2), 88–94.
<https://doi.org/10.1111/j.1600-0838.2010.01194.x>
- Wyshak, G., & Frisch, R. E. (1982). Evidence for a secular trend in age of menarche. *The New England Journal of Medicine*, 306(17), 1033–1035.
<https://doi.org/10.1056/NEJM198204293061707>
- Yang, X., Telama, R., Hirvensalo, M., Viikari, J. S. A., & Raitakari, O. T. (2009). Sustained participation in youth sport decreases metabolic syndrome in adulthood. *International Journal of Obesity*, 33(11), 1219–1226.
<https://doi.org/10.1038/ijo.2009.171>
- Yeager, K. K., Agostini, R., Nattiv, A., & Drinkwater, B. (1993). The female athlete triad: Disordered eating, amenorrhea, osteoporosis. *Medicine & Science in Sports & Exercise*, 25(7), 775–777.
<https://doi.org/10.1249/00005768-199307000-00003>
- Yermachenko, A., & Dvornyk, V. (2014). Nongenetic determinants of age at menarche: A systematic review. *BioMed Research International*, 2014, Article 371583. <https://doi.org/10.1155/2014/371583>
- Zanker, C. L., & Swaine, I. L. (1998). Bone turnover in amenorrhoeic and eumenorrhoeic women distance runners. *Scandinavian Journal of Medicine & Science in Sports*, 8(1), 20–26. <https://doi.org/10.1111/j.1600-0838.1998.tb00224.x>
- Zarów, R., & Cichocka, B. A. (2008). A comparative analysis of estimation of age at menarche by various methods in women participating in the Krakow Longitudinal Growth Study, Poland. *American Journal of Human Biology*, 20(2), 146–148. <https://doi.org/10.1002/ajhb.20701>
- Zhang, Q., Greenbaum, J., Zhang, W.-D., Sun, C.-Q., & Deng, H.-W. (2018). Age at menarche and osteoporosis: A Mendelian randomization study. *Bone*, 117, 91–97. <https://doi.org/10.1016/j.bone.2018.09.015>
- Zhang, Y.-Y., Liu, P.-Y., & Deng, H.-W. (2003). The impact of reproductive and menstrual history on bone mineral density in Chinese women. *Journal of Clinical Densitometry*, 6(3), 289–296. <https://doi.org/10.1385/jcd:6:3:289>
- Zhang, Z.-X., Lei, S.-F., Deng, F.-Y., Zhang, F., Liu, Y.-J., Recker, R. R., Papasian, C. J., & Deng, H.-W. (2009). Bivariate genome-wide linkage analysis for traits BMD and AAM: Effect of menopause on linkage signals. *Maturitas*, 62(1), 16–20. <https://doi.org/10.1016/j.maturitas.2008.10.001>
- Zipfel, S., Giel, K. E., Bulik, C. M., Hay, P., & Schmidt, U. (2015). Anorexia nervosa: Aetiology, assessment, and treatment. *The Lancet Psychiatry*, 2(12), 1099–1111. Scopu. [https://doi.org/10.1016/S2215-0366\(15\)00356-9](https://doi.org/10.1016/S2215-0366(15)00356-9)



ORIGINAL PAPERS

I

MENSTRUAL DYSFUNCTION AND BODY WEIGHT DISSATISFACTION AMONG FINNISH YOUNG ATHLETES AND NON - ATHLETES

by

Ravi, S., Waller, B., Valtonen, M., Villberg, J., Vasankari, T., Parkkari, J.,
Heinonen, O. J., Alanko, L., Savonen, K., Vanhala, M., Selänne, H., Kokko, S.,
& Kujala, U. M. 2021

Scandinavian Journal of Medicine and Science in Sports, 31(2), 405-417

<https://doi.org/10.1111/sms.13838>

Reproduced with kind permission by Wiley.



MISS SUVI RAVI (Orcid ID : 0000-0001-9706-5449)

DR MAARIT VALTONEN (Orcid ID : 0000-0001-8883-2255)

DR TOMMI VASANKARI (Orcid ID : 0000-0001-7209-9351)

Article type : Original Article

Title

Menstrual dysfunction and body weight dissatisfaction among Finnish young athletes and non-athletes

Running title

Menstrual dysfunction in young athletes

Authors

Suvi Ravi¹, Benjamin Waller², Maarit Valtonen³, Jari Villberg¹, Tommi Vasankari⁴, Jari Parkkari⁵, Olli J. Heinonen⁶, Lauri Alanko^{7,8}, Kai Savonen^{9,10}, Marja Vanhala¹¹, Harri Selänne¹², Sami Kokko¹, Urho M. Kujala¹

¹Faculty of Sport and Health Sciences, University of Jyväskylä, Jyväskylä, Finland

²Physical Activity, Physical Education, Sport and Health Research Centre, Sports Science Department, School of Social Sciences, Reykjavik University, Reykjavik, Iceland

³Research Institute for Olympic Sports, Jyväskylä, Finland

⁴UKK Institute for Health Promotion Research, Tampere, Finland

⁵Tampere Research Center of Sports Medicine, Tampere, Finland

⁶Paavo Nurmi Centre & Unit for Health and Physical Activity, University of Turku, Turku, Finland

This article has been accepted for publication and undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the [Version of Record](#). Please cite this article as [doi: 10.1111/sms.13838](https://doi.org/10.1111/sms.13838)

This article is protected by copyright. All rights reserved

⁷Sports Medicine Clinic, Foundation for Sports and Exercise Clinic, Helsinki, Finland

⁸Central Finland Central Hospital, Jyväskylä, Finland

⁹Kuopio Research Institute of Exercise Medicine, Kuopio, Finland

¹⁰Department of Clinical Physiology and Nuclear Medicine, Kuopio University Hospital, Kuopio, Finland

¹¹Department of Sports and Exercise Medicine, Oulu Deaconess Institute Foundation, Oulu, Finland

Center for Life Course Health Research, Faculty of Medicine, University of Oulu, Oulu, Finland

Medical Research Center, Oulu University Hospital and University of Oulu, Oulu, Finland

¹²Faculty of Education and Psychology, Department of Psychology, University of Jyväskylä, Finland

Corresponding author: Suvi Ravi, Faculty of Sport and Health Sciences, University of Jyväskylä, Jyväskylä, Finland. Email: suvi.m.ravi@jyu.fi

Acknowledgments

The authors thank all the sports clubs and schools who participated, the young people who took part in the study, and the Finnish Ministry of Education and Culture for funding the study (grant numbers: 6/091/2011, 67/626/2015).

Conflict of interest

The authors have no conflicts of interest.

ABSTRACT

To determine the prevalence of menstrual dysfunction (MD; i.e. oligomenorrhea or amenorrhea) and attitudes towards body weight among athletes and non-athletes, we studied a cohort of athletes and non-athletes, in adolescence (14–16 years) and subsequently in young adulthood (18–20

years). We further studied the differences between athletes reporting MD and eumenorrheic athletes at both time periods and identified physical and behavioural characteristics that might predict MD in young adulthood. Data were collected using questionnaires, accelerometers, and a pre-participation screening. In adolescence, the athletes reported current primary amenorrhea more often than the non-athletes (4.7% vs. 0%, $p=0.03$). In young adulthood, athletes reported MD more frequently than non-athletes (38.7% vs. 5.6%, $p<0.001$). Athletes had less desire than non-athletes to lose weight at both time points, and in adolescence athletes were more satisfied with their weight. However, about one fifth of the athletes and about 40% of the non-athletes experienced body weight dissatisfaction at both time points. In adolescence, athletes reporting MD had lower BMI than eumenorrheic athletes. In young adulthood, athletes with MD were more physically active than eumenorrheic athletes. The only longitudinal predictor of MD in young adulthood was MD in adolescence. Our findings indicate that MD is relatively frequent among young Finnish athletes. However, athletes appear to have a smaller tendency to experience body weight dissatisfaction than their non-athletic peers. MD seems to track from adolescence to adulthood, suggesting that there is a need to focus on possible causes at the earliest feasible phase of an athlete's career.

Key words: menstrual dysfunction, amenorrhea, body weight dissatisfaction, exercising women, young athletes

Introduction

Menstrual dysfunction (MD) is one of the components of the Female Athlete Triad/Relative Energy Deficiency in Sport (RED-S) consisting of an irregular or absent menstrual cycle as well as subclinical MD with no perceptible symptoms, such as luteal phase deficiency and anovulation.¹⁻³ MD is fairly common among female athletes and it is suggested to be more common in athletes competing in sports that emphasise leanness.^{2,4,5} MD prevalence rates vary between studies depending on the diagnostic criteria and the population studied, with studies usually investigating only clinically detectable MD.⁴⁻¹⁴ Nevertheless, the results of studies comparing the prevalence of MD in athletes and non-athletes have been inconsistent. Hoch et al.¹² and Muia et al.⁶ found that MD was more common in runners and athletes competing in several different sports, respectively, than in sedentary controls. Contrastingly, in other studies the MD prevalence between athletes

competing in different sports and controls has been similar.^{4,7} Additionally, Fogelholm and Hiilloskorpi⁸ found no difference in the prevalence of MD between athletes and controls, although there was a trend towards a lower prevalence of MD in the control group. It has further been observed that there is a difference in menarcheal age between athletes and non-athletes with the former attaining menarche later.^{4,12}

Although there are several different reasons for MD, in short-term clinical studies, low energy availability (EA) has been found to cause metabolic and endocrine changes, indicating that it is the primary reason for MD in the Female Athlete Triad/RED-S.^{1-3,15} Nevertheless, the impact of low EA on MD is modified by several factors, including genetic and psychological factors.^{16,17} Low EA, and consequently MD, can result from intentionally or unintentionally decreased energy intake with a possible combination of increased energy expenditure.^{1,18} Thus, disordered eating (DE)/eating disorder (ED) can also result in low EA,¹⁻³ especially in sports emphasising leanness.¹⁹ One risk factor for DE/ED is body weight dissatisfaction (i.e. the discrepancy between actual and desired weight).²⁰ It has previously been observed that body weight dissatisfaction is present among young female adults²¹ and adolescents.²² Being an athlete can protect from or cause body weight dissatisfaction. On the one hand, athletes may experience pressures to change their body weight in order to meet the “ideal” body shape or for perceived performance improvements.²³ On the other hand, it has been established that lower BMI and physical activity are associated with lower body dissatisfaction.²⁴

Few studies have investigated training volume/physical activity between athletes with MD and eumenorrheic athletes. Torstveit and Sundgot-Borgen⁴ found that self-reported training volume was not associated with MD in elite athletes, while in the study conducted by Henriksson et al.,²⁵ athletes with MD reported higher training hours than eumenorrheic athletes. In addition, Tornberg et al.²⁶ found that aerobic exercise volume assessed by training logs and heart rate monitors did not differ between endurance athletes with MD and eumenorrheic endurance athletes, although amenorrheic athletes performed more resistance training. Furthermore, other studies have found no difference between amenorrheic and eumenorrheic athletes with regard to exercise energy expenditure, based on self-reported physical activity,²⁷ or on objectively measured non-exercise physical thermogenesis and exercise energy expenditure.²⁸ However, more research is needed to investigate differences in objectively measured physical activity between athletes with MD and eumenorrheic athletes.

Our study was designed to compare the prevalence of self-reported MD (irregular or absent menstrual cycles), and attitudes towards body weight in a cohort of young Finnish athletes and non-athletes at two life stages, namely adolescence (ages 14–16) and young adulthood (ages 18–20). We also analysed attitudes towards body weight by different weight categories (underweight, normal weight, and overweight). In addition, we investigated whether there were differences in BMI, objectively measured physical activity, self-reported training hours, or attitudes towards body weight among athletes reporting MD and eumenorrheic athletes. Finally, we conducted analyses to determine whether BMI, MD, attitudes towards body weight, physical activity, or age at menarche in adolescence might predict MD in young adulthood.

Materials and methods

Participants

This study formed part of the *Finnish Health Promoting Sports Club* (FHPSC) study.²⁹ In the present study, baseline and follow-up surveys were used. The baseline data were collected in 2013 in two rounds separately for participants in winter and summer sports aged 14–16 years. Detailed information on the recruiting process has been presented elsewhere.²⁹ In brief, 272 sports clubs from the 10 most popular sports disciplines in Finland were approached and from these, 156 clubs (85 winter disciplines and 71 summer disciplines) agreed to participate in the study. Comparative data for non-sports club members (non-athletes) were collected at the same time from schools. The sample for the present study consisted of the girls who underwent pre-participation screening of the FHPSC study, where medical history as well as height and weight was assessed. Participants for the pre-participation screening were randomly invited from both the sports club participants and non-participants who replied to the questionnaires administered to the whole sample of the FHPSC study. In the present study, girls who participated in a sports club and exercised at least four times a week in the training season were considered as athletes. Thus, sports club participants who exercised less than four times a week in the training season were excluded (Figure 1). There was no difference in the prevalence of MD between the excluded sports club members and the members included in the study ($p=0.48$), but the excluded members had a lower age at menarche (12.2 vs. 12.7, $p=0.02$). The final sample size at baseline (i.e. at adolescence) was 283 (178 athletes, 105 non-athletes).

The follow-up data for the FHPSC study were collected in 2017–2018 when the participants were 18–20 years of age. All the participants who had undergone the pre-participation screening at adolescence and who had given their consent to be re-contacted were invited to participate for the follow-up. In total, 227 (72.1%) of the 315 girls invited, agreed to participate. Those who refused to participate had a similar MD prevalence at adolescence to those who agreed to participate in the follow-up (23.8% and 20.0%, respectively, $p=0.61$). Sports club members who reported exercising less than four times a week in the training season were excluded (Figure 1). Those excluded had a similar prevalence of MD ($p=0.15$) and age at menarche ($p=0.11$) to the athletes included in the study. The final sample size at follow-up (in young adulthood) was 211 (52 athletes, 159 non-athletes).

Sports disciplines were categorised by modifying the criteria of Sundgot-Borgen and Torstveit³⁰ as follows: endurance (cross-country skiing, orienteering, swimming, race walking, and canoe sprint), aesthetic (synchronised skating, figure skating, synchronized swimming, gymnastics, and sport dance), technical (horse riding), ball games (soccer, basketball, floorball, badminton, water ball, ice-hockey, and ringette), and power/anti-gravitation sports (track and field, speed skating, and taekwondo).

The declaration of Helsinki was followed throughout the study, and the study was approved by the Ethics Committee of the Health Care District of Central Finland. All the participants gave written informed consent before entering the study. In addition, informed consent was requested from a guardian if a participant was under 18 years of age.

Data collection

The baseline data and the follow-up data were collected by with an identical protocol, explained elsewhere,²⁹ using structured questionnaires, pre-participation screening, and accelerometers.

Questionnaires:

Menstrual status questions included: age at menarche (if occurred), duration of the menstrual cycle, number of periods in the preceding year, and hormonal contraceptive (e.g. oral contraceptives, implants, injections, transdermal patches, vaginal rings, or intrauterine systems) use. In the case of an absence of periods, inquiry was made of the number of consecutively missed

cycles. The participants' gynaecological age was determined by subtracting the age at menarche from their chronological age. Participants who did not use hormonal contraceptives were then classified into the MD group (menarche not occurred by the age of 15, i.e. primary amenorrhea, having ≤ 9 periods in a preceding year, duration of the menstrual cycle >35 days, or missing periods in ≥ 3 previous months after menarche),¹ or the eumenorrhea group. Participants whose menstrual cycle was 36–89 days and whose gynaecological age was one year or less were excluded from this analysis; this was due to the fact that it takes some time to achieve a regular menstrual cycle, and there is great variation in durations between the first and second menstrual cycle. However, menstrual cycles exceeding 90 days are not normal, even when they occur in the first gynaecological year.³¹

The following yes/no questions were asked regarding participants' attitudes towards body weight: 'Are you satisfied with your present weight?', 'Do you think that you should lose weight?', and 'Do you think that you should gain weight?'

Weekly training hours were reported separately in the training and competition season, in addition to weekly rest days during the training and competition season.

Pre-participation screening:

In the pre-participation screening, height and weight, without shoes, were measured in the laboratory to the nearest 0.5cm and 0.1kg, respectively. BMI was calculated on the basis of these data. The adolescents' BMI was age-adjusted based on age- and gender-specific BMI international cut-off points, using the participants' mean age (15 years).^{32,33} Hence, BMI $<17.5\text{kg/m}^2$ was classified as underweight, BMI $=17.5\text{--}23.9\text{kg/m}^2$ was classified as normal weight, and BMI $>23.9\text{kg/m}^2$ was classified as overweight. At the young adulthood stage, the cut-off points were: underweight = BMI $<18.5\text{kg/m}^2$; normal weight = BMI $18.5\text{--}24.9\text{kg/m}^2$; overweight = BMI $\geq 25.0\text{kg/m}^2$. In the current study, obese participants (2 and 13 in adolescence and in young adulthood, respectively) were included in the overweight group.

Accelerometer data:

Physical activity was measured with a hip-worn, light triaxial accelerometer (Hookie AM20, Traxmeet Ltd., Espoo, Finland). The accelerometer was set up during the pre-participation screening and subsequently returned or mailed back to the research unit. Participants were asked

to wear the accelerometer during waking hours (excluding activities in water) for seven consecutive days. The analysis of the raw acceleration data was based on novel algorithms that employed the mean amplitude deviation (MAD) of the resultant acceleration in 6-s epochs, and the angle for posture estimation (APE) of the body. The analysis further incorporated consistent and accurate assessments of the intensity, volume, and daily distribution of physical activity, plus the physical activity volume for sedentary and stationary behaviours, along with its daily distribution.³⁴⁻³⁶

The MAD metric has been validated through directly measured incident oxygen uptake (VO_2) during walking or running on an indoor track.³⁶ This strong association made it possible to convert MAD values to incident energy consumption (MET). The MET was smoothed by calculating the one-minute exponential moving average of MAD values for each 6s epoch. Following recommended use,³⁷ cut-points for different activities were set as 3–6 MET for moderate activities, and over 6 MET for vigorous activities, with corresponding mean daily total times also determined. The mean times spent in moderate and vigorous activity per day were summed to obtain the total mean time spent in moderate-to-vigorous physical activity (MVPA) per day. The average daily step count during the monitoring week was also documented.

Statistical analyses

All the continuous variables were tested for normality prior to statistical analysis. Continuous variables are presented as means and standard deviations, and categorical variables as percentages and counts. The differences between groups were investigated using the two independent samples t-test for distributed data or Mann-Whitney U-test for non-distributed data. In addition, crosstabs, chi-square, and Fisher's exact test were used when appropriate for categorical data. Longitudinal analyses were performed using McNemar's test when analysing the differences in proportions of participants with MD at both time points and a binary logistic regression model when investigating the associations between adolescent's BMI, MD, attitudes towards body weight, physical activity, and age at menarche and MD in young adulthood.

Statistical analyses were conducted using IBM SPSS Statistics version 24 (Armonk, NY). The significance level was set at <0.05 , two-tailed.

Results

Based on the questionnaire, menstrual status could be determined in 149 (83.7%) adolescent athletes and 97 (92.4%) adolescent non-athletes. Out of those, 8 (5.4%) athletes and 17 (17.5%) non-athletes used hormonal contraceptives. Only participants whose menstrual status could be determined and who did not use hormonal contraceptives were included in the analysis regarding MD (Figure 1). However, all the participants were included in the analysis of other variables such as age at menarche, gynaecological age, and attitudes towards body weight.

In adolescence, the number of athletes in each sports categories were as follows: endurance $n=53$ (29.8% of the athletes), aesthetic $n=62$ (34.8%), technical $n=3$ (1.7%), ball games $n=42$ (23.6%), and power/anti-gravitation $n=18$ (10.1%).

Menstrual status in the young adult population was available for 50 (96.2%) athletes and 130 (81.8%) non-athletes. Out of those, 19 (38.0%) athletes and 59 (45.4%) non-athletes used hormonal contraceptives. Participants who used hormonal contraceptives or whose menstrual status could not be determined from the questionnaires were excluded from the analysis regarding MD.

In young adulthood, athletes were distributed to the sports categories as follows: endurance $n=22$ (42.3% of the athletes), aesthetic $n=12$ (23.1%), ball games $n=10$ (19.2%), and power/anti-gravitation $n=8$ (15.4%). There were no athletes in the technical sports group in young adulthood.

Athletes and non-athletes

Table 1 presents the characteristics of the athletes and non-athletes at adolescence and in young adulthood. At adolescence, the groups were similar in chronological age, but athletes were slightly younger in gynaecological age than non-athletes, since menarche had occurred later in athletes. Athletes also exhibited lower BMI, more MVPA, and a higher daily step count than non-athletes. There was no difference in the prevalence of MD between athletes and non-athletes at adolescence. However, primary amenorrhea (both current and lifetime) was more common among athletes than non-athletes. Non-athletes were less satisfied with their weight and had more desire

than athletes to lose weight. By contrast, athletes wished to gain weight more often than non-athletes.

In young adulthood, athletes and non-athletes were similar in both chronological and gynaecological age, and there were no differences between groups in terms of age at menarche, weight, height, or BMI. Athletes had more MVPA and daily steps than non-athletes. The prevalence of MD differed between groups, since 39% of the athletes reported MD, as opposed to 6% of the non-athletes. The groups were similar in body weight satisfaction, but there was a trend towards more satisfaction among athletes ($p=0.07$). Furthermore, non-athletes were more willing to lose weight than athletes.

Figure 2 presents attitudes towards body weight by different weight groups (underweight, normal weight, or overweight) among athletes and non-athletes in adolescence. Among the athletes, higher weight satisfaction was associated with lower BMI ($p=0.01$), while a higher desire to lose weight was associated with higher BMI ($p=0.02$). There was no association between a desire to gain weight and BMI among the athletes ($p=0.28$). Among non-athletes, lower weight satisfaction was associated with higher BMI ($p<0.001$), and a higher desire to lose weight was associated with higher BMI ($p<0.001$). Among non-athletes in adolescence, there was no difference between weight groups in terms of a desire to gain weight ($p=0.20$). Normal weight athletes were more satisfied with their weight ($p=0.006$) than normal weight non-athletes. They also wished to lose weight less often ($p=0.01$) and wanted to gain weight more often ($p=0.04$) than normal weight non-athletes. Similarly, overweight athletes were more satisfied with their weight ($p=0.004$) than overweight non-athletes. They also showed less desire to lose weight ($p=0.004$). There was no difference in the desire to gain weight between overweight athletes and non-athletes. The attitudes of underweight athletes did not differ from those of underweight non-athletes.

Among young adult athletes, there were no differences in attitudes towards weight between the three weight groups (Figure 3). Among non-athletes, higher weight satisfaction was associated with lower BMI ($p<0.001$), a higher desire to lose weight was associated with higher BMI ($p<0.001$), and a lower desire to gain weight was associated with higher BMI ($p<0.001$). Overweight athletes were more satisfied with their weight ($p=0.005$) and had less desire to lose weight ($p=0.001$) than overweight non-athletes. Athletes did not differ from non-athletes in the normal and underweight groups.

Athletes reporting MD and eumenorrhic athletes

Table 2 presents the characteristics of athletes reporting MD and eumenorrhic athletes. At adolescence, the gynaecological age of athletes with MD was lower than that of the eumenorrhic athletes. In addition, athletes with MD had a higher age at menarche and lower BMI than eumenorrhic athletes. The groups showed similar amounts of measured MVPA and steps per day; however, eumenorrhic athletes reported a higher training volume during the training season than athletes in the MD group.

In young adulthood, the only differences between athletes with MD and eumenorrhic athletes were seen in objectively measured MVPA and in step counts. Athletes with MD had on average 40 minutes more MVPA and almost 5000 more steps than the eumenorrhic athletes (Table 2). There were no significant differences in attitudes towards body weight between the MD and the eumenorrhea groups, at either adolescence or young adulthood.

There were no differences in MD prevalence between the different sports categories either in adolescence or in young adulthood.

Longitudinal analysis

In the longitudinal analysis, MD in adolescence predicted MD in young adulthood among all participants (OR 11.33, 95% CI 3.26–39.34, $p < 0.001$), and among those who had been athletes in adolescence (OR 18.00, 95% CI 3.69–87.70, $p = 0.001$). BMI, attitudes towards body weight, physical activity, or age at menarche reported in adolescence did not predict MD in young adulthood. McNemar's test showed that the MD proportions did not differ between adolescence and young adulthood, either when an analysis was conducted of all the participants ($p = 0.61$), or purely of those who had been athletes in adolescence ($p = 1.00$).

Discussion

A higher percentage of adolescent athletes than non-athletes reported primary amenorrhea. We also found that adolescent female athletes had a higher mean age at menarche than non-athletes of

similar age. In young adulthood, MD was more common in athletes than in non-athletes, and the prevalence of MD in athletes was relatively high (38.7%). In adolescence, athletes reporting MD had a lower BMI and a higher age at menarche than eumenorrheic athletes. To our knowledge, this is the first study to compare physical activity levels assessed by accelerometers in eumenorrheic athletes and athletes with MD. We found that young adult athletes with MD performed more MVPA and steps per day than eumenorrheic athletes, despite the fact that there was no difference in self-reported training volume. Body weight satisfaction was more common in adolescent athletes, and young adult athletes showed a trend towards higher body satisfaction than non-athletes. The only factor predicting MD in young adulthood was MD in adolescence, while there was no relationship between BMI, attitudes towards body weight, physical activity, or age at menarche in adolescence and MD in young adulthood.

Prevalence of menstrual dysfunction

Studies that have used the same criteria for MD as the current study are mainly consistent with our findings showing that MD in adolescent athletes ranges between 18% and 28%.^{5,9-11,13} However, in our study, the prevalence of current primary amenorrhea among adolescent athletes was 4.7%, which is higher than in several other studies conducted in athletes of a similar age.^{5,7,11,13} The characteristics and definition of an ‘athlete’ may contribute to the differences between the studies.

The prevalence of MD in young adult athletes in the present study was 38.7%, which is lower than has been observed among elite endurance athletes.³⁸ However, in a large study conducted among Norwegian elite athletes,⁴ the prevalence of MD was lower than in the present study. The difference applied to all the Norwegian subjects (with an MD prevalence of 16.5%), and also to the lean sports athletes (where the MD prevalence in endurance and aesthetic athletes was 30.5% and 30.0%, respectively) regardless of the fact that lean sports athletes tend to have higher rates of MD than athletes in other disciplines.^{2,4,5} In addition, in a study conducted among university athletes in all types of sports, the prevalence of MD was lower (26.8%) than in the present study.¹⁴

In a previous Finnish study,⁸ the prevalence of MD among national-level athletes was 27–37% depending on the type of sports, which is in accordance with the present study. However, among high-level Finnish endurance athletes, the reported prevalence of amenorrhea was higher than that observed in the present study.³⁹

Studies comparing the prevalence of MD in athletes and non-athletes have reported conflicting findings. Studies conducted among adolescent elite runners⁶ and varsity athletes from high school¹² found that athletes had a higher prevalence of MD than controls, which is in accordance with our findings among participants in young adulthood. By contrast, studies conducted among young elite athletes in a range of sports^{4,7} did not find any difference in MD prevalence between athletes and controls. This is consistent with our results among participants in adolescence.

In the present study, the mean age at menarche assessed in adolescence was 12.7 years in athletes, which is consistent with other studies.^{5,11,13} In addition, our finding that athletes' age at menarche was higher than that of non-athletes is in agreement with the studies conducted by Torstveit and Sundgot-Borgen⁴ and by Hoch et al.¹²

Attitudes towards body weight

We found that non-athletes had more desire to lose weight than athletes, both in adolescence and in young adulthood. The finding is consistent with the study conducted by Fogelholm and Hiilloskorpi.⁸ Our figure for body weight satisfaction among non-athletes at adolescence is about the same as has been previously reported among Finnish adolescents.²² Our finding that body weight dissatisfaction was more common among adolescent non-athletes than among athletes of the same age is consistent with a meta-analysis conducted by Hausenblas and Downs,⁴⁰ which found that athletes reported a more positive body image than their non-athletic peers. Physical activity may help athletes to maintain a healthier body composition, in line with indications that physically active persons have fewer problems related to eating control than their less active counterparts.⁴¹ It should also be noted that in our study, adolescent athletes had a lower BMI than non-athletes, and this could be one reason for the fact that athletes were more satisfied with their weight than non-athletes.

When we took weight status into account, we observed that higher weight satisfaction was associated with lower BMI in all groups except young adult athletes. The finding of an association between higher weight satisfaction and lower BMI is in accordance with other studies.^{21,24} In adolescent and young adult non-athletes, and also in adolescent athletes, an increased desire to lose weight was associated with higher BMI – a finding consistent with the results of Neighbors and Sobal.²¹ As regards our measurements of athletes in young adulthood, we may have lacked the

statistical power to detect possible effects operating between BMI, weight satisfaction, and a desire to lose or gain weight, due to the small numbers of underweight and overweight athletes.

In the current study, athletes tended to exhibit a smaller tendency to lose weight and to be more satisfied with their weight than non-athletes in the same weight group. We did not measure body composition, but it can be speculated that athletes had more lean mass than non-athletes, and that this could affect body satisfaction and the desire to lose weight.

Some normal weight participants showed a desire to lose weight, both in adolescence and in young adulthood. In fact, it has been demonstrated that many underweight and normal weight females misclassify themselves as overweight, and that some weight loss efforts among women result from body image problems, rather than any actual need to lose weight.⁴² This might have been the case among some of our participants also. It is known that some elite female athletes try to lose weight in order to improve their performance, or to meet the 'ideal' body weight and composition of an athlete.²³ In the present study, at both time-points, about one fifth of the athletes and about 40% of the non-athletes experienced body weight dissatisfaction. Since it is known that body weight dissatisfaction is a risk factor for disordered eating,²⁰ it would be important to pay attention to young people who are dissatisfied with their weight.

Athletes reporting MD and eumenorrheic athletes

Some, but not all studies have shown that amenorrheic athletes have lower BMI than their eumenorrheic peers.^{27,28,43} This was the case also in the current study, where the adolescent athletes reporting MD had lower BMI than the eumenorrheic athletes. Nevertheless, the average BMI in our MD group remained within the normal limits. Furthermore, in young adulthood, no difference in BMI between the athletes with MD and eumenorrheic athletes was seen. Low BMI does not in itself seem to cause MD; however, it may be an indication of low EA, bearing in mind that low EA has been shown to be the reason for MD in the Female Athlete Triad/RED-S, as opposed to low BMI or low body fat.⁴⁴

To our knowledge, this is the first study to compare objectively measured physical activity levels in eumenorrheic athletes and athletes with MD. Torstveit and Sundgot-Borgen⁴ found that self-reported training volume was not associated with MD in elite athletes in multiple sports

disciplines, which is in accordance with the results of the present study regarding adolescence. In addition, in the study conducted by Tornberg et al.,²⁶ there was no difference in aerobic training volume between amenorrheic and eumenorrheic athletes even though amenorrheic athletes performed more resistance training. However, Henriksson et al.²⁵ found that endurance runners with MD had more training hours per week than eumenorrheic runners. In the present study, young adult athletes with MD had more MVPA than eumenorrheic athletes and their step count was greater – this despite the fact that there was no difference between the groups in self-reported training hours. Previous studies have indicated that training volume *per se* does not cause MD, but that vigorous and regular exercise increases energy expenditure. Furthermore, increased energy expenditure could lead to low EA, and consequently to MD.⁴⁴ It is possible that this was also the case among our MD participants. We did not ask the participants about their attitudes towards eating, so we cannot know if the possible low EA resulted from DE or ED. Future studies should investigate whether the association between MD and objectively measured physical activity can be replicated with larger samples.

We did not find a difference in MD prevalence between different sports categories, which is contrary to some^{4,5} but not all⁹ previous findings. However, the statistical power of this analysis was low due to the small number of participants in different sports groups.

Longitudinal analysis

Relatively few studies have investigated the factors predicting MD in athletes. To et al.⁴⁵ conducted a prospective study in Chinese collegiate dance students and found that a decline in BMI or body fat did not explain MD in a year-long follow-up. Wiksten-Almströmer et al.⁴⁶ studied women diagnosed with MD in adolescence and found that MD was still present in 62% of subjects not using hormonal contraceptives after six years of follow-up. The only significant factor predicting resumption of regular menses was previous eating disorder (anorexia nervosa). Those who were anorexic in adolescence had the highest rate of resumption of regular menses. In our study, the only variable predicting MD in young adulthood was MD in adolescence. Further research is needed to confirm these results. However, it is important to pay attention to adolescents with MD to prevent future problems in health and/or performance.

Limitations

There are some limitations in the current study. We investigated MD on the basis of self-reported data, without additional laboratory measurements. The possible other causes for MD, such as polycystic ovary syndrome (PCOS), pregnancy, or thyroid disorders were not recorded in a structured way in our study. In addition, the range of menstrual cycles in adolescence is wider than in adulthood,³¹ complicating interpretation of the data regarding MD in adolescence. Participants were asked if they had had MD only within the preceding 12 months; hence there is a possibility that the participants had experienced secondary amenorrhea or oligomenorrhea at a previous time in their life. Furthermore, we did not measure body composition or ask about weight fluctuation. It was thus not possible to determine whether there was an association between body fat percentage and weight satisfaction, regardless of BMI. Moreover, we did not obtain data on whether a participant had recently lost or gained weight. Nor did we ask about eating disorders or assess energy intake. Finally, there is a possibility of recall bias in the reports at each time point.

Perspective

While previous studies have shown that MD is a common problem in elite and high school athletes in many countries,^{4,14,38} the findings of the present study indicate that young Finnish athletes, too, exhibit fairly high levels of MD. Our results also suggest that young female athletes may be more satisfied with their weight than their non-athletic peers. This is consistent with an earlier meta-analysis,⁴⁰ which showed that athletes had a more positive body image than non-athletes. The present study also found that young adult athletes reporting MD performed more physical activity than eumenorrheic athletes – a finding contrary to most of the previous studies investigating training volumes or exercise energy expenditure.^{4,26-28} Furthermore, it has been shown that efforts to lose weight are not necessarily related to any actual need for weight loss,⁴² as body image problems are common among young females.^{21,22} In the current study, weight dissatisfaction was seen among both athletes and non-athletes. Thus, irrespective of one's status as an athlete or non-athlete, it would be important to support young people who are dissatisfied with their weight, in order to identify or forestall disordered eating, which can result from body weight dissatisfaction.¹⁷ Finally, we found that the only factor predicting MD in young adulthood was MD in adolescence. This finding highlights the importance of identifying athletes with MD as early as possible. Increasing knowledge on the high prevalence of MD as well as body weight

dissatisfaction seen in young females is important to prevent future health problems related to MD and disordered eating and to decrease the stigma surrounding these issues.

References

1. Nattiv A, Locks A, Manore M, Sanborn C, Sundgot-Borgen J, Warren M. The female athlete triad. *Medicine & Science in Sports & Exercise*. 2007;39(10):1867-1882.
2. De Souza MJ, Nattiv A, Joy E, et al. 2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad: 1st International Conference held in San Francisco, California, May 2012 and 2nd International Conference held in Indianapolis, Indiana, May 2013. *Br J Sports Med*. 2014;48(4):289.
3. Mountjoy M, Sundgot-Borgen J, Burke L, et al. The IOC consensus statement: Beyond the female athlete triad—Relative energy deficiency in sport (RED-S). *Br J Sports Med*. 2014;48(7):491-497.
4. Torstveit MK, Sundgot-Borgen J. Participation in leanness sports but not training volume is associated with menstrual dysfunction: A national survey of 1276 elite athletes and controls. *Br J Sports Med*. 2005;39(3):141-147.
5. Nichols JF, Rauh MJ, Barrack MT, Barkai H, Pernick Y. Disordered eating and menstrual irregularity in high school athletes in lean-build and nonlean-build sports. *Int J Sport Nutr Exerc Metab*. 2007;17(4):364-377.
6. Muia EN, Wright HH, Onywera VO, Kuria EN. Adolescent elite Kenyan runners are at risk for energy deficiency, menstrual dysfunction and disordered eating. *J Sports Sci*. 2016;34(7):598-606.
7. Martinsen M, Bratland-Sanda S, Eriksson AK, Sundgot-Borgen J. Dieting to win or to be thin? A study of dieting and disordered eating among adolescent elite athletes and non-athlete controls. *Br J Sports Med*. 2010;44(1):70-76.
8. Fogelholm M, Hiilloskorpi H. Weight and diet concerns in Finnish female and male athletes. *Med Sci Sports Exerc*. 1999;31(2):229-235.

9. Thein-Nissenbaum JM, Rauh MJ, Carr KE, Loud KJ, McGuine TA. Associations between disordered eating, menstrual dysfunction, and musculoskeletal injury among high school athletes. *Journal of orthopaedic & sports physical therapy*. 2011;41(2):60-69.
10. Rauh MJ, Barrack M, Nichols JF. Associations between the female athlete triad and injury among high school runners. *International journal of sports physical therapy*. 2014;9(7):948.
11. Rauh MJ, Tenforde AS, Barrack MT, Rosenthal MD, Nichols JF. Associations between sport specialization, running-related injury, and menstrual dysfunction among high school distance runners. *Athletic Training and Sports Health Care*. 2018;10(6):260-269.
12. Hoch AZ, Pajewski NM, Moraski L, et al. Prevalence of the female athlete triad in high school athletes and sedentary students. *Clinical journal of sport medicine: official journal of the Canadian Academy of Sport Medicine*. 2009;19(5):421.
13. Thein-Nissenbaum JM, Rauh MJ, Carr KE, Loud KJ, McGuine TA. Menstrual irregularity and musculoskeletal injury in female high school athletes. *Journal of athletic training*. 2012;47(1):74-82.
14. Tenforde AS, Carlson JL, Chang A, et al. Association of the female athlete triad risk assessment stratification to the development of bone stress injuries in collegiate athletes. *Am J Sports Med*. 2017;45(2):302-310.
15. Loucks A, Thuma J. Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. *The Journal of Clinical Endocrinology & Metabolism*. 2003;88(1):297-311.
16. Williams NI, Statuta SM, Austin A. Female athlete triad: Future directions for energy availability and eating disorder research and practice. *Clin Sports Med*. 2017;36(4):671-686.
17. Shi J, Zhang B, Choi J, et al. Age at menarche and age at natural menopause in East Asian women: A genome-wide association study. *Age*. 2016;38(5-6):513-523.
18. Loucks AB, Kiens B, Wright HH. Energy availability in athletes. *J Sports Sci*. 2011;29(sup1):S7-S15.

19. Mountjoy M, Sundgot-Borgen JK, Burke LM, et al. IOC consensus statement on relative energy deficiency in sport (RED-S): 2018 update. *Br J Sports Med*. 2018;bjsports-099193.
20. Stice E, Shaw HE. Role of body dissatisfaction in the onset and maintenance of eating pathology: A synthesis of research findings. *J Psychosom Res*. 2002;53(5):985-993.
21. Neighbors LA, Sobal J. Prevalence and magnitude of body weight and shape dissatisfaction among university students. *Eating Behav*. 2007;8(4):429-439.
22. Mikkilä V, Lahti-Koski M, Pietinen P, Virtanen SM, Rimpelä M. Associates of obesity and weight dissatisfaction among Finnish adolescents. *Public Health Nutr*. 2003;6(1):49-56.
23. Sundgot-Borgen J. Risk and trigger factors for the development of eating disorders in female elite athletes. *Medicine & Science in Sports & Exercise*. 1994;26(4):414-419.
24. Coelho EM, Fonseca SC, Pinto GS, Mourão-Carvalho MI. Factors associated with body image dissatisfaction in Portuguese adolescents: Obesity, sports activity and TV watching. *Motricidade*. 2016;12(2):18-26.
25. Henriksson GB, Schnell C, Hirschberg AL. Women endurance runners with menstrual dysfunction have prolonged interruption of training due to injury. *Gynecol Obstet Invest*. 2000;49(1):41-46.
26. Tornberg ÅB, Melin A, Koivula FM, et al. Reduced neuromuscular performance in amenorrhoeic elite endurance athletes. *Medicine & Science in Sports & Exercise*. 2017;49(12):2478-2485.
27. Zanker CL, Swaine IL. Bone turnover in amenorrhoeic and eumenorrhoeic women distance runners. *Scand J Med Sci Sports*. 1998;8(1):20-26.
28. Clark LR, Dellogono MJ, Mangano KM, Wilson TA. Clinical menstrual dysfunction is associated with low energy availability but not dyslipidemia in division I female endurance runners. *Journal of Exercise Physiology Online*. 2018;21(2):265-277.

29. Kokko S, Selänne H, Alanko L, et al. Health promotion activities of sports clubs and coaches, and health and health behaviours in youth participating in sports clubs: The health promoting sports club study. *BMJ open sport & exercise medicine*. 2015;1(1):e000034.
30. Sundgot-Borgen J, Torstveit MK. Prevalence of eating disorders in elite athletes is higher than in the general population. *Clinical Journal of Sport Medicine*. 2004;14(1):25-32.
31. American Academy of Pediatrics, American College of Obstetricians and Gynecologists. Menstruation in girls and adolescents: Using the menstrual cycle as a vital sign. *Pediatrics*. 2006;118(5):2245-2250.
32. Cole TJ, Flegal KM, Nicholls D, Jackson AA. Body mass index cut offs to define thinness in children and adolescents: International survey. *BMJ*. 2007;335(7612):194.
33. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: International survey. *BMJ*. 2000;320(7244):1240.
34. Vähä-Ypyä H, Vasankari T, Husu P, Suni J, Sievänen H. A universal, accurate intensity-based classification of different physical activities using raw data of accelerometer. *Clinical physiology and functional imaging*. 2015;35(1):64-70.
35. Vähä-Ypyä H, Husu P, Suni J, Vasankari T, Sievänen H. Reliable recognition of lying, sitting, and standing with a hip-worn accelerometer. *Scand J Med Sci Sports*. 2018;28(3):1092-1102.
36. Vähä-Ypyä H, Vasankari T, Husu P, et al. Validation of cut-points for evaluating the intensity of physical activity with accelerometry-based mean amplitude deviation (MAD). *PloS one*. 2015;10(8):e0134813.
37. Sievänen H, Kujala UM. Accelerometry—Simple, but challenging. *Scand J Med Sci Sports*. 2017;27(6):574-578.
38. Melin A, Tornberg ÅB, Skouby S, et al. Energy availability and the female athlete triad in elite endurance athletes. *Scand J Med Sci Sports*. 2015;25(5):610-622.

39. Heikura IA, Uusitalo AL, Stellingwerff T, Bergland D, Mero AA, Burke LM. Low energy availability is difficult to assess but outcomes have large impact on bone injury rates in elite distance athletes. *Int J Sport Nutr Exerc Metab.* 2018;28(4):403-411.
40. Hausenblas HA, Downs DS. Comparison of body image between athletes and nonathletes: A meta-analytic review. *Journal of applied sport psychology.* 2001;13(3):323-339.
41. Rintala M, Lyytikäinen A, Leskinen T, et al. Leisure-time physical activity and nutrition: A twin study. *Public Health Nutr.* 2011;14(5):846-852.
42. Lowry R, Galuska DA, Fulton JE, Wechsler H, Kann L, Collins JL. Physical activity, food choice, and weight management goals and practices among US college students. *Am J Prev Med.* 2000;18(1):18-27.
43. McCall LM, Ackerman KE. Endocrine and metabolic repercussions of relative energy deficiency in sport (RED-S). *Current Opinion in Endocrine and Metabolic Research.* 2019.
44. Loucks AB. Energy availability, not body fatness, regulates reproductive function in women. *Exerc Sport Sci Rev.* 2003;31(3):144-148.
45. To WW, Wong MW, Lam IY. Hormonal predisposition to menstrual dysfunction in collegiate dance students. *Acta Obstet Gynecol Scand.* 2000;79(12):1117-1123.
46. Wiksten-Almströmer M, Hirschberg AL, Hagenfeldt K. Prospective follow-up of menstrual disorders in adolescence and prognostic factors. *Acta Obstet Gynecol Scand.* 2008;87(11):1162-1168.

Table 1. Characteristics of the cohort of the athletes and non-athletes in adolescence and in young adulthood.

	Adolescence (14–16 years)					Young adulthood (18–20 years)				
	n	Athletes	n	Non-athletes	p-value	n	Athletes	n	Non-athletes	p-value
Chronological age, y	178	14.9 (0.6)	105	15.0 (0.4)	0.43	52	18.8 (0.7)	159	18.9 (0.7)	0.71
Gynaecological age, y	165	2.4 (1.2)	104	2.8 (1.1)	0.002	50	5.9 (1.6)	159	6.3 (1.4)	0.06
Age at menarche, y	165	12.7 (1.1)	104	12.2 (1.1)	0.001	50	12.9 (1.5)	159	12.5 (1.3)	0.16
Height, cm	178	166.2 (6.0)	105	165.7 (6.4)	0.45	52	168.7 (6.9)	158	167.3 (6.4)	0.19
Weight, kg	178	57.9 (7.6)	105	59.9 (9.7)	0.08	52	63.1 (8.0)	158	65.5 (11.4)	0.13
BMI, kg/m ²	178	20.9 (2.2)	105	21.8(3.1)	0.01	52	22.2 (2.4)	158	23.4 (3.9)	0.06
MVPA per day, h:mm	164	1:19 (0:28)	87	0:59 (0:23)	<0.001	39	1:21 (0:38)	130	0:54 (0:30)	<0.001
Daily step count	164	9310 (2816)	87	7674 (2535)	<0.001	39	10117 (4273)	130	7236 (3179)	<0.001
Current MD	141	17.7% (25)	80	17.5% (14)	0.97	31	38.7% (12)	71	5.6% (4)	<0.001
Current/previous primary amenorrhea	172	8.1% (14)	102	0.0% (0)	0.003	50	12.0% (6)	159	7.5% (12)	0.39
Current primary amenorrhea	172	4.7% (8)	102	0.0% (0)	0.03	50	0.0% (0)	159	0.0% (0)	-
Menarche occurred	178	92.7% (165)	104	100.0% (104)	0.003	52	100.0% (52)	159	100.0% (159)	-
Hormonal contraceptive users	178	5.1% (9)	105	17.1% (18)	0.001	51	40.4% (21)	157	49.7% (79)	0.24
Satisfied with weight	174	81.6% (142)	104	55.8% (58)	<0.001	51	76.5% (39)	157	62.4% (98)	0.07
Desire to lose weight	171	19.3% (33)	103	43.7% (45)	<0.001	51	29.4% (15)	155	46.5% (72)	0.03
Desire to gain weight	172	14.0% (24)	100	4.0% (4)	0.009	48	2.1% (1)	148	5.3% (8)	0.69

Sports category	178	-	-	52	-	-
<i>Endurance</i>	29.8% (53)	-	-	42.3% (22)	-	-
<i>Aesthetic</i>	33.7% (60)	-	-	23.1% (12)	-	-
<i>Technical</i>	2.8% (5)	-	-	0.0% (0)	-	-
<i>Ball games</i>	23.6% (42)	-	-	19.2% (10)	-	-
<i>Power/anti-gravitation</i>	10.1% (18)	-	-	15.4% (8)	-	-

The data are presented as means (standard deviations) or percentages (counts).

Table 2. Characteristics of the athlete cohort divided into menstrual dysfunction (MD) and eumenorrhea groups, in adolescence and in young adulthood.

	Adolescence (14–16 years)					Young adulthood (18–20 years)				
	n	MD [†]	n	Eumenorrhea	p-value	n	MD	n	Eumenorrhea	p-value
Chronological age, y	31	15.2 (0.5)	116	15.0 (0.6)	0.11	12	18.5 (0.5)	19	18.8 (0.8)	0.22
Gynaecological age, y	23	2.0 (1.2)	116	2.6 (1.0)	0.04	12	5.5 (1.6)	18	6.1 (1.0)	0.21
Age at menarche, y	23	13.3 (1.4)	116	12.5 (1.0)	0.008	12	13.0 (1.5)	18	12.7 (0.9)	0.61
Height, cm	31	167.8 (5.9)	116	166.2 (6.1)	0.19	12	169.8 (9.6)	19	169.0 (6.8)	0.71
Weight, kg	31	56.8 (7.4)	116	59.4 (7.5)	0.08	12	65.1 (10.2)	19	64.5 (8.3)	0.64

BMI, kg/m ²	31	20.1 (2.0)	116	21.5 (2.2)	0.002	12	22.6 (3.3)	19	22.6 (2.5)	0.99
MVPA per day, h:mm	26	1:17 (0:30)	107	1:18 (0:26)	0.91	9	1:37 (0:22)	14	0:57 (0:26)	0.001
Daily step count	26	8869 (2825)	107	9168 (2713)	0.62	9	12172 (2980)	14	7326 (2287)	<0.001
Self-reported weekly training hours in the training season	27	8.9 (3.5)	113	10.5 (4.3)	0.04	12	12.7 (4.6)	19	10.9 (4.3)	0.29
Self-reported weekly training hours in the competition season	27	9.0 (3.3)	113	10.6 (5.1)	0.10	12	11.3 (4.6)	19	10.1 (4.4)	0.82
Self-reported weekly rest days in the training season	27	1.9 (0.8)	112	1.8 (0.9)	0.53	12	1.5 (0.7)	19	1.5 (0.9)	0.82
Self-reported weekly rest days the in the competition season	27	1.9 (0.8)	112	1.6 (0.8)	0.06	12	1.5 (0.7)	19	1.6 (0.7)	0.89
Satisfied with weight	30	80.0% (24)	112	81.3% (91)	1.00	12	75.0% (9)	19	78.9% (15)	1.00
Desire to lose weight	30	13.3% (4)	109	20.2% (22)	0.34	12	33.3% (4)	19	26.3% (5)	0.70
Desire to gain weight	30	10.0% (3)	112	13.4% (15)	0.76	11	0.0% (0)	18	0.0% (0)	-
Sports category	31		116		0.674	12		19		0.300
<i>Endurance</i>		25.0% (10)		75.0% (30)			30.8% (4)		69.2% (9)	
<i>Aesthetic</i>		16.7% (9)		83.3% (45)			28.6% (2)		71.4% (5)	
<i>Technical</i>		0.0% (0)		100.0% (3)			0.0% (0)		0.0% (0)	

<i>Ball games</i>	27.0% (10)	73.0% (27)	71.4% (5)	28.6% (2)
<i>Power/anti-gravitation</i>	15.4% (2)	84.6% (11)	25.0% (1)	75.0% (3)

The data are presented as means (standard deviations) or percentages (counts). † Current MD ($n=25$) or history of primary amenorrhea within the preceding year ($n=6$).

Figure legends:

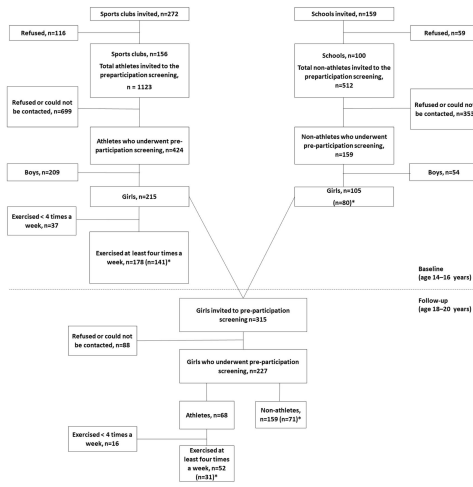
Figure 1. Data collection and final study groups.

* Number of girls who did not use hormonal contraceptives and who answered the questions regarding menstruation in such a way that it was possible to determine their menstrual status. Only these girls were included in the MD analysis.

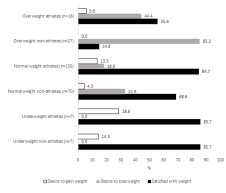
Five of the girls participating in the study in adolescence were not invited to the follow-up, as they did not want to be re-contacted.

Figure 2. Attitudes towards body weight by weight groups among athletes and non-athletes in adolescence (14–16 years of age). Bars indicate the percentages of the participants who had a desire to gain or lose weight, or who were satisfied with their weight. The sum of the percentages may deviate from 100, as the participants responded to each item separately. Some reported satisfaction with their weight, but still indicated a desire to lose or gain weight.

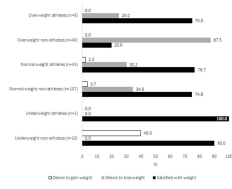
Figure 3. Attitudes towards body weight by weight groups among athletes and non-athletes in young adulthood (18–20 years of age). Bars indicate the percentages of the participants who had a desire to gain or lose weight, or who were satisfied with their weight. The sum of percentages may deviate from 100, as the participants responded to each item separately. Some reported satisfaction with their weight, but still indicated a desire to lose or gain weight.



sms_13838_f1.tif



sms_13838_f2.tif



sms_13838_f3.tif



II

ADOLESCENT SPORT PARTICIPATION AND AGE AT MENARCHE IN RELATION TO MIDLIFE BODY COMPOSITION, BONE MINERAL DENSITY, FITNESS, AND PHYSICAL ACTIVITY

by

Ravi, S., Kujala, U. M., Tammelin, T. H., Hirvensalo, M., Kovanen, V.,
Valtonen, M., Waller, B., Aukee, P., Sipilä, S., & Laakkonen, E. K. 2020

Journal of Clinical Medicine, 9(12), Article 3797

<https://doi.org/10.3390/jcm9123797>

Reproduced with kind permission by MDPI.



Article

Adolescent Sport Participation and Age at Menarche in Relation to Midlife Body Composition, Bone Mineral Density, Fitness, and Physical Activity

Suvi Ravi ^{1,*}, Urho M. Kujala ¹, Tuija H. Tammelin ², Mirja Hirvensalo ¹,
Vuokko Kovanen ³, Maarit Valtonen ⁴, Benjamin Waller ⁵, Pauliina Aukee ⁶, Sarianna Sipilä ³
and Eija K. Laakkonen ³

¹ Faculty of Sport and Health Sciences, University of Jyväskylä, 40014 Jyväskylä, Finland; urho.m.kujala@jyu.fi (U.M.K.); mirja.hirvensalo@jyu.fi (M.H.)

² LIKES Research Centre for Physical Activity and Health, 40700 Jyväskylä, Finland; tuija.tammelin@likes.fi

³ Gerontology Research Center and Faculty of Sport and Health Sciences, University of Jyväskylä, 40014 Jyväskylä, Finland; vukekovanen@gmail.com (V.K.); sarianna.sipila@jyu.fi (S.S.); eija.k.laakkonen@jyu.fi (E.K.L.)

⁴ Research Institute for Olympic Sports, 40700 Jyväskylä, Finland; maarit.valtonen@kihu.fi

⁵ Physical Activity, Physical Education, Sport and Health Research Centre, Sports Science Department, School of Social Sciences, Reykjavik University, 102 Reykjavik, Iceland; benw@ru.is

⁶ Department of Obstetrics and Gynecology, Pelvic Floor Research and Therapy Unit, Central Finland Central Hospital, 40620 Jyväskylä, Finland; pauliina.aukee@ksshp.fi

* Correspondence: suvi.m.ravi@jyu.fi; Tel.: +358-406707282

Received: 26 October 2020; Accepted: 21 November 2020; Published: 24 November 2020



Abstract: This study aimed to investigate the associations of competitive sport participation in adolescence and age at menarche (AAM) with body composition, femoral neck bone mineral density (BMD), physical performance, and physical activity (PA) in middle-aged women. 1098 women aged 47–55 years formed the sample of this retrospective study. Participants self-reported their PA level at age 13–16 years and AAM. The protocol also included dual-energy X-ray absorptiometry, physical performance tests, and accelerometer-measured PA. Participants were divided into three groups according to their PA level at the age of 13–16 (no exercise, regular PA, and competitive sport) and according to their AAM (≤ 12 , 13, and ≥ 14 years). After adjusting for potential confounding factors, participation in competitive sport at age 13–16 was associated with higher midlife lean mass and BMD, and better physical performance compared to groups with no exercise or regular PA. Individuals with AAM ≥ 14 years had lower midlife BMI and fat mass than participants in the other AAM groups and pre- and perimenopausal women with AAM ≥ 14 years had lower BMD than those with AAM ≤ 12 . The findings indicate that participation in competitive sport in adolescence is associated with healthier body composition, higher BMD, and better physical performance in midlife, but BMD might be impaired if menarche occurs late.

Keywords: adolescent athlete; female athlete; age at menarche; body composition; bone mineral density; physical performance; physical activity

1. Introduction

Physical activity (PA) provides many health benefits, especially during adolescence [1]. Adolescence is a critical time for skeletal growth and mineralization [2], which is enhanced with weight-bearing PA [3]. Studies with female participants have shown that sport participation in childhood/adolescence is associated with better bone health in early [4–6] and late adulthood [7–9],

and former athletes have more lean mass and less fat mass than their sedentary controls in midlife [7]. PA tracks from adolescence to adulthood among women, although only at low to moderate level [10–12] and organised sport participation in adolescence is associated with healthy lifestyle habits later in life [13]. Despite these positive effects of PA, some female athletes suffer from conditions such as low energy availability, eating disorders or disordered eating, menstrual dysfunction and low bone mineral density (BMD), which can have adverse long-term health effects [14,15]. Further, athletes may be exposed to injuries, and consequently to later life pain and/or disabilities [16–19], but data on the prevalence of musculoskeletal problems in former female athletes compared to the general population is limited. There is also a lack of knowledge regarding fractures in former female athletes compared to non-athletes [20].

In addition to sport participation in adolescence, age at menarche (AAM) has been shown to be associated with bone health. AAM is associated negatively with BMD [21–24] and positively with fracture risk later in life [25,26]. Although female athletes tend to attain menarche later than their non-athletic peers [27–29], investigating the association between AAM and bone health in adulthood rarely take into account adolescence PA. Moreover, while there is evidence that AAM is inversely associated with fat percentage and BMI in adulthood [30–32], it is not known if AAM is associated with lean mass and physical performance in midlife.

Since, to our knowledge, no previous study has investigated long-term consequences of both sport participation in adolescence and AAM on midlife characteristics, further information on these links would provide important information for understanding these relationships. Thus, with this retrospective descriptive study, we aimed to investigate the associations between competitive sport participation in adolescence (i.e., at age 13–16) and AAM with middle-aged body composition, BMD, physical performance, and PA in 47–55-year-old women. Furthermore, we investigated if the lifetime occurrence of fractures, current musculoskeletal problems, and the lifetime occurrence of anorexia nervosa differed until middle age according to adolescent physical activity status or AAM.

2. Materials and Methods

2.1. Participants

In the current study, data collected in the Estrogenic Regulation of Muscle Apoptosis (ERMA) study [33] were utilised. A written invitation was sent to 6878 women representing a random sample of 47–55-year-old women living in Jyväskylä or neighbouring municipalities in Central Finland. The response rate was 47%. From the eligible participants ($n = 1627$), 1393 gave blood samples and 1102 of them provided full questionnaire data. Due to technical error, four questionnaires were lost. Thus, the sample size of the present study was 1098. Of the whole sample, 988 provided information about their physical activity (PA) level at age 13–16 and 1081 responded to the question regarding age at menarche (AAM) (Figure 1).

In the ERMA study, the exclusion criteria were conditions and use of medications affecting ovarian function, obesity (self-reported BMI $> 35 \text{ kg/m}^2$), or conditions hindering daily physical or mental functioning.

The study was approved by the Ethics Committee of the Central Finland Health Care District (K-S shp Dnro 8U/2014) and all the participants provided written informed consent. The study protocol is described in detail in Kovanen et al. [33].

2.2. Assessment Methods

2.2.1. Adolescent Predictors

PA Level

Participants described their PA level at age 13–16 as one of four possible options: no exercise, regular independent leisure-time PA, regular other supervised PA in a sport club etc., and regular competitive

sport and training related to that sport (modified by Hirvensalo et al. [34]). Regular independent leisure-time PA was defined as regular PA during a journey to or from school/work (>2 km/one way) or as regular PA causing sweating that is not organised by a school, sports club, fitness centre, etc.

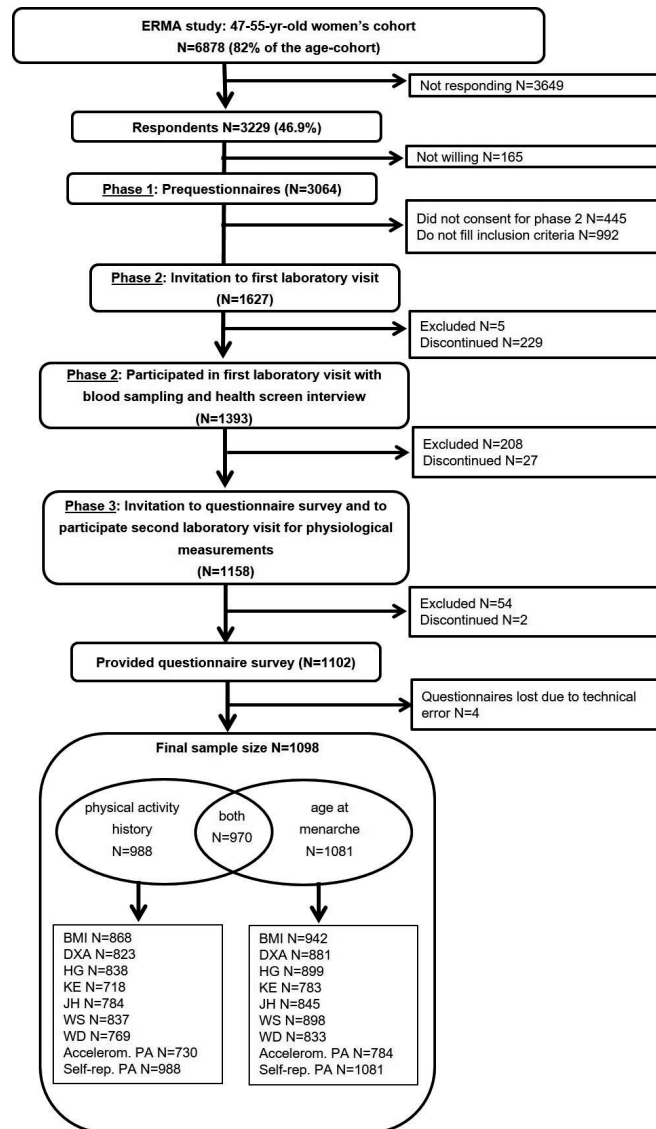


Figure 1. Flow chart of the recruitment of the participants. BMI = body mass index; DXA = Dual-energy X-ray absorptiometry; HG = hand grip force; KE = knee extension force; JH = jumping height; WS = walking speed; WD = walking distance; accelerom. PA = accelerometer-measured physical activity; self-rep. PA = self-reported physical activity. Some of the participants were excluded for some or all physical performance tests for the health reasons.

The definition for supervised PA was as follows: all regular non-competitive PA, organised by a sports club, fitness centre, Girl Scouts, etc. Competitive sport and related training was defined as regular, goal-oriented competitive sport within a sports club etc., and competing and training in that discipline.

Participants were classified into three groups based on their answers: no exercise group, regular PA group, which includes participants who reported performing independent or supervised or both but

did not report being competing in any sport, and competitive sport group. Participants were also asked what sports disciplines they were engaged in and they were instructed to underline the disciplines in which they competed. PA level at other age stages 7–12, 17–19, 20–29, 30–39, and 40–50 years was similarly assessed to show the tracking of PA.

AAM

Participants self-reported the age of their first menstruation (age at menarche; AAM) and were subsequently categorised in approximately equal groups as follows: ≤ 12 years, 13 years, and ≥ 14 years.

2.2.2. Middle Age Characteristics

Anthropometrics and Body Composition

All of the anthropometrics and body composition measurements were assessed between 7:00 and 10:00 a.m. after overnight fasting. Participants' weight was measured in their underwear to the nearest 0.01 kg with a beam scale. Height was measured to the nearest 1.0 cm with a stadiometer. BMI was calculated as weight in kilograms divided by height in meters squared. Femoral neck BMD and body composition were assessed by dual x-ray absorptiometry (DXA, LUNAR; GE Healthcare, Chicago, IL, USA) [35]. Fat percentage was calculated as total fat mass divided by total mass multiplied by 100. Fat and lean mass indexes were calculated as fat mass (kg) and lean mass (kg), respectively, divided by height squared (m^2). Appendicular lean mass index (ALMI) was calculated as arms lean mass (kg) plus legs lean mass (kg) divided by height squared (m^2).

Physical Performance Tests

Muscle performance measurements were performed as presented earlier in Bondarev et al. 2018 [36]. Briefly, hand grip force was assessed on the dominant arm in a sitting position with the elbow flexed at 90° angle using an adjustable dynamometer chair (Good Strength, Metitur, Jyväskylä, Finland). Participants were instructed to squeeze the handle as forcefully as possible and maintain the contraction for 2–3 s. Maximal isometric knee extension force was measured in a sitting position, knee fixed in 60 degrees of flexion, on a custom-made dynamometer chair (Good Strength; Metitur Oy, Palokka, Finland) from the side of the dominant hand. Participants were instructed to extend the knee towards full extension to produce maximal force. In both the abovementioned tests, the trunk was stabilized to exclude the compensation of other muscles. Lower body muscle power was assessed with a countermovement jump performed on a custom-made contact mat. Flight time (t) was recorded and vertical jumping height (cm) was calculated as $(g \times t^2) / 8 \times 100$, where g is the acceleration of gravity (9.81 m/s^2) [37]. In all of the three aforementioned muscle force and power tests, three to five maximal efforts were performed, and the highest value was recorded.

Walking speed was assessed via a 10-m walk in a laboratory corridor using photocells, with five meters allowed for acceleration prior to measurement [38]. Participants performed two trials with the fastest accepted as their final result. The 6-min walking test was performed on a 20-m indoor track and the participants were instructed to walk as many laps as possible in 6 min. This test was used to assess submaximal exercise tolerance and aerobic capacity [39]. The rest between the different physical performance tests and between repetitions were separated by an approximately one-minute pause. The same protocol was applied in every participant (i.e., the instructions for the second tests were given in the similar way) resulting in approximately similar time between the tests in each participant.

Accelerometer-Measured PA

Accelerometer-measured PA was assessed using GT3X+ and wGT3X+ accelerometers (ActiGraph, Pensacola, FL, USA) [40]. Participants were instructed to wear the accelerometer on their right hip during waking hours (excluding activities in water) for seven consecutive days. Each participant was personally individually instructed regarding the use of the accelerometer and was also provided with

a diary accompanied with the instructions to record their wake-up time, working hours, and periods when the accelerometer was removed for over 30 min. Raw acceleration data were collected at 60 Hz and consequently filtered and converted into 60 s epoch counts. Further data analysis was conducted with Excel-based customised program. Tri-axial vector magnitude cut-off -points of moderate and vigorous physical activity were >2690 to ≤ 6166 cpm and >6166 cpm, respectively [40,41]. The mean times spent in moderate and vigorous activity per day were summed to obtain the total mean time spent in moderate-to-vigorous physical activity (MVPA) per day. The mean daily step count was also documented.

Self-Reported PA

Self-reported PA was inquired using a series of modified structured questions [33,42]. Recently, this tool has been compared with accelerometer-based data in this same cohort and has found to demonstrate similar association between accelerometer-measured PA compared to other widely used self-reported PA questionnaires [43]. Participants were asked about their leisure physical activity, including monthly frequency, mean duration, and mean intensity of PA sessions; and PA during the journey to and from work. Volume of activity was calculated using an activity metabolic equivalent (MET) index by assigning a multiple of resting metabolic rate (MET score) to each activity and calculating the product of intensity \times duration \times frequency of activity [42].

The following MET values were used (work metabolic rate divided by resting metabolic rate): 4 (for exercise intensity corresponding to walking and for physical activity during the work journey), 6 (vigorous walking to jogging), 10 (jogging), and 13 (running) [44]. The activity MET index was expressed as the score of leisure MET hours per day and has been validated by Waller et al. [45].

Background Variables

Participants reported their level of education by choosing from eight answer options of primary school to doctoral level. Participants were classified into two groups based on their answers: those with bachelor level or higher education and those with education lower than bachelor level.

Participants also reported their number of live births, if they had ever used oral contraceptive pills and in what year they had started and finished using them. Participants also reported use of hormonal contraceptives (HC) during the preceding 10 years.

The menopausal status determination procedure followed the Stages of Reproductive Aging Workshop +10 guidelines [46]. Participants kept a menstrual diary for 6–12 months for menstrual cycle assessment. Serum samples to measure FSH concentrations were obtained during menstrual days 1–5 for all women who had detectable menstrual bleeding at the onset of study. For participants who had undergone hysterectomy or were using progesterone-containing contraceptives, the menopausal group assignment was based solely on the FSH level, but with stricter cut-off values than used for participants with natural menstrual bleeding [33].

Participants were also asked if they have had one or more fractures in their wrist or forearm, tibia, femur, or at some other location. Participants were also questioned if they had some musculoskeletal disorders or symptoms. Additionally, participants were asked to report if they had ever had a diagnosis of anorexia nervosa and in which year the diagnosis was given.

2.3. Statistical Analysis

All the continuous variables were tested for normality prior to statistical analysis. Continuous variables are presented as means and 95% confidence intervals, and categorical variables as frequencies and percentages. Differences between groups were investigated using one-way analysis of variance (ANOVA) with Bonferroni post hoc pairwise comparisons or Kruskal-Wallis rank sum test with subsequent pairwise comparisons for continuous variables, and Pearson's chi-square for categorical variables. Analysis of covariance (ANCOVA) was used to adjust for potential confounding factors (self-reported leisure-time PA in adolescence and in midlife, age, AAM, menopausal status, number of

parities, education, and HC use during the 10 preceding years). As we assumed that BMD in postmenopausal women regardless of AAM may be lower than in women within the other menopausal status groups, and therefore potentially skew results or cloud an association between AAM and BMD, we investigated the association between AAM and BMD also in pre-, early perimenopausal, and late perimenopausal women only. Statistical analyses were conducted using IBM SPSS Statistics version 24 (Armonk, NY, USA). The significance level was set at <0.05, two-tailed.

3. Results

3.1. PA Participation from Childhood to Midlife

The most common sport disciplines (during 1970s and 1980s) in the competitive sport group were track and field (28.7%), volleyball (20.6%), cross-country skiing (18.4%), running (16.9%), Finnish baseball (7.4%), and gymnastics (7.4%). Most of the participants who engaged in competitive sport at age 13–16 reported having competed in two or more sport disciplines ($n = 98, 72.1\%$).

For each age stage, PA level was compared to the individual’s PA level at age 13–16 years (i.e., adolescence). At every age stage except the latest (i.e., age 40–50 years), those who engaged in competitive sport at age 13–16 years formed the larger proportion of those in competitive sport compared to those that reported regular PA or no exercise at age 13–16 years (p for all <0.001). Conversely, those who engaged in competitive sport at age 13–16 years formed the smaller proportion of those with no exercise at all at other age stages except the latest compared to those that reported regular PA or no exercise at age 13–16 years (p for all <0.001). There was no difference in the PA level groups between ages 13–16 and 40–50 ($p = 0.272$) (Table 1). The proportion of the participants engaged in competitive sport was highest at age 13–16 and gradually decreased thenceforth. Furthermore, the proportion of those reporting regular PA increased at each age stage after the age of 13–16 (Figure S1). The correlation coefficients between PA level at age 13–16 and PA level at ages 7–12, 17–19, 20–29, 30–39, and 40–50 were 0.54, 0.60, 0.29, 0.19 ($p < 0.001$ for all), and -0.02 ($p = 0.463$), respectively.

Table 1. Proportion (n) of participants of different physical activity (PA) level at each age stages according to the PA level at the age of 13–16.

Age	Competitive Sport (CS) at Age 13–16 ($n = 136$)			Regular PA (RPA) at Age 13–16 ($n = 689$)			No Exercise (NE) at Age 13–16 ($n = 163$)		
	CS	RPA	NE	CS	RPA	NE	CS	RPA	NE
7–12	50.7% (69)	45.6% (62)	3.7% (5)	5.8% (40)	85.5% (589)	8.7% (60)	3.7% (6)	36.8% (60)	59.5% (97)
17–19	48.5% (66)	47.1% (64)	4.4% (6)	2.0% (14)	88.0% (606)	10.0% (69)	1.8% (3)	29.4% (48)	68.7% (112)
20–29	22.8% (31)	70.6% (96)	6.6% (9)	2.0% (14)	87.2% (601)	10.7% (74)	2.5% (4)	65.6% (107)	31.9% (52)
30–39	8.8% (12)	85.3% (116)	5.9% (8)	1.6% (11)	89.3% (615)	9.1% (63)	0.6% (1)	76.7% (125)	22.7% (37)
40–50	1.5% (2)	91.2% (124)	7.4% (10)	1.2% (8)	94.0% (647)	4.9% (34)	0.6% (1)	95.1% (155)	4.3% (7)

3.2. Midlife Characteristics According to Adolescence PA

Those who were engaged in competitive sport at age 13–16 were slightly younger and had used HC more often during the preceding 10 years than participants in the other two groups. Participants in the competitive sport group also had a higher education level in midlife and later AAM than participants with no exercise (Table 2).

Table 2. Midlife characteristics of participants according to physical activity (PA) level at the age of 13–16.

Variable	n	Competitive Sport	n	Regular PA	n	No Exercise	p-Value	Model 1: p-Value ^{a,b}	Model 2: p-Value ^{c,d}
Background variables									
Age (y)	136	50.9 (50.5–51.2) ^{R,N}	689	51.4 (51.3–51.6)	163	51.6 (51.3–51.9)	0.005 ^e		
Age at menarche (y)	134		677		162		0.010		
≤12		35.1% (47) ^N		35.2% (238) ^N		38.9% (63)			
13		30.6% (41)		34.3% (232)		43.2% (70)			
≥14		34.3% (46)		30.6% (207)		17.9% (29)			
Bachelor or higher education	136	48.5% (66) ^N	689	41.5% (286)	163	34.4% (56)	0.045		
Number of parities	136	2.0 (1.8–2.1)	687	2.1 (2.0–2.2) ^N	163	1.9 (1.7–2.1)	0.017 ^f		
Used OCP at some point of life	136	6.6% (9)	689	9.9% (68)	163	11.0% (18)	0.399		
Used HC during the preceding 10 years	136	62.5% (85) ^{R,N}	689	50.8% (350)	163	49.1% (80)	0.031		
Menopausal status	136		689		161		0.893		
PRE		30.1% (41)		27.4% (189)		24.5% (40)			
EPM		19.1% (26)		18.6% (128)		19.0% (31)			
LPM		LPM 21.3% (29)		19.3% (133)		20.9% (34)			
POST		POST 29.4% (40)		34.7% (239)		35.6% (58)			
Body composition									
Height (cm)	113	166.4 (165.4–167.4)	609	165.6 (165.1–166.0)	146	164.7 (163.8–165.7)	0.074 ^e	0.188 ^a	0.257 ^c
Weight (m)	113	70.3 (68.3–72.3)	609	70.3 (69.4–71.2)	146	68.5 (66.8–70.1)	0.182 ^e	0.046 ^a	0.038 ^c
BMI (kg/m ²)	113	25.4 (24.7–26.1)	609	25.6 (25.3–25.9)	146	25.2 (24.7–25.8)	0.431 ^e	0.151 ^a	0.124 ^c
Total fat mass (kg)	101	24.0 (22.4–25.5)	584	25.5 (24.8–26.2)	138	24.4 (23.1–25.7)	0.141 ^e	0.041 ^a	0.037 ^c
Fat percentage (%)	101	33.3 (31.8–34.7) ^R	584	35.4 (34.8–36.0)	138	35.0 (33.8–36.2)	0.028 ^e	0.022 ^a	0.026 ^c
Fat mass index (kg/m ²)	101	8.7 (8.1–9.3)	584	9.3 (9.0–9.5)	138	9.0 (8.5–9.7)	0.143 ^e	0.065 ^a	0.062 ^c
Total lean mass (kg)	101	43.7 (42.8–44.6) ^{R,N}	584	42.1 (41.7–42.4)	138	41.4 (40.7–42.1)	<0.001 ^e	0.001 ^a	0.001 ^c
Lean mass index (kg/m ²)	101	15.8 (15.5–16.0) ^{R,N}	584	15.3 (15.2–15.4)	138	15.2 (15.0–15.5)	0.002 ^e	0.002 ^a	0.001 ^c
ALMI (kg/m ²)	101	6.9 (6.7–7.0) ^{R,N}	584	6.6 (6.6–6.7)	138	6.6 (6.5–6.7)	<0.001 ^e	0.001 ^a	0.001 ^c

Table 2. Cont.

Variable	n	Competitive Sport	n	Regular PA	n	No Exercise	p-Value	Model 1: p-Value ^{a,b}	Model 2: p-Value ^{c,d}
<i>BMD</i>									
FN BMD (g/cm ²)	101	1.00 (0.98–1.03) ^{R,N}	584	0.96 (0.95–0.97)	138	0.95 (0.93–0.97)	<0.001^e	0.001^a	0.002^c
FN T score < −1	101	14.9% (15) ^{R,N}	584	25.7% (150)	138	28.3% (39)	0.039		
FN T score ≤ −2.5	101	0.0% (0)	584	0.9% (5)	138	0.7% (1)	1.000		
<i>Physical performance</i>									
Hand grip force (N)	106	333.5 (322.6–344.4) ^{R,N}	590	313.5 (308.7–318.4)	142	305.5 (296.4–314.5)	0.001^e	0.002^a	0.002^c
Knee extension force (N)	92	509.1 (488.4–529.8) ^{R,N}	508	460.6 (452.7–468.4)	118	442.3 (425.6–458.9)	<0.001^e	<0.001^a	<0.001^c
Jumping height (cm)	101	21.3 (20.5–22.1) ^{R,N}	547	19.0 (18.6–19.3)	136	18.7 (18.0–19.4)	<0.001^e	<0.001^a	<0.001^c
Walking speed (m/s)	106	2.9 (2.8–3.0) ^{R,N}	591	2.6 (2.6–2.7)	140	2.6 (2.5–2.6)	<0.001^e	<0.001^a	<0.001^c
Walking distance in 6 min (m)	95	696.7 (684.8–708.5) ^{R,N}	543	667.5 (662.3–672.7)	131	660.7 (651.1–670.2)	<0.001^e	<0.001^a	0.001^c
<i>Self-reported PA</i>									
Leisure-time PA (MET-h/d)	136	4.9 (4.2–5.6) ^{R,N}	689	4.2 (3.9–4.4)	163	3.6 (3.2–4.0)	<0.001^f	0.008^b	0.007^d
<i>Accelerometer-measured PA</i>									
Leisure-time MVPA (min/d)	88	47.6 (41.6–53.6)	516	43.1 (41.1–45.2)	126	40.7 (36.6–44.7)	0.166 ^f	0.178 ^b	0.271 ^d
Leisure-time step count (steps/d)	88	7277 (6676–7878)	516	6843 (6601–7084)	126	6763 (6334–7192)	0.339 ^e	0.302 ^b	0.384 ^d
Total MVPA (min/d)	88	54.3 (47.7–60.7)	516	50.3 (48.0–52.5)	126	46.7 (42.6–50.8)	0.188 ^f	0.220 ^b	0.312 ^d
Total step count (steps/d)	88	8903 (8291–9515)	516	8698 (8450–8947)	126	8510 (8087–8933)	0.646 ^f	0.743 ^b	0.761 ^d

Values are presented as means (95% confidence intervals) or percentages (counts). R and N indicate significant difference ($p < 0.05$) from regular PA and no exercise groups, respectively. OCP = oral contraceptive pills; HC = hormonal contraceptive; PRE = premenopausal; EPM = early perimenopausal; LPM = late perimenopausal; POST = postmenopausal; BMI = body mass index; ALMI = appendicular lean mass index; FN = femoral neck; BMD = bone mineral density; MET = metabolic equivalent; MVPA = moderate-to-vigorous physical activity. ^a Adjusted for self-reported leisure-time PA in midlife, age, menarcheal age, and menopausal status. ^b Adjusted for age, menarcheal age, and menopausal status. ^c Adjusted for self-reported leisure-time PA in midlife, age, menarcheal age, menopausal status, number of parities, education, and hormonal contraceptive use during the 10 preceding years. ^d Adjusted for age, menarcheal age, menopausal status, number of parities, education, and hormonal contraceptive use during the 10 preceding years. ^e ANOVA. ^f Kruskal-Wallis test. Sample sizes for both adjusted models were 111, 598, and 149 for competitive sport, regular PA, and no exercise groups, respectively, in height, weight, and BMI. Corresponding sample sizes for body composition measurements and BMD were 99, 573, and 137, for physical performance measurements they were 104, 579, and 141 (hand grip force), 99, 538, and 135 (jumping height), 104, 580, and 139 (walking speed), 93, 534, and 131 (walking distance in 6 min), and for physical activity measurements they were 134, 677, and 162 (self-reported PA) and 86, 510, and 125 (accelerometer measured PA). Statistically significant p -values are indicated in bold.

Participants in the competitive sport group had more lean mass, higher lean mass index, ALMI, and BMD than those with no exercise or regular PA. They also performed better in all physical performance tests than participants in the other two groups. Self-reported PA at midlife was higher in the competitive sport group than in the other groups but no difference was found in accelerometer-measured PA between the groups. Differences in total lean mass, lean mass index, ALMI, BMD, physical performance, and self-reported leisure-time PA between the competitive sport group and the other groups remained after adjustment for potential confounding factors (Table 2). Participants who did not respond to the question regarding PA at age 13–16 (11.1% of the whole sample, $n = 110$) were similar to those who completed all variables except in hand grip force (non-responders 297.9 N vs. responders 314.7 N, $p = 0.018$) and in menopausal status (proportions of premenopausal, early perimenopausal, late perimenopausal, and postmenopausal among non-responders 30.9%, 11.8%, 11.8%, and 45.5%, respectively and among responders 27.3%, 18.7%, 19.8%, and 34.1%, respectively, $p = 0.018$).

3.3. AAM and Midlife Characteristics

AAM among participants ranged between 9 and 19 years (Table S1) with mean age 12.95 years (95% CI 12.9–13.0). The most common menarcheal ages were 13 (34.9%), 12 (24.1%), and 14 years (17.8%). 102 (9.3%) of the participants reported that they had used OCP at some point of their life with starting age between 16–54 years. No participants reported starting OCP use before menarche and 15 of the OCP users did not provide starting year.

The group that reported AAM ≥ 14 years included the smallest portion of participants with no exercise and the greatest portion of participants with regular PA and competitive sport at age 13–16. There was a significant linear downward trend in weight, BMI, total fat mass, fat percentage, and fat mass index across the AAM groups, all of which decreased as AAM increased. All pairwise comparisons were statistically significant except for fat percentage between the group reporting AAM ≤ 12 years and the group reporting AAM = 13 years. BMD was higher in the group reporting AAM ≤ 12 years compared to the group reporting AAM ≥ 14 years and differences in physical performance were found in jump height, with the group of AAM ≥ 14 years jumping higher than the group of AAM ≤ 12 years. Participants in the group reporting AAM ≥ 14 years took more steps than those in the other groups, but no other differences in PA between the groups were found. The differences in weight, BMI, fat mass, fat mass index, ALMI, jump height, and leisure-time step count persisted when adjusted for confounding factors (Table 3). Exclusion of postmenopausal women from this analysis demonstrated an inverse association between AAM and BMD even when adjusted for PA level at age of 13–16, self-reported leisure-time PA at midlife, age, and menopausal status ($p = 0.040$). The inverse association was found also after further adding number of parities, education, and hormonal contraceptive use during the 10 preceding years as covariates in the model ($p = 0.041$).

16.2% ($n = 178$) of the participants reported that they have had at least one fracture during their life, while 37.6% ($n = 413$) reported having current musculoskeletal disorder/symptom. Six participants (0.5%) reported that they have received a diagnosis of anorexia nervosa, while one participant did not answer this question. Age at the time of the diagnosis varied between 13 and 39 (mean 21.5, 95% CI 10.5–32.5) and two participants had received the diagnosis before AAM. No differences were found in the amount of lifetime fractures and anorexia nervosa prevalence, or current musculoskeletal problems between the different PA level in adolescence (Table S2) or AAM groups (Table S3). The participants who did not respond to the question regarding AAM (1.5%, $n = 17$) were similar to those who did in terms of all the outcome variables.

Table 3. Midlife characteristics of the participants according to different menarcheal age.

Variable	n	AAM ≤ 12	n	AAM = 13	n	AAM ≥ 14	p-Value	Model 1: p-Value ^{a,b}	Model 2: p-Value ^{c,d}
Background variables									
Age (y)	391	51.3 (51.1–51.5)	377	51.4 (51.2–51.6)	313	51.4 (51.2–51.6)	0.465 ^e		
Bachelor or higher education (%)	391	41.2% (161)	377	41.9% (158)	313	40.9% (128)	0.961		
Number of parities	391	2.0 (1.9–2.2)	377	2.0 (1.9–2.1)	313	2.0 (1.9–2.2)	0.937 ^f		
Used OCP at some point of life	391	7.9% (31) ^H	377	7.7% (29) ^H	313	13.1% (41)	0.025		
Used HC during the preceding 10 years	391	54.2% (212)	377	48.0% (181)	313	53.7% (168)	0.172		
Menopausal status									
PRE	390	24.8% (97)	377	29.7% (112)	313	29.4% (92)	0.373		
EPM		20.2% (79)		17.2% (65)		16.3% (51)			
LPM		21.7% (85)		17.2% (65)		18.2% (57)			
POST		33.2% (130)		35.8% (135)		36.1% (113)			
PA at age 13–16	346		343		281		0.014		
CS		13.5% (47) ^H		12.0% (41) ^H		16.4% (46)			
RPA		68.6% (238)		67.9% (233)		73.3% (206)			
NE		17.9% (62)		20.1% (69)		10.3% (29)			
Body composition									
Height (cm)	335	165.3 (164.7–165.9)	341	165.3 (164.7–166.0)	266	166.3 (165.6–166.9)	0.073 ^e	0.122 ^a	0.135 ^c
Weight (m)	335	71.7 (70.5–72.8) ^{M,H}	341	69.6 (68.4–70.7)	266	68.0 (66.8–69.3)	<0.001 ^e	<0.001 ^a	<0.001 ^c
BMI (kg/m ²)	335	26.2 (25.8–26.6) ^{M,H}	341	25.4 (25.0–25.8) ^H	266	24.6 (24.2–25.0)	<0.001 ^e	<0.001 ^a	<0.001 ^c
Total fat mass (kg)	315	26.6 (25.7–27.5) ^{M,H}	319	24.9 (24.0–25.9) ^H	247	23.1 (22.0–24.2)	<0.001 ^e	<0.001 ^a	<0.001 ^c
Fat percentage (%)	315	36.4 (35.6–37.1) ^H	319	35.1 (34.3–35.9) ^H	247	33.1 (32.1–34.0)	<0.001 ^e	<0.001 ^a	<0.001 ^c
Fat mass index (kg/m ²)	315	9.7 (9.4–10.1) ^{M,H}	319	9.1 (8.8–9.4) ^H	247	8.4 (8.0–8.8)	<0.001 ^e	<0.001 ^a	<0.001 ^c
Total lean mass (kg)	315	42.3 (41.8–42.8)	319	41.8 (41.4–42.3)	247	42.3 (41.8–42.9)	0.105 ^e	0.099 ^a	0.195 ^c
Lean mass index (kg/m ²)	315	15.5 (15.4–15.6)	319	15.3 (15.2–15.5)	247	15.3 (15.2–15.5)	0.135 ^e	0.032 ^a	0.047 ^c
ALMI (kg/m ²)	315	6.7 (6.6–6.7)	319	6.6 (6.5–6.7)	247	6.6 (6.5–6.7)	0.237 ^e	0.075 ^a	0.124 ^c
BMD									
FN BMD (g/cm ²)	315	0.97 (0.96–0.98) ^H	319	0.96 (0.95–0.97)	247	0.94 (0.93–0.96)	0.034 ^e	0.056 ^a	0.056 ^c
FN T score < −1	315	24.2% (76)	319	23.5% (75)	247	29.6% (73)	0.214		
FN T score ≤ −2.5	315	0.3% (1)	319	1.3% (4)	247	0.8% (2)	0.404		
Physical performance									
Hand grip force (N)	321	314.0 (307.2–320.8)	324	315.0 (308.6–321.5)	254	311.0 (304.0–318.1)	0.715 ^e	0.279 ^a	0.195 ^c
Knee extension force (N)	282	468.8 (457.7–480.0)	271	457.8 (447.1–468.5)	230	459.7 (446.6–472.7)	0.344 ^e	0.419 ^a	0.442 ^c
Jumping height (cm)	300	18.7 (18.3–19.2) ^H	302	19.2 (18.7–19.7)	243	19.8 (19.2–20.4)	0.013 ^e	0.019 ^a	0.013 ^c
Walking speed (m/s)	319	2.6 (2.6–2.7)	324	2.7 (2.6–2.7)	255	2.7 (2.6–2.7)	0.443 ^e	0.508 ^a	0.507 ^c
Walking distance in 6 min (m)	300	665.5 (658.5–672.5)	293	669.8 (663.2–676.3)	240	674.1 (666.0–682.1)	0.259 ^e	0.155 ^a	0.093 ^c

Table 3. Cont.

Variable	n	AAM ≤ 12	n	AAM = 13	n	AAM ≥ 14	p-Value	Model 1: p-Value ^{a,b}	Model 2: p-Value ^{c,d}
Self-reported PA									
Leisure time PA (MET-h/d)	391	4.4 (4.0–4.8)	377	4.0 (3.6–4.3)	313	4.2 (3.8–4.5)	0.178 ^f	0.288 ^b	0.285 ^d
Accelerometer-measured PA									
Leisure-time MVPA (min/d)	282	42.4 (39.6–45.2)	277	41.0 (38.4–43.6)	225	46.9 (43.4–50.4)	0.054 ^f	0.053 ^b	0.036 ^d
Leisure-time step count (steps/d)	282	6812 (6495–7128) ^H	277	6586 (6282–6892) ^H	225	7365 (6986–7744)	0.005 ^f	0.025 ^b	0.018 ^d
Total MVPA (min/d)	282	48.6 (45.6–51.6)	277	48.3 (45.6–51.1)	225	53.6 (49.8–57.5)	0.117 ^f	0.117 ^b	0.093 ^d
Total step count (steps/d)	282	8541 (8214–8868) ^H	277	8492 (8179–8804) ^H	225	9066 (8677–9455)	0.030 ^f	0.166 ^b	0.169 ^d

Values are presented as means (95% confidence intervals) or percentages (counts). M and H indicate significant difference ($p < 0.05$) from the middle and the latest menarcheal group, respectively. AAM = age at menarche; PRE = premenopausal; EPM = early perimenopausal; LPM = late perimenopausal; POST = postmenopausal; PA = physical activity; NE = no exercise; RPA = regular physical activity; CS = competitive sport; BMI = body mass index; ALMI = appendicular lean mass index; FN = femoral neck; BMD = bone mineral density; MET = metabolic equivalent; MVPA = moderate-to-vigorous physical activity. ^a Adjusted for PA level at age of 13–16, self-reported leisure-time PA in midlife, age, and menopausal status. ^b Adjusted for PA level at age 13–16, age, and menopausal status. ^c Adjusted for PA level at ages 13–16, self-reported leisure-time PA in midlife, age, menopausal status, number of parities, education, and hormonal contraceptive use during the 10 preceding years. ^d Adjusted for PA level at age 13–16, age, menopausal status, number of parities, education, and hormonal contraceptive use during the 10 preceding years. ^e ANOVA. ^f Kruskal-Wallis test. Sample sizes for both adjusted models were 299, 312, and 243 for groups reporting AAM ≤ 12 years, = 13 years, and ≥ 14 years, respectively, in height, weight, and BMI. Corresponding sample sizes for body composition measurements and BMD were 286, 295, and 228, for physical performance measurements they were 291, 299, and 234 (hand grip force), 252, 246, and 210 (knee extension force), 271, 278, and 223 (jumping height), 289, 299, and 235 (walking speed), 270, 268, and 220 (walking distance in 6 min), and for physical activity measurements they were 348, 343, and 282 (self-reported PA) and 257, 254, and 210 (accelerometer-measured PA). Statistically significant p-values are indicated in bold.

4. Discussion

This is the first study to describe midlife body composition, BMD, physical performance, and PA according to PA in adolescence and AAM in middle-aged women. We found that those who had been engaged in competitive sport at age 13–16 had more lean mass, higher BMD in the femoral neck, and better physical fitness in midlife than participants with regular PA or no exercise, even when adjusted for potential confounding factors. Participants in the competitive sport group had later AAM than those with no exercise in adolescence. Furthermore, participants with later AAM had lower BMI and less fat mass than those with earlier AAM, and BMD was lower in the group with $AAM \geq 14$ compared to those with $AAM \leq 12$. These associations persisted when adjusted for potential confounding factors, except for the difference in BMD. However, when postmenopausal participants were excluded from this analysis, an inverse relationship between AAM and BMD was found regardless of the confounding factors.

4.1. PA Participation from Childhood to Midlife

We observed declining correlation coefficients between PA level at age 13–16 and PA level at other age stages with advancing age, which is consistent with a meta-analysis conducted by Craigie et al. [11] who found that the strength of tracking of PA declined when the duration of the follow-up increased. In our study, participation in competitive sport in adolescence was associated with higher self-reported leisure-time PA in middle-age, which is in line with an earlier study showing that participation in competitive sport in adolescence predicts maintenance of a high PA level in old age [34]. However, we found no association between PA in adolescence and accelerometer-measured PA in midlife. We may, however, have lacked sufficient power to detect these differences as the sample sizes were smaller in accelerometer-measured compared to self-reported PA.

4.2. Association between PA in Adolescence and AAM

Previous studies have stated that adolescent athletes tend to attain menarche later than their less physically active counterparts [28,29], and similarly in the present study, participants in the competitive sport group and regular PA group had later AAM compared to the no exercise group. Studies have shown that although AAM is largely modified by genes [47–49], it is influenced by environmental factors, including PA [50]. However, since evidence regarding an association between PA before menarche and later AAM is limited [28], it is not known if there is a causal relationship between these variables, if selection bias was present or if other behavioural differences between athletes and non-athletes explain the reported association [27,28].

4.3. Competitive Sport in Adolescence and Midlife Characteristics

We found no difference in fat mass between the adolescent PA groups, in contrast lower fat mass in former gymnasts, runners, and swimmers compared to controls in midlife has been reported [7,9]. The inconsistency between the studies may be due to different study population, with aesthetic and endurance athletes tending to have lower BMI than athletes in other sport disciplines [27], with our study representing a variety of sporting disciplines. Furthermore, our finding concerning higher midlife lean mass in participants in the competitive sports group is in agreement with one [7] but in contrast to another [9] study.

Consistent with some [7,9] but not all studies [51], we found that there was an association between competitive sport in adolescence and higher BMD in midlife. In our study, this association persisted after adjusting for potential confounding factors. This is contrary to the findings of Nilsson et al. [52], who reported that among women aged 75–80 years PA at age 10–30 was positively associated with BMD, but this association was not seen when adjusted for current PA. Therefore, in older women, current PA might be more strongly associated with BMD than PA in adolescence [53]. Further, and again in contrast to our findings, these authors found no association between PA at age 10–30 and current

hand grip strength among women aged 75–80 [52]. These inconsistencies may be due to the fact that participants in our study were younger and PA history was studied in narrower age-range than in the study conducted by Nilsson and colleagues.

While it has been found that the risk of osteoarthritis of the hip and knee is greater in former female athletes compared to the general population [54] and that former athletes have higher risk of knee problems and higher rates of Achilles tendon problems in later life compared to controls [55], we did not find an association between sport participation in adolescence and musculoskeletal problems in middle age. As we did not ask participants what kind of musculoskeletal problems they had, it is possible that in our study, specific types of problems were more common in different groups.

In contrast to our findings, Tveit et al. found that a risk of fracture was lower among former male athletes than among controls [56]. However, we did not differentiate fractures occurred during the sport career from those occurred after retirement from sport. In addition to the fact that participants in the study conducted by Tveit et al. [56] were males, they were also older than those in our study. The incidence of fractures has been reported to be higher among older women compared to our study population [57,58] and thus, fracture rates among women in our study were relatively low.

Due to low number of anorexia nervosa cases in our study, no conclusion in terms of possible association between anorexia and PA in adolescence can be made. Other studies have suggested that eating disorders in general are more common in athletes than in non-athletes [59,60]. The lifetime prevalence of anorexia nervosa in our study was lower than recently found among middle aged women living in UK [61], young adult women living in Finland [62], and also slightly lower than in women living in U.S. [63]. Incidence of eating disorders are highest in adolescence and in young adulthood [64], and our participants were at that age in 1970s and 1980s. It has been reported that anorexia nervosa has become more common since that time [65], which may explain the differences between the results of abovementioned studies and our findings. However, it is suggested that the increased prevalence rates may be due to increased detection of anorexia nervosa rather than true increase of incidence rates [65]. Thus, there might have been some undiagnosed anorexia nervosa cases among our study population as well. Furthermore, in our study, diagnosis of anorexia nervosa was self-reported and asked by a single question, which may further underestimate the actual prevalence rate.

4.4. AAM and Midlife Characteristics

In line with previous observational evidence [31,32,66–73], our results show that AAM is inversely associated with BMI, fat mass, and fat percentage in middle-age. The inverse relationship between AAM and BMI in adulthood was also recently supported by Mendelian randomisation studies [48,74,75]. Fewer studies have investigated the association between AAM and midlife lean mass. Similar to our finding, Kirchengast et al. [66] did not find an association between AAM and lean mass either in pre- or postmenopausal women.

The observed inverse relationship between AAM and midlife BMD in the current study is consistent with findings of some studies conducted in women with a mean age over 40 years [21,76] and also in postmenopausal women only [22,24,77]. However, not all studies have found this association [78–84] and also in our study, the relationship was abolished after adjusting models for potential confounding factors. While shared genetic factors have shown to influence both AAM and BMD [76,85,86], postmenopausal bone loss may interfere with this relationship; the association between early menarche and higher BMD may be stronger in premenopausal than in postmenopausal women [85]. Our finding showing a relationship, regardless of confounding factors but excluding postmenopausal women, between AAM and midlife femoral neck BMD in pre- and perimenopausal supports this hypothesis. However, higher body mass is known to associate with higher BMD [87,88], which also may affect the associations between AAM and BMD that were found in our study. Therefore, further studies are needed to clarify the effects of AAM on midlife BMD. In our study population, there were few participants with extremely late AAM (e.g., >16 years) and thus, there was no statistical power to compare that group with earlier AAM. Thus, more research is needed to investigate if there is

even greater difference in midlife BMD between former athletes with the current criterion of primary amenorrhea (i.e., menarche occurred at the age of 15 or older) and those with AAM < 15 years.

In contrast to our findings, late AAM has been linked to increased fracture risk in later life [26,89–92]. A possible explanation for the controversy between earlier studies compared to ours may be that in our study participants were younger than those in the studies demonstrating this association. Furthermore, again in contrast to our findings, it has been suggested that early menarche is associated with chronic widespread musculoskeletal complaints in later life [93]. No conclusion regarding the association between anorexia nervosa and AAM can be made based on the current study due to only few anorexia nervosa cases in our study population. However, in several other studies anorexia nervosa has been linked to later menarche [94].

4.5. Limitations

We acknowledge some limitations. PA level in adolescence and AAM were self-reported retrospectively and thus susceptible to response or recall bias. It is reasonable to assume that those who had been competitive athletes in their adolescence did not have difficulties in assigning themselves in an accurate PA category. However, differentiation between no exercise and regular PA may have been overlapping with the possibility that participants had different perceptions for PA. In addition, it has been reported that long-term recall of AAM is fairly valid [95–98] and that the accuracy of recall is increased when AAM is categorised into groups [98]. We did not ask the precise month the participant attained menarche, which may have caused some inaccuracy; however, few middle-aged women are able to recall the precise month they started to menstruate [96,97]. Furthermore, the study design does not enable causal interpretations. Although we adjusted our analysis for potential confounding factors, residual confounding cannot be completely ruled out. It is possible that other factors, such as genetic or different lifestyle habits, affected the observed associations.

No information on menstrual history, such as history of menstrual dysfunction, was collected and thus, we do not know if the participants have had regular menses after menarche. On the other hand, recall of menstrual regularity is less accurate than recall of AAM in women near menopause [95]. In addition, we did not collect information on training volume in adolescence and thus, cannot be sure if the participants engaged in competitive sport in adolescence actually differed from those who exercised regularly in terms of training volume and intensity. Furthermore, our study sample consisted of Caucasian relatively healthy individuals with no health conditions jeopardizing physical or mental conditioning, which can limit the generalisability of the results. Finally, the sample size did not allow us to investigate interaction effects between AAM and sport participation on BMD and body composition. However, it is possible that such interactions exist. Thus, based on our results, we cannot say if, for example, individuals who participated in competitive sport during adolescence but had an AAM \leq 12 years have better midlife BMD than those in a competitive sport group with an AAM \geq 14 years.

It is also important to bear in mind that our participants engaged in competitive sport in their adolescence, approximately 40 years ago. Since then, sport specialisation at early age has increased globally [99] and participation in organised sport club has become more frequent in Finnish girls [100]. Early sport specialisation can lead to physical and mental harm as it has been linked to reduced motor skill development, increased injury rates, sport dropout, burnout, eating disorders, and menstrual dysfunction [99,101–103]. Thus, generalisability of our results to those who are currently engaged in competitive sport should be done with caution.

5. Conclusions

This large, retrospective study suggests that participation in competitive sport at age 13–16 is associated with higher midlife lean mass and femoral neck BMD, and better physical performance compared to regular PA or no exercise during adolescence. In addition, we found that competitive sport at age 13–16 was associated with later menarche compared to no exercise at that age. The current

study also showed that AAM was negatively associated with midlife BMI and fat mass regardless of confounding factors in all participants, and with femoral neck BMD in pre- and perimenopausal women. Thus, competitive sport in adolescence seems to be associated with healthier body composition, higher BMD, and better performance in midlife, but late AAM might be associated with low BMD. Further research is needed to investigate whether middle-aged or older individuals with primary amenorrhea (i.e., AAM \geq 15 years) differ from individual with AAM < 15 years especially in terms of BMD or fractures. Furthermore, future studies should investigate what is the volume and intensity of the exercise needed in adolescence with respect to positive outcomes in middle age seen in our study.

Supplementary Materials: The following are available online at <http://www.mdpi.com/2077-0383/9/12/3797/s1>, Figure S1: Proportions of the participants in different physical activity level groups across the age categories, Table S1: Age at menarche among participants ($n = 1081$), Table S2: Proportion (n) of the participants reporting fractures, musculoskeletal disorders/symptoms, and anorexia nervosa diagnosis according to physical activity (PA) level at the age of 13–16, Table S3: Proportion (n) of the participants reporting fractures, musculoskeletal disorders/symptoms, and anorexia nervosa diagnosis according to age at menarche (AAM) groups.

Author Contributions: Conceptualization, S.R., U.M.K., T.H.T., M.H., V.K., P.A., S.S., and E.K.L.; methodology, S.R., U.M.K., S.S. and E.K.L.; formal analysis, S.R.; investigation, U.M.K., V.K., S.S. and E.K.L.; resources, V.K. and E.K.L.; data curation, E.K.L.; writing—original draft preparation, S.R., U.K.M., T.H.T., S.S., and E.K.L.; writing—review and editing, S.R., U.M.K., T.H.T., M.H., V.K., M.V., B.W., P.A., S.S. and E.K.L.; visualization, S.R. and E.K.L.; supervision, U.M.K., M.V., B.W. and E.K.L.; project administration, E.K.L.; funding acquisition, V.K. and E.K.L. All authors have read and agreed to the published version of the manuscript.

Funding: This research was funded by the Academy of Finland (VK: grant 275323, EKL: grants 309504, 314181) and the Juho Vainio Foundation (EKL).

Acknowledgments: We thank all the study participants who contributed their time and information. In addition, we are grateful for the staff members at the Faculty of Sport and Health Sciences, University of Jyväskylä and at the LIKES Research Centre for Physical Activity and Health who helped with the study.

Conflicts of Interest: The authors declare no conflict of interest. The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, or in the decision to publish the results.

References

- Haskell, W.L.; Lee, I.-M.; Pate, R.R.; Powell, K.; Blair, S.N.; Franklin, B.; Macera, C.; Heath, G.W.; Thompson, P.D.; Bauman, A.; et al. Physical Activity and Public Health: Updated Recommendation for Adults From the American College of Sports Medicine and the American Heart Association. *Circulation* **2007**, *116*, 1081–1093. [CrossRef] [PubMed]
- Theintz, G.; Buchs, B.; Rizzoli, R.; Slosman, D.; Clavien, H.; Sizonenko, P.C.; Bonjour, J.-P. Longitudinal monitoring of bone mass accumulation in healthy adolescents: Evidence for a marked reduction after 16 years of age at the levels of lumbar spine and femoral neck in female subjects. *J. Clin. Endocrinol. Metab.* **1992**, *75*, 1060–1065. [CrossRef]
- Kohrt, W.M.; Bloomfield, S.A.; Little, K.D.; Nelson, M.E.; Yingling, V.R. Physical Activity and Bone Health. *Med. Sci. Sports Exerc.* **2004**, *36*, 1985–1996. [CrossRef] [PubMed]
- Zanker, C.L.; Osborne, C.; Oldroyd, B.; Truscott, J.G.; Cooke, C.B. Bone density, body composition and menstrual history of sedentary female former gymnasts, aged 20–32 years. *Osteoporos. Int.* **2004**, *15*, 145–154. [CrossRef] [PubMed]
- Bass, S.; Pearce, G.; Bradney, M.; Hendrich, E.; Delmas, P.D.; Harding, A.; Seeman, E. Exercise Before Puberty May Confer Residual Benefits in Bone Density in Adulthood: Studies in Active Prepubertal and Retired Female Gymnasts. *J. Bone Miner. Res.* **1998**, *13*, 500–507. [CrossRef] [PubMed]
- Erlandson, M.C.; Kontulainen, S.; Chilibeck, P.D.; Arnold, C.M.; Faulkner, R.; Baxter-Jones, A.D.G. Higher premenarcheal bone mass in elite gymnasts is maintained into young adulthood after long-term retirement from sport: A 14-year follow-up. *J. Bone Miner. Res.* **2011**, *27*, 104–110. [CrossRef] [PubMed]
- Andreoli, A.; Celi, M.; Volpe, S.L.; Sorge, R.; Tarantino, U. Long-term effect of exercise on bone mineral density and body composition in post-menopausal ex-elite athletes: A retrospective study. *Eur. J. Clin. Nutr.* **2011**, *66*, 69–74. [CrossRef] [PubMed]

8. Khan, K.M.; Bennell, K.; Hopper, J.L.; Flicker, L.; Nowson, C.; Sherwin, A.J.; Crichton, K.J.; Harcourt, P.R.; Wark, J.D. Self-reported ballet classes undertaken at age 10–12 years and hip bone mineral density in later life. *Osteoporos. Int.* **1998**, *8*, 165–173. [CrossRef] [PubMed]
9. Pollock, N.K.; Laing, E.M.; Modlesky, C.M.; O'Connor, P.J.; Lewis, R.D. Former college artistic gymnasts maintain higher BMD: A nine-year follow-up. *Osteoporos. Int.* **2006**, *17*, 1691–1697. [CrossRef]
10. Telama, R. Tracking of Physical Activity from Childhood to Adulthood: A Review. *Obes. Facts* **2009**, *2*, 187–195. [CrossRef]
11. Craigie, A.M.; Lake, A.A.; Kelly, S.A.; Adamson, A.J.; Mathers, J.C. Tracking of obesity-related behaviours from childhood to adulthood: A systematic review. *Maturitas* **2011**, *70*, 266–284. [CrossRef]
12. Telama, R.; Yang, X.; Leskinen, E.; Kankaanpää, A.; Hirvensalo, M.; Tammelin, T.; Viikari, J.S.A.; Raitakari, O.T. Tracking of Physical Activity from Early Childhood through Youth into Adulthood. *Med. Sci. Sports Exerc.* **2014**, *46*, 955–962. [CrossRef]
13. Palomäki, S.; Hirvensalo, M.; Smith, K.; Raitakari, O.; Männistö, S.; Hutri-Kähönen, N.; Tammelin, T.H. Does organized sport participation during youth predict healthy habits in adulthood? A 28-year longitudinal study. *Scand. J. Med. Sci. Sports* **2018**, *28*, 1908–1915. [CrossRef] [PubMed]
14. De Souza, M.J.; Nattiv, A.; Joy, E.; Misra, M.; Williams, N.; Mallinson, R.J.; Gibbs, J.C.; Olmsted, M.; Goolsby, M.; Matheson, G.; et al. 2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad: 1st International Conference held in San Francisco, California, May 2012 and 2nd International Conference held in Indianapolis, Indiana, May 2013. *Br. J. Sports Med.* **2014**, *48*, 289. [CrossRef] [PubMed]
15. Mountjoy, M.; Sundgot-Borgen, J.K.; Burke, L.M.; Ackerman, K.; Blauwet, C.; Constantini, N.; Lebrun, C.; Lundy, B.; Melin, A.K.; Meyer, N.L.; et al. IOC consensus statement on relative energy deficiency in sport (RED-S): 2018 update. *Br. J. Sports Med.* **2018**, *52*, 687–697. [CrossRef] [PubMed]
16. Kujala, U.M.; Orava, S.; Parkkari, J.; Kaprio, J.; Sarna, S. Sports Career-Related Musculoskeletal Injuries. *Sports Med.* **2003**, *33*, 869–875. [CrossRef] [PubMed]
17. Friery, K.B. Incidence of injury and disease among former athletes: A review. *J. Exerc. Physiol. Online* **2008**, *11*, 26–45.
18. Schwenk, T.L.; Gorenflo, D.W.; Dopp, R.R.; Hipple, E. Depression and Pain in Retired Professional Football Players. *Med. Sci. Sports Exerc.* **2007**, *39*, 599–605. [CrossRef]
19. Tveit, M.; Rosengren, B.E.; Nilsson, J.-Å.; Karlsson, M.K. Former Male Elite Athletes Have a Higher Prevalence of Osteoarthritis and Arthroplasty in the Hip and Knee Than Expected. *Am. J. Sports Med.* **2011**, *40*, 527–533. [CrossRef]
20. Meczekalski, B.; Katulski, K.; Czyzyk, A.; Podfigurna-Stopa, A. Health in older women athletes. *Maturitas* **2014**, *79*, 357–361. [CrossRef]
21. Chevalley, T.; Bonjour, J.-P.; Ferrari, S.; Rizzoli, R. Deleterious Effect of Late Menarche on Distal Tibia Microstructure in Healthy 20-Year-Old and Premenopausal Middle-Aged Women. *J. Bone Miner. Res.* **2009**, *24*, 144–152. [CrossRef] [PubMed]
22. Mohammadi, F.; Iranagh, J.A.; Motalebi, S.A.; Hamid, T.A. Reproductive factors influencing bone mineral density in postmenopausal women. *Women Health* **2018**, *59*, 145–154. [CrossRef] [PubMed]
23. Gerdhem, P.; Obrant, K.J. Bone mineral density in old age: The influence of age at menarche and menopause. *J. Bone Miner. Metab.* **2004**, *22*, 372–375. [CrossRef]
24. Varenna, M.; Binelli, L.; Zucchi, F.; Ghiringhelli, D.; Gallazzi, M.; Sinigaglia, L. Prevalence of Osteoporosis by Educational Level in a Cohort of Postmenopausal Women. *Osteoporos. Int.* **1999**, *9*, 236–241. [CrossRef]
25. Paganini-Hill, A.; Atchison, K.A.; Gornbein, J.A.; Nattiv, A.; Service, S.K.; White, S.C. Menstrual and Reproductive Factors and Fracture Risk: The Leisure World Cohort Study. *J. Womens Health* **2005**, *14*, 808–819. [CrossRef]
26. Cooper, G.S.; Sandler, D.P. Long-term effects of reproductive-age menstrual cycle patterns on peri- and postmenopausal fracture risk. *Am. J. Epidemiol.* **1997**, *145*, 804–809. [CrossRef]
27. Torstveit, M.K.; Sundgot-Borgen, J. Participation in leanness sports but not training volume is associated with menstrual dysfunction: A national survey of 1276 elite athletes and controls. *Br. J. Sports Med.* **2005**, *39*, 141–147. [CrossRef]
28. Calthorpe, L.; Brage, S.; Ong, K.K. Systematic review and meta-analysis of the association between childhood physical activity and age at menarche. *Acta Paediatr.* **2019**, *108*, 1008–1015. [CrossRef] [PubMed]

29. Hoch, A.Z.; Pajewski, N.M.; Moraski, L.; Carrera, G.F.; Wilson, C.R.; Hoffmann, R.G.; Schimke, J.E.; Gutterman, D.D. Prevalence of the Female Athlete Triad in High School Athletes and Sedentary Students. *Clin. J. Sport Med.* **2009**, *19*, 421–428. [CrossRef] [PubMed]
30. Bubach, S.; Menezes, A.M.B.; Barros, F.C.; Wehrmeister, F.C.; Gonçalves, H.; Assunção, M.C.F.; Horta, B.L. Impact of the age at menarche on body composition in adulthood: Results from two birth cohort studies. *BMC Public Health* **2016**, *16*, 1007. [CrossRef] [PubMed]
31. Trikudanathan, S.; Pedley, A.; Massaro, J.M.; Hoffmann, U.; Seely, E.W.; Murabito, J.M.; Fox, C.S. Association of Female Reproductive Factors with Body Composition: The Framingham Heart Study. *J. Clin. Endocrinol. Metab.* **2013**, *98*, 236–244. [CrossRef]
32. Prentice, P.; Viner, R.M. Pubertal timing and adult obesity and cardiometabolic risk in women and men: A systematic review and meta-analysis. *Int. J. Obes.* **2013**, *37*, 1036–1043. [CrossRef]
33. Kovanen, V.; Aukee, P.; Kokko, K.; Finni, T.; Tarkka, I.M.; Tammelin, T.; Kujala, U.M.; Sipilä, S.; Laakkonen, E.K. Design and protocol of Estrogenic Regulation of Muscle Apoptosis (ERMA) study with 47 to 55-year-old women's cohort: Novel results show menopause-related differences in blood count. *Menopause* **2018**, *25*, 1020–1032. [CrossRef] [PubMed]
34. Hirvensalo, M.; Lintunen, T.; Rantanen, T. The continuity of physical activity—a retrospective and prospective study among older people. *Scand. J. Med. Sci. Sports* **2000**, *10*, 37–41. [CrossRef] [PubMed]
35. Van Der Ploeg, G.E.; Withers, R.T.; Laforgia, J. Percent body fat via DEXA: Comparison with a four-compartment model. *J. Appl. Physiol.* **2003**, *94*, 499–506. [CrossRef] [PubMed]
36. Bondarev, D.; Laakkonen, E.K.; Finni, T.; Kokko, K.; Kujala, U.M.; Aukee, P.; Kovanen, V.; Sipilä, S. Physical performance in relation to menopause status and physical activity. *Menopause* **2018**, *25*, 1432–1441. [CrossRef]
37. Bosco, C.; Luhtanen, P.; Komi, P.V. A simple method for measurement of mechanical power in jumping. *Eur. J. Appl. Physiol.* **1983**, *50*, 273–282. [CrossRef]
38. Ronkainen, P.H.A.; Kovanen, V.; Alén, M.; Pöllänen, E.; Palonen, E.-M.; Ankarberg-Lindgren, C.; Hämäläinen, E.; Turpeinen, U.; Kujala, U.M.; Puolakka, J.; et al. Postmenopausal hormone replacement therapy modifies skeletal muscle composition and function: A study with monozygotic twin pairs. *J. Appl. Physiol.* **2009**, *107*, 25–33. [CrossRef]
39. ATS. Committee on Proficiency Standards for Clinical Pulmonary Function Laboratories ATS Statement: Guidelines for the six-minute walk test. *Am. J. Respir. Crit. Care Med.* **2002**, *166*, 111–117. [CrossRef]
40. Laakkonen, E.K.; Kulmala, J.; Aukee, P.; Hakonen, H.; Kujala, U.M.; Lowe, D.A.; Kovanen, V.; Tammelin, T.; Sipilä, S. Female reproductive factors are associated with objectively measured physical activity in middle-aged women. *PLoS ONE* **2017**, *12*, e0172054. [CrossRef]
41. Sasaki, J.; John, D.; Freedson, P.S. Validation and comparison of ActiGraph activity monitors. *J. Sci. Med. Sport* **2011**, *14*, 411–416. [CrossRef] [PubMed]
42. Kujala, U.M.; Kaprio, J.; Sarna, S.; Koskenvuo, M. Relationship of Leisure-Time Physical Activity and Mortality: The Finnish twin cohort. *JAMA* **1998**, *279*, 440–444. [CrossRef] [PubMed]
43. Hyvärinen, M.; Sipilä, S.; Kulmala, J.; Hakonen, H.; Tammelin, T.H.; Kujala, U.M.; Kovanen, V.; Laakkonen, E.K. Validity and Reliability of a Single Question for Leisure-Time Physical Activity Assessment in Middle-Aged Women. *J. Aging Phys. Act.* **2020**, *28*, 231–241. [CrossRef] [PubMed]
44. Ainsworth, B.; Haskell, W.L.; Herrmann, S.D.; Meckes, N.; Bassett, D.R.; Tudor-Locke, C.; Greer, J.L.; Vezina, J.; Whitt-Glover, M.C.; Leon, A.S. 2011 Compendium of Physical Activities: A Second Update of Codes and MET Values. *Med. Sci. Sports Exerc.* **2011**, *43*, 1575–1581. [CrossRef]
45. Waller, K.; Kaprio, J.; Kujala, U.M. Associations between long-term physical activity, waist circumference and weight gain: A 30-year longitudinal twin study. *Int. J. Obes.* **2007**, *32*, 353–361. [CrossRef]
46. Harlow, S.D.; Gass, M.; Hall, J.E.; Lobo, R.; Maki, P.M.; Rebar, R.W.; Sherman, S.S.; Sluss, P.M.; de Villiers, T.J.; STRAW + 10 Collaborative Group. Executive Summary of the Stages of Reproductive Aging Workshop + 10: Addressing the Unfinished Agenda of Staging Reproductive Aging. *J. Clin. Endocrinol. Metab.* **2012**, *97*, 1159–1168. [CrossRef]
47. He, C.; Kraft, P.; Chen, C.; Buring, J.E.; Paré, G.; Hankinson, S.E.; Chanock, S.J.; Ridker, P.M.; Hunter, D.J.; Chasman, D.I. Genome-wide association studies identify loci associated with age at menarche and age at natural menopause. *Nat. Genet.* **2009**, *41*, 724–728. [CrossRef]

48. Day, F.R.; Thompson, D.J.; Helgason, H.; Chasman, D.I.; Finucane, H.; Sulem, P.; Ruth, K.S.; Whalen, S.; Sarkar, A.K.; Albrecht, E.; et al. Genomic analyses identify hundreds of variants associated with age at menarche and support a role for puberty timing in cancer risk. *Nat. Genet.* **2017**, *49*, 834–841. [CrossRef]
49. Zhu, J.; Kusa, T.O.; Chan, Y.-M. Genetics of pubertal timing. *Curr. Opin. Pediatr.* **2018**, *30*, 532–540. [CrossRef]
50. Yermachenko, A.; Dvornyk, V. Nongenetic Determinants of Age at Menarche: A Systematic Review. *BioMed Res. Int.* **2014**, *2014*, 1–14. [CrossRef]
51. Valdimarsson, Ö.; Alborg, H.G.; Düppe, H.; Nyquist, F.; Karlsson, M. Reduced Training Is Associated with Increased Loss of BMD. *J. Bone Miner. Res.* **2005**, *20*, 906–912. [CrossRef] [PubMed]
52. Nilsson, M.; Sundh, D.; Mellström, D.; Lorentzon, M. Current Physical Activity Is Independently Associated With Cortical Bone Size and Bone Strength in Elderly Swedish Women. *J. Bone Miner. Res.* **2016**, *32*, 473–485. [CrossRef] [PubMed]
53. Sipilä, S.; Törmäkangas, T.; Sillanpää, E.; Aukee, P.; Kujala, U.M.; Kovanen, V.; Laakkonen, E.K. Muscle and bone mass in middle-aged women: Role of menopausal status and physical activity. *J. Cachex-Sarcopenia Muscle* **2020**, *11*, 698–709. [CrossRef] [PubMed]
54. Spector, T.D.; Harris, P.A.; Hart, D.; Cicuttini, F.M.; Nandra, D.; Etherington, J.; Wolman, R.L.; Doyle, D.V. Risk of osteoarthritis associated with long-term weight-bearing sports: A radiologic survey of the hips and knees in female ex-athletes and population controls. *Arthritis Rheum.* **1996**, *39*, 988–995. [CrossRef]
55. Haljaste, K.; Unt, E. Relationships between physical activity and musculoskeletal disorders in former athletes. *Coll. Antropol.* **2010**, *34*, 1335–1340. [PubMed]
56. Tveit, M.; Rosengren, B.E.; Nyquist, F.; Nilsson, J.-Å.; Karlsson, M.K. Former Male Elite Athletes Have Lower Incidence of Fragility Fractures Than Expected. *Med. Sci. Sports Exerc.* **2013**, *45*, 405–410. [CrossRef] [PubMed]
57. Bergström, U.; Jönsson, H.; Gustafson, Y.; Pettersson, U.; Stenlund, H.; Svensson, O. The hip fracture incidence curve is shifting to the right. *Acta Orthop.* **2009**, *80*, 520–524. [CrossRef]
58. Burge, R.; Dawson-Hughes, B.; Solomon, D.H.; Wong, J.B.; King, A.; Tosteson, A. Incidence and Economic Burden of Osteoporosis-Related Fractures in the United States, 2005–2025. *J. Bone Miner. Res.* **2007**, *22*, 465–475. [CrossRef]
59. Bratland-Sanda, S.; Sundgot-Borgen, J. Eating disorders in athletes: Overview of prevalence, risk factors and recommendations for prevention and treatment. *Eur. J. Sport Sci.* **2013**, *13*, 499–508. [CrossRef]
60. Martinsen, M.; Sundgot-Borgen, J. Higher Prevalence of Eating Disorders among Adolescent Elite Athletes than Controls. *Med. Sci. Sports Exerc.* **2013**, *45*, 1188–1197. [CrossRef]
61. Micali, N.; Martini, M.G.; Thomas, J.J.; Eddy, K.T.; Kothari, R.; Russell, E.; Bulik, C.M.; Treasure, J. Lifetime and 12-month prevalence of eating disorders amongst women in mid-life: A population-based study of diagnoses and risk factors. *BMC Med.* **2017**, *15*, 12. [CrossRef] [PubMed]
62. Keski-Rahkonen, A.; Hoek, H.W.; Susser, E.S.; Linna, M.S.; Sihvola, E.; Raevuori, A.; Bulik, C.M.; Kaprio, J.; Rissanen, A. Epidemiology and Course of Anorexia Nervosa in the Community. *Am. J. Psychiatry* **2007**, *164*, 1259–1265. [CrossRef] [PubMed]
63. Hudson, J.I.; Hiripi, E.; Pope, H.G.; Kessler, R.C. The Prevalence and Correlates of Eating Disorders in the National Comorbidity Survey Replication. *Biol. Psychiatry* **2007**, *61*, 348–358. [CrossRef] [PubMed]
64. Jacobi, C.; Hayward, C.; De Zwaan, M.; Kraemer, H.C.; Agras, W.S. Coming to Terms With Risk Factors for Eating Disorders: Application of Risk Terminology and Suggestions for a General Taxonomy. *Psychol. Bull.* **2004**, *130*, 19–65. [CrossRef] [PubMed]
65. Keski-Rahkonen, A.; Mustelin, L. Epidemiology of eating disorders in Europe: Prevalence, incidence, comorbidity, course, consequences, and risk factors. *Curr. Opin. Psychiatry* **2016**, *29*, 340–345. [CrossRef] [PubMed]
66. Kirchengast, S.; Gruber, D.; Sator, M.; Huber, J. Impact of the age at menarche on adult body composition in healthy pre- and postmenopausal women. *Am. J. Phys. Anthr.* **1998**, *105*, 9–20. [CrossRef]
67. Pierce, M.B.; Leon, D. Age at menarche and adult BMI in the Aberdeen Children of the 1950s Cohort Study. *Am. J. Clin. Nutr.* **2005**, *82*, 733–739. [CrossRef] [PubMed]
68. Lakshman, R.; Forouhi, N.G.; Sharp, S.J.; Luben, R.; Bingham, S.A.; Khaw, K.-T.; Wareham, N.J.; Ong, K.K. Early Age at Menarche Associated with Cardiovascular Disease and Mortality. *J. Clin. Endocrinol. Metab.* **2009**, *94*, 4953–4960. [CrossRef] [PubMed]

69. Newby, P.K.; Dickman, P.W.; Adami, H.-O.; Wolk, A. Early anthropometric measures and reproductive factors as predictors of body mass index and obesity among older women. *Int. J. Obes.* **2005**, *29*, 1084–1092. [CrossRef] [PubMed]
70. Wang, W.; Zhao, L.-J.; Liu, Y.-Z.; Recker, R.R.; Deng, H. Genetic and environmental correlations between obesity phenotypes and age at menarche. *Int. J. Obes.* **2006**, *30*, 1595–1600. [CrossRef] [PubMed]
71. Hardy, R.; Maddock, J.; Ghosh, A.K.; Hughes, A.D.; Kuh, D. The relationship between pubertal timing and markers of vascular and cardiac structure and function in men and women aged 60–64 years. *Sci. Rep.* **2019**, *9*, 1–10. [CrossRef] [PubMed]
72. Magnus, M.C.; Lawlor, D.A.; Iliodromiti, S.; Padmanabhan, S.; Nelson, S.M.; Fraser, A. Age at Menarche and Cardiometabolic Health: A Sibling Analysis in the Scottish Family Health Study. *J. Am. Heart Assoc.* **2018**, *7*. [CrossRef] [PubMed]
73. Farahmand, M.; Tehrani, F.R.; Khalili, D.; Cheraghi, L.; Azizi, F. Is there any association between age at menarche and anthropometric indices? A 15-year follow-up population-based cohort study. *Eur. J. Pediatr.* **2020**, *179*, 1379–1388. [CrossRef] [PubMed]
74. Gill, D.; Brewer, C.F.; Fabiola Del Greco, M.; Sivakumaran, P.; Bowden, J.; Sheehan, N.A.; Minelli, C. Age at menarche and adult body mass index: A Mendelian randomization study. *Int. J. Obes.* **2018**, *42*, 1574–1581. [CrossRef]
75. Bell, J.A.; Carslake, D.; Wade, K.H.; Richmond, R.C.; Langdon, R.J.; Vincent, E.E.; Holmes, M.V.; Timpson, N.J.; Smith, G.D. Influence of puberty timing on adiposity and cardiometabolic traits: A Mendelian randomisation study. *PLoS Med.* **2018**, *15*, e1002641. [CrossRef]
76. Guo, Y.; Zhao, L.-J.; Shen, H.; Deng, H. Genetic and Environmental Correlations between Age at Menarche and Bone Mineral Density at Different Skeletal Sites. *Calcif. Tissue Int.* **2005**, *77*, 356–360. [CrossRef]
77. Parker, S.E.; Troisi, R.; Wise, L.A.; Palmer, J.R.; Titus-Ernstoff, L.; Strohsnitter, W.C.; Hatch, E.E. Menarche, menopause, years of menstruation, and the incidence of osteoporosis: The influence of prenatal exposure to diethylstilbestrol. *J. Clin. Endocrinol. Metab.* **2013**, *99*, 594–601. [CrossRef]
78. Hagemans, M.L.; Van Der Schouw, Y.T.; De Kleijn, M.J.; Van Staveren, W.A.; Pop, V.J.; Leusink, G.L.; Grobbee, D.E. Indicators for the total duration of premenopausal endogenous estrogen exposure in relation to BMD. *Hum. Reprod.* **2004**, *19*, 2163–2169. [CrossRef]
79. Blum, M.; Harris, S.S.; Must, A.; Phillips, S.M.; Rand, W.M.; Dawson-Hughes, B. Weight and Body Mass Index at Menarche are Associated with Premenopausal Bone Mass. *Osteoporos. Int.* **2001**, *12*, 588–594. [CrossRef]
80. Nakaoka, D.; Sugimoto, T.; Kaji, H.; Kanzawa, M.; Yano, S.; Yamauchi, M.; Sugishita, T.; Chihara, K. Determinants of Bone Mineral Density and Spinal Fracture Risk in Postmenopausal Japanese Women. *Osteoporos. Int.* **2001**, *12*, 548–554. [CrossRef] [PubMed]
81. Sioka, C.; Fotopoulos, A.; Georgiou, A.; Xourgia, X.; Papadopoulos, A.; Kalef-Ezra, J.A. Age at menarche, age at menopause and duration of fertility as risk factors for osteoporosis. *Climacteric* **2010**, *13*, 63–71. [CrossRef] [PubMed]
82. Hassa, H.; Tanir, H.; Senses, T.; Öge, T.; Sahin-Mutlu, F. Related factors in bone mineral density of lumbal and femur in natural postmenopausal women. *Arch. Gynecol. Obstet.* **2005**, *273*, 86–89. [CrossRef] [PubMed]
83. Zhang, Y.-Y.; Liu, P.-Y.; Deng, H. The impact of reproductive and menstrual history on bone mineral density in Chinese women. *J. Clin. Densitom.* **2003**, *6*, 289–296. [CrossRef]
84. Ozdemir, F.; Demirbag, D.; Rodoplu, M. Reproductive Factors Affecting the Bone Mineral Density in Postmenopausal Women. *Tohoku J. Exp. Med.* **2005**, *205*, 277–285. [CrossRef] [PubMed]
85. Zhang, Z.-X.; Lei, S.-F.; Deng, F.; Zhang, F.; Liu, Y.-J.; Recker, R.R.; Papasian, C.J.; Deng, H. Bivariate genome-wide linkage analysis for traits BMD and AAM: Effect of menopause on linkage signals. *Maturitas* **2009**, *62*, 16–20. [CrossRef] [PubMed]
86. Chevalley, T.; Bonjour, J.P.; Ferrari, S.; Rizzoli, R. The Influence of Pubertal Timing on Bone Mass Acquisition: A Predetermined Trajectory Detectable Five Years before Menarche. *J. Clin. Endocrinol. Metab.* **2009**, *94*, 3424–3431. [CrossRef] [PubMed]
87. Liel, Y.; Edwards, J.; Shary, J.; Spicer, K.; Gordon, L.; Bell, N.H. The Effects of Race and Body Habitus on Bone Mineral Density of the Radius, Hip, and Spine in Premenopausal Women*. *J. Clin. Endocrinol. Metab.* **1988**, *66*, 1247–1250. [CrossRef] [PubMed]
88. Edelstein, S.L.; Barrett-Connor, E. Relation between Body Size and Bone Mineral Density in Elderly Men and Women. *Am. J. Epidemiol.* **1993**, *138*, 160–169. [CrossRef] [PubMed]

89. Fujiwara, S.; Kasagi, F.; Yamada, M.; Kodama, K. Risk Factors for Hip Fracture in a Japanese Cohort. *J. Bone Miner. Res.* **1997**, *12*, 998–1004. [CrossRef] [PubMed]
90. Roy, D.; O'Neill, T.; Finn, J.; Lunt, M.; Silman, A.; Felsenberg, D.; Armbrecht, G.; Banzer, D.; Benevolenskaya, L.; Bhalla, A.; et al. Determinants of incident vertebral fracture in men and women: Results from the European Prospective Osteoporosis Study (EPOS). *Osteoporos. Int.* **2003**, *14*, 19–26. [CrossRef] [PubMed]
91. Silman, A.J. Risk factors for Colles' fracture in men and women: Results from the European Prospective Osteoporosis Study. *Osteoporos. Int.* **2003**, *14*, 213–218. [CrossRef] [PubMed]
92. Johnell, O.; Gullberg, B.; Kanis, J.A.; Allander, E.; Elffors, L.; Dequeker, J.; Dilsen, G.; Gennari, C.; Lopes Vaz, L.A.; Lyritis, G.; et al. Risk factors for hip fracture in european women: The MEDOS study. *J. Bone Miner. Res.* **2009**, *10*, 1802–1815. [CrossRef] [PubMed]
93. Kvalheim, S.; Sandvik, L.; Winsvold, B.S.; Hagen, K.; Zwart, J.-A. Early menarche and chronic widespread musculoskeletal complaints- Results from the HUNT study. *Eur. J. Pain* **2015**, *20*, 458–464. [CrossRef]
94. Roze, C.; Doyen, C.; Le Heuzey, M.-F.; Armoogum, P.; Mouren, M.-C.; Léger, J. Predictors of late menarche and adult height in children with anorexia nervosa. *Clin. Endocrinol.* **2007**, *67*, 462–467. [CrossRef] [PubMed]
95. Must, A.; Phillips, S.; Naumova, E.; Blum, M.; Harris, S.; Dawson-Hughes, B.; Rand, W.M. Recall of Early Menstrual History and Menarcheal Body Size: After 30 Years, How Well Do Women Remember? *Am. J. Epidemiol.* **2002**, *155*, 672–679. [CrossRef] [PubMed]
96. Casey, V.; Dwyer, J.T.; Coleman, K.A.; Krall, E.A.; Gardner, J.; Valadian, I. Accuracy of recall by middle-aged participants in a longitudinal study of their body size and indices of maturation earlier in life. *Ann. Hum. Biol.* **1991**, *18*, 155–166. [CrossRef] [PubMed]
97. Żarów, R.; Cichoń, B.A. A comparative analysis of estimation of age at menarche by various methods in women participating in the Krakow Longitudinal Growth Study, Poland. *Am. J. Hum. Biol.* **2008**, *20*, 146–148. [CrossRef]
98. Cooper, R.; Blell, M.; Hardy, R.; Black, S.; Pollard, T.M.; Wadsworth, M.E.J.; Pearce, M.S.; Kuh, D. Validity of age at menarche self-reported in adulthood. *J. Epidemiol. Commun. Health* **2006**, *60*, 993–997. [CrossRef]
99. Mostafavifar, A.M.; Best, T.M.; Myer, G.D. Early sport specialisation, does it lead to long-term problems? *Br. J. Sports Med.* **2013**, *47*, 1060–1061. [CrossRef]
100. Mathisen, F.K.S.; Kokko, S.; Tynjälä, J.; Torsheim, T.; Wold, B. Leisure-time physical activity and participation in organized sports: Changes from 1985 to 2014 in Finland and Norway. *Scand. J. Med. Sci. Sports* **2019**, *29*, 1232–1242. [CrossRef]
101. Myer, G.D.; Jayanthi, N.; DiFiori, J.P.; Faigenbaum, A.D.; Kiefer, A.W.; Logerstedt, D.; Micheli, L.J. Sport Specialization, Part I: Does Early Sports Specialization Increase Negative Outcomes and Reduce the Opportunity for Success in Young Athletes? *Sports Health* **2015**, *7*, 437–442. [CrossRef] [PubMed]
102. Sundgot-Borgen, J. Risk and trigger factors for the development of eating disorders in female elite athletes. *Med. Sci. Sports Exerc.* **1994**, *26*, 414–419. [CrossRef] [PubMed]
103. Rauh, M.J.; Tenforde, A.S.; Barrack, M.T.; Rosenthal, M.D.; Nichols, J.F. Associations between Sport Specialization, Running-Related Injury, and Menstrual Dysfunction Among High School Distance Runners. *Athl. Train. Sports Health Care* **2018**, *10*, 260–269. [CrossRef]

Publisher's Note: MDPI stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.



© 2020 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<http://creativecommons.org/licenses/by/4.0/>).

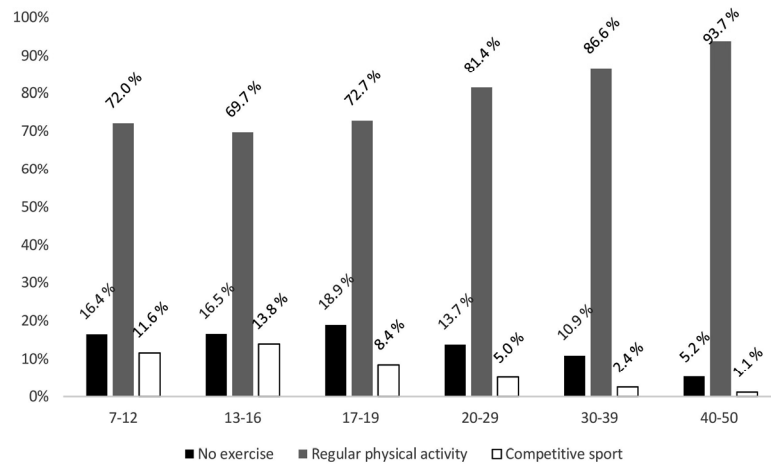


Figure S1. Proportions of the participants in different physical activity level groups across the age categories.

Table S1. Age at menarche among participants ($n = 1081$).

Age at menarche	Number of participants	Proportion of participants
9	2	0.2%
10	15	1.4%
11	113	10.5%
12	261	24.1%
13	377	34.9%
14	192	17.8%
15	81	7.5%
16	30	2.8%
17	8	0.7%
18	1	0.1%
19	1	0.1%

Table S2. Proportion (n) of the participants reporting fractures, musculoskeletal disorders/symptoms, and anorexia nervosa diagnosis according to physical activity (PA) level at the age of 13–16.

	Competitive sport ($n = 136$)	Regular PA ($n = 689$)	No exercise ($n = 163$)	p -value
Fracture	17.6% (24)	15.5% (107)	17.2% (28)	0.761
Musculoskeletal disorder/symptom	43.4% (59)	37.4% (258)	31.9% (52)	0.123
Anorexia nervosa	0.7% (1)	0.6% (4)	0.0% (0)	0.641

Data are presented as percentages (frequencies).

Table S3. Proportion (n) of the participants reporting fractures, musculoskeletal disorders/symptoms, and anorexia nervosa diagnosis according to age at menarche (AAM) groups.

	AAM ≤ 12 ($n = 391$)	AAM = 13 ($n = 377$)	AAM ≥ 14 ($n = 313$)	p -value
Fracture	16.6% (65)	17.2% (65)	14.4% (45)	0.571
Musculoskeletal disorder/symptom	36.6% (143)	37.9% (143)	38.0% (119)	0.901
Anorexia nervosa	0.0% (0) ^a	0.5% (2) ^b	1.3% (4)	0.069

Data are presented as percentages (frequencies). ^a $n = 390$; ^b $n = 376$.



III

SELF-REPORTED RESTRICTIVE EATING, EATING DISORDERS, MENSTRUAL DYSFUNCTION, AND INJURIES IN ATHLETES COMPETING AT DIFFERENT LEVELS AND SPORTS

by

Ravi, S., Ihalainen, J. K., Taipale-Mikkonen, R. S., Kujala, U. M., Waller, B.,
Mierlahti, L., Lehto, J., & Valtonen, M. 2021





Nutrients, 13(9), Article 3275

<https://doi.org/10.3390/nu13093275>

Reproduced with kind permission by MDPI.

Article

Self-Reported Restrictive Eating, Eating Disorders, Menstrual Dysfunction, and Injuries in Athletes Competing at Different Levels and Sports

Suvi Ravi ^{1,*}, Johanna K. Ihalainen ², Ritva S. Taipale-Mikkonen ³, Urho M. Kujala ¹, Benjamin Waller ⁴, Laura Mierlahti ⁵, Johanna Lehto ⁶ and Maarit Valtonen ⁶

¹ Faculty of Sport and Health Sciences, University of Jyväskylä, 40014 Jyväskylä, Finland; urho.m.kujala@jyu.fi

² Neuromuscular Research Center, Biology of Physical Activity, Faculty of Sport and Health Sciences, University of Jyväskylä, 40014 Jyväskylä, Finland; johanna.k.ihalainen@jyu.fi

³ Sports Technology Unit, Faculty of Sport and Health Sciences, University of Jyväskylä, 88610 Vuokatti, Finland; ritva.s.taipale@jyu.fi

⁴ Sport and Health Research Centre, Sports Science Department, School of Social Sciences, Physical Activity, Physical Education, Reykjavik University, 102 Reykjavik, Iceland; ben.waller@jamk.fi

⁵ Paavo Nurmi Centre & Unit for Health and Physical Activity, University of Turku, 20520 Turku, Finland; laura.mierlahti@utu.fi

⁶ Research Institute for Olympic Sports, 40700 Jyväskylä, Finland; johanna.lehto@kihu.fi (J.L.); maarit.valtonen@kihu.fi (M.V.)

* Correspondence: suvi.m.ravi@jyu.fi; Tel.: +358-406707282



Citation: Ravi, S.; Ihalainen, J.K.; Taipale-Mikkonen, R.S.; Kujala, U.M.; Waller, B.; Mierlahti, L.; Lehto, J.; Valtonen, M. Self-Reported Restrictive Eating, Eating Disorders, Menstrual Dysfunction, and Injuries in Athletes Competing at Different Levels and Sports. *Nutrients* **2021**, *13*, 3275. <https://doi.org/10.3390/nu13093275>

Academic Editor: Andrew John Hill

Received: 17 August 2021

Accepted: 16 September 2021

Published: 19 September 2021

Publisher's Note: MDPI stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.



Copyright: © 2021 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<https://creativecommons.org/licenses/by/4.0/>).

Abstract: The purpose of this study was to investigate the prevalence of self-reported restrictive eating, current or past eating disorder, and menstrual dysfunction and their relationships with injuries. Furthermore, we aimed to compare these prevalences and associations between younger (aged 15–24) and older (aged 25–45) athletes, between elite and non-elite athletes, and between athletes competing in lean and non-lean sports. Data were collected using a web-based questionnaire. Participants were 846 female athletes representing 67 different sports. Results showed that 25%, 18%, and 32% of the athletes reported restrictive eating, eating disorders, and menstrual dysfunction, respectively. Higher rates of lean sport athletes compared with non-lean sport athletes reported these symptoms, while no differences were found between elite and non-elite athletes. Younger athletes reported higher rates of menstrual dysfunction and lower lifetime prevalence of eating disorders. Both restrictive eating (OR 1.41, 95% CI 1.02–1.94) and eating disorders (OR 1.89, 95% CI 1.31–2.73) were associated with injuries, while menstrual dysfunction was associated with more missed participation days compared with a regular menstrual cycle (OR 1.79, 95% CI 1.05–3.07). Our findings indicate that eating disorder symptoms and menstrual dysfunction are common problems in athletes that should be managed properly as they are linked to injuries and missed training/competition days.

Keywords: female athlete; eating disorder; disordered eating; menstrual irregularity; sports injury

1. Introduction

Athletes may feel pressure to alter their weight in order to improve performance or subjective appearance, which can lead to dieting, restrictive and disordered eating, or even a clinical eating disorder [1]. Indeed, attitudes toward eating and body image can be seen as a continuum from energy balance and healthy body image to disordered eating, culminating with a clinical eating disorder, such as anorexia nervosa or bulimia nervosa [2]. Disordered eating is defined as abnormal eating behaviors, including, for example, restrictive eating, fasting, skipping meals, and vomiting [3]. Clinical eating disorders and disordered eating, especially restrictive eating, are often associated with low energy availability, which may consequently result in suppression of the hypothalamus–pituitary–ovarian axis and further lead to menstrual dysfunction [3].

Disordered eating, eating disorders, and menstrual dysfunction are common among female athletes [4–6]. For example, De Souza and colleagues [5] reported that the prevalence of disordered eating in female collegiate and elite athletes varies from 6% to 42%, while eating disorder prevalence among female athletes ranges from 2% to 33% [4]. Furthermore, the prevalence of menstrual dysfunction in female athletes is reported to vary from 9% to 60% [6]. Meanwhile, the prevalence of disordered eating, eating disorders, and menstrual dysfunction appears to depend on the type of sport, with athletes competing in sports emphasizing leanness being at higher risk than non-lean sport athletes [3,7,8]. Relatively few studies have, however, investigated if the level of sport participation is linked to disordered eating or menstrual dysfunction prevalence among athletes. Existing evidence suggests that elite athletes exhibit higher levels of eating disorder symptoms compared to non-elite athletes or to physically active females that are not competing [9].

Participation in competitive sport is associated with multiple health benefits but also with injury risk [10]. Injuries have been found to be more common in adolescent athletes with disordered eating than among those with healthy attitudes toward body image and eating [11–13]. For example, Thein-Nissenbaum et al. [12] reported that athletes with disordered eating had an odds ratio (95% confidence interval) of 2.3 (1.4–4.0) for an injury (overuse and traumatic injuries combined) compared to athletes with no disordered eating. Furthermore, Scheid and Stefanik [11] found that athletes with a high drive for thinness (i.e., disordered eating) had a 69% increase in the number of injuries compared with those reporting a low drive for thinness. However, few studies have investigated the issue in athletes above adolescent age. Recently, Ackerman et al. [14] found no relationship between disordered eating and injury rate in 15–30 year old athletes (mean 18.9 years, $SD \pm 3.3$), although endurance athletes with disordered eating (assessed by questionnaires) trended toward higher injury rates than endurance athletes without disordered eating.

Studies investigating menstrual dysfunction and musculoskeletal injuries in athletes have produced conflicting results [12,15–20]. While Rauh et al. [17] found that athletes with menstrual dysfunction had higher odds of injuries (OR 2.9, 95% CI 1.4–6.1) than eumenorrheic athletes, other studies have found no association between menstrual dysfunction and injuries [12,15,16,19]. In some cases, menstrual dysfunction has even been associated with fewer injuries, as von Rosen et al. [20] found that athletes with menstrual dysfunction had fewer current injuries than those with a regular menstrual cycle (21.9% vs. 38%, $p = 0.024$).

To understand the role of the participation level, age, and type of sport on eating problems and menstrual dysfunction, and to evaluate their relationships with injuries, the aim of this study was twofold. First, we aimed to determine the prevalence of self-reported (i) restrictive eating, (ii) current or past eating disorder, and (iii) menstrual dysfunction in a group of female athletes, and to compare the prevalence of these issues in (i) elite and non-elite athletes, (ii) younger and older athletes, and (iii) lean and non-lean sport athletes. Secondly, our purpose was to investigate if there is a relationship of (i) restrictive eating, (ii) current or past eating disorder, and/or (iii) menstrual dysfunction with injuries in this group of athletes and to explore if these associations are different (i) in elite and non-elite athletes, (ii) in younger and older athletes, or (iii) in athletes competing in lean and non-lean sports.

2. Materials and Methods

2.1. Study Design

The current study was part of a larger cross-sectional survey-based study implemented using an online questionnaire constructed with Webropol 3.0 Online Survey and Reporting Tool (Webropol Oy 2020, Helsinki, Finland). The questionnaire gathered data from six areas: (i) participant characteristics, (ii) low energy availability-related issues (i.e., injuries, gastrointestinal function, and menstrual cycle) using the Low Energy Availability in Females Questionnaire (LEAF-Q) [21], (iii) disordered eating (including restrictive eating) and eating disorders using the female athlete triad screening questionnaire [22], (iv) menstrual cycle function and hormonal contraception, (v) athletes' perception of the

effects of menstrual cycle on performance, and (vi) communication around menstrual cycle. This study primarily used data on disordered eating/eating disorders, menstrual function, and injuries. The questionnaire was tested in a small group of athletes before data collection, and only minor changes (i.e., changes in the order of the questions and a few misspellings) were made on the basis of their feedback.

2.2. Recruitment Strategy and Participants

A link to the questionnaire was promoted by the Finnish Olympic Committee, national sports federations, and sports academies, and it was also distributed via social media (e.g., Twitter, Instagram). The survey was available between May 2020 and August 2020.

All female athletes at least 15 years of age were eligible for participation in the study, regardless of the participation level. The questionnaire was offered only in Finnish. The study was evaluated by the Ethical Committee of University of Jyväskylä. Before filling in the questionnaire, participants were informed about the aims of the study and the content of the questionnaire. They were informed that participation was voluntary and that they were free to drop out or leave questions unanswered at any point. No personal identification information was collected; however, participants were given the opportunity to provide their email address in order to give permission for further contact by the research group.

Initially, 926 athletes filled in the questionnaire. Thirty-two questionnaires were excluded due to incomplete data (responders answered only questions concerning menstrual function). For the purpose of the current study, all athletes over 45 years of age ($n = 23$) or those who did not report their age ($n = 9$) were excluded in an attempt to include only premenopausal women. In addition, those who did not provide information on their sports discipline ($n = 7$) or participation level ($n = 9$) were excluded. The final sample size of the current study was 846. Participants represented 67 different sports (Table S1).

2.3. Data Collection

Participants reported their participation level by choosing one of the following options: recreational athlete, regional/district-level athlete, national-level athlete, or international-level athlete. Recreational athletes and athletes competing at regional/district level were assigned into the non-elite athletes group and those competing at national or international level composed the elite athletes group.

Participants self-reported their year of birth, and their age was calculated as 2020 minus the year of birth. Participants were classified as younger (15–24 years old) and older athletes (25–45 years old).

Participants reported their main sports and were categorized into lean sport athletes and non-lean sport athletes. Lean sport athletes competed in sports where leanness or weight are considered important (endurance, aesthetic, weight class, and antigravitation sports) and non-lean sport athletes in sports where these factors are not considered to have a great impact on performance (ball games, as well as technical and power sports) [8]. For details of this sports type classification, see Table S1.

Participants' BMI was calculated from reported height and weight. In addition, participants reported their training volume as training hours in the preceding year.

Questions from the female athlete triad screening questionnaire (Mountjoy et al., 2015) were utilized when assessing restrictive eating and eating disorders. Participants were assigned into the restrictive eating group if they answered "yes" to the following question: "Do you limit or carefully control the foods that you eat?" They were classified into the eating disorder group if they answered "yes" to the following question: "Do you currently or have you ever suffered from an eating disorder?"

Questions from the LEAF-Q [21], as well as questions designed by our own research group, were utilized as means to assess menstrual function and hormonal contraceptive use. A participant was classified into the menstrual dysfunction group if her menstrual cycle was >35 days, she had not had periods in the preceding three months, she had had <9 periods in the preceding year, she had not yet reached menarche despite being

≥ 15 years old (primary amenorrhea) [3,23], or she reported using hormonal contraceptives because otherwise menstruation stops [24]. Participants using some form of hormonal contraceptives (oral contraceptives, implants, injections, transdermal patches, vaginal rings, or intrauterine systems) for any other reason (contraception, reduction in menstruation pains, reduction in bleeding, to regulate the menstrual cycle in relation to performance, etc.) were excluded from the analysis regarding menstrual dysfunction ($n = 344$).

Regarding injuries, participants were asked if they had had absences from training or competitions during the preceding year due to sports injuries and were classified as injured and non-injured athletes on the basis of their responses. Injured athletes were asked to report how many training or competition days they had missed in the preceding year due to injuries from the following options: 1–7 days, 8–14 days, 15–21 days, or 22 days or more [21]. Participants were assigned into two groups on the basis of their responses: those who had missed ≥ 22 and those who had missed < 22 participation days. In the current study, the definition of injury included both acute and overuse injuries.

2.4. Statistical Analysis

The continuous variables were tested for normality prior to statistical analysis. Descriptive statistics are presented as means and standard deviations (SD) for normally distributed data, medians and interquartile ranges (IQR) for non-normally distributed data, and percentages and counts for categorical data. Differences in prevalences between groups were analyzed using chi-square or Fisher's exact tests for categorical variables and two-independent-sample *t*-test or Mann–Whitney U test for continuous variables. Logistic regression analysis was used to determine crude and adjusted odds ratios (ORs) and their 95% confidence intervals (95% CIs) for the association of restrictive eating, eating disorders, and menstrual dysfunction with injury occurrence and missed participation days. Statistical analyses were conducted using IBM SPSS Statistics version 24 (Armonk, NY, USA). The significance level was set at $p < 0.05$, two-tailed.

3. Results

3.1. Prevalence of Restrictive Eating, Eating Disorders, and Menstrual Dysfunction in Different Groups of Athletes

The proportion of athletes reporting restrictive eating, current or past eating disorder, menstrual dysfunction, primary amenorrhea, injuries, and ≥ 22 missed participation days are presented in Table 1. No differences in any of the abovementioned variables between elite and non-elite athletes were found. Younger athletes reported lower rates of current or previous eating disorders than older athletes, while the prevalence of menstrual dysfunction was lower in the older athletes compared with the younger. Lean sport athletes reported more restrictive eating, eating disorders, and menstrual dysfunction than non-lean sport athletes; however, more non-lean sport athletes had sustained at least one injury during the preceding year than lean sport athletes. No differences were found in current or past primary amenorrhea between elite and non-elite athletes or between lean and non-lean athletes, but there was a trend toward higher rates of primary amenorrhea in elite athletes compared with non-elite athletes ($p = 0.08$) and in lean sport athletes compared with non-lean sport athletes ($p = 0.06$) (Table 1).

Table 1. Proportion of athletes reporting restrictive eating (RE), current or past eating disorder (ED), menstrual dysfunction (MD), current or past primary amenorrhea (PA), one or more injuries in the preceding year, and ≥ 22 missed participation days (from training or competition) due to injuries.

	All Participants	Non-Elite Athletes	Elite Athletes	<i>p</i> -Value	Odds Ratio (95% CI)	Younger Athletes	Older Athletes	<i>p</i> -Value	Odds Ratio (95% CI)	Lean Sport Athletes	Non-Lean Sport Athletes	<i>p</i> -Value	Odds Ratio (95% CI)
RE	24.6% (207/841)	24.7% (54/219)	24.6% (153/622)	0.986	1.00 (0.70–1.43)	23.5% (116/494)	26.2% (91/347)	0.363	0.86 (0.63–1.19)	26.9% (146/542)	20.4% (61/299)	0.035	1.44 (1.03–2.02)
ED	18.4% (155/843)	18.1% (40/221)	18.5% (115/622)	0.898	0.97 (0.66–1.45)	14.6% (72/494)	23.8% (83/349)	0.001	0.55 (0.39–0.78)	20.6% (112/543)	14.3% (43/300)	0.024	1.55 (1.06–2.28)
MD	31.6% (160/506)	27.0% (33/122)	33.1% (127/384)	0.213	0.75 (0.48–1.18)	35.0% (111/317)	25.9% (49/189)	0.033	1.54 (1.03–2.29)	35.6% (115/323)	24.6% (45/183)	0.010	1.70 (1.13–2.55)
PA	15.5% (129/832)	11.9% (26/219)	16.8% (103/613)	0.084	0.67 (0.42–1.06)	15.7% (77/490)	15.2% (52/342)	0.842	1.04 (0.71–1.52)	17.3% (93/538)	12.2% (36/294)	0.055	1.50 (0.99–2.27)
Injury	55.3% (468/846)	50.7% (112/221)	57.0% (356/625)	0.106	0.78 (0.57–1.06)	55.6% (276/496)	54.9% (192/350)	0.820	1.03 (0.78–1.36)	50.8% (277/545)	63.5% (191/301)	<0.001	0.60 (0.45–0.79)
≥ 22 missed days ^a	30.8% (143/464)	29.7% (33/111)	31.2% (110/353)	0.776	0.94 (0.59–1.49)	32.2% (88/273)	28.8% (55/191)	0.430	1.18 (0.79–1.760)	29.6% (81/274)	32.6% (62/190)	0.481	0.87 (0.58–1.29)

^a From injured athletes. CI = confidence interval. Statistically significant *p*-values are indicated in bold.

Athletes reporting restrictive eating did not differ from those not reporting restrictive eating in terms of BMI (median 22.5 kg/m² (IQR 20.6–24.8) vs. 22.6 kg/m² (IQR 21.0–24.3), respectively, $p = 0.741$) or training volume (median 600 (IQR 364–800) vs. 520 (IQR 390–720), respectively $p = 0.092$). Athletes reporting a current or past eating disorder had similar BMI (median 22.3 kg/m² (IQR 20.6–24.1) vs. 22.6 kg/m² (IQR 21.0–24.4), respectively, $p = 0.198$) and training hours (median 555 (IQR 366–726) vs. 540 (IQR 400–730), respectively, $p = 0.983$) to those reporting no eating disorder. Athletes reporting menstrual dysfunction had lower BMI (median 21.6 kg/m² (IQR 20.1–23.6) vs. 22.7 kg/m² (IQR 21.0–24.3), $p < 0.001$) and higher training volume (median 627 h (IQR 414–832) vs. 550 h (IQR 400–720), $p = 0.041$) than athletes reporting regular menstrual cycles. For additional descriptive data of the participants, see Table S2.

3.2. Restrictive Eating, Eating Disorders, Menstrual Dysfunction, and Injuries

Odds ratios for injury occurrence and ≥ 22 missed participation days by restrictive eating, eating disorders, and menstrual dysfunction among all participants are presented in Table 2. Athletes with restrictive eating or current or past eating disorder were more likely to report an injury than those with no restrictive eating or eating disorder. No differences were found in injury occurrence among athletes by the menstrual status, or in missed participation days by restrictive eating or eating disorder. However, athletes reporting menstrual dysfunction were more likely to report ≥ 22 missed participation days than those reporting regular menstrual cycles.

Table 2. Frequencies and proportions of athletes with one or more injury during the preceding year and athletes with ≥ 22 missed participation days due to injuries during the preceding year, along with crude odds ratios by restrictive eating (RE), eating disorders (ED), and menstrual dysfunction (MD).

Variable	<i>n</i>	One or More Injury			Variable	<i>n</i>	≥ 22 Missed Participation Days Due to Injuries ^a		
		Proportion (%)	OR	95% CI			Proportion (%)	OR	95% CI
No RE	339	53.5	1.00	Reference	No RE	104	30.8	1.00	Reference
RE	128	61.8	1.41	1.02–1.94	RE	39	31.2	1.02	0.66–1.59
No ED	362	52.6	1.00	Reference	No ED	117	32.6	1.00	Reference
ED	105	67.7	1.89	1.31–2.73	ED	26	25.0	0.69	0.42–1.13
No MD	187	54.0	1.00	Reference	No MD	50	27.3	1.00	Reference
MD	87	54.4	1.01	0.70–1.48	MD	35	40.2	1.79	1.05–3.07

^a Only injured participants were included in this analysis. OR = odds ratio; CI = confidence interval. Statistically significant differences are indicated in bold.

The association between restrictive eating and injury occurrence was seen in non-elite athletes (OR 1.90, 95% CI 1.01–3.57) and in lean sport athletes (OR 1.68, 95% CI 1.14–2.47, but not in elite, non-lean, younger, and older athletes. The association between eating disorder and injuries was observed in elite (OR 1.91, 95% CI 1.24–2.93) and non-elite athletes (OR 2.41, 95% CI 1.11–5.23), in older athletes (OR 2.46, 95% CI 1.44–4.19), and in lean (OR 1.91, 95% CI 1.24–2.93) and non-lean athletes (OR 2.41, 95% CI 1.11–5.23). The relationship between menstrual dysfunction and ≥ 22 missed participation days was observed in elite (OR 2.37, 95% CI 1.28–4.36) and lean sport athletes (OR 2.07, 95% CI 1.05–4.11).

The relationships of restrictive eating and eating disorder with one or more injuries during the preceding year among all participants still existed when adjusted for BMI, training hours in the preceding year, and age. However, no association between menstrual dysfunction and missed participation days among all athletes was found when adjusting for the abovementioned confounders (Table 3).

Table 3. Adjusted odds ratios for one or more injury during the preceding year and ≥ 22 missed training days due to injury during the preceding year by restrictive eating (RE), current or past eating disorder (ED), and menstrual dysfunction (MD).

Risk Factor	One or More Injury		Risk Factor	≥ 22 Missed Participation Days due to Injuries ^a	
	OR ^b	95% CI		OR ^b	95% CI
RE	1.50	1.05–2.14	RE	0.92	0.57–1.49
ED	2.29	1.52–3.44	ED	0.64	0.38–1.08
MD	1.10	0.72–1.67	MD	1.63	0.91–2.91

^a Only injured participants were included in this analysis; ^b Adjusted for BMI, training hours in the preceding year, and age. OR = odds ratio; CI = confidence interval. Statistically significant differences are indicated in bold.

4. Discussion

The main findings of this large survey-based study were as follows: (1) 25%, 18%, and 32% of the athletes reported restrictive eating, current or past eating disorder, and menstrual dysfunction, respectively; (2) lean sport athletes had higher rates of restrictive eating, eating disorders, and menstrual dysfunction than non-lean sport athletes, while no differences in any of these variables were seen between elite and non-elite athletes; (3) younger athletes reported higher rates of menstrual dysfunction and lower rates of current or past eating disorders than older athletes; (4) restrictive eating and eating disorders were associated with injury occurrence; (5) athletes reporting menstrual dysfunction were more likely to report more missed participation days due to injuries than athletes reporting regular menstrual cycles.

4.1. Prevalence of Restrictive Eating, Eating Disorders, and Menstrual Dysfunction

In the present study, 24.6% of the athletes reported restrictive eating, which is higher than the 11.1% reported by Hinton and Beck [25] in female collegiate athletes, but lower than the 39.6% reported by Bennell et al. [26] in female 17–26 year old track-and-field athletes. The lifetime prevalence of self-reported eating disorder in our sample was 18.1%, which is similar to the 17.9% reported by Silén et al. [27] in a Finnish female community-based sample and slightly higher than reports in French athletes (11.2%) [28]. However, the lifetime prevalence of eating disorder observed in this study was lower than self-reported current eating disorder in a sample of Norwegian athletes (27.3%) [29]. Menstrual dysfunction prevalence in the current study among all participants was 31.6%, which is lower than what we recently found in Finnish athletes aged 18–20 years (38.9%) [30], but higher than what was found in high-school athletes (18.8–20.1%) [12,31] and in Finnish athletes aged 14–16 years (17.7%) [30]. The prevalence of a current or past primary amenorrhea in the present study among all participants was 15.5%, which is higher than reported earlier [8,30]. Differences in study populations and methodologies are likely to explain the discrepancies between studies.

In the present study, there were no differences in restrictive eating or eating disorder prevalence between elite and non-elite athletes, i.e., in those competing at national or international level vs. in those competing at regional/district level, which contrasts with the findings of Kong and Harris [9] who found that elite athletes (athletes competing at international or national level and training at least 12 h per week) had higher levels of eating disorder symptoms than noncompetitive athletes or those competing at local to national level and training less than 12 h per week. The differences between our findings and those of Kong and Harris [9] may be attributed to, as with the whole sample, different methodologies and/or study populations. Our findings indicate that, in addition to elite athletes, athletes competing at lower levels are also at high risk for eating disorder symptoms.

While we did not find any relationship between menstrual dysfunction prevalence and the participation level, higher training volume was associated with menstrual dysfunction. This finding is consistent with previous studies [16,32]. However, not all studies have found this association [8]. We recently showed that self-reported weekly training volume was not associated with menstrual dysfunction in athletes aged 18–20 years; however, athletes reporting menstrual dysfunction exhibited higher rates of accelerometer-measured

moderate-to-vigorous physical activity than athletes that reported regular menstrual cycles. This association was not, however, observed in athletes aged 14–16 years [30]. The results of the present study indicate that it is not the participation level per se that is associated with menstrual dysfunction, but training volume. Higher training hours may lead to low energy availability, which has been shown to cause alterations in the hypothalamic–pituitary–ovarian axis, which may lead to menstrual dysfunction [3,33].

The older athletes in our sample reported higher rates of current or past eating disorders than the younger ones, while no differences in restrictive eating between the age groups were found. We did not ask participants at what age they received the diagnosis, nor did we differentiate between current and past eating disorders. Thus, the prevalence of a current eating disorder is not known for either group. In earlier studies, the incidence of eating disorders was shown to be highest in adolescence and early adulthood [34].

In the present study, younger athletes reported higher rates of menstrual dysfunction than older athletes, which contradicts the findings of Torstveit and Sundgot-Borgen [8], who found no difference in menstrual dysfunction prevalence in athletes or controls divided into three age groups (16–19, 20–29, and 30–39 years). However, it has been suggested that the risk of menstrual dysfunction decreases with advanced gynecological age (chronological age minus age at menarche) [35], which might explain our findings.

Our results are consistent with a study conducted by Torstveit et al. [29] showing that eating disorder prevalence is higher in lean sport athletes compared with non-lean sport athletes. In addition, eating disorder symptoms, including restrictive eating, have been shown to be higher in lean sport athletes compared with non-lean sport athletes [7,36–38], which is in accordance with our findings. Lean sport athletes are reported to have higher desire and external social pressures to be thin, as well as higher rates of body dissatisfaction than non-lean sport athletes [37,38], which may explain these findings. Furthermore, we found that menstrual dysfunction was more common in lean sport athletes compared with non-lean sport athletes, which is consistent with previous studies [8,31,39]. Lean sport athletes might be more susceptible to dieting and unhealthy body image than non-lean sport athletes as they may feel more pressures to maintain a low weight and fat percentage for aesthetic, performance, or sociocultural reasons [1] and, thus, may suffer from low energy availability and also menstrual dysfunction.

4.2. Restrictive Eating, Eating Disorders, and Injuries

In the current study, both restrictive eating and eating disorders were associated with injuries, which is in accordance with previous findings [11–13]. While previous studies have been conducted in adolescent athletes [11–13], our study showed that the association between eating disorders and injuries applies also in older athletes, which suggests that increasing age does not protect athletes from injuries associated with restrictive eating and eating disorders. With respect to restrictive eating and injuries in older athletes, as well as both restrictive eating and eating disorders and injuries in younger athletes, we may have lacked statistical power to reveal significant differences between the groups. Because we did not ask the participants if they are still suffering from an eating disorder or have already recovered from it, we cannot determine if a previous eating disorder was associated with the risk of injuries in these athletes.

4.3. Menstrual Dysfunction and Injuries

We found no relationship between menstrual dysfunction and injury occurrence, which is in contrast with the studies conducted by Rauh et al. [17,18] but in agreement with some other studies [12,15,16,19]. However, in the present study, menstrual dysfunction was associated with more missed participation days, which is in accordance with studies conducted by Beckvid Henriksson et al. [16] and Ihalainen et al. [40]. It is known that menstrual dysfunction often results from low energy availability in athletes [3]. Indeed, athletes with menstrual dysfunction in the present study had lower BMI, although median BMI was still within the normal range (i.e., 21.6 kg/m²), and they trained more than

participants reporting regular menstrual cycles. Thus, it is possible that athletes with menstrual dysfunction in the present study might have had lower energy availability than those with regular cycles. Furthermore, the association between menstrual dysfunction and more missed participation days was observed in elite and lean sport athletes who also trained more and were leaner than non-elite and non-lean sport athletes. This and the fact that no association between menstrual dysfunction and missed participation days was found when adjusting for BMI and training hours might indicate that BMI and training hours mediate the association between those variables.

4.4. Limitations

Our study was not without limitations. First, as this was a cross-sectional study, any causal inferences could not be made. Second, we used self-reported data instead of clinical examination, whereas nuance and details regarding restrictive eating and eating disorders were not examined. Third, there was a possibility of self-selection bias, which could have affected our findings. Fourth, we were not able to provide a response rate as we used an open survey, and it is not known how many athletes were reached. Lastly, our study did not include a non-athletic control group and, thus, we could not compare the prevalence of restrictive eating, eating disorders, and menstrual dysfunction in athletes to nonathletes. It should also be noted that the data were collected during the COVID-19 pandemic and restrictions. We cannot exclude the possibility that this influenced some of the answers given by the participants. Despite these limitations, our large sample size and utilization of validated questionnaires enabled us to show the prevalences of restrictive eating, eating disorders, and menstrual dysfunction and their relationships with injuries in different-aged Finnish athletes competing at different levels and sports.

5. Conclusions

The prevalence of self-reported restrictive eating, current or previous eating disorder, and menstrual dysfunction in a sample of 15–45 years old Finnish elite and non-elite athletes was 25%, 18%, and 32%, respectively. Lean sport athletes reported higher levels of those symptoms than non-lean sport athletes, while younger athletes (aged 15–24 years) exhibited lower prevalence of current or previous eating disorder and higher prevalence of menstrual dysfunction than older athletes (aged 25–45 years). We found no differences between elite and non-elite athletes regarding restrictive eating, eating disorders, or menstrual dysfunction. Restrictive eating and current or past eating disorder were associated with injury occurrence, and athletes with menstrual dysfunction reported more missed participation days due to injuries than regularly menstruating athletes. Athletes and those working with them should understand the role of eating disorder symptoms and menstrual dysfunction in injury risk, in addition to other consequences for health and performance that have been associated with eating disorders/disordered eating and menstrual dysfunction [3,41]. Eating disorder symptoms and menstrual dysfunction should be taken seriously, and athletes presenting with these symptoms should receive medical evaluation and appropriate treatment. In addition, an athlete's readiness to participate in sports should be evaluated [41]. Our results can also motivate athletes to seek help for problems with eating and menstrual cycle as our findings indicate that these problems are associated with more injuries/fewer training days.

Supplementary Materials: The following are available online at <https://www.mdpi.com/article/10.3390/nu13093275/s1>: Table S1. List of sports reported by the athletes; Table S2. Characteristics of the participants and comparisons between the athletes classified by their competition level, age, and type of sport.

Author Contributions: Conceptualization, S.R. and J.K.I.; methodology, S.R.; formal analysis, S.R.; investigation, J.K.I. and R.S.T.-M.; resources, J.K.I. and R.S.T.-M.; data curation, J.K.I.; writing—original draft preparation, S.R.; writing—review and editing, S.R., J.K.I., R.S.T.-M., U.M.K., B.W., L.M., J.L., and M.V.; visualization, S.R.; supervision, J.K.I., R.S.T.-M., U.M.K., B.W., and M.V.; project

administration, J.K.I.; funding acquisition, J.K.I. All authors read and agreed to the published version of the manuscript.

Funding: This research was funded by Urheiluoopistosäätiö, grant number 20190110.

Institutional Review Board Statement: The study was conducted according to the guidelines of the Declaration of Helsinki and evaluated by the Ethical Committee of University of Jyväskylä (06/2020).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: Data presented in this study are available on reasonable request from the corresponding author.

Acknowledgments: The authors want to thank all the athletes participating in this study. Furthermore, the authors want to express their gratitude to the Finnish Olympic Committee and all the national sports federations and sports academies who promoted the study to athletes.

Conflicts of Interest: The authors declare no conflict of interest.

References

1. Sundgot-Borgen, J.; Torstveit, M.K. Aspects of disordered eating continuum in elite high-intensity sports. *Scand. J. Med. Sci. Sports* **2010**, *20* (Suppl. 2), 112–121. [CrossRef]
2. Shisslak, C.M.; Crago, M.; Estes, L.S. The Spectrum of Eating Disturbances. *Int. J. Eat. Disord.* **1995**, *18*, 209–219. [CrossRef]
3. Nattiv, A.; Loucks, A.B.; Manore, M.M.; Sanborn, C.F.; Sundgot-Borgen, J.; Warren, M.P. American College of Sports Medicine Position Stand. The Female Athlete Triad. *Med. Sci. Sports Exerc.* **2007**, *39*, 1867–1882. [CrossRef]
4. Bratland-Sanda, S.; Sundgot-Borgen, J. Eating disorders in athletes: Overview of prevalence, risk factors and recommendations for prevention and treatment. *Eur. J. Sport Sci.* **2013**, *13*, 499–508. [CrossRef] [PubMed]
5. De Souza, M.J.; Koltun, K.J.; Etter, C.V.; Southmayd, E.A. Current Status of the Female Athlete Triad: Update and Future Directions. *Curr. Osteoporos. Rep.* **2017**, *15*, 577–587. [CrossRef] [PubMed]
6. Gibbs, J.; Williams, N.I.; DE Souza, M.J. Prevalence of Individual and Combined Components of the Female Athlete Triad. *Med. Sci. Sports Exerc.* **2013**, *45*, 985–996. [CrossRef] [PubMed]
7. Mancine, R.P.; Gusfa, D.W.; Moshrefi, A.; Kennedy, S.F. Prevalence of disordered eating in athletes categorized by emphasis on leanness and activity type—A systematic review. *J. Eat. Disord.* **2020**, *8*, 1–9. [CrossRef]
8. Torstveit, M.K.; Sundgot-Borgen, J. Participation in leanness sports but not training volume is associated with menstrual dysfunction: A national survey of 1276 elite athletes and controls. *Br. J. Sports Med.* **2005**, *39*, 141–147. [CrossRef]
9. Kong, P.; Harris, L.M. The Sporting Body: Body Image and Eating Disorder Symptomatology Among Female Athletes from Leanness Focused and Nonleanness Focused Sports. *J. Psychol.* **2014**, *149*, 141–160. [CrossRef]
10. Maffulli, N.; Longo, U.G.; Gougoulas, N.; Caine, D.; Denaro, V. Sport injuries: A review of outcomes. *Br. Med. Bull.* **2010**, *97*, 47–80. [CrossRef]
11. Scheid, J.L.; Stefanik, M.E. Drive for Thinness Predicts Musculoskeletal Injuries in Division II NCAA Female Athletes. *J. Funct. Morphol. Kinesiol.* **2019**, *4*, 52. [CrossRef]
12. Thein-Nissenbaum, J.M.; Rauh, M.J.; Carr, K.E.; Loud, K.J.; McGuine, T.A. Associations Between Disordered Eating, Menstrual Dysfunction, and Musculoskeletal Injury Among High School Athletes. *J. Orthop. Sports Phys. Ther.* **2011**, *41*, 60–69. [CrossRef]
13. Thomas, J.J.; Keel, P.K.; Heatherton, T.F. Disordered eating and injuries among adolescent ballet dancers. *Eat. Weight. Disord.-Stud. Anorex. Bulim. Obes.* **2011**, *16*, e216–e222. [CrossRef] [PubMed]
14. Ackerman, K.E.; Holtzman, B.; Cooper, K.M.; Flynn, E.F.; Bruinvels, G.; Tenforde, A.S.; Popp, K.L.; Simpkin, A.; Parziale, A.L. Low energy availability surrogates correlate with health and performance consequences of Relative Energy Deficiency in Sport. *Br. J. Sports Med.* **2018**, *53*, 628–633. [CrossRef]
15. Beals, K.A.; Manore, M.M. Disorders of the female athlete triad among collegiate athletes. *Int. J. Sport Nutr. Exerc. Metab.* **2002**, *12*, 281–293. [CrossRef] [PubMed]
16. Henriksson, G.B.; Schnell, C.; Hirschberg, A.L. Women Endurance Runners with Menstrual Dysfunction Have Prolonged Interruption of Training due to Injury. *Gynecol. Obstet. Investig.* **2000**, *49*, 41–46. [CrossRef]
17. Rauh, M.J.; Nichols, J.F.; Barrack, M.T. Relationships Among Injury and Disordered Eating, Menstrual Dysfunction, and Low Bone Mineral Density in High School Athletes: A Prospective Study. *J. Athl. Train.* **2010**, *45*, 243–252. [CrossRef]
18. Rauh, M.J.; Barrack, M.; Nichols, J.F. Associations between the female athlete triad and injury among high school runners. *Int. J. Sports Phys. Ther.* **2014**, *9*, 948–958. [PubMed]
19. Tenforde, A.S.; Sayres, L.C.; McCurdy, M.L.; Collado, H.; Sainani, K.L.; Fredericson, M. Overuse Injuries in High School Runners: Lifetime Prevalence and Prevention Strategies. *PM&R* **2011**, *3*, 125–131. [CrossRef]
20. Von Rosen, P.; Heijne, A.; Frohm, A.; Fridén, C. Menstrual irregularity and use of oral contraceptives in female adolescent athletes in Swedish National Sports High Schools. *Int. J. Adolesc. Med. Health* **2017**, *32*. [CrossRef]

21. Melin, A.; Tornberg, A.B.; Skouby, S.O.; Faber, J.; Ritz, C.; Sjödin, A.; Sundgot-Borgen, J. The LEAF questionnaire: A screening tool for the identification of female athletes at risk for the female athlete triad. *Br. J. Sports Med.* **2014**, *48*, 540–545. [CrossRef] [PubMed]
22. Mountjoy, M.; Hutchinson, M.; Cruz, L.; Lebrun, C. Female Athlete Triad screening questionnaire. Female athlete Triad coalition. 2015.
23. Elliott-Sale, K.J.; Minahan, C.L.; de Jonge, X.A.K.J.; Ackerman, K.E.; Sipilä, S.; Constantini, N.W.; Lebrun, C.M.; Hackney, A.C. Methodological Considerations for Studies in Sport and Exercise Science with Women as Participants: A Working Guide for Standards of Practice for Research on Women. *Sports Med.* **2021**, *51*, 843–861. [CrossRef] [PubMed]
24. Koltun, K.J.; Williams, N.I.; De Souza, M.J. Female Athlete Triad Coalition cumulative risk assessment tool: Proposed alternative scoring strategies. *Appl. Physiol. Nutr. Metab.* **2020**, *45*, 1324–1331. [CrossRef] [PubMed]
25. Hinton, P.; Beck, N.C. Nutrient intakes of men and women collegiate athletes with disordered eating. *J. Sports Sci. Med.* **2005**, *4*, 253–262. [PubMed]
26. Bennell, K.L.; Malcolm, S.A.; Thomas, S.; Ebeling, P.R.; McCrory, P.R.; Wark, J.D.; Brukner, P.D. Risk Factors for Stress Fractures in Female Track-and-Field Athletes: A Retrospective Analysis. *Clin. J. Sport Med.* **1995**, *5*, 229–235. [CrossRef]
27. Silén, Y.; Sipilä, P.N.; Raevuori, A.; Mustelin, L.; Marttunen, M.; Kaprio, J.; Keski-Rahkonen, A. DSM-5 eating disorders among adolescents and young adults in Finland: A public health concern. *Int. J. Eat. Disord.* **2020**, *53*, 790–801. [CrossRef]
28. Schaal, K.; Tafflet, M.; Nassif, H.; Thibault, V.; Pichard, C.; Alcotte, M.; Guillet, T.; El Helou, N.; Berthelot, G.; Simon, S.; et al. Psychological Balance in High Level Athletes: Gender-Based Differences and Sport-Specific Patterns. *PLoS ONE* **2011**, *6*, e19007. [CrossRef]
29. Torstveit, M.K.; Rosenvinge, J.H.; Sundgot-Borgen, J. Prevalence of eating disorders and the predictive power of risk models in female elite athletes: A controlled study. *Scand. J. Med. Sci. Sports* **2007**, *18*, 108–118. [CrossRef]
30. Ravi, S.; Waller, B.; Valtonen, M.; Villberg, J.; Vasankari, T.; Parkkari, J.; Heinonen, O.J.; Alanko, L.; Savonen, K.; Vanhala, M.; et al. Menstrual dysfunction and body weight dissatisfaction among Finnish young athletes and non-athletes. *Scand. J. Med. Sci. Sports* **2020**, *31*, 405–417. [CrossRef]
31. Nichols, J.F.; Rauh, M.J.; Barrack, M.T.; Barkai, H.-S.; Pernick, Y. Disordered Eating and Menstrual Irregularity in High School Athletes in Lean-Build and Nonlean-Build Sports. *Int. J. Sport Nutr. Exerc. Metab.* **2007**, *17*, 364–377. [CrossRef] [PubMed]
32. Cobb, K.L.; Bachrach, L.K.; Greendale, G.; Marcus, R.; Neer, R.M.; Nieves, J.; Sowers, M.F.; Brown, B.W.; Gopalakrishnan, G.; Luetters, C.; et al. Disordered Eating, Menstrual Irregularity, and Bone Mineral Density in Female Runners. *Med. Sci. Sports Exerc.* **2003**, *35*, 711–719. [CrossRef] [PubMed]
33. Loucks, A.B.; Verdun, M.; Heath, E.M. Low energy availability, not stress of exercise, alters LH pulsatility in exercising women. *J. Appl. Physiol.* **1998**, *84*, 37–46. [CrossRef] [PubMed]
34. Jacobi, C.; Hayward, C.; De Zwaan, M.; Kraemer, H.C.; Agras, S. Coming to Terms with Risk Factors for Eating Disorders: Application of Risk Terminology and Suggestions for a General Taxonomy. *Psychol. Bull.* **2004**, *130*, 19–65. [CrossRef]
35. Williams, N.I.; Statuta, S.M.; Austin, A. Female Athlete Triad: Future Directions for Energy Availability and Eating Disorder Research and Practice. *Clin. Sports Med.* **2017**, *36*, 671–686. [CrossRef] [PubMed]
36. McDonald, A.H.; Pritchard, M.; McGuire, M.K. Self-reported eating disorder risk in lean and non-lean NCAA Collegiate Athletes. *Eat. Weight Disord.-Stud. Anorex. Bulim. Obes.* **2020**, *25*, 745–750. [CrossRef]
37. Wells, E.K.; Chin, A.D.; Tacke, J.A.; Bunn, J.A. Risk of Disordered Eating Among Division I Female College Athletes. *Int. J. Exerc. Sci.* **2015**, *8*, 256–264.
38. Reinking, M.F.; Alexander, L.E. Prevalence of Disordered-Eating Behaviors in Undergraduate Female Collegiate Athletes and Nonathletes. *J. Athl. Train.* **2005**, *40*, 47–51.
39. Beals, K.A.; Hill, A.K. The Prevalence of Disordered Eating, Menstrual Dysfunction, and Low Bone Mineral Density among US Collegiate Athletes. *Int. J. Sport Nutr. Exerc. Metab.* **2006**, *16*, 1–23. [CrossRef]
40. Ihalainen, J.K.; Kettunen, O.; McGawley, K.; Solli, G.S.; Hackney, A.C.; Mero, A.A.; Kyröläinen, H. Body Composition, Energy Availability, Training, and Menstrual Status in Female Runners. *Int. J. Sports Physiol. Perform.* **2021**, *16*, 1043–1048. [CrossRef]
41. Mountjoy, M.; Sundgot-Borgen, J.K.; Burke, L.M.; Ackerman, K.E.; Blauwet, C.; Constantini, N.; Lebrun, C.; Lundy, B.; Melin, A.; Meyer, N.L.; et al. IOC consensus statement on relative energy deficiency in sport (RED-S): 2018 update. *Br. J. Sports Med.* **2018**, *52*, 687–697. [CrossRef] [PubMed]

Supplemental material

Table S1. List of sports reported by the athletes (number of athletes in parentheses).

Lean sports (n = 545, 64.4% of the whole sample)	Non-lean sports (n = 301, 35.6% of the whole sample)
Biathlon (1)	Agility (1)
Brazilian jiu-jitsu (10)	Alpine skiing (2)
Cheerleading (43)	American football (22)
Climbing (2)	Badminton (3)
Cross-country skiing (42)	Bandy (7)
Cycling (8)	Basketball (2)
Dancing (23)	Beach volley (3)
Diving (2)	Crossfit (17)
Figure skating (51)	Disc golf (14)
Fitness (11)	Fencing (1)
Gymnastic (21)	Finnish baseball (10)
Hurdling (3)	Flag football (2)
Judo (7)	Floorball (36)
Kickboxing (1)	Futsal (2)
Middle- and long-distance running (34)	Handball (6)
Mixed martial arts (1)	Horseback riding (5)
Mushing (2)	Ice hockey (33)
Orienteering (30)	Motorsport (2)
Powerlifting (9)	Ringette (15)
Race walking (2)	Roller derby (1)
Rowing (4)	Rugby (2)
Skating (1)	Sailing (2)
Steeplechase (1)	Shooting (1)
Street workout (1)	Snowboarding (1)
Strongman (2)	Soccer (78)
Swimming (115)	Speed skating (3)
Swimrun (1)	Sprint (3)
Synchronized swimming (14)	Taido (1)
Taekwondo (6)	Tennis (1)
Thai boxing (1)	Ultimate (1)
Track and field (antigravitation sports) (17)	Underwater rugby (1)
Triathlon (24)	Volleyball (17)
Weightlifting (51)	Water polo (6)
Wrestling (4)	

Table S2. Characteristics of the participants and comparisons between the athletes classified by their competition level, age, and type of sport.

	All participants	Non-elite athletes	Elite athletes	Younger athletes	Older athletes	Lean sport athletes	Non-lean sport athletes
Age (years), mean	24.3 (7.5)	27.0 (8.6)	23.3 (6.8)	19.0 (2.6)	31.8 (5.4)	24.3 (5.8)	24.4 (6.9)
(SD)	(n = 846)	(n = 221)	(n = 625)	(n = 496)	(n = 350)	(n = 545)	(n = 301)
Height (cm), mean	167.8 (6.5)	167.3 (5.9)	168.0 (6.6)	168.2 (6.5)	167.3 (6.4)	167.4 (6.4)*	168.7 (6.6)*
(SD)	(n = 846)	(n = 221)	(n = 625)	(n = 496)	(n = 350)	(n = 545)	(n = 301)
Weight (kg), median (IQR)	64.0 (58–70) (n = 841)	64.0 (58–70) (n = 219)	64.0 (58–70) (n = 622)	63.0 (57–68)* (n = 491)	65.0 (59–72)* (n = 350)	63.0 (57–68)* (n = 544)	65.0 (61–73)* (n = 297)
BMI (kg/m ²), median (IQR)	22.6 (20.8–24.3) (n = 841)	22.8 (21.0–25.1) (n = 219)	22.5 (20.8–24.2) (n = 622)	22.5 (20.7–23.8)* (n = 491)	23.2 (21.1–25.6)* (n = 350)	22.3 (20.6–24.1)* (n = 544)	23.2 (21.6–25.4)* (n = 297)
% BMI < 18.5kg/m ²	2.6 (22) (n = 841)	2.23 (5) (n = 219)	2.7 (17) (n = 622)	3.1 (15) (n = 491)	2.0 (7) (n = 350)	3.3 (18) (n = 544)	1.3 (4) (n = 297)
Training hours ^a , median (IQR)	550 (384–728) (n = 726)	484 (250–574)* (n = 189)	600 (450–786)* (n = 537)	624 (436–850)* (n = 413)	467 (300–600)* (n = 313)	540 (390–730) (n = 484)	561 (380–728) (n = 242)

^a During the preceding year.

* Statistically significant difference ($p < 0.05$) compared with the comparison group.

SD = standard deviation; IQR = interquartile range



IV

EATING BEHAVIOURS, MENSTRUAL HISTORY, AND THE ATHLETIC CAREER: A RETROSPECTIVE SURVEY FROM ADOLESCENCE TO ADULTHOOD IN FEMALE ENDURANCE ATHLETES

by

Ravi, S., Valtonen, M., Ihalainen, J.K., Holopainen, E., Kosola, S., Heinonen,
S., Waller, B., Kujala, U.M., & Parkkari J. 2023

BMJ Open Sport & Exercise Medicine, 9(1), Article e001489

<https://doi.org/10.1136/bmjsem-2022-001489>

Reproduced with kind permission by BMJ Journals.

Eating behaviours, menstrual history and the athletic career: a retrospective survey from adolescence to adulthood in female endurance athletes

Suvi Ravi ¹, Maarit Valtonen ², Johanna K Ihalainen ¹,
Elina Holopainen ³, Silja Kosola ⁴, Saara Heinonen ¹, Ben Waller ^{5,6},
Urho M Kujala ¹, Jari Parkkari ¹

To cite: Ravi S, Valtonen M, Ihalainen JK, *et al.* Eating behaviours, menstrual history and the athletic career: a retrospective survey from adolescence to adulthood in female endurance athletes. *BMJ Open Sport & Exercise Medicine* 2023;**9**:e001489. doi:10.1136/bmjsem-2022-001489

► Additional supplemental material is published online only. To view, please visit the journal online (<http://dx.doi.org/10.1136/bmjsem-2022-001489>).

Accepted 17 February 2023



© Author(s) (or their employer(s)) 2023. Re-use permitted under CC BY-NC. No commercial re-use. See rights and permissions. Published by BMJ.

For numbered affiliations see end of article.

Correspondence to
Suvi Ravi; suvi.m.ravi@jyu.fi

ABSTRACT

Aim To evaluate differences in menstrual and pubertal history and trends in eating behaviours among women with and without a competitive sports background. Additionally, we investigated if menstrual history and eating behaviours are associated with sports career-related factors.

Methods This retrospective study was conducted on 100 women with a competitive endurance sports background and their age-matched, gender-matched and municipality-matched controls (n=98). Data were collected using a questionnaire using previously validated instruments. Generalised estimating equations were used to calculate associations of menstrual history and eating behaviours with outcome variables (career length, participation level, injury-related harms and career termination due to injury).

Results Athletes reported higher rates of delayed puberty and menstrual dysfunction than controls. No differences between the groups were observed in the Eating Disorder Examination Questionnaire short form (EDE-QS) scores at any age. Previous disordered eating (DE) was associated with current DE in both groups. Among athletes, higher EDE-QS scores during the sports career were associated with a shorter career (B=-0.15, 95% CI -0.26 to -0.05). Secondary amenorrhoea was associated with lower participation level (OR 0.51, 95% CI 0.27 to 0.95), injury-related harms during the career (OR 4.00, 95% CI 1.88 to 8.48) and career termination due to injury (OR 1.89, 95% CI 1.02 to 3.51).

Conclusion The findings indicate that DE behaviours and menstrual dysfunction, specifically secondary amenorrhoea, have a disadvantageous relationship with a sports career in women competing in endurance sports. DE during the sports career is associated with DE after the career.

INTRODUCTION

Participation in sports during adolescence is associated with mental,¹ metabolic² and bone³ health benefits later in life. Nevertheless, sports-related musculoskeletal injuries are common in athletes.⁴ While many of these

WHAT IS ALREADY KNOWN ON THIS TOPIC

- ⇒ Disordered eating behaviours and menstrual dysfunction are commonly observed in female endurance sports athletes.
- ⇒ Associations of disordered eating behaviours and menstrual function with sports career-related factors, such as length, success and injuries during the career, remain understudied.
- ⇒ Whether athletes with disordered eating behaviour during their sports career present with disordered eating after their career remains unknown.

WHAT THIS STUDY ADDS

- ⇒ Among women with a competitive sports background and a mean age of 40, higher scores on the Eating Disorder Examination Questionnaire short form during the sports career were associated with a shorter sports career.
- ⇒ Secondary amenorrhoea during the sports career was associated with lower participation level, injury-related harms and sports career termination due to injury.
- ⇒ Athletes with disordered eating during their career were more likely to report disordered eating also after their career than were athletes who did not report disordered eating during their career.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

- ⇒ Female athletes and those working with them should acknowledge that disturbances in eating behaviours and in the menstrual cycle could have a harmful relationship on an athletic career.

injuries heal completely and cause no later health problems, some may induce pain and functional limitations in later life.⁴⁻⁶

Some sports-related factors are associated with a heightened risk for disordered eating (DE) behaviours. Athletes can experience pressures to have a certain body weight or composition, and thus, restrict their eating or engage in other unhealthy eating behaviours



used to control weight.⁷ DE can result in low energy availability (LEA), that is, insufficient energy intake in relation to exercise energy expenditure. LEA, in turn, may lead to several health and performance issues in women, including menstrual dysfunction, impaired bone health, injuries and decreased performance.^{8,9} LEA, however, can also present without DE.⁹ Young athletes may be especially vulnerable to interruptions in their menstrual cycle if their energy availability is insufficient.¹⁰ LEA may also contribute to delayed puberty or primary amenorrhoea.⁹ However, the long-term consequences of LEA/DE and menstrual dysfunction in young female athletes remain poorly explored.

Findings regarding the prevalence of subclinical DE behaviours and clinical eating disorders (ED) in athletes and non-athletes are inconsistent.^{11–14} The risk of DE/ED seems to be higher among lean sports (ie, sports where weight and/or leanness are considered important) than among non-lean sports athletes.¹¹ Whether individuals with a competitive (lean) sports background differ from their non-athletic peers after the sports career regarding DE behaviours remains unreported.

This study aimed to compare menstrual and pubertal history and trends in eating behaviours between women with a competitive sports background and their non-athletic peers. The second aim was to investigate associations of menstrual history and eating behaviours during the sports career with career length, participation level, sports injury-related harms and career termination due to sports injury in women who had engaged in competitive endurance sports in their adolescence.

MATERIALS AND METHODS

This is an observational study with retrospectively collected questionnaire data.

Participants

Inclusion criteria for athletes were as follows: (1) placement among the top 30 in cross-country running national championships in the division for girls aged 16 or 17 between the years 1990–2005 or (2) placement among the top 10 in adolescents' track and field national championships in 2000 metre running in the division for girls aged 14 or 15 between the years 1992–2003. Athletes' names and dates of birth were collected from the result lists, and their addresses were drawn from the population registry. Two controls, matched for sex, age and municipality, were randomly selected for each available athlete from the general population. The final number of invited athletes and controls were 270 and 551, respectively. Controls who reported a history of competing in sports were excluded from the analyses. The flow chart of the recruitment process is presented in figure 1.

Data collection

Questionnaire data were collected using the Webropol V.3.0 Online Survey and reporting tool (Webropol Oy 2022, Helsinki, Finland) between May 2022 and July

2022. The research team developed the questionnaire. It consisted of questions on demographics, menstrual and pubertal history and eating behaviours at different age stages along with sports career and injuries (only athletes). Questions from previously validated questionnaires^{15,16} concerning eating behaviours and menstrual function, along with other previously used questions,^{17–19} were included in the questionnaire. Current and previous DE behaviours were assessed using the Eating Disorder Examination Questionnaire (EDE-Q) short form (EDE-QS), which is a 12-item version of the 28-item EDE-Q²⁰ and has demonstrated high internal consistency (Cronbach's alpha=0.913) and convergent validity with the EDE-Q both among people with ($r=0.91$) and without ($r=0.82$) an ED.¹⁵ EDE-QS captures respondents' DE behaviours and perceptions, such as unhealthy weight management behaviours (eg, dietary restriction or vomiting), lack of control while eating and body weight and shape dissatisfaction on a 4-point scale. Total scores of the EDE-QS range from 0 to 36, with higher scores indicating more unhealthy attitudes toward eating and body image. In this study, the participants were asked to complete the EDE-QS several times and to recall their thinking at different age periods, that is, at ages 13–15, 16–18, 19–21, 22–25 and at present. EDE-QS scores were calculated for each age period. A score of ≥ 15 was used as a cut-off for DE behaviour.²¹ The Cronbach's alphas in this study among athletes and controls at different ages varied between 0.91 and 0.94.

Participants were also asked if they were ever diagnosed with an ED by a medical doctor. In the case of an ED, participants provided the exact diagnosis and year of diagnosis.

Questions concerning menarche and menstrual history were developed by modifying the Low Energy Availability in Females Questionnaire.¹⁶ Athletes reported on their menstrual cycle length and regularity during their sports career, while controls were asked to answer the menstrual cycle-related questions throughout their whole life. Participants were assigned to the secondary amenorrhoea group if they reported missing periods for at least three consecutive months for any reason other than pregnancy or hormonal contraceptive use.²² For primary amenorrhoea, age at menarche ≥ 16 years was used as a criterion because this criterion was effective when study participants were adolescents.^{9,23} Participants were also asked if they were examined by a medical doctor or other medical personnel due to delayed puberty.

The effect of injuries and/or pain on the sports career (ie, injury-related harms) was assessed by asking the athletes if injury or musculoskeletal pain hindered their sports career. The response options were as follows: (1) not at all or very little: I usually did not have to skip competitions or pause normal training because of injury or pain, (2) somewhat: I sometimes had to skip competitions and/or pause normal training because of injury or pain, (3) quite a lot: I regularly had to skip competitions and/or pause normal training for a long time because

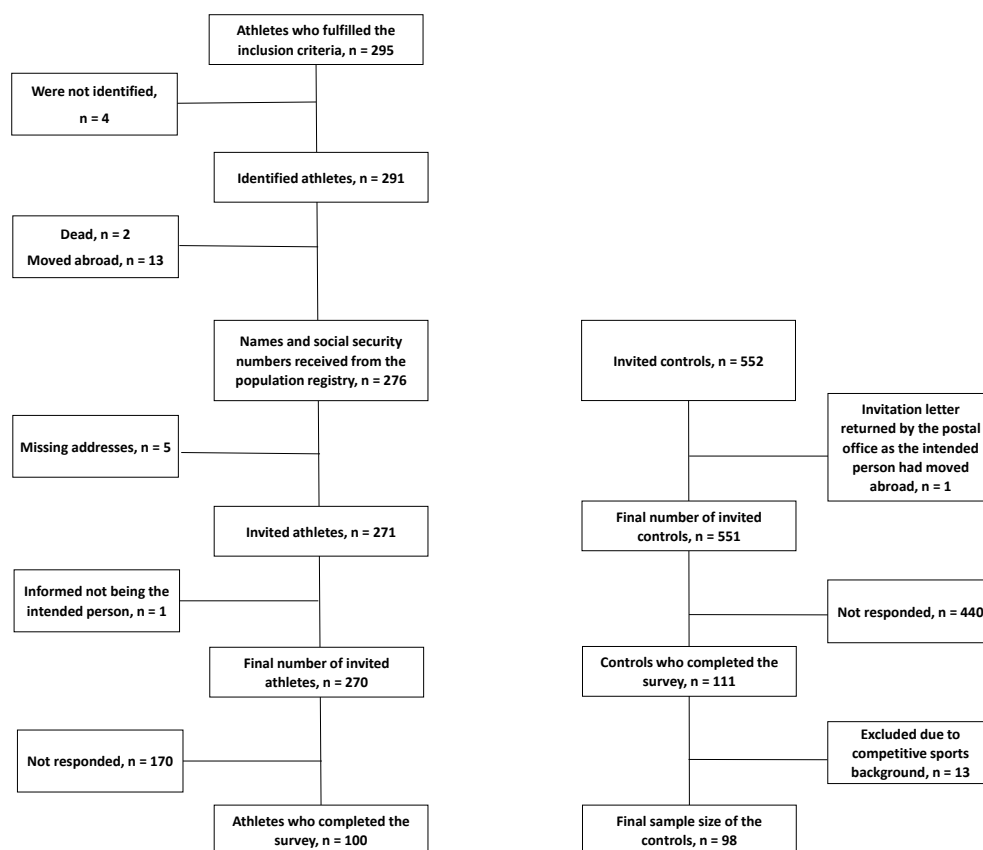


Figure 1 Flow chart of the recruitment process.

of injury or pain and (4) significant amount: I often had to skip competitions and pause training for a long time because of injury or pain. Athletes were classified into two groups based on their responses (response options 1 and 2 vs response options 3 and 4).

Retired athletes were asked if any specific injury or several injuries impacted their decision to terminate their sports career at a level they had practised and actively competed in.¹⁷ Response options were: (1) not at all, (2) an injury or several injuries contributed to sports career termination and (3) an injury or several injuries was/were the main cause for terminating the sports career. Response options 2 or 3 were combined for analyses.

Athletes also reported if an acute or overuse injury impacted their decision to terminate their sports career. An acute injury was defined as an injury that occurs suddenly or accidentally, interrupting training or ability to compete or causing an identifiable trauma. An overuse injury was defined as an injury that causes worsening pain during or after exercise without any noticeable external cause of injury.¹⁷ In addition, athletes provided information on the type of career-ending injury/injuries, which

were then categorised by tissue, as suggested by Bahr *et al.*²⁴ Athletes were also asked when they started goal-oriented training (at least twice weekly) in their sports and ended their sports career at the highest level they had competed in.¹⁷ Career length for each participant was calculated by subtracting the starting age from the age at career termination or the current age if an athlete was still active.

Athletes reported the highest level they had competed at and were grouped into national and international level athletes (Tiers 3 and 4), as recently recommended by McKay and colleagues.²⁵ One athlete reported that her highest competition level had been at the regional/district level, and she was combined with the national-level athletes in the analysis.

Statistical analyses

Descriptive statistics are presented as means with SD and counts with percentages. Differences between the athletes and the controls were analysed with a t-test (normal distribution) and Mann-Whitney U test (skewed distribution) or χ^2 test in case of categorical variables. Longitudinal

analyses for the EDE-QS scores were performed using Friedman's analysis of variance with post hoc pairwise comparisons. Associations between menstrual history, EDE-QS scores and sports career-related outcomes were tested using generalised estimating equations (GEE). The models used primary amenorrhoea, secondary amenorrhoea, EDE-QS scores and age phase, that is, time, as independent variables. Missing data were not imputed. In the primary analysis, only EDE-QS scores at the ages of each individual's sports career were considered. As 75% of the athletes had terminated their sports career by the age of 23, only ages 13–15, 16–18 and 19–21 were included in the models. The secondary analysis used EDE-QS scores at ages 13–15, 16–18 and 19–21 from all athletes.

Statistical analyses were conducted with IBM SPSS Statistics V.26 (Armonk, New York, USA) and R Project for Statistical Computing V.4.0.2 (Vienna, Austria). The significance level was set at 0.05.

Patient and public involvement

The questionnaire was piloted among former athletes and non-athletes before data collection, and small changes to the questionnaire were made based on their feedback.

RESULTS

Response rate and characteristics of the participants

One hundred athletes (response rate 37.0%) and 111 controls (response rate 20.1%) completed the questionnaire. Thirteen controls had participated in competitive sports and were excluded. Thus, the final control group included 98 women. The groups were similar in age

First sports discipline, n (%)	
Distance running	64 (64)
Track and field (middle-distance and long-distance running)	16 (16)
Cross-country skiing	12 (12)
Other (orienteering, swimming, gymnastic, Finnish baseball)	8 (8)
Retired from sports, n (%)	94 (94)
Career length (years), mean±SD*	12.0±5.8
Highest competition level, n (%)†	
International (Tier 4)	34 (34)
National (Tier 3)	65 (65)
Local (Tier 2)	1 (1)
High amount of injury-related harm during the sports career, n (%)	16 (16)
Sports career termination due to injury, n (%)*	42 (44.7)

*Only retired athletes, n=94
 †Tiers, as suggested by McKay *et al*²⁴ are used when classifying athletes.

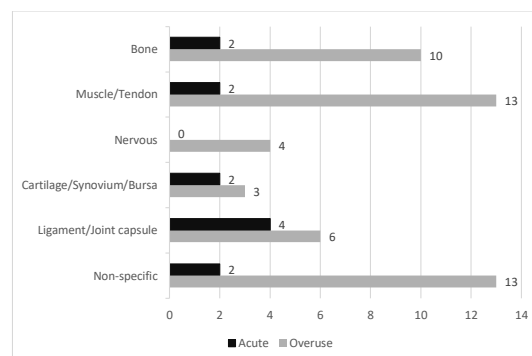


Figure 2 Types of career-ending injuries reported by the athletes. The counts present the number of injured athletes.

(mean 39.6±4.5 years and 39.4±4.5 years in athletes and controls, respectively, $p=0.801$).

Characteristics of the athletes are presented in table 1. All track and field athletes had participated in middle-distance and long-distance running, and one had also competed in jumping disciplines. Participants who reported commencing their sports career in gymnastics or Finnish baseball had later changed to endurance sports.

Of the 42 athletes who had terminated their career due to injury, 10 (23.8%) reported that an acute injury contributed to their retirement. In contrast, 34 (81.0%) reported that an overuse injury impacted their career termination. Two athletes reported both acute and overuse injuries that contributed to career termination. Twenty (47.6%) athletes who had terminated their career due to injury were able to identify one single contributing injury (or bilateral injury). The remaining 22 (52.4%) athletes reported several injuries that affected career termination. Figure 2 presents the number of athletes with career-ending injuries by injured tissue and type of injury (acute/overuse). Secondary amenorrhoea was present in 5 of the 9 athletes (56%) who reported bone stress-related career-ending injuries and 7 of the 21 athletes (33%) who reported other career-ending injuries ($p=0.418$).

Menstrual and pubertal history and DE behaviours

Menstrual and pubertal history, ED diagnoses and EDE-QS scores at different age stages among the athletes and the controls are presented in table 2. Athletes were older at menarche, achieved regular menstruation later and had higher rates of primary and secondary amenorrhoea. No statistically significant differences between the groups were found in ED diagnoses or EDE-QS scores at any age stage.

Changes in EDE-QS scores over time in both athletes ($\chi^2(4) = 24.7, p<0.001$) and controls ($\chi^2(4) = 9.8, p=0.044$) were statistically significant. Post hoc analyses are presented in table 3. Trends in EDE-QS scores over

Table 2 Menstrual and pubertal history and trends in eating behaviours among the athletes and the controls

	Athletes (n=100)	Controls (n=98)	P value
Age at menarche (years), mean±SD	14.0±2.0	12.5 ± 1.2 ^a	<0.001‡
Age at attaining regular menses (years), mean±SD	16.0±3.7 ^b	14.7±2.9 ^b	0.005‡
Primary amenorrhoea, n (%)	20 (20.0)	2 (2.0)	<0.001‡
Spontaneous menarche*, n (%)	93 (93.0)	98 (100.0)	0.014‡
Examined due to delayed puberty, n (%)	13 (13.0)	2 (2.0)	<0.001‡
Secondary amenorrhoea, n (%)†	31 (34.1) ^c	20 (20.4)	0.035‡
Regular menses always/nearly always, n (%)†	50 (54.3) ^d	72 (78.3) ^d	<0.001‡
Cycle length 21–35 days, n (%)†	55 (64.7) ^e	81 (89.0) ^c	<0.001‡
Eating disorder diagnosis, n (%)	7 (7.1) ^f	8 (8.4) ^g	0.740
EDE-QS, mean±SD			
Age 13–15	6.3±7.3	6.4±7.4	0.943
Age 16–18	8.4±7.9	7.3±7.7	0.340
Age 19–21	8.0 ± 8.0 ^h	7.4±7.8	0.589
Age 22–25	7.2±7.4 ⁱ	7.5±7.8	0.737
Currently	5.6±6.4	6.0 (6.2)	0.697
EDE-QS≥15, n (%)			
Age 13–15	16 (16.0)	15 (15.3)	0.893
Age 16–18	20 (20.0)	18 (18.4)	0.771
Age 19–21	19 (19.8) ^h	15 (15.3)	0.411
Age 22–25	17 (18.9) ⁱ	13 (13.3)	0.293
Currently	12 (12.0)	10 (10.2)	0.688

^a n=97; ^b n=82; ^c n=91; ^d n=92; ^e = 85; ^f n=98; ^g n=95; ^h n=96; ⁱ n=90.
 *Menarche had occurred spontaneously, that is, without any treatments.
 †During the sports career (athletes) or ever (controls).
 ‡p<0.05
 EDE-QS, the Eating Disorder Examination Questionnaire short form scores.

Table 3 Post hoc analyses of EDE-QS scores at different age stages among athletes and controls

Age group comparisons	Athletes (n=100)		Controls (n=98)	
	P value	Adjusted p value*	P value	Adjusted p value*
13–15 vs 16–18	<0.001‡	0.002‡	0.090	0.903
13–15 vs 19–21	0.004‡	0.040‡	0.119	1.00
13–15 vs 22–25	0.109	1.00	0.013‡	0.130
13–15 vs currently	0.832	1.00	0.718	1.00
16–18 vs 19–21	0.423	1.00	0.892	1.00
16–18 vs 22–25	0.038‡	0.381	0.429	1.00
16–18 vs currently	0.001‡	0.005‡	0.183	1.00
19–21 vs 22–25	0.203	1.00	0.354	1.00
19–21 vs currently	0.008‡	0.077	0.231	1.00
22–25 vs currently	0.164	1.00	0.034‡	0.337

*Adjusted for the Bonferroni correction for multiple tests.
 ‡p<0.05

time were more stable among controls than among athletes (figure 3).

Nineteen (19%) of the athletes reported DE during their careers (ie, had EDE-QS scores of 15 or more), and 17 (89%) of them had ended their careers. Current DE was reported by 35% (n=6) of the retired athletes with DE during their career compared with 8% (n=6) of the retired athletes without DE during their sports career (p=0.003). Twenty-one (21%) controls reported DE during their adolescence or young adulthood (between ages 13 and 21), and current DE was reported by 43% (n=9) of them compared with one (1%) control with current DE but without DE at adolescence/young adulthood (p<0.001).

Associations of menstrual history and history of DE behaviours with a sports career

No differences were found between athletes with and without primary or secondary amenorrhoea in sports career length or participation level or between those with and without DE at ages 13–15, 16–18 or 19–21 in participation level. Athletes with DE at age 16–18 had a shorter

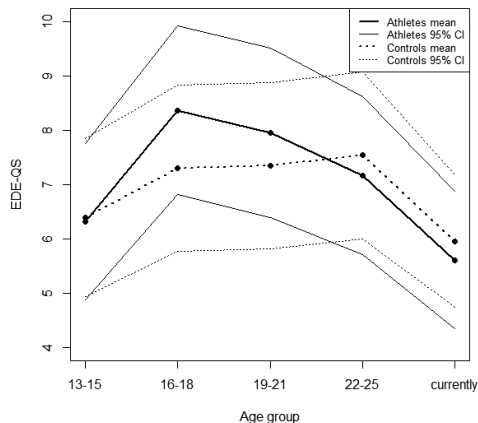


Figure 3 Eating Disorder Examination Questionnaire short form (EDE-QS) scores over time among athletes and controls.

career than those without DE at that age (10.5 years vs 14.0 years, $p=0.020$), while no differences in career length were found between those with and without DE at ages 13–15 and 19–21.

The GEE models for the associations of menstrual history and eating behaviours during the sports career with sports career-related factors among athletes are presented in table 4. EDE-QS scores were associated with sports career length, with one point in the EDE-QS score being associated with a 0.15-year shorter career. Those reporting secondary amenorrhoea during their sports career had 0.5 times lower odds of being competed at the international level (vs national level), 4.0 times higher odds of reporting that injuries/pain negatively affected their sports career and 1.9 times higher odds of reporting that an injury had affected their sports career termination. The secondary analysis, where EDE-QS scores at ages 13–15, 16–18 and 19–21 from all athletes were used, is presented in online supplemental file 1.

DISCUSSION

We found no statistically significant differences in eating behaviours between the athletes and the controls, but menstrual dysfunction and delayed puberty were more common among the athletes. Among the athletes, current EDE-QS scores were lower than earlier in life, indicating a healthier attitude toward eating and body image in adulthood than in adolescence. In contrast, among the controls, trends in EDE-QS scores over time were more stable. DE behaviour earlier in life was associated with current DE in both groups. Higher EDE-QS scores, that is, more unhealthy attitudes toward eating and body image during the sports career, were associated with

Table 4 Associations of menstrual history and eating behaviours during the sports career with career length, participation level, injury-related harms during the career and career termination due to injury

Sports career length (n=100)			
	B	95% CI	P value
Primary amenorrhoea	2.28	−0.55 to 5.11	0.115
Secondary amenorrhoea	−0.16	−2.05 to 1.72	0.864
EDE-QS	−0.15	−0.26 to −0.05	0.003*
Time (age 16–18 vs age 13–15)	0.63	−1.47 to 2.73	0.555
Time (age 19–21 vs age 13–15)	3.01	0.64 to 5.38	0.013*
Participation level (international vs national) (n=100)			
	OR	95% CI	P value
Primary amenorrhoea	1.09	0.54 to 2.21	0.808
Secondary amenorrhoea	0.51	0.27 to 0.95	0.033*
EDE-QS	0.99	0.95 to 1.03	0.544
Time (age 16–18 vs age 13–15)	0.06	0.56 to 2.00	0.086
Time (age 19–21 vs age 13–15)	1.74	0.87 to 3.49	0.117
Injury-related harms during the sports career (n=100)			
	OR	95% CI	P value
Primary amenorrhoea	1.22	0.53 to 2.81	0.639
Secondary amenorrhoea	4.00	1.88 to 8.48	<0.001*
EDE-QS	0.97	0.93 to 1.01	0.187
Time (age 16–18 vs age 13–15)	1.14	0.49 to 2.66	0.758
Time (age 19–21 vs age 13–15)	1.40	0.57 to 3.47	0.461
Sports career termination due to injury (n=94)			
	OR	95% CI	P value
Primary amenorrhoea	0.61	0.30 to 1.23	0.168
Secondary amenorrhoea	1.89	1.02 to 3.51	0.045*
EDE-QS	1.01	0.97 to 1.05	0.598
Time (age 16–18 vs age 13–15)	0.90	0.48 to 1.69	0.747
Time (age 19–21 vs age 13–15)	1.16	0.57 to 2.37	0.683

* $p<0.05$

EDE-QS, the Eating Disorder Examination Questionnaire short form scores.

a shorter career. In addition, secondary amenorrhoea during the career was associated with lower participation level, higher rates of injury-related harms and a higher likelihood of career termination due to injury.

Menstrual history and attitudes toward eating

The athletes had higher age at menarche, higher rates of delayed puberty and menstrual dysfunction and less

spontaneously occurring menarche than controls, which agrees with other studies.^{13 26 27} While the mechanisms behind these associations are incompletely understood, they may be related to intensive exercise and/or LEA in athletes, which leads to decreased leptin concentrations and alterations in other peptides. This may result in disturbances in gonadotrophin-releasing hormone secretion, which further suppresses the function of the hypothalamus-pituitary-gonadal axis.^{28 29}

Evidence regarding the difference in the prevalence of eating problems between athletes and non-athletes is inconclusive. A meta-analysis conducted in 2000 found that DE was more common in athletes than non-athletes, but the effect size was small.¹⁴ In a recent meta-analysis, no differences were found in ED psychopathology between athletes and non-athletes.¹² This parallels our findings of no statistically significant differences in DE or ED between the groups. However, our athletes were lean sport athletes, among whom eating problems are thought to be more common than in non-lean sports.^{12 30} The lifetime prevalence of clinical ED diagnoses in our athletes was 7.1%, lower than in our previous cross-sectional study conducted in a mixed-sport sample of Finnish athletes (18.4%).³¹ The detection of EDs has improved over time,³² which might explain the differences between the findings.

Although no differences in the EDE-QS scores between athletes and controls were found, the trends of the scores differed between the groups. Among athletes, EDE-QS scores increased significantly after the age of 13–15, while controls' scores remained more constant over time. This could be related to sports-specific pressures of controlling body weight or composition, which can lead to DE.⁷ In addition, some personality traits, especially perfectionism, may increase the likelihood of both competitive environments and DE.^{7 33} ED incidence generally peaks in adolescence and young adulthood.³⁴ In line with this, EDE-QS scores were higher in adolescence than currently also in our athletes.

A history of DE was associated with current DE among athletes and controls. While several studies have investigated DE in athletes, few studies have focused on former athletes. Oltmans and colleagues³⁵ found that at the baseline of their prospective study, 28% of the former elite Dutch athletes presented with DE. In another study,³⁶ 5.8% of former collegiate athletes reported DE. In our study, the prevalence of present DE was 12%, which falls between previously reported prevalence rates.

Associations of menstrual history and DE with a sports career

A 10-point increase in the EDE-QS score was associated with a 1.5-year shorter sports career. We are unaware of other studies investigating the association between career length and DE, and prospective studies are needed to confirm our findings. One possible mechanism behind this relationship is decreased bone mineral density (BMD) associated with DE.^{8 9} Low BMD may be a risk factor for bone stress injuries, which in turn may cause

long time loss from training and thus predispose to career termination. However, as the present study did not find an association of EDE-QS scores with either injury-related harms during the sports career or career termination due to injury, a possible explanation for the association between higher EDE-QS scores and shorter career length may be related to other factors than injuries. Further research with a prospective design is also needed to investigate the relationship between participation level and menstrual dysfunction, as we cannot demonstrate causality with our retrospective study design.

Secondary amenorrhoea during the sports career was associated with both injuries/pain during the sports career and career termination due to injury. These results support and extend the findings of Rauh and colleagues,³⁷ who found that menstrual dysfunction was associated with running-related injuries among female runners in a model adjusted for bone mineral density less than -2 SDs. Menstrual dysfunction may also be associated with a longer healing time for injuries.^{31 38} The mechanism behind these associations is unclear but might be related to LEA, which increases the risk for menstrual dysfunction, poor bone quality and injuries.⁸ However, not all studies have found an association between menstrual dysfunction and injuries^{39–41} and thus, further studies are needed to confirm the findings.

STRENGTHS AND LIMITATIONS

Our study is the first to investigate trends over time in eating behaviours among athletes compared with controls and the first to assess the associations of menstrual history and eating behaviours with sports career length, participation level and athletic retirement due to injury among adult women with a competitive sports background. Moreover, as we recruited athletes who had succeeded in their adolescence, our sample included those who had terminated their sports careers early.

The greatest limitation of our study is that recall bias may exist because participants were asked to respond based on their memory. Athletes may recall their menstrual cycle-related attributes better than controls, at least in case of delayed puberty and/or absence of menstruation. Sampling bias is also possible because people interested in the research topic may have been more likely to participate. Moreover, our sample size was relatively small, which limited statistical power. The low response rate, especially among the controls, may limit the generalisability of our findings. Finally, due to our study design, we cannot infer causality, and some external confounding factors not considered in the analysis, such as lifestyle or personality, may have influenced the associations found in this study.

Conclusions

Athletes reported higher rates of delayed puberty and menstrual dysfunction than controls, but no differences between the groups were found in eating behaviours at any age stage. Athletes with DE during their sports career



were more likely to report DE also after their career than athletes without DE during their career. EDE-QS scores were negatively associated with sports career length. In addition, secondary amenorrhoea during the sports career was associated with lower participation level, higher rates of injury-related harms during the career, and sports career termination due to injury. These findings indicate that a regular menstrual cycle and healthy attitudes toward eating and body image may benefit an athlete's health and career. Female athletes and those working with them should be aware that disturbances in eating patterns and menstrual function could have a harmful association on a sports career. The study's findings underline the sports community's potential role in influencing female athletes' long-term health by promoting positive body image and eating behaviour.

Author affiliations

¹Faculty of Sport and Health Sciences, University of Jyväskylä, Jyväskylä, Finland

²KIHU Research Institute for Olympic Sports, Jyväskylä, Finland

³Obstetrics and Gynecology, University of Helsinki and Helsinki University Hospital, Helsinki, Finland

⁴Pediatric Research Center, New Children's Hospital, Helsinki University Hospital and University of Helsinki, Helsinki, Finland

⁵Sports Science Department, Reykjavik University, Reykjavik, Iceland

⁶Institute of Rehabilitation, Jyväskylä University of Applied Sciences, Jyväskylä, Finland

Twitter Suvi Ravi @suvmara

Acknowledgements The authors want to thank all the participants for voluntary participation in this study and those former athletes and non-athletes who provided valuable feedback on the questionnaire when piloting the survey. We also want to thank Dr Daniela Eklund for translation of the questionnaire and research information into Swedish.

Contributors UMK and MV conceived the work, and it was further developed by SR, JKI, EH and SK. Survey development and acquisition of the data was completed by SR. Survey questions were reviewed and further developed by MV, JKI, EH, SK, UMK and JP. Data analysis and interpretation were conducted by SR and SH. SR drafted the manuscript, which was critically revised by all the authors. All authors approved the final manuscript. JP is the study guarantor.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient and public involvement Patients and/or the public were involved in the design, or conduct, or reporting, or dissemination plans of this research. Refer to the Methods section for further details.

Patient consent for publication Not applicable.

Ethics approval The study was approved by the Human Sciences Ethics Committee of the University of Jyväskylä (1641/13.00.04.00/2021). Participants were informed of the purpose of the study and that participation in the study was voluntary. All participants provided electronic informed consent before completing the questionnaire.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement The data are not publicly available due to containing information that could compromise the privacy and consent of the participants, but anonymised data are available on reasonable request from the corresponding author.

Supplemental material This content has been supplied by the author(s). It has not been vetted by BMJ Publishing Group Limited (BMJ) and may not have been peer-reviewed. Any opinions or recommendations discussed are solely those of the author(s) and are not endorsed by BMJ. BMJ disclaims all liability and responsibility arising from any reliance placed on the content. Where the content includes any translated material, BMJ does not warrant the accuracy and reliability

of the translations (including but not limited to local regulations, clinical guidelines, terminology, drug names and drug dosages), and is not responsible for any error and/or omissions arising from translation and adaptation or otherwise.

Open access This is an open access article distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited, appropriate credit is given, any changes made indicated, and the use is non-commercial. See: <http://creativecommons.org/licenses/by-nc/4.0/>.

ORCID iDs

Suvi Ravi <http://orcid.org/0000-0001-9706-5449>

Maarit Valtonen <http://orcid.org/0000-0001-8883-2255>

Johanna K Ihalaainen <http://orcid.org/0000-0001-9428-4689>

Elina Holopainen <http://orcid.org/0000-0003-3572-1337>

Silja Kosola <http://orcid.org/0000-0002-2881-8299>

Saara Heinonen <http://orcid.org/0009-0009-9972-3834>

Ben Waller <http://orcid.org/0000-0002-0738-0670>

Urho M Kujala <http://orcid.org/0000-0002-9262-1992>

Jari Parkkari <http://orcid.org/0000-0001-5211-9845>

REFERENCES

- Appelqvist-Schmidlechner K, Vaara J, Häkkinen A, *et al*. Relationships between youth sports participation and mental health in young adulthood among Finnish males. *Am J Health Promot* 2018;32:1502–9.
- Yang X, Telama R, Hirvensalo M, *et al*. Sustained participation in youth sport decreases metabolic syndrome in adulthood. *Int J Obes (Lond)* 2009;33:1219–26.
- Ravi S, Kujala UM, Tammelin TH, *et al*. Adolescent sport participation and age at menarche in relation to midlife body composition. *Bone Mineral Density, Fitness, and Physical Activity J Clin Med* 2020;9:3797.
- Maffulli N, Longo UG, Gougoulas N, *et al*. Sport injuries: a review of outcomes. *Br Med Bull* 2011;97:47–80.
- Kujala UM, Orava S, Parkkari J, *et al*. Sports career-related musculoskeletal injuries. *Sports Medicine* 2003;33:869–75.
- Palmer D, Cooper DJ, Emery C, *et al*. Self-Reported sports injuries and later-life health status in 3357 retired olympians from 131 countries: a cross-sectional survey among those competing in the games between London 1948 and pyeongchang 2018. *Br J Sports Med* 2021;55:46–53.
- Wells KR, Jeacocke NA, Appaneal R, *et al*. The Australian Institute of sport (AIS) and national eating disorders collaboration (NEDC) position statement on disordered eating in high performance sport. *Br J Sports Med* 2020;54:1247–58.
- Mountjoy M, Sundgot-Borgen J, Burke L, *et al*. The IOC consensus statement: beyond the female athlete triad -- relative energy deficiency in sport (RED-S). *Br J Sports Med* 2014;48:491–7.
- Nattiv A, Loucks AB, Manore MM, *et al*. American College of sports medicine position stand. the female athlete triad. *Med Sci Sports Exerc* 2007;39:1867–82.
- Williams NI, Statuta SM, Austin A. Female athlete triad: future directions for energy availability and eating disorder research and practice. *Clin Sports Med* 2017;36:671–86.
- Bratland-Sanda S, Sundgot-Borgen J. Eating disorders in athletes: overview of prevalence, risk factors and recommendations for prevention and treatment. *Eur J Sport Sci* 2013;13:499–508.
- Chapa DAN, Johnson SN, Richson BN, *et al*. Eating-disorder psychopathology in female athletes and non-athletes: a meta-analysis. *Int J Eat Disord* 2022;55:861–85.
- Coelho GM de O, Soares E de A, Ribeiro BG. Are female athletes at increased risk for disordered eating and its complications? *Appetite* 2010;55:379–87.
- Smolak L, Murnen SK, Ruble AE. Female athletes and eating problems: a meta-analysis. *Int J Eat Disord* 2000;27:371–80.
- Gideon N, Hawkes N, Mond J, *et al*. Development and psychometric validation of the EDE-QS, a 12 item short form of the eating disorder examination questionnaire (EDE-Q). *PLoS One* 2016;11:e0152744.
- Melin A, Tornberg AB, Skouby S, *et al*. The leaf questionnaire: a screening tool for the identification of female athletes at risk for the female athlete triad. *Br J Sports Med* 2014;48:540–5.
- Ristolainen L, Kettunen JA, Kujala UM, *et al*. Sport injuries as the main cause of sport career termination among Finnish top-level athletes. *European Journal of Sport Science* 2012;12:274–82.
- Wang Y-X, Arvizu M, Rich-Edwards JW, *et al*. Menstrual cycle regularity and length across the reproductive lifespan and

- risk of premature mortality: prospective cohort study. *BMJ* 2020;371:m3464.
- 19 Nicodemus KK, Folsom AR, Anderson KE. Menstrual history and risk of hip fractures in postmenopausal women. The Iowa women's health study. *Am J Epidemiol* 2001;153:251–5.
 - 20 Fairburn CG, Beglin SJ. Assessment of eating disorders: interview or self-report questionnaire? *Int J Eat Disord* 1994;16:363–70.
 - 21 Prnjak K, Mitchison D, Griffiths S, et al. Further development of the 12-Item EDE-QS: identifying a cut-off for screening purposes. *BMC Psychiatry* 2020;20:146.
 - 22 Elliott-Sale KJ, Minahan CL, de Jonge XAKJ, et al. Methodological considerations for studies in sport and exercise science with women as participants: a working guide for standards of practice for research on women. *Sports Med* 2021;51:843–61.
 - 23 Practice committee of the american society for reproductive medicine. current evaluation of amenorrhea. *Fertil Steril* 2004;82:266–72.
 - 24 Bahr R, Clarsen B, Derman W, et al. International olympic committee consensus statement: methods for recording and reporting of epidemiological data on injury and illness in sports 2020 (including the STROBE extension for sports injury and illness surveillance (STROBE-SIIS)). *Orthop J Sports Med* 2020;8:2325967120902908.
 - 25 McKay AKA, Stellingwerff T, Smith ES, et al. Defining training and performance caliber: a participant classification framework. *Int J Sports Physiol Perform* 2022;17:317–31.
 - 26 Warren MP. The effects of exercise on pubertal progression and reproductive function in girls. *J Clin Endocrinol Metab* 1980;51:1150–7.
 - 27 Cannavò S, Curtò L, Trimarchi F. Exercise-related female reproductive dysfunction. *J Endocrinol Invest* 2001;24:823–32.
 - 28 Martos-Moreno GA, Chowen JA, Argente J. Metabolic signals in human puberty: effects of over and undernutrition. *Mol Cell Endocrinol* 2010;324:70–81.
 - 29 Muñoz-Calvo MT, Argente J. Nutritional and pubertal disorders. *Endocr Dev* 2016;29:153–73.
 - 30 Torstveit MK, Rosenvinge JH, Sundgot-Borgen J. Prevalence of eating disorders and the predictive power of risk models in female elite athletes: a controlled study. *Scand J Med Sci Sports* 2008;18:108–18.
 - 31 Ravi S, Ihalainen JK, Taipale-Mikkonen RS, et al. Self-Reported restrictive eating, eating disorders, menstrual dysfunction, and injuries in athletes competing at different levels and sports. *Nutrients* 2021;13:3275.
 - 32 Keski-Rahkonen A, Mustelin L. Epidemiology of eating disorders in Europe: prevalence, incidence, comorbidity, course, consequences, and risk factors. *Curr Opin Psychiatry* 2016;29:340–5.
 - 33 Thomas JJ, Keel PK, Heatherton TF. Disordered eating attitudes and behaviors in ballet students: examination of environmental and individual risk factors. *Int J Eat Disord* 2005;38:263–8.
 - 34 Jacobi C, Hayward C, de Zwaan M, et al. Coming to terms with risk factors for eating disorders: application of risk terminology and suggestions for a general taxonomy. *Psychol Bull* 2004;130:19–65.
 - 35 Oltmans E, Confectioner K, Jonkers R, et al. A 12-month prospective cohort study on symptoms of mental health disorders among Dutch former elite athletes. *Phys Sportsmed* 2022;50:123–31.
 - 36 Kerr G, Berman E, Souza MJD. Disordered eating in women's gymnastics: perspectives of athletes, coaches, parents, and judges. *Journal of Applied Sport Psychology* 2006;18:28–43.
 - 37 Rauh MJ, Barrack M, Nichols JF. Associations between the female athlete triad and injury among high school runners. *Int J Sports Phys Ther* 2014;9:948–58.
 - 38 Beckvid Henriksson G, Schnell C, Lindén Hirschberg A. Women endurance runners with menstrual dysfunction have prolonged interruption of training due to injury. *Gynecol Obstet Invest* 2000;49:41–6.
 - 39 Beals KA, Manore MM. Disorders of the female athlete triad among collegiate athletes. *Int J Sport Nutr Exerc Metab* 2002;12:281–93.
 - 40 Tenforde AS, Sayres LC, McCurdy ML, et al. Overuse injuries in high school runners: lifetime prevalence and prevention strategies. *PM R* 2011;3:125–31.
 - 41 Thein-Nissenbaum JM, Rauh MJ, Carr KE, et al. Associations between disordered eating, menstrual dysfunction, and musculoskeletal injury among high school athletes. *J Orthop Sports Phys Ther* 2011;41:60–9.

Supplemental table 1. Associations of eating behaviours at ages 13–15, 16–18, and 19–21 and menstrual history with sports career length, participation level, injury-related harms during the sports career, and sports career termination due to an injury.

<i>Sports career length (n = 100)</i>			
	B	95% CI	P-value
Primary amenorrhea	2.49	-0.14, 5.12	0.063
Secondary amenorrhea	-0.03	-1.74, 1.68	0.972
EDE-QS	-0.19	-0.28, -0.11	< 0.001
Time (age 16-18 vs. age 13-15)	0.43	-1.63, 2.49	0.684
Time (age 19-21 vs. age 13-15)	0.66	-1.41, 2.73	0.531
<i>Participation level (international vs national) (n = 100)</i>			
	OR	95% CI	P-value
Primary amenorrhea	1.11	0.57, 2.14	0.762
Secondary amenorrhea	0.62	0.35, 1.11	0.106
EDE-QS	0.98	0.94, 1.02	0.261
Time (age 16-18 vs age 13-15)	1.04	0.56, 1.96	0.887
Time (age 19-21 vs age 13-15)	1.01	0.54, 1.90	0.978
<i>Injury-related harms during the sports career (n = 100)</i>			
	OR	95% CI	P-value
Primary amenorrhea	1.17	0.52, 2.62	0.704
Secondary amenorrhea	3.55	1.73, 7.27	< 0.001
EDE-QS	0.96	0.92, 1.00	0.076
Time (age 16-18 vs age 13-15)	1.08	0.47, 2.50	0.850
Time (age 19-21 vs age 13-15)	1.11	0.48, 2.57	0.799
<i>Sports career termination due to injury (n = 94)</i>			
	OR	95% CI	P-value
Primary amenorrhea	0.54	0.28, 1.06	0.074
Secondary amenorrhea	1.87	1.06, 3.29	0.030
EDE-QS	1.01	0.97, 1.04	0.708
Time (age 16-18 vs age 13-15)	0.98	0.53, 1.82	0.960
Time (age 19-21 vs age 13-15)	1.03	0.55, 1.92	0.923

OR = odds ratio; CI = confidence interval; EDE-QS = the Eating Disorder Examination Questionnaire short form scores