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Titta Kontro

Relationships between Physical Activity, Smoking, Alcohol Use, and Health among Finnish Male Former Elite Athletes

UNIVERSITY OF JYVÄSKYLÄ
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ABSTRACT

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Alcohol- and smoking-related health problems are global issues. Finnish former athletes have lower morbidity than the general population, but little is known about how the continuation of physical activity (PA), smoking, and alcohol use contributes to former athletes' health in later life. The purpose was to investigate how a former elite-level sports career was associated with alcohol use and smoking and health. Furthermore, the aim was to investigate whether all-cause mortality and health habits differ between former athletes and their brothers.

Using national registers, the occurrence of alcohol- or smoking-related diseases and deaths among Finnish male former elite athletes (n=2202) and matched controls (n=1403) alive in 1970 was followed from 1970 to 2015. In 1985, 1995, 2001, and 2008, the surviving participants questionnaire-reported their PA, alcohol use, and smoking. The brothers' genealogy and data were collected via a questionnaire sent to them in 2001.

Former athletes were more physically active and smoked less than controls in all questionnaires. In 1985, former athletes used alcohol more than controls, especially if their careers had ended by sports injury. Five latent profiles were found. Smoking decreased in all profiles and PA decreased in three profiles, while alcohol use also increased in some profiles. The cross-lagged path model indicated that the associations of alcohol use and PA were weak at most. The risk of alcohol-related morbidity did not differ between former athletes and controls, but the risk of chronic pulmonary diseases or deaths was lower among former athletes. In 2001 health habits were better among former athletes and they survived 2–3 years longer than their brothers.

Although the risk of excessive alcohol use increased in individuals whose athletic careers ended by sports injury, overall PA and alcohol use affected each other's development only modestly during the follow-up. In 1985 former athletes reported higher alcohol use than controls, but there was no difference in alcohol-related morbidity. The ability to compete at the highest level of sports in young adulthood is associated with a reduced risk of chronic pulmonary disease in later life. Genetic differences between former athletes and brothers, aerobic training for elite endurance sports, and a healthier lifestyle may all contribute to reduced mortality.

Keywords: alcohol, chronic disease(s), cohort study, former athlete(s), morbidity, mortality, physical activity, smoking

TIIVISTELMÄ (FINNISH ABSTRACT)

Kontro, Titta
Entisten suomalaisten mieshuippu-urheilijoiden fyysisen aktiivisuuden, alkoholinkäytön ja tupakoinnin väliset yhteydet sekä niihin liittyvät sairaudet ja kuolleisuus.
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Tupakointi ja haitallinen alkoholinkäyttö ovat globaaleja ongelmia. Tutkimuksen tarkoituksena oli tutkia entisten huippu-urheilijoiden alkoholinkäytön, fyysisen aktiivisuuden ja tupakoinnin välisiä yhteyksiä sekä niihin liittyviä sairauksia ja kuolleisuutta. Lisäksi tavoitteena oli selvittää, eroavatko entisten huippu-urheilijoiden ja heidän veljiensä kuolleisuus tai elintavat toisistaan.

Alkoholi- ja keuhkosairauksia tutkittiin kansallisten sairaalapoistorekistereiden avulla 1970-2015, vuonna 1970 elossa oli entisiä huippu-urheilijoita (n=2202) ja verrokkeja (n=1403). Entiset huippu-urheilijat ja verrokit raportoivat alkoholinkulutustaan, tupakointi- ja liikuntatottumuksiaan kyselytutkimuksissa vuosina 1985, 1995, 2001 ja 2008. Veljien aineisto kerättiin rekistereistä ja he osallistuivat myös kyselytutkimukseen vuonna 2001.

Entiset huippu-urheilijat olivat fyysisesti aktiivisempia ja tupakoivat verrokkeja vähemmän kaikissa kyselytutkimuksissa. Vuoden 1985 kyselyssä urheilijat käyttivät verrokkeja enemmän alkoholia. Alkoholinkulutus oli runsasta etenkin niillä urheilijoilla, joiden urheilu-ura oli päättynyt loukkaantumisen vuoksi. Kaksimuuttujaisen polkumallin mukaan yhteydet alkoholinkulutuksen ja fyysisen aktiivisuuden välillä olivat pääosin heikkoja. Aineistosta löydettiin viisi profiilia. Seurannan aikana tupakointi väheni kaikissa profiileissa ja fyysinen aktiivisuus väheni kolmessa, kun taas alkolinkäyttö myös lisääntyi joissakin profiileissa. Entisten huippu-urheilijoiden ja verrokkien alkoholisairauksien tai -kuolemien riski ei eronnut toisistaan, mutta urheilijoiden keuhkosairauksien riski oli verrokkeja alhaisempi. Vuonna 2001 entisten huippu-urheilijoiden elintavat olivat terveellisempiä ja he elivät 2-3 vuotta pidempään kuin heidän veljensä.

Vaikka entisten huippu-urheilijoiden alkoholinkulutus kasvoi urheilu-uran päättyessä loukkaantumiseen, fyysinen aktiivisuus ei näyttänyt ennustavan alkoholinkäyttöä seurannan aikana. Urheilijat raportoivat käyttävänsä enemmän alkoholia vuonna 1985, mutta alkoholisairauksien riski ei eronnut verrokeista. Kyky kilpaurheilla varhaisaikuisuudessa näyttäisi olevan yhteydessä alhaisempaan kroonisten keuhkosairauksien riskiin myöhemmällä iällä. Entisten huippu-urheilijoiden ja veljien väliset geneettiset eroavaisuudet, kestävyysurheilijoiden aerobinen harjoittelu ja terveellisemmät elintavat saattavat olla yhteydessä alhaisempaan kuolleisuuteen.

Avainsanat: alkoholi, entinen huippu-urheilija, fyysinen aktiivisuus, kohorttitutkimus, krooninen sairaus, kuolleisuus, sairastuvuus, tupakointi

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Titta Kontro

LIST OF ORIGINAL PUBLICATIONS

I Kontro, T.K., Tolvanen, A., Sarna, S., Kaprio, J. & Kujala, U.M. 2020. Physical activity, use of alcohol and smoking in middle-aged and aging men. A longitudinal study among Finnish male former athletes and controls. European Journal of Sport Science, 1-10. doi: 10.1080/17461391.2020.1761889.

II Kontro, T.K., Sarna, S., Kaprio, J. & Kujala, U.M. 2017. Use of alcohol and alcohol-related morbidity in Finnish former elite athletes. Medicine & Science in Sports & Exercise 49(3), 492-499. doi: 10.1249/MSS.0000000000001137.

III Kontro, T.K., Sarna, S., Kaprio, J. & Kujala, U.M. 2019. The difference in risk of chronic pulmonary disease morbidity and mortality between former elite athletes and ordinary men in Finland. European Journal of Sport Science, 1-10. doi: 10.1080/17461391.2019.1697375.

IV Kontro, T.K., Sarna, S., Kaprio, J. & Kujala, U.M. 2018. Mortality and health-related habits in 900 Finnish former elite athletes and their brothers. British Journal Sports Medicine 52(2), 89-95. doi: 10.1136/bjsports-2017-098206.

Under the supervisors' guidance, the author contributed to the original publications listed above as follows. In all *Studies I–IV,* the author was privileged to use pre-existing data. The register-based data sets (*Studies II–IV*) were updated. All data sets in *Studies I–IV* (basic information and register- and questionnaire-based data sets) were modified rigorously and combined by the author. All *Studies I–IV* utilized a basic information data set that was combined with other data sets. The author analyzed the data and wrote the first drafts of the manuscripts. The author finalized the manuscripts after input from all coauthors.

FIGURES

TABLES

ABBREVIATIONS

AHA	The American Heart Association
AHEI	alternate healthy eating index
ACC	The American College of Cardiology
AdjLMR	the adjusted Lo–Mendell–Rubin likelihood ratio test
ADL	activities of daily living
AHR	airway hyperresponsiveness
APA	American Psychiatric Association
ACSM	The American College of Sports Medicine
AUDIT	Alcohol Use Disorders Identification Test
AvePP	the average posterior probability
BCH	Bose–Chaudhuri–Hocquenghem
BMI	body mass index
BSI-53	the Brief Symptom Inventory-53
CB	chronic bronchitis
CHD	coronary heart disease
CI	confidence interval
COPD	chronic obstructive pulmonary disease
CPD	cigarettes per day
CRD	chronic respiratory disease
CRF	cardiorespiratory fitness
CVD	cardiovascular disease
EE	expenditure of energy
EIA	exercise-induced asthma
EIB	exercise-induced bronchoconstriction
ESC	The European Society of Cardiology
ICD	International Classification of Diseases
FTND	the Fagerström Test for Nicotine Dependence
GINA	the Global Initiave for Asthma
GOLD	the Global Initiative for Chronic Obstructive Lung Disease
GWAS	genome-wide association study
HDO	heavy drinking occasion
HSI	heaviness of smoking index
HR	heart rate
HR	hazard ratio
HRQoL	health-related quality of life
IADL	instrumental activities of daily living
kg	kilogram
LDL	low-density lipoprotein
LPA	latent profile analysis
MAR	missing at random
MET	metabolic equivalent
min	minute

NFL	National Football League
OA	osteoarthritis
OKM	Ministry of Education and Culture in Finland
PA	physical activity
PRS	polygenic risk score
SAMHSA	The Substance Abuse and Mental Health Services Administration
SD	standard deviation
SES	socioeconomic status
SRH	self-rated health
TBI	traumatic brain injury
TDEE	total daily energy expenditure
THL	Finnish Institute for Health and Welfare
URTI	upper respiratory tract infections
VO_2	oxygen uptake
WHO	World Health Organization

CONTENTS

1 INTRODUCTION

There is a widespread scientific and public health policy consensus that certain lifestyle habits, such as hazardous alcohol drinking, cigarette smoking, and low physical activity (PA), significantly contribute to morbidity and mortality (Fine et al. 2004, Lim et al. 2012, WHO 2014c) and are associated with socioeconomic status (SES). Epidemiologic studies using a variety of SES measures have consistently shown that, in the general population, mortality risk increases as SES decreases (Mackenbach et al. 2008, Hajat et al. 2011). Furthermore, self-rated health (SRH) strongly and reliably indicates overall morbidity (Manderbacka, Lundberg & Martikainen 1999) and is a good predictor of mortality (Idler & Benyamini 1997). Previous studies on PA and SRH have shown that PA is related to better SRH (Abu-Omar, Rütten & Robine 2004, Södergren et al. 2008, Engberg et al. 2015).

Correspondingly, regular PA is associated with better functioning and well-being, especially at an older age (Heikkinen et al. 1993, King et al. 2000, Heikkinen 2005, Paterson & Warburton 2010), and a lower risk of morbidity (Kujala et al. 2003a, Gajewski & Poznanska 2008) and all-cause mortality (Sarna et al. 1993, Kujala et al. 1998, Samitz, Egger & Zwahlen 2011, Eijsvogels et al. 2016). However, very intense exercise patterns may increase the risk of cardiovascular morbidity and mortality compared to light to moderate amounts of exercise (Eijsvogels et al. 2016, Kim & Baggish 2017). Furthermore, there is no irrefutable evidence for a causal relationship between PA in adulthood and mortality, either in animal experiments or in randomized controlled intervention studies with healthy individuals (Karvinen et al. 2015).

Cross-sectional and longitudinal studies have found that continuous PA compared to habitual physical inactivity during late adolescence predicts a lower prevalence of smoking during young adulthood even after familial factors are considered (Audrain-McGovern, Rodriguez & Moss 2003, Kujala, Kaprio & Rose 2007). Correspondingly, alcohol drinkers are much more likely to smoke (Carmody et al. 1985, Bobo & Husten 2000), and dependence on tobacco and alcohol is also correlated (Grant et al. 2004, Li et al. 2007). The healthcare expenditure on treating smoking-attributable diseases is 5.7 % of the global health expenditure,

a heavy economic burden that occurs particularly in Europe and North America (Goodchild, Nargis & Tursand'Espaignet 2018). Consistently, cigarette smoke exposure has been highly correlated mortality and the development of chronic obstructive pulmonary disease (COPD) (Mannino et al. 2000, Hooper et al. 2012). Furthermore, in the clinical setting, patients who present with a combination of COPD and asthma-related traits are quite common (Hedman et al. 1999, Guerra 2009, Kauppi et al. 2011).

Epidemiological studies suggest that self-reported and physician-diagnosed asthma are twice as common in Finnish athletes (Alaranta et al. 2004) and elite Norwegian athletes (Nystad, Harris & Borgen 2000) than in control populations. However, Kujala et al. (1996) reported that the lifetime occurrence of asthma or other pulmonary diseases was not increased in former elite athletes in later life. Additionally, it has been observed that current (Wechsler et al. 1997, Peretti-Watel et al. 2003) and former athletes (Sarna et al. 1993, Fogelholm, Kaprio & Sarna 1994, Bäckmand et al. 2010) smoke less than nonathletes.

Some studies have emphasized sports participation as a possible protective factor against excessive alcohol and other substance use in young individuals (Hellandsjø-Bu et al. 2002, Lorente et al. 2004), while many studies indicate that athletic participation is associated with more excessive alcohol consumption (O'Brien & Lyons 2000, Nelson & Wechsler 2001, Martens et al. 2006a, Dietze, Fitzgerald & Jenkinson 2008) and alcohol dependence (Partington et al. 2013). However, there is a paucity of scientific data on the use of alcohol among former athletes, because most studies are only focused on drinking habits among younger and current athletes (O'Brien & Lyons 2000, Nelson & Wechsler 2001, Martens et al. 2006a, Dietze, Fitzgerald & Jenkinson 2008, Partington et al. 2013).

Studies have found that retirement can lead to increased alcohol use among older people, who may use alcohol to counter boredom and combat loneliness (Menninger 2002, Klimstra & Mahgoub 2010, Blazer & Wu 2011). During the last decades, athletic retirement has begun to be studied (Alfermann, Stambulova & Zemaityte 2004, Stephan, Torregrosa & Sanchez 2007), and it has been recognized that sports career termination causes significant changes in an athlete's personal and social life (Wylleman, Alfermann & Lavallee 2004). Some studies have focused on the importance of social, emotional, and economic support in the adaptation to athletic retirement (Stephan et al. 2003). If an active sports career ends unexpectedly, and an individual has a high athletic identity, then adaptation to forced athletic retirement will be challenging (Alfermann, Stambulova & Zemaityte 2004) and might lead to alcohol-related problems (Chambers 2002).

The health status of former Finnish athletes has been previously widely studied, and it has been observed that former athletes have lower morbidity and mortality than the general population, which may be due to their genetic backgrounds, increased PA levels, or other better health habits (Sarna et al. 1993, Fogelholm, Kaprio & Sarna 1994, Kujala et al. 2003a, Bäckmand et al. 2010, Kettunen et al. 2015). However, less is known about how the continuation of PA, smoking habits, and the use of alcohol contribute to former athletes' health in later years.

The first aim of the present dissertation was to gain new information on the effects of an athletic career and long-term habitual PA on the behavioral health–related factors, such as alcohol consumption and smoking, of middle-aged and aging men. The second aim was to examine the use of alcohol and alcohol-related morbidity and mortality in Finnish former elite athletes and controls. The third aim was to study how a former competitive sports career and smoking were associated with the incidence and mortality of any chronic pulmonary diseases. The fourth aim was to investigate whether all-cause mortality and the development of behavioral and biological risk factors differ between former athletes and their brothers in later life. As stated earlier, regular PA plays an important role in the prevention and treatment of many chronic diseases and in the reduction of disabilities, especially among elderly people. Alcohol- and smoking-related health problems are global issues, and both of these addictions cause distress and great expenditures in a society. Therefore, the present dissertation was significant from an individual and a public health viewpoint.

2 REVIEW OF THE LITERATURE

2.1 Physical activity and sports participation

2.1.1 Definitions

There are different definitions and classifications of physical activity (PA), athlete and sports in literature.

2.1.1.1 Physical activity

PA means any bodily movement produced by the skeletal muscles that requires the expenditure of energy (EE) (Caspersen, Powell & Christenson 1985, Bouchard, Blair & Haskell 2007). PA is generally described by its duration, frequency, and intensity and can be regarded as a continuum from physical inactivity to extreme activity (Shephard 2003). PA may consist of activities for leisure or fitness, with a purpose such as fitness, social interaction, or maintaining or improving physical or psychological health (Caspersen, Powell & Christenson 1985, Armstrong, Bauman, & Davies 2000, Tudor-Locke & Myers 2001). Exercise is defined as planned and structured PA (Fogelholm et al. 2007). Correspondingly, physical fitness is characterized as a set of attributes that individuals have, or that they achieve, that is related to their ability to perform PA (Caspersen, Powell & Christenson 1985). Genetic contributions to cardiorespiratory fitness (CRF) are important, but PA is considered as the principal determinant for CRF (Blair, Cheng & Holder 2001).

2.1.1.2 Athlete

Applying data on the same cohort as the present dissertation, former elite athletes were defined as men who had represented Finland at least once in the Olympic Games, European or World championships, or international contests between two or three countries between 1920 and 1965 (Sarna et al. 1993). Former athletes were categorized into three groups according to the type of training needed to

achieve optimal results: endurance, mixed, and power sports (Åstrand & Rodahl 1986, Sarna et al. 1993). According to Sarna et al. (1993), endurance sports included long- and middle-distance running and cross-country skiing. Mixed sports consisted of soccer, ice hockey, basketball, high jumping, pole vaulting, long jumping, triple jumping, hurdling, short-distance running, and the decathlon. Power sports included weightlifting, boxing, wrestling, and shot-putting as well as discus, javelin, and hammer throwing (Sarna et al. 1993). Correspondingly, Araújo and Scharhag (2016) found in their study that there are several criteria or types for the classification of athletes in the literature, given the sport or modality, the position or characteristics, the sex, age, and nature of participation (recreational or competitive), and the type of predominant metabolic pathway for energy production or muscle action (endurance versus strength).

Competitive and elite athletism is characterized by aiming at international success and ethically sustainable individual or group sports (Robinson & France 2011, OKM 2014). Correspondingly, according to the Merriam-Webster Dictionary, an athlete is "*a person who is trained or skilled in exercises, sports, or games requiring physical strength, agility, or stamina*." Furthermore, the American Heart Association (AHA) (Maron et al. 2004) and the American College of Cardiology (ACC) (Maron, Zipes & Kovacs 2015) utilize the following description for a competitive athlete: "*One who participates in an organized team or individual sport that requires regular competition against others as a central component, places a high premium on excellence and achievement, and requires some form of systematic (and usually intense) training*." This definition is line with the European Society of Cardiology (ESC)'s description of competitive athletes: "*individuals of young and adult age, either amateur or professional, who are engaged in exercise training on a regular basis and participate in official sports competition*"(Pelliccia et al. 2005).

There was a need to modernize and standardize the use of the term "*athlete*" in medical and health sciences research (Araújo & Scharhag 2016). According to the Araújo and Scharhag (2016), to be considered an athlete, four criteria should be simultaneously fulfilled (Table 1).

TABLE 1 Four criteria for athletes and sports.

An athlete should (Araújo & Scharhag 2016):	**A sport should (SportAccord 2011):**
(1) train in sports, aiming to improve his/her performance or results	(1) have an element of competition
(2) be actively participating in sports competitions	(2) be in no way harmful to any living creature
(3) be formally registered as a competitor in a local, regional, or national sport federation	(3) not rely on equipment provided by a single supplier (excluding proprietary games such as arena football)
(4) have sports training and competition as his/her major activity or focus of interest, habitually devoting several hours to these sports activities on most days, exceeding the time allocated to other professional or leisure activities	(4) not rely on any "luck" element specifically designed into the sport.

2.1.1.3 Sports

The definition of *sports* underscores clear goals, quality, and a voluminous amount of exercises (Finnish Olympic Committee 2014). Dictionary.com defines *sport* as "*an athletic activity requiring skill or physical prowess*." It goes on to specifically mention racing, baseball, tennis, golf, bowling, wrestling, hunting, and fishing as sports. Correspondingly, the Oxford Dictionary characterizes *sport* as "*an activity involving physical exertion and skill in which an individual or a team competes against another or others for entertainment.*" By the latter definition, hunting does not qualify as a sport because it does not involve competition. Furthermore, according to SportAccord (2011), sports should fulfill four criteria (Table 1). However, all sports do not fulfill these criteria.

2.1.2 Physical activity, health, and functionality

PA has various positive health effects on adult chronic diseases (Warburton, Nicol & Bredin 2006, Physical Activity Guidelines Advisory Committee 2008, Booth, Roberts & Laye 2012, Reiner et al. 2013) and on psychological health (Booth, Roberts & Laye 2012, Reiner et al. 2013). PA also has an essential role in the treatment of many chronic diseases (Pedersen & Saltin 2006, Kujala 2009). Those who comply with general PA recommendations have a 20–30 % lower risk

of all-cause mortality compared to individuals who are insufficiently physically active (WHO 2014c). Physical inactivity is a major and global problem. It has been estimated, mainly on the basis of observational associations, that insufficient PA contributes to 69.3 million disability-adjusted life-years and 3.2 million deaths each year (WHO 2014c). A physically inactive person is often described as an individual who does not meet the recommended level of PA (Tudor-Locke & Myers 2001). It has been reported that almost every third adult is physically inactive and, thus, physical inactivity contributes to the global noncommunicable disease epidemic (Hallal et al. 2012). Activities performed in a sitting or lying position are generally decribed as sedentary behavior (Pate, O'Neill & Lobelo 2008), which is a distinct concept from physical inactivity (Tudor-Locke & Myers 2001, Sedentary Behaviour Research Network 2012).

It has been observed that some PA is better than none, but to achieve optimal health outcomes, it is recommended that PA has a longer duration, higher intensity, or greater frequency. The general guidelines for weekly PA for healthy adults are: to perform at least 150 minutes of moderate or 75 minutes of vigorous-intensity aerobic activity or an equivalent combination of moderate- and vigorous-intensity aerobic activity every week and muscle strengthening activities on two or more days per week (Physical Activity Guidelines Advisory Committee 2008, WHO 2010). To gain more health benefits, moderate-intensity aerobic PA should be increased to 300 minutes weekly or vigorous PA to 150 minutes weekly (Physical Activity Guidelines Advisory Committee 2008, WHO 2010). Furthermore, the Physical Activity Guidelines Advisory Committee (2018) recommends that individuals should avoid a sedentary lifestyle by being physically active as much as possible and taking breaks from sitting during the day. Individuals should also get enough sleep (Physical Activity Guidelines Advisory Committee 2018).

It has been shown that different life events have an effect on PA. For instance, having a child and remarrying may decrease PA levels, whereas retirement may increase PA (Engberg et al. 2012). PA maintains also functionality in daily activities among older people (Heikkinen et al. 1993, King, Rejeski & Buchner 2000, Heikkinen 2005, Paterson & Warburton 2010). Cross-sectional studies have found that PA is associated with better quality of life, functionality, independent living, successful aging, and locomotion (Grimby et al. 1992, Frändin et al. 1995, Strawbridge et al. 1998, Brach et al. 2004). According to longitudinal studies, physically active individuals retain their independent functionality and locomotion longer (Mor et al. 1989, LaCroix et al. 1993, Seeman et al. 1995, Laukkanen, Kauppinen & Heikkinen 1998). Physically active people at older ages have reported higher levels of physical function and well-being in correlational and prospective longitudinal studies, whereas randomized intervention studies have not consistently supported this relationship (Spirduso & Cronin 2001).

2.1.3 Physical activity assessment

PA has many dimensions and components, so there are different methods for assessing it. A total dose of PA, consisting of the duration, frequency, and intensity, is commonly used to assess PA (Warren et al. 2010). The assessment of a total dose of PA contributes the ability to calculate the EE associated with the PA. The body oxygen consumption (VO_2) among healthy normal-weight individuals is approximately 3.5 ml/kg per min at rest (Ainsworth et al. 2011), and this refers to 1 metabolic equivalent (MET) (Warren et al. 2010, Ainsworth et al. 2011). Movement intensity is often categorized based on METs, including sleep (1.0 MET), sedentary behavior (1.1–1.4 METs), light PA (1.5–2.9 METs), moderate PA (3.0–5.9 METs), and vigorous PA (over 6.0 METs) (Warren et al. 2010, Ainsworth et al. 2011).

There are a wide variety of subjective and objective methods for the assessment of PA, such as questionnaires, observation, and physiological markers such as calorimetry, heart rate (HR), and motion sensors (Westerterp 2009). Subjective assessment methods such as self-report questionnaires are commonly used tools for assessing PA; these methods are inexpensive and practical ways to assess PA in a large population (Sallis & Saelens 2000, Warren et al. 2010). However, some inherent limitations of subjective methods are that such approaches may be prone to under- or overreporting and recall bias (Warren et al. 2010, Hills, Mokhtar & Byrne 2014). Using objective methods to assess PA recall bias can be avoided (Warren et al. 2010). Furthermore, objective assessment methods, such as motion sensors (accelerometers and pedometers) and HR monitors, are often used to assess PA, as they are relatively inexpensive, easy to wear, and they can be used in free-living conditions (Westerterp 2009), whereas calorimetry (the doubly labeled water method), the gold standard for assessing PA (Westerterp 2009, McArdle, Katch & Katch 2010), is too costly and laborious to be considered applicable for large population studies (Melanson, Freedson & Blair 1996).

It should be noted that the results are directional, and all the subjective and objective methodologies have a number of limitations; as stated earlier, a low to moderate validity for self-report and monitoring measures of PA has been found (Kohl, Fulton & Caspersen 2000). In both the subjective and objective methods, the limitations include using methods for a limited number of days, which may not be representative of the individual's habitual PA, and the possibility of reactivity based on the individual knowing they are being monitored (Hills, Mokhtar & Byrne 2014). The use of multiple complementary methods is recommended because any single technique cannot quantify all aspects of PA under free-living conditions (Brage et al. 2005, Hills, Mokhtar & Byrne 2014).

Usually, self-reported instruments such as questionnaires show a moderate to good reliability and a poor to moderate criterion validity, whereas the absolute validity is often poor (Warren et al. 2010). Use of questionnaires and diaries may lead to overestimation of EE (Irwin, Ainsworth & Conway 2001). One study indicated that the PA questionnaire had adequate levels of test–retest reliability and validity in the assessment of the total daily energy expenditure (TDEE) and

weekly PA in epidemiological studies (Suzuki, Kawakami & Shimiza 1998). Additionally, studies show that the convergent validity was strong between objective methods, pedometers, and accelerometers but weaker between these and self-report (Harris et al. 2009).

The major advantages of accelerometers are their small size, lack of visual feedback to the individual wearing the device, and capacity to continuously record data over an extended period, but accelerometers cannot register static exercise and have a low sensitivity to sedentary activities (Hills, Mokhtar & Byrne 2014). For some studies, pedometers may be preferred to accelerometers due to their lower cost (Warren et al. 2010) and that their intra- and interunit reliability are also consistently high (McNamara, Hudson & Taylor 2010). Nevertheless, recording only the number of steps per day does not provide accurate information on EE (Kumahara, Tanaka & Schutz 2009). Additionally, the estimation of PA and EE via HR monitoring is popular, relatively inexpensive, convenient, noninvasive, and versatile and provides accurate information on the frequency, intensity, duration, and EE of free-living PA. However, there are also some limitations, for example, that the relationship between HR–VO_2 consumption differs between lower body and upper body activities (Hills, Mokhtar & Byrne 2014).

In summary, with many approaches available, the accurate quantification of EE and the assessment of PA can be very challenging. It is important to appreciate that irrespective of the techniques' apparent sophistication, both subjective and objective methods have inherent limitations and strengths. Objective measures have a better construct validity (Warren et al. 2010), being more strongly associated with established PA determinants, and thus offered better value to the researchers than the questionnaire, but the latter provided useful details on the activity type, so it could be beneficial to combine those methods in assessing PA (Harris et al. 2009, Hills, Mokhtar & Byrne 2014). There is a need for accurate, reliable, and valid methods of measuring PA to clarify the relationships between PA and health outcomes (Prince et al. 2008). However, it is important to evaluate both activity and sedentary sessions during the day (Hills, Mokhtar & Byrne 2014). Furthermore, there is evidence that accelerometers and pedometers could be used as a motivating method for increasing an individual's PA (Tudor-Locke & Bassett 2004, Barwais 2013). All daily activities are very important to an individual's health (Ryen 2014).

2.2 Physical activity and sports continuation among former athletes in later life

Competitive sports are often associated with high levels of PA (Sorenson et al. 2015), whereas the evidence on the long-term PA behavior of former competitive athletes is conflicting. It has been found that former athletes are more physically active after their athletic careers than their controls (Sarna et al. 1993, Fogelholm, Kaprio & Sarna 1994, Sarna et al. 1997, Kujala et al. 1998, Bäckmand et al. 2006,

Bäckmand et al. 2010, Laine et al. 2014), though the findings of some studies are contrary (Reifsteck, Gill & Brooks 2013, Sorenson et al. 2015). Presumably, former athletes prefer to maintain physically active behavior after their athletic career termination as an adaptive approach to aging (Atchley 1989) and to maintaining their identity (Burke & Reitzes 1991) as well as to achieve positive goals and to avoid negative health outcomes in later life (Markus & Nurius 1986), because they are motivated to do so (Bandura 1986, Deci & Ryan 1985). However, based on previous studies in PA, former competitive athletes' behavior as compared to that of the general population may result invarious barriers to PA (Reifsteck, Gill & Brooks 2013, Sorenson et al. 2015).

Studies have shown that former athletes have a significant decline in PA after retirement from competitive sports (Reifsteck, Gill & Brooks 2013, Sorenson et al. 2015). In fact, due to the higher risk of sports injuries throughout their sports careers, athletes may have various psychological (such as fear of injury; Kvist et al. 2005, Simon & Docherty 2014) and physical (such as pain, osteoarthritis (OA); Kujala et al. 2003b, Lohmander et al. 2007, Meir, Weatherby & Rolfe 2007, Schwenk et al. 2007, Huffman et al. 2008,Tveit et al. 2011) barriers to PA as compared to the general population. It has been observed that an overemphasized athletic identity, an excessively specific sport goal orientation, and a high focus on sport-specific training may decrease PA in former athletes after their active athletic careers (Tracey & Elcombe 2004).

It has been found that former athletes may become more sedentary individuals who have a higher fat percentage, perform worse on physical fitness tests, and are generally less physically active compared to controls (Simon & Docherty 2017). This could be explained by a previous sports injury or a reduced interest in participating in PA or by a loss of identity after an active athletic career (Grove, Lavallee & Gordon 1997, Lavallee, Gordon & Grove 1997, Lam et al. 2017). It has been observed that transitioning from being a high-level elite athlete to being a recreational athlete is challenging (Simon & Docherty 2017). Many athletes may want to continue to participate at that high level of sports activity but are unable to because of injury, pain, or lack of competitive sport leagues (Grove, Lavallee & Gordon 1997, Lavallee, Gordon & Grove 1997). According to Simon and Docherty (2017), former athletes may lose their will to exercise if they cannot compete at that high level. It has been observed that athletic identity is positively related to PA and significantly predicts participation in PA (Reifsteck, Gill & Lappan 2016).

Studies have shown that aerobic activity ability in aging individuals declines by 5 % to 15 % per decade (Rogers et al. 1990, Trappe et al. 1996). However, it may decline less than 5 % among those individuals who can maintain high-level PA training (Pollock et al. 1987, Pollock et al. 1997). There is a higher than average reduction in aerobic capacity with age among intensively trained athletes who become more sedentary in later life (Trappe et al. 1996, Simon & Docherty 2017). A decline in aerobic capacity is related to a significant reduction in training intensity and exercise volume (Jackson et al. 1995, Kasch et al. 1995,

Trappe et al. 1996). Furthermore, former athletes have performed worse in muscle strength tests compared to nonathletes, and this may be due to pain associated with previous injuries or to other physical limitations (Simon & Docherty 2017). Consequently, former athletes who want to regularly participate in vigorous activities may question whether engaging in sports increases their risk of developing OA (Kujala, Kaprio & Sarna 1994, Kujala et al. 2003b, Simon & Docherty 2017). However, it has been shown that moderate habitual exercise may not increase the risk of developing OA, and that carefully selected exercises improve muscle strength and mobility among older people and even people with mild or moderate OA (Lequesne, Dang & Lane 1997, Gelber et al. 2000).

Obesity is a worldwide public health crisis (NCD Risk Factor Collaboration 2016). Causal analyses support the effects of a higher body mass index (BMI) on coronary heart disease (CHD) and its intermediates of glucose, blood pressure, cholesterol, and low-density lipoprotein (LDL) (Würtz et al. 2014, Dale et al. 2017). Saarni et al. (2006) observed that repeated cycles of weight loss among athletes may be harmful to weight control and may increase the risk of obesity. Furthermore, studies have found that high school (Wang et al. 1993), collegiate (Noel et al. 2003, Secora et al. 2004), and professional (Snow, Millard-Stafford & Rosskopf 1998) football players have gradually increased in size over the past several decades, and that the cardiovascular mortality risk was elevated among National Football League (NFL) players with the highest playing-time BMI (Lincoln et al. 2018). Correspondingly, retired NFL players revealed that linemen have a 52 % greater risk of cardiovascular-related death compared to the general population (Baron & Rinsky 1994). It is suggested that if individuals can maintain moderate to high PA, their total body weight will remain stable during the life-span, with their body fat percentage increasing 2 % over 20 years (Pollock et al. 1997). However, former athletes who had a decline in their PA and participated in low-intensity exercise increased their total weight and body fat percentage (Pollock et al. 1997, Simon & Docherty 2017). It has also been discovered that former athletes who competed with a BMI greater than 30 kg/m^2 had two times the risk of mortality due to cardiovascular disease (CVD) compared to other players (Baron et al. 2012).

In summary, it is important to note that when the high-level regular training stimulus and the sense of competition are removed, there is an impact on former athletes psychologically and physiologically, e.g. former athletes may not want to participate in any PA (Simon & Docherty 2017). Negative consequences such as injuries may lead to physical inactivity (Russell et al. 2018). Studies have shown that reduced PA significantly and explicitly increases an individual's risk for developing conditions such as obesity, type 2 diabetes, and CVD (Manson, Nathan & Krolewski 1992, Paffenbarger, Hyde & Wing 1993, Tuomilehto, Lindstrom & Eriksson 2001), while PA reduces the risks not only for obesity but also for several chronic diseases (Warburton, Nicol & Bredin 2006, Physical Activity Guidelines Advisory Committee 2008, Booth, Roberts & Laye 2012, Reiner et al. 2013). According to Russell et al. (2018), further research should conduct longitudinal studies of the psychological responses to injury and the relationship

to PA behavior. Hence, researchers could better understand former elite athletes' PA behavior and, furthermore, healthcare professionals could better facilitate a transition from competitive sports to a lifetime of enjoyable, meaningful, and beneficial PA (Russell et al. 2018). Overall, as the former athletes performed worse on the physical fitness tests, this may indicate that those areas should be targeted first when creating prevention and treatment programming (Simon & Docherty 2017).

2.3 Alcohol use and alcohol-related diseases and deaths among current and former athletes

2.3.1 Alcohol use

The World Health Organization (WHO) recorded that the total alcohol consumption has remained quite stable since 1990, including relative stability in all WHO regions (WHO 2011). However, the total global alcohol consumption increased after a relatively stable phase in 2005 (WHO 2018), though the percentage of drinkers worldwide decreased from 47.6 % to 43.0 % from 2000 to 2016 (WHO 2018). According to statistics from the Finnish National Institute for Health and Welfare (THL), the total alcohol use in Finland increased up to 2007 and then decreased by nearly a fifth until 2016. Between 2008 and 2016, both the percentages of weekly drinkers and of the total alcohol consumption among Finnish men aged between 15 and 69 years have decreased. Furthermore, the long-term growth trend in alcohol use by retired people appears to have reversed (THL 2018a). Generally, changes in alcohol-related habits are closely connected to changes in culture and society (Addictionlink.fi 2009).

Several major determinants have an impact on the levels and patterns of alcohol use. Some are individual risk factors such as age and gender, and others are environmental factors (e.g., the availability of alcohol, the policy environment, the economic status of a country) (WHO 2018). One consistent pattern that has been shown in survey studies is an age-related decline in alcohol use starting in young adulthood (Adams & Schoenborn 2006). It has been found that, for men, the mean alcohol use increased sharply during adolescence, peaked at around 25 years, and then declined and plateaued during midlife, before declining at around 60 years (Britton et al. 2015). So, the drinking behavior among aging populations may reflect the effectiveness of public health efforts (Shaw et al. 2011). However, daily alcohol consumption became more common during mid to older age, most significantly among men, reaching over 50 % of men (Britton et al. 2015).

2.3.2 Alcohol use among current and former athletes

In some studies, sports participation is identified as a possible protective factor against excessive alcohol and other substance use in young individuals (Hellandsjø-Bu et al. 2002, Lorente et al. 2004, Woitas-Slubowska 2009), while other studies indicate that athletic participation is associated with more excessive alcohol consumption (Leichliter et al. 1998, Garry & Morrissey 2000, O'Brien& Lyons 2000, Nelson & Welcher 2001, Martens et al. 2006a, Dietze, Fitzgerald & Jenkinson 2008, O'Brien & Kypri 2008, Wichstrøm & Wichstrøm 2009, O'Farrell et al. 2010, O'Brien et al. 2011, Bedendo et al. 2013, Sønderlund et al. 2014) and alcohol dependence (Partington et al. 2013). Green et al. (2014) found that participation in sports is associated with binge drinking despite gender or nationality. Excessive use of alcohol is a common problem, especially among young male athletes (Martens et al. 2006b, Weiss 2010). Athletes estimate that their nonathlete friends use more alcohol compared to the athletes themselves (Dams-O'Connor, Martin & Marten 2007). Lorente et al. (2003) found that athletes drink less often but have more cases of alcohol poisoning (Lorente et al. 2003). Correspondingly, athletes have reported more binge-drinking episodes than nonathletes (Nelson & Wechsler 2001).

Studies have found that despite gender, heavier alcohol use is more common among participants in team and contact sports compared to those in other sports (Merlo, Hong & Cottler 2010, O'Farrel et al. 2010, Bedendo et al. 2013). Correspondingly, team sports athletes are associated with higher levels of alcohol consumption compared to individual sports athletes (Peretti-Watel et al. 2003, Brenner & Swanik 2007, Partington et al. 2013), but there is no consistent evidence across different sports. It has been reported that heavy drinking occasions (HDO) are quite common among football players in Brazil and Ireland (O'Farrel et al. 2010, Bedendo et al. 2013). According to Ford (2007), male icehockey players and female football players use the most alcohol, whereas male basketball players use the least. However, some studies have found that swimmers and divers use even more alcohol than team sports athletes (Green et al. 2001, Martens et al. 2006b). In contrast, risky alcohol consumption is more uncommon among endurance and track and field athletes (Martens et al. 2006b, Gmel, Kuendig & Daeppen 2009).

There is less scientific data on alcohol use among former athletes, and many studies only focus on drinking habits among college or university athletes (Nelson & Wechsler 2001, Martens et al. 2006a, Dams-O'Connor, Martin & Marten 2007, Lisha & Sussman 2010, Cadigan et al. 2013, Partington et al. 2013). An essential limitation of these cross-sectional studies is that they cannot differentiate the longitudinal course of risky drinking among individuals with variable degrees of athletic participation (Martens et al. 2006a). However, it has been observed that intercollegiate athletes who were more athletically involved demonstrated sharper increases in problem drinking (heavy drinking, alcohol-related problems) during and after their competitive sports careers (Cadigan et al. 2013). Alcohol-related problems were associated with disability and adaptation to life

after an athletic career among former icehockey players (Chambers 2002). It has also been found that alcohol use increased among former baseball players during their athletic careers, but alcohol consumption seemed to decrease during the last few years of their athletic careers (Mahoney 2002).

Presumably, most alcohol-related risk factors among the general population, such as sociodemographic and familial factors, are also applicable to athletes (Martens et al. 2006a). There is limited evidence on why athletes use more alcohol than nonathletes (Durkin, Wolfe & Clark 2005, Grossbard et al. 2005, Ford 2007, Yusko et al. 2008, Weaver et al. 2013), but there are some motivational factors behind athletes and their reasons for engaging in risky alcohol consumption behaviors (Martens et al. 2008, Martens et al. 2010). Sports-induced anxiety or pressure, the competitive nature of athletes, peer- or teammate-induced influence, and cultural relations between athletes and alcohol might be related to risky alcohol consumption (Brewer, VanRaalte & Linder 1993, Thombs & Hamilton 2002, Wilson, Pritchard & Schaffer 2004, Martens et al. 2006a, Dams-O'Connor, Martin & Marten 2007, Lisha & Sussman 2010, O'Brien et al. 2011). Correspondingly, stress can lead to burnout and hence to increased alcohol consumption among athletes (Gustafsson et al. 2007).

Furthermore, an athletic identity and the social environment are associated with alcohol use among athletes (Brewer, VanRaalte & Linder 1993, Leichliter et al. 1998, Thombs & Hamilton 2002). It is also generally known that alcohol, especially beer, distributors support sports, and commercials for alcohol are commonly used (Madden & Grube 1994, Green et al. 2001, Martens et al. 2006a). Externalizing behaviors may be associated with higher alcohol consumption and dependence (Grant et al. 2004, Barr & Dick 2019). Studies have suggested that team sports athletes are more extroverted and neurotic than endurance sports athletes (Allen, Greenlees & Jones 2013), whereas contact sports athletes score high on the antisocial and histrionic scales (Sohrabi, Atashak & Aliloo 2011). Bäckmand et al. (2001) observed that Finnish former power, combat, and team sports athletes were more extroverted compared to controls. Additionally, shooters and endurance sport athletes had lower neuroticism scores than controls (Bäckmand et al. 2001).

The same factors, especially personal and psychological factors, may be related to heavy drinking during and after an active athletic career. However, different factors can predispose individuals to alcohol-related problems at different ages (Lee et al. 2012). Studies have found that retirement can lead to increased alcohol use among older people, who use alcohol to combat loneliness, relieve tension, and counter boredom (Menninger 2002, Klimstra & Mahgoub 2010, Blazer & Wu 2011). Aside from loneliness, sociality can increase binge drinking, especially among young athletes (Lisha & Sussman 2010). It has been recognized that sports career termination causes significant changes in an athlete's personal and social life (Wylleman, Alfermann & Lavallee 2004). Adaptation to athletic retirement will be challenging if an individual has a high athletic identity and an active sports career that ends unexpectedly (Alfermann, Stambulova &

Zemaityte 2004). This might predispose the individual to alcohol-related problems (Chambers 2002). Conversely, it was observed that senior-year students who ended their participation in intercollegiate athletics consumed less alcohol than consistent athletes (Cadigan et al. 2013).

2.3.3 Alcohol-related diseases

The excessive use of alcohol is harmful globally, and alcohol consumption is the world's third largest risk factor for diseases (Lim et al. 2012, WHO 2014b, WHO 2014c). The increased mortality related to heavy alcohol consumption is associated with cirrhosis, pancreatitis, certain cancers, stroke, accidents, and external causes of death (Corrao et al. 2004, Di Castelnuovo et al. 2006, Lim et al. 2012). Furthermore, heavy alcohol use is especially associated with poor mental functioning (Salonsalmi et al. 2017).

Alcohol use is related to an increasingly higher risk of sport injuries compared to other injuries (O'Brien & Lyons 2000), especially among women (Gmel, Kuendig & Daeppen 2009). Furthermore, studies have shown that addictions, anxiety, depression, amnesia, and insomnia are associated with heavy alcohol consumption (Corrao et al. 2004, Smith & Randall 2012, Costin & Miles 2014). It has also been found that both binge and problem drinking are related to all-cause disability retirement (Salonsalmi et al. 2012). On the contrary, moderate alcohol use may decrease the risk of CHD (Corrao et al. 2004, Di Castelnuovo et al. 2006, Jayasekara et al. 2014, Roerecke & Rehm 2014, Kaprio 2015) and protect from myocardial infarct (Brügger-Andersen et al. 2009), but the research findings are conflicting. Yoon et al. (2020) observed in their meta-analysis that light to moderate alcohol use may protect from CVD, if individuals were younger than 41 years old, and they did not have more than two comorbidities. However, according to a Mendelian randomization meta-analysis, the reduction of alcohol use even among moderate drinkers is good for their cardiovascular health (Holmes et al. 2014).

Alcohol abuse can be classified into three categories: risky alcohol use, harmful alcohol use, and alcohol dependence (Table 2). The risk limits of alcohol consumption mean that amount of alcohol, which probably increases adverse effects for health (Seppä 2003). One standard alcoholic drink includes 12 g of pure alcohol (Alkoholiongelmaisen hoito 2015). According to the Finnish Institute for Health and Welfare (THL) (2014), in Finland, health counseling is recommended for women when their daily alcohol use is a minimum of 20 g of pure alcohol (about two standard drinks), or when binge drinking (minimum five standard drinks at time) occurs weekly. Correspondingly, for men, the limits are 40 g (about four standard drinks) per day or seven standard drinks at a time, occurring weekly (THL 2014). According to the Finnish Current Care Guidelines (2015), a high risk level among women and men means 12–16 and 23–24 standard drinks weekly, respectively. Further, a woman who uses 7 standard drinks per week and a man who uses 14 belong to the moderate risk group (Alkoholiongelmaisen hoito 2015).

TABLE 2 Classification, definition, and ICD-code for alcohol use.

Class	Definition* (ICD-code)
Risky alcohol use	The limits for risky alcohol use have been exceeded, but there are neither significant signs of harmful alcohol use nor dependence.
Harmful alcohol use	Individual has clearly recognizable and defined alcohol-related adverse physical and psychological effects but no dependence (F 10.1).
Alcohol dependence	A syndrome that is related to compulsive alcohol use, increased tolerance, and withdrawals and continues despite adverse effects (F 10.2).

*(DeWit et al. 2000, Weiss & Porrino 2002, Aalto 2010, Alkoholiongelmaisen hoito 2015)

The WHO developed the International Statistical Classification of Diseases and Related Health Problems (ICD), which has been used since 1996 and is currently in its tenth revision (i.e., ICD-10). In Appendix 1 are presented alcohol-related diseases according to their ICD-codes (ICD-8, ICD-9, or ICD-10): *Mental and behavioral disorders due to alcohol use* (ICD-10, F10.00–10.99), *alcohol dependence* (ICD-10, F10.20–10.29), *alcohol-related liver diseases* (ICD-10, K70.0–70.4, 70.9), *alcohol-related pancreatitis* (ICD-10, K86.00–86.01, 86.08), and *other alcohol-related diseases* (ICD-10, G31.2, G40.51, G62.1, G72.1, I42.6, K29.2). Alcohol-related diseases have been investigated for several decades, so both older and newer scientific articles and literature have been used in this section.

2.3.3.1 Mental and behavioral disorders due to alcohol use

Alcohol-related disorders can increase the risk of mental and behavioral disorders or alcohol psychosis (Ramsey et al. 2004, Nunes et al. 2006, Caton et al. 2007). It is important to note that when discussing the prevalence or incidence of mental and behavioral disorders due to alcohol use, primary alcohol psychoses, which are part of alcohol use, should be considered separately from the developmental processes of mental disorders or symptoms (Petrakis et al. 2002, Shivani, Goldsmith & Anthenelli 2002, Bhullar et al. 2013). To reliably diagnose other coincidental mental disorders usually requires four weeks without alcohol or that the symptoms occur before alcohol use (American Psychiatric Association, APA 2013). The most common alcohol psychoses are delirium tremens and alcohol-related hallucinations (Tienari 1993, Mäkelä 2003, Aalto 2010, Alkoholiongelmaisen hoito 2015). Generally, Wernicke–Korsakoff syndrome is categorized as an alcohol-related mental disorder, as it causes motor and cognitive disorders (Mäkelä 2003, Aalto 2010).

Binge drinking can cause anxiety, depression, amnesia, and insomnia among all individuals (Shivani et al. 2002, Smith & Randall 2012, Costin & Miles

2014). Conversely, alcohol can momentarily decrease anxiety by releasing endorphins, hence causing satisfaction (Vengeliene et al. 2008, Spanagel 2009, Costin & Miles 2014). Alcohol can affect an individual's personality by increasing aggressiveness and causing delusional disorders and bipolarity (Driessen et al. 1998, Shivani, Goldsmith & Anthenelli 2002, Bhullar et al. 2013, Engelhard et al. 2015) and is associated with a higher risk of schizophrenia (Niemi-Pynttäri et al. 2013). Alcohol use and schizophrenia may increase aggression (Räsänen et al. 1998). Alcohol psychosis is also related to a higher risk of aggressive behavior (Tiihonen et al. 1997).

2.3.3.2 Alcohol dependence syndrome

Alcohol dependence syndrome, alcoholism, is a chronic disease in which the body is addicted to alcohol use (Mäkelä 2003, Caetano & Babor 2006, Aalto 2010) and is a large public health problem (Holder et al. 2000, Boening et al. 2001, Kuoppasalmi, Heinälä & Lönnqvist 2019). Kiiskinen et al. (2020) observed that the polygenic risk score (PRS) for alcohol use is related to all-cause and alcohol-related morbidity. According to Gohen-Gilbert et al. (2015), genetic factors are significantly related to alcoholism, especially at young age. More advanced language abilities in childhood may predict heavy alcohol use in adolescence (Latvala et al. 2014). A good tolerance to alcohol may predispose an individual to alcohol dependence syndrome (Gohen-Gilbert et al. 2015).

Alcoholism is usually related to physiological dependence when the tolerance to alcohol is increased, and the individual has withdrawals (DeWit et al. 2000, Weiss & Porrino 2002, Aalto 2010). Biologically compulsive alcohol use is related to long-term stress, causing changes in the functionality of the central nervous system (Gianoulakis 2001, Weiss & Porrino 2002, Mäkelä 2003). Many mental disorders increase harmful alcohol use and the risk of alcohol dependence syndrome because alcohol is often used to relieve different anxiety, fear, and depression symptoms (Alcaniz et al. 2015).

2.3.3.3 Alcoholic liver disease

Heavy alcohol use causes harmful effects to the digestive tract, mouth, esophagus, stomach, intestine, liver, and pancreas (WHO 2014b). In 2003, alcoholic liver disease as a primary diagnosis caused 3002 hospital admissions in Finland (THL 2014). The most common alcohol-related liver diseases are fatty liver, hepatitis, and liver cirrhosis (Mann, Smart & Govoni 2003, Salaspuro 2003, Jiménez-Romero et al. 2015). Among both men and women, alcohol-related liver cirrhosis is typically developed through regular alcohol use for approximately over ten years (Rodes, Salaspuro & Sorensen 1993). Harmful alcohol use can cause hepatitis or other liver disease, but all heavy users will not contract liver disease (Hart et al. 2010, Stickel & Hampe 2012).

Alcoholic liver disease is caused by alcohol and by alcohol metabolism or metabolites (acetaldehyde, acetate) (Lieber 1985, lshak, Zimmerman & Ray 1991, Salaspuro 1993). Acetaldehyde can bond to many proteins by changing their function, which predisposes an individual to developing liver damage (Seppä,

Alho & Kiianmaa 2013). Another important factor in the developmental process of liver damage is the release of endotoxins into the systemic circulation and liver, which releases cytokines and hence causes inflammation that could damage liver cells (Seppä, Alho & Kiianmaa 2013).

2.3.3.4 Alcoholic pancreatic diseases

Alcohol is the most common cause of acute and chronic pancreatitis (Kristiansen et al. 2008), and acute pancreatitis is a common alcohol-related disease in Finland (Seppä, Alho & Kiianmaa 2013). However, the reasons for chronic pancreatitis are not well-known, but the most important risk factors are alcohol, gallstone disease, trauma, and genetic factors (Vonlaufen 2007). In 2013, alcoholic pancreatic disease as a primary diagnose caused 1903 hospital admissions in Finland (THL 2014).

The association between heavy alcohol use and pancreatitis has been known for many decades (Singh & Simsek 1990, Pikkarainen 1993, Steinberg & Tenner 1994). The most important factors due to pancreatitis are a blockage of the sinusoidal sphincter (Goff 1993), the enzymes in pancreas hypersecretion (Singh & Simsek 1990), and the pancreatic toxicity of alcohol or subsequently its metabolites (Nordback et al. 1991). Alcohol damages pancreatic cells, and subsequently as the trypsine enzyme is already activated in the pancreas, this causes inflammation (Pikkarainen 1993, Salaspuro 2003, Seppä, Alho & Kiianmaa 2013).

2.3.3.5 Other alcohol-related diseases

Alcohol affects the circulation in many ways (Kamsa-Ard et al. 2014). It dilates the peripherical blood vessels and increases blood pressure; it also increases sympathetic nervous system activation, heart rate, and minute volume (Gaziano et al. 2000, Kamsa-Ard et al. 2014, Mathews, Liedenberg & Mathews 2015). Binge drinking and hangover predispose individuals to arrythmias (Cohen, Kiatsky & Armstrong 1988), even among young people who do not have heart problems (Gaziano et al. 2000), whereas long-term and heavy alcohol use cause morphological changes inthe myocardium, such as cardiomyopathy, which causes heart failure (Regan 1990, Kupari 2003, Holmes et al. 2014).

According to the ICD-10 classification, other alcohol-related diseases include gastritis, polyneuropathy, alcohol-related dementia, and nervous system degeneration. Gastritis is often related to Helicobacter infection, alcohol use, or the use of anti-inflammatory drugs such as ibuprofen (Lee et al. 2008). Heavy alcohol use increases the risk of polyneuropathy because alcohol damages the peripheral nerves (Juntunen 1993, Peters et al. 2006, Chopra & Tiwari 2011). Alcohol-related dementia can develop as a result of long-term alcohol use, and cognitive disorders are associated with central nervous system degeneration (Hilbom 1993, Crews 2008, Ridley, Draper & Withall 2013, Costin & Miles 2014). Furthermore, alcohol is related to cancers (Vertio 1993, Corrao et al. 2004), hormonal disorders (Välimäki 1993), immunity insufficiency (Mutchnick & Lee 1988), and embryogenesis disorders (Spohr, Wilims & Steinhausen 1993).

2.3.4 Alcohol-related diseases and deaths among current and former athletes

There is a paucity of scientific data on alcohol-related diseases and deaths among current and former athletes. However, it has been found that alcohol-related harms are more common among college athletes than nonathletes of the same age (Nelson & Wechsler 2001, Dams-O'Connor, Martin & Marten 2007, Yusko et al. 2008). Correspondingly, athletes had higher mean Alcohol Use Disorders Identification Test (AUDIT) scores than nonathletes, and gaming disorder was associated with alcohol dependence more often among athletes compared to non-athletes (Weiss & Rundell 2011). This was consistent with later findings which additionally showed team sports athletes seemed to have higher mean AUDIT scores than individual sports athletes (Partington et al. 2013).

The ACSM Position Stand (1982) concludes that acute alcohol consumption may decrease cardiovascular and muscular endurance, strength, power, and speed. Furthermore, athletes who drank alcohol at least once weekly had a yearly injury rate of 54.8 %, whereas nondrinking athletes had an injury rate of 23.5 % (O'Brien & Lyons 2000). Studies have found that low to moderate doses of alcohol do not positively impact performance, rather endurance performance is decreased (McNaughton & Preece 1986, Kendrick, Affrime & Lowenthal 1994, Lecoultre & Schutz 2009). It is less clear whether alcohol use has an influence on measures of muscle strength, but studies have found myopathy among rats (Preedy & Peters 1988) and the erosion of lean body mass (Preedy, Salisbury & Peters 1994). Alcohol use might also be associated with a decline in muscle strength because muscle mass is positively associated with muscle strength (Kim et al. 2016). Correspondingly, it has been found that heavy alcohol use was associated with a greater decline in muscle strength in the Japanese population (Cui et al. 2019). However, Baumeister et al. (2018) found that moderate drinkers seemed to have better fitness compared to individuals with higher average alcohol use or current abstainers. Nevertheless, the results of the existing cross-sectional population-based studies investigating the association between CRF and alcohol use have been inconsistent (Montoye, Gayle & Higgins 1980, Tobita et al. 2003, Laukkanen et al. 2009). Although, heavy alcohol use among athletes is a general problem globally, there is a lack of intervention studies focused on prevention or management (Kingsland et al. 2016).

2.4 Smoking and chronic pulmonary diseases and deaths among current and former athletes

2.4.1 Smoking

Tobacco smoking is estimated to lead to the premature deaths of approximately 6 million people worldwide (WHO 2013). Most smoking-related deaths arise from respiratory diseases (mainly COPD), CVDs (mainly CHD), and cancers

(mainly lung cancer) (Action on Smoking and Health 2016). Furthermore, smoking increases the risk of blindness, deafness, osteoporosis, back pain, and peripheral vascular disease (may lead to amputation) (Peto 1994, US Department of Health and Human Services 2004, Jemel et al. 2008). Smoking is also a major risk factor for brain damage, such as stroke, vascular dementia, and Alzheimer's disease (US Department of Health and Human Services 2004, Ferri et al. 2011).

Finnish men's and women's tobacco smoking has decreased since the 1980s, but women's smoking seems to have remained at the same level since the mid-2000s. In 2017, the prevalence of daily smoking was 13 % for Finnish men and 10 % for women aged 20 to 84 years (THL 2018b). In the USA in 2018, smoking seemed to be most common among divorced people, people with low SESs, and people aged 25–44 years and 45–64 years (Creamer et al. 2019). It has also been observed that parents' smoking behavior increases the risk of smoking among their children (Sargent et al. 2001). Correspondingly, a lower childhood SES is associated with a lower PA and higher prevalence of smoking in later life (Puolakka et al. 2018).

2.4.2 Smoking among current and former athletes

Smoking is associated with less leisure-time PA in cross-sectional and longitudinal studies (Escobedo et al. 1993, Pate et al. 1996, Audrain-McGovern et al. 2003). Habitual physical inactivity during late adolescence as compared to continuous PA through adolescence predicts a higher prevalence of smoking during young adulthood even after familial, including genetic, factors are considered (Kujala, Kaprio & Rose 2007). It has been shown that current athletes smoke less than nonathletes (Wechsler et al. 1997, Peretti-Watel et al. 2003, Martinsen & Sundgot-Borgen 2014). Elite athletes also smoke less after concluding their competitive careers (Sarna et al. 1993, Fogelholm, Kaprio & Sarna 1994, Bäckmand et al. 2010). However, smokeless tobacco with nicotine is generally used to enhance performance, especially among younger athletes, but it is very addictive and increases the risk of cardiovascular disorders (Chagué et al. 2015).

The association between cigarette smoking and sports injuries has been studied less (Sacks & Nelson 1994, Wen et al. 2005, Iacobelli et al. 2008). However, it has been reported that smokers have up to a 2.5 times higher risk of injury compared to nonsmokers (Tsai et al. 1990, Sacks & Nelson 1994, Leistikow et al. 1998). Smoking decreases CRF (Stea et al. 2009, Hamari et al. 2010) and, furthermore, evidence of adverse effects on cartilage, ligaments, tendons, and muscles has been also found (AL-Bashaireh et al. 2018). Smoking and low fitness are both independent risk factors for sports injury, but the combined influence on low fitness and smoking may result in an even greater risk for sports injury (Brooks et al. 2019).

2.4.3 Chronic pulmonary diseases

Chronic respiratory diseases (CRDs) are diseases of the airways and other structures of the lung (WHO 2019b). Airway obstruction increases the risk for all-

cause mortality according to the severity of the Global Initiative for Chronic Obstructive Lung Disease (GOLD) stage (Mattila et al. 2015, for more details of GOLD stages, see Appendix 2). The two most prevalent obstructive lung diseases are asthma and COPD, which affect millions of individuals worldwide, and their incidences are rising globally (GINA 2012, GOLD 2013). The development of COPD and mortality are highly correlated with cigarette smoke exposure (Mannino et al. 2000, Hooper et al. 2012). Current smoking and previously diagnosed asthma are the strongest risk factors for airway obstruction (Vasankari et al. 2019), whereas the decreased risk for airway obstruction is associated with a high alternate healthy eating index (AHEI) (Vasankari et al. 2019).

Both COPD and asthma are characterized by chronic airway inflammation and airflow limitation, but systemic inflammation may also be present (Barnes & Celli 2009). Both diseases are heterogeneous and multifactorial, displaying a variety of inflammation and symptoms (Rennard & Drummord 2015, Kleinert & Horton 2018). However, there is also evidence that the inflammatory processes in asthma and COPD differ. Findings suggest that there is no strong common genetic component in asthma and COPD (Smolonska et al. 2014), although a genetic correlation between the two has been discovered (Hobbs et al. 2017). Furthermore, it has been found that a low SES, as measured by educational and income levels, is a risk factor for both asthma and COPD (Kanervisto et al. 2011).

In the clinical setting, patients who present with a combination of asthma and COPD-related traits are common (Hedman et al. 1999, Guerra 2009, Kauppi et al. 2011). Overlap syndrome can develop when there is an accelerated decline in lung function or incomplete lung growth or both (Gibson & Simpson 2009). Patients with overlapping asthma and COPD are associated with a low health-related quality of life (HRQoL) (Kauppi et al. 2011). The significant risk factors for accelerated decline in lung function and incomplete lung growth are aging, tobacco smoke exposure, bronchial hyperresponsiveness, asthma, and lower respiratory exacerbations or infections (Gibson & Simpson 2009).

In Appendix 3 are presented chronic pulmonary diseases according to their ICD-codes (ICD-8, ICD-9, or ICD-10): *asthma, chronic bronchitis, emphysema, other obstructive pulmonary diseases (COPD), and bronchiectasia.* The classification and definition of COPD has changed over the last decades and is likely to change in the future.

2.4.3.1 Asthma

Asthma is a chronic pulmonary disease described as wheezing and recurrent attacks of breathlessness (WHO 2019a). However, the Lancet Commission has argued that asthma is not an adequate name because comorbidities, lifestyle, and environmental factors should be considered, and the underlying treatable traits should be recognized (Kleinert & Horton 2018). Asthma is generally related to two major pathological features: airway inflammation and bronchial hyperresponsiveness (GINA 2012). Bronchial hyperresponsiveness due to asthmatic airway inflammation causes a reversible and variable airway obstruction typical of asthma and leads to recurrent episodes of breathlessness, wheezing, and chest

tightness (GINA 2012). Generally, asthmatic airway inflammation is associated with tissue eosinophilia and an IgE-mediated allergy, but there are also other types of asthma (Pavord 2012, Wenzel 2012).

As previously mentioned, asthma is a heterogenic and multifactorial disease that has different symptoms (Kleinert & Horton 2018). Asthma occurs worldwide, with an estimated 300 million patients, and globally, it`s prevalence ranges from 1 % to 18 % of the population in different countries (Masoli et al. 2004). Furthermore, asthma is the most common chronic disease among children (WHO 2019a). In Finland, the prevalence of self-reported, physician-diagnosed asthma in the adult population was 6.8 % in 1996 as compared to a prevalence of 9.4 % in 2007 (Pallasaho et al. 2011). However, asthma is still underdiagnosed and -treated, creating a substantial burden to individuals and families and possibly restricting individuals' activities for a lifetime (WHO 2019a).

2.4.3.2 Chronic obstructive pulmonary disease (COPD)

COPD is a slowly progressive lung disease causing non-reversible airway obstruction and leading to major morbidity and mortality worldwide (Keuhkoahtaumatauti 2014, WHO 2017). As previously stated, COPD is a heterogeneous collection of diseases with differing pathogenic mechanisms, causes, and physiological effects (Rennard & Drummord 2015). Various classification systems and phenotypes of COPD have been proposed (Manian 2019). An awareness of the four predominant clinical COPD phenotypes can lead to more accurate diagnostics and treatments specifically targeted for a specific subpopulation (Mazur, Laitinen & Kinnula 2013). Conventionally, COPD is subdivided into several entities, including chronic bronchitis (CB) and emphysema (Shapiro et al. 2010), small-airway obstruction, and systemic inflammation (Barnes 2004, Sinden & Stockley 2010, GOLD 2013).

COPD is the fourth leading global cause of death, but the WHO predicts that it will rise to third place by 2030 (WHO 2014a). COPD was the third leading cause of death in the United States in 2013 (Kochanek et al. 2014), and in Finland, about 1000 individuals die from COPD yearly (Keuhkoahtaumatauti 2014). The prevalence of COPD stood at 10.1 % in an international study (Doll et al. 2004), and at 3.6 % in one Finnish study (Kainu et al. 2013); another study placed the prevalence in Finland at 4.3 % in men and 3.1 % in women (Vasankari et al. 2010). A cohort study showed significant differences related to gender in the clinical presentation of COPD: men had more advanced airway obstruction but less severe gas transfer impairment than women (Laitinen et al. 2009).

Smoking is well-established as the main cause of COPD, and cigarette smoke exposure has been highly correlated with the development of COPD and mortality (Franklin et al. 1956). It has been found that COPD patients were more frequently heavy former or current smokers compared to patients with asthma (Hirvonen et al. 2019). Furthermore, significant risk factors for COPD in many countries are occupational exposures to noxious particles or gases and to both outdoor and indoor air pollutants (GOLD 2013). Exposure to tobacco smoke or

other triggering factors induces pulmonary and low-grade systemic inflammation (Sinden & Stockley 2010). The chronic pulmonary inflammation in COPD causes small-airway injury and fibrosis, leading to irreversible airway obstruction and emphysema (Barnes 2004). The systemic inflammation in COPD is associated with cardiovascular diseases, cachexia, skeletal muscle dysfunction, osteoporosis, and depression (Agusti et al. 2003, Barnes & Celli 2009), which are typical comorbidities in COPD. Thus, the term *chronic systemic inflammatory syndrome* would be better for describing this disease (Fabbri & Rabe 2007). Because of the large social and economic burden of COPD, it is good to remember that it is preventable and treatable (GOLD 2013), and much can be done to help patients to cope with this multidimensional disease. For instance, an exercise program tailored to each patient's physical capabilities is beneficial to COPD patients (Katajisto et al. 2012), PA decreases the number of exacerbations and short-acting bronchodilator use among COPD patients (Katajisto et al. 2015).

As previously mentioned, COPD is a heterogeneous collection of diseases, with CB at one end, and emphysema at the other, and most patients have some characteristics of both. Epidemiologic studies have used different definitions of CB during the years, but the classic definition comprises sputum production and a chronic cough for at least 3 months per year for two consecutive years (Pauwels et al. 2001). CB has various clinical consequences, including an accelerated decline in lung function, an increased exacerbation rate, a worse HRQoL, and a possibly increased mortality (Vestbo et al. 1996, Burgel et al. 2009, Kim et al. 2011). Smoking is the primary risk factor for CB (Pelkonen et al. 2006). However, it should be noted that CB is described in 4 % to 22 % of never-smokers (Miravitlles et al. 2006, Pelkonen et al. 2006), suggesting that other risk factors exist. Other potential risk factors are dusts, chemical fumes, and inhalational exposures to biomass fuels (Trupin et al. 2003, Matheson et al. 2005). The presence of gastroesophageal reflux is another potential risk factor for CB, possibly via pulmonary aspiration of refluxed gastric contents, producing acid-induced injury and infection or neurally mediated reflex bronchoconstriction secondary to irritation of the esophageal mucosa (Barish et al. 1985, Smyrnios, Irwin & Curley 1995). CB is seen among 3.4 % to 22.0 % of adults (Lange et al. 1989, Pallasaho et al. 1999, Sobradillo et al. 1999, von Hertzen et al. 2000, Miratvilles et al. 2006), and its prevalence is higher in individuals with COPD, affecting 14 % to 74 % of these individuals (Burgel et al. 2009, Agusti et al. 2010, Kim et al. 2011, de Oca et al. 2012).

Pulmonary emphysema is a phenotypic component of COPD, bearing a poor prognosis and substantial lung function impairment (Coxson et al. 2013, Fernandez-Bussy, Labarca & Herth 2018, Dunlap et al. 2019, Rustagi et al. 2019). Emphysemic patients have a worse quality of life, lower BMI, and less exercise tolerance compared to nonemphysemic ones (Makita et al. 2007, Miniati et al. 2008, Pistolesi et al. 2008, Ogawa et al. 2009). In addition, emphysema is a strong predictor of reduced survival (Johannessen et al. 2013), independently of coexisting cardiovascular or metabolic disorders (Miniati et al. 2014). Protease/antiprotease imbalance, triggered by chronic lung inflammation, has been found as a pathogenetic mechanism of emphysema among smokers (Snider 1992).

2.4.3.3 Bronchiectasis

Bronchiectasis is a chronic respiratory disease of airway dilatation, where the patient typically suffers from respiratory infections, sputum, cough, dyspnea, fatigue, and poor quality of life (Spinou et al. 2016, Guan et al. 2018). Over the past years, this condition has received increased interest, with important developments in establishing national and international patient registries (Chalmers et al. 2016, Araújo et al. 2018, Visser et al. 2019), disease-specific health status questionnaires (Quittner et al. 2014, Spinou et al. 2017), and randomized controlled trials of new treatments (Aksamit et al. 2018, De Soyza et al. 2018, Haworth et al. 2019). A number of new treatment approaches have been proposed, including immune-modulating drugs and long-term antibiotic therapies (Altenburg et al. 2013, De Soyza et al. 2015, Aksamit et al. 2018, De Soyza et al. 2018). Most healthcare professionals agree that airway clearance and exercise are the most important parts of caring for bronchiectasis, but this area has received the least attention (Herrero-Cortina et al. 2016, Wong, Sullivan & Jayaram 2018), as the evidence base for respiratory physiotherapy has not been advanced at the same pace as other aspects of bronchiectasis treatment (Chalmers & Chotirmall 2018).

2.4.4 Chronic pulmonary diseases and deaths among current and former athletes

The positive effect of exercise and a physically active lifestyle has been documented in respiratory diseases (Donesky-Cuenco et al. 2007), including asthma (Welsh, Kemp & Roberts 2005, Counil & Voisin 2006). Moderate exercise has an immunostimulatory effect, in contrast to intense exercise, which temporarily decreases immune system function (Nieman 2003). Previous studies have found that the intense exercise necessary in competitive sports is related to a three times higher incidence of upper respiratory tract infections (URTI) (Spence et al. 2007). Moreover, polymorphisms in cytokine genes may have an effect on the risk for URTI among some athletes (Cox et al. 2010). Respiratory problems are common among athletes of all abilities and, although regular PA promotes health and well-being, there is a legitimate concern that certain environmental conditions (such as indoor swimming) with repeated and frequent periods of high ventilation might be detrimental to an individual's respiratory health (Weiss & Rundell 2011). It has been observed that the prevalence of asthma among swimmers was higher compared to the general population, but there was no difference between swimmers and other endurance sports athletes (Päivinen, Keskinen & Tikkanen 2010).

Despite large variations in the prevalence of between sports and reports, exercise-induced asthma (EIA) and exercise-induced bronchoconstriction (EIB) are some of the most common chronic medical findings among Olympic athletes (Moreira, Delgado & Carlsen 2011, Fitch 2012). In particular, airway hyperresponsiveness (AHR) and asthma are the most common chronic medical conditions experienced by both Summer and Winter Olympic athletes (Fitch 2012). Correspondingly, Turcotte et al. (2003) and Helenius et al. (2005) found that

asthma, AHR, and EIB appear to be more common in elite athletes than in less-trained controls. During high-intensity exercise, the risk for airway epithelium injury is increased among those athletes who sustain high ventilation during exercise and/or those who are exposed to unfavorable environmental conditions (such as chlorinated indoor pools or polluted or cold, dry air) (Bougault & Boulet 2012, Rundell 2012, Sue-Chu 2012). It has been found that the epithelium's repeated injury and repair process could be the underlying cause for the development of AHR/asthma (Kippelen & Anderson 2012).

Correspondingly, studies suggest that self-reported and physician-diagnosed asthma are twice as common in Finnish athletes (Alaranta et al. 2004) and elite Norwegian athletes (Nystad, Harris & Borgen 2000) than in randomly selected age-matched and sex-matched control populations. The higher prevalence of asthma reported in athletes may be a result of overdiagnosis, particularly because a diagnosis of asthma is often made on the basis of the individual's history alone (Parsons et al. 2006), and athletes often experience symptoms only during intensive exercise. However, there is a lack of studies focused on the risk of chronic pulmonary diseases among former athletes several decades after their peak sporting performance. Kujala et al. (1996) reported that the lifetime occurrence of asthma or other pulmonary diseases was not increased in former elite athletes, and exercise alone, even in a cold environment, was not associated with a greater prevalence of asthma in later life.

In summary, respiratory disorders are often a cause of morbidity in top-level endurance athletes, more often than that compromising their performance and rarely being a cause of death (Bussotti, Di Marco & Marchese 2014). However, there is a lack of consensus on whether vigorous PA in a cold environment predisposes individuals to chronic pulmonary diseases. Some studies have found that subjects that routinely perform strenuous exercise in cold conditions have a high prevalence of chronic airway inflammation and hyperreactivity (Sue-Chu, Larsson & Bjermer 1996); further, cold weather exercise can lead to an asthma-like airway disease (Davis et al. 2005). Whether early prevention of airway injury in elite athletes can prevent or reduce progression to AHR/asthma remains to be seen, but in particular, environmental interventions are the most important (Kippelen et al. 2012). Additionally, it has been suggested that genetic analyses may have a predictive value in the identification of individuals who are more likely to experience recurrent infection when exposed to high physical stress (Zehsaz 2015). Interactions between genetic factors and early environmental exposure patterns may explain the early development of chronic obstructive lung diseases (Postma, Bush & Van den Berge 2015). In particular, genome-wide association studies (GWASs) have identified genetic variants in the development of lung function in early life and in the later progression to asthma and COPD (Hall, Hall & Sayers 2019). Finally, the prevention of chronic pulmonary diseases should be a major public health goal among athletes and nonathletes (Rennard & Drummord 2015, Kleinert & Horton 2018).

2.5 Mortality among current and former athletes compared to the general population

Observational studies have found that regular PA is associated with better functioning and well-being, especially at an older age (King et al. 2000, Paterson & Warburton 2010), a lower risk for morbidity (Kujala et al. 2003a, Gajewski & Poznanska 2008), and all-cause mortality (Sarna et al. 1993, Kujala et al. 1998, Samitz, Egger & Zwahlen 2011, Eijsvogels et al. 2016). Samitz et al. (2011) reported that the increase in death risk reduction per unit of time was the largest for vigorous exercise. Long-term vigorous exercise training is associated with increased survival rates in specific groups of athletes (Teramoto & Bungum 2010). Clarke et al. (2015) found that Olympic medalists live longer than the general population.

In particular, former endurance sports athletes have a higher longevity and lower mortality than the general population (Sarna et al. 1993, Kettunen et al. 2015). For instance, previous studies have reported inconsistent results for power sports athletes (Clarke et al. 2015), and NFL players had a 52 % greater risk of cardiovascular-related death than the general population (Baron & Rinsky 1994). In general, runners have a 25 % - 40 % reduced risk of premature mortality and live approximately 3 years longer than nonrunners (Lee et al. 2017). However, some epidemiological studies also suggest that very intense patterns of exercise may increase the risk for cardiovascular morbidity and mortality compared to light to moderate amounts of exercise (Eijsvogels et al. 2016, Kim & Baggish 2017). Although a high lifelong exercise volume among surviving athletes is associated with coronary artery calcifications, the atherosclerotic plaques of the most active athletes have a more benign composition (Aengevaeren et al. 2017).

There is no irrefutable evidence for a causal relationship between PA in adulthood and mortality, either in animal experiments or in randomized controlled intervention studies with healthy individuals (Karvinen et al. 2015). A high level of CRF is associated with a lower risk of all-cause and CVD mortality in both rats (Koch et al. 2011) and in humans (Kodama et al. 2009). It has been previously widely reported that former athletes have a lower morbidity and mortality compared to the general population (Sarna et al. 1993), which may be due to their genetic backgrounds, increased PA levels, or other better health habits (Kujala et al. 2003a, Gajewski & Poznanska 2008, Kettunen et al. 2015). However, the evidence is mostly based on observational follow-up studies, one limitation of which is the difficulty of controlling for genetic or other selection bias. It is debated whether high-intensity exercise is beneficial for reducing mortality risk in later life (Blair et al. 1992, Blair & Connelly 1996). Most studies have focused on professional athletes and used the general population as a control group. The findings from these studies differ: some studies showed a lower mortality in athletes than in their nonathletic counterparts from the general population (Karvonen et al. 1974, Menotti et al. 1990, Sarna et al. 1993, Kujala et al. 2001, Lee & Skerrett 2001, Gajewski & Poznanska 2008, Teramoto & Bungum 2010, Garatachea, Santos-Lozano & Sanchis-Gomar 2014, Kettunen et al. 2015),

whereas other studies did not find a survival benefit (Schnohr 1971, Beaglehole & Stewart 1983, Waterbor et al. 1988, Belli & Vanacore 2005, Kuss, Kluttig & Greiser 2011, DeKosky, Jaffee & Bauer 2018, Nguyen, Zafonte & Chen 2019).

In summary, it is generally accepted that leisure-time PA is associated with better health, well-being, and a reduced risk of cardiovascular and all-cause mortality. This association has been found in various studies, most of which already reflect these results in meta-analyses or systematic reviews (Erlichman, Kerbey & James 2002, Bauman 2004, Melzer, Kayser & Pichard 2004, Garatachea, Santos-Lozano & Sanchis-Gomar 2014). The lower mortality risk of professional athletes compared to the general population could be due to specific social and psychometric characteristics, and whether high intensity exercise brings an increased mortality risk or a survival benefit for athletes remains to be elaborated. Zwiers et al. (2012) found that engagement in sports with high-intensity exercise did not result in a survival benefit compared to sports with low-intensity exercise among former Olympic athletes. Furthermore, it is not clear whether vigorous-intensity activity gives an additional benefit beyond its contribution to PA volume when compared to moderate-intensity activity (Lee & Skerrett 2001).

3 AIMS OF THE STUDY

The health status of former Finnish male athletes has been previously widely studied, and it has been observed that former athletes have a lower morbidity and mortality than the general population, which may be due to their genetic backgrounds, higher PA levels, or other better health habits (Sarna et al. 1993, Fogelholm, Kaprio & Sarna 1994, Kujala et al. 1996, Kujala et al. 2003a, Bäckmand et al. 2010, Kettunen et al. 2015). However, there is a paucity of longitudinal studies focused on the risk of alcohol-related diseases or deaths and alcohol consumption among former athletes several decades after their peak athletic performance. Correspondingly, little is known about how the continuation of PA, smoking habits, and alcohol use contributes to former athletes' health in later years. Very little is known about the associations between participation in vigorous sports, health habits, familial factors, and subsequent mortality. So, the present dissertation includes novel data and findings that have not been previously analyzed from the Finnish former athlete cohort or, to author's knowledge, from any other comparable cohort worldwide.

The present dissertation had four specific objectives (*Studies I–IV*):

I. *The first aim was to examine the effects of long-term habitual physical activity (PA) and sports groups on the behavioral health–related factors, such as alcohol consumption and smoking, of middle-aged and aging men (Study I).*

Regular PA plays an important role in the prevention and treatment of many chronic diseases and in the reduction of disabilities in elderly people. The purpose of *Study I* was to investigate the direction and magnitude of the associations between PA, alcohol use, and smoking at four time points (the 1985, 1995, 2001, and 2008 questionnaire studies) across middle age and older age after an active sports career among former elite athletes and controls. *Study I* also investigated other background factors, such as reasons for quitting a sports career, related to the use of alcohol and smoking. Furthermore, it was examined whether the characteristics

of longitudinal latent profiles related to alcohol use, smoking and PA during the follow-up.

II. *The second aim was to examine alcohol use and alcohol-related morbidity and mortality in Finnish former elite athletes and their matched controls (Study II).*

Retirement from elite sports has been investigated over the last few decades (Alfermann, Stambulova & Zemaityte 2004, Stephan et al. 2007), and it has been recognized that sports career termination causes significant changes in an athlete's personal and social life (Wylleman et al. 2004). There is much discussion on binge drinking among former athletes in media, but there is a paucity of evidence on the impact of a history of competitive sports on later alcohol use and the occurrence of alcohol-related diseases. Furthermore, it was appropriate to investigate the existence of differences in alcohol consumption and alcohol-related diseases or deaths between former athletes from different sports.

III. *The third aim was to explore the risk and determinants (PA and smoking) of chronic pulmonary diseases among former elite athletes in later life (Study III).*

There is little evidence about the way in which vigorous PA during young adulthood is associated with the risk of chronic pulmonary diseases in later life. *Study III* had a different focus compared to the previous study of the same cohort (Kujala et al. 1996), e.g. updated registration data, and several decades' longer follow-up time. *Study III* had three aims, the first of which was to examine how a former competitive sports career and smoking were associated with the incidence and mortality of any chronic pulmonary diseases during three decades of follow-up. Second, these risks were compared between different sports groups. Third, it was studied how different factors, such as PA, were associated with smoking among former athletes after their active athletic careers.

IV. *The fourth aim was to investigate whether all-cause mortality and the development of behavioral and biological risk factors differ between former athletes and their brothers in later life (Study IV).*

There is no scientific data on the differences in behavioral health–related factors, such as PA, alcohol use, and smoking, among former elite athletes and their brothers. Therefore, *Study IV* examined whether the development of former elite athletes' behavioral and biological risk factors differed from those of other athletes and their brothers in later life.

4 PARTICIPANTS AND METHODS

4.1 Participants

According to Sarna et al. (1993), an original cohort of former elite athletes (n=2657) was formed by identifying men who had represented Finland at least once in the Olympic Games, European or World championships, or international contests between two or three countries between 1920 and 1965. A control cohort (n=1712) was selected from Finnish men who at the age of 20 years had been identified as healthy in the medical inspection for enlisting in ordinary military service. The control cohort was formed by matching the same age groups and area of residence with those of the former elite athletes (Sarna et al. 1993). The athletes' brothers' (n=2674) data were collected from local parish registry data in early 1980s.

Studies I, II, and *III* consisted of male former athletes and matched controls, and *Study IV* also included former athletes' age-matched brothers. In *Studies I, III,* and *IV*, the former athletes were grouped into three groups based on the type of training needed to achieve optimal results: endurance, mixed, and power sports (Åstrand & Rodahl 1986). For the purpose of *Study II*, the former male athletes were classified differently, and shooters were also included. According to Sarna et al. (1993), endurance sports included long- and middle-distance running and cross-country skiing. Mixed sports consisted of soccer, ice hockey, basketball, high jumping, pole vaulting, long jumping, triple jumping, hurdling, short-distance running, and the decathlon. Power sports included weightlifting, boxing, wrestling, and shot-putting as well as discus, javelin, and hammer throwing (Sarna et al. 1993).

As with the target group (participants), there were differences in the follow-up time and the classification into sports groups because of the different aims of *Studies I–IV*. For example, because of the study's purpose and the literature review (studies have shown that team sports athletes use more alcohol than other athletes), it was more appropriate to investigate more specific sports groups

(such as individual versus team sports) in *Study II* compared to the three previously mentioned sports groups in Studies *I, III,* and *IV*.

Study I Participants who had died before the follow-up started on January 1, 1985, were excluded from this study. Thus, the final study population (n=2732) consisted of 1633 former athletes and 1099 matched control participants. The male former elite athletes were divided into three groups according to the type of training needed to achieve optimal results: endurance (n=287), mixed (n=769), and power sports (n=577) (Åstrand & Rodahl 1986). Endurance sports included long- (n=128) and middle-distance running (n=66) and cross-country skiing (n=93). Mixed sports consisted of soccer (n=199), ice hockey (n=144), basketball (n=80), high jumping (n=39), pole vaulting (n=43), long jumping (n=26), triple jumping (n=30), hurdling (n=74), short-distance running (n=99), and the decathlon (n=35). Power sports included weightlifting (n=91), boxing (n=177), wrestling (n=182), and shot-putting (n=29) as well as discus (n=29), javelin (n=41), and hammer throwing (n=28). The former elite athletes (n=1255, 82 % response rate in 1985) and controls (n=764, 76 % response rate in 1985) who answered the PA-, alcohol-, or smoking-related questions in any of the questionnaires were included in the statistical analysis.

Study II Participants who had died before the register-based follow-up started on January 1, 1970, were excluded from this study. Thus, the final study population (n=3605) consisted of 2202 former male athletes and 1403 matched control participants. For the purpose of this study, the former male athletes were classified into the following groups according to sports and their specific physical loading types: short-& middle-distance running (n=202), endurance sports (n=280, including long-distance running and cross-country skiing), jumping & hurdling (n=251, including high jump, pole vault, long jump, and triple jump), throwing & decathlon (n=211, including javelin, discus, shot-putting, and hammer), weightlifting (n=111), combat sports (n=488, including wrestling and boxing), team sports (n=488, including soccer, ice hockey, and basketball), and shooting (n=171).

Study III Participants who had died before the register-based follow-up started on January 1, 1970, were excluded from this study. Thus, the final study population (n=3531) consisted of 2078 former male athletes and 1453 matched control participants. The male former elite athletes were divided into three groups according to the type of training needed to achieve optimal results: endurance (n=359), mixed (n=924), and power sports (n=795) (Åstrand & Rodahl 1986). Endurance sports included long- (n=162) and middle-distance running (n=79) and cross-country skiing (n=118). Mixed sports consisted of soccer (n=262), ice hockey (n=156), basketball (n=84), high jumping (n=46), pole vaulting (n=54), long jumping (n=35), triple jumping (n=34), hurdling (n=85), short-distance running (n=123), and the decathlon (n=45). Power sports included weightlifting

(n=115), boxing (n=253), wrestling (n=261), and shot-putting (n=38) as well as discus (n=35), javelin (n=56), and hammer throwing (n=37).

Study IV Due to missing or uncertain information on date of birth or date of death or due to death before 1936, 219 athletes and 1774 brothers were excluded. Correspondingly, brothers who had died before the time when the athlete started an elite athletic career were excluded. After including only athletes who had brothers with complete data, the final study population (n=2755) consisted of 900 former male athletes and their 1855 brothers. Former elite athletes (n=900) and their individually matched brothers who were closest in age (n=900) were included in the primary mortality analysis (paired Cox regression model). The former male athletes were classified into the following three groups according to sports and sports-specific aerobic fitness characteristics: endurance sports (n=217), mixed sports (n=307), and power sports (n=376) (Åstrand & Rodahl 1986). Endurance sports included long-(n=95) and middle-distance running (n=45) and cross-country skiing (n=77). Mixed sports consisted of soccer (n=75), ice hockey (n=33), basketball (n=22), high jumping (n=17), pole vaulting (n=23), long jumping (n=14), triple jumping (n=15), hurdling (n=36), short-distance running (n=46), and the decathlon (n=26). Power sports included weightlifting (n=43), boxing (n=94), wrestling (n=155), and shot-putting (n=18) as well as discus (n=15), javelin (n=32), and hammer throwing (n=19). A sensitivity analysis based on former elite athletes (n=900) and all of their brothers (n=1855) was also conducted.

4.2 Study designs and methods

4.2.1 Study designs

This dissertation utilized several data sets, which were originally collected to primarily answer research questions other than those of the present dissertation. The register-based data sets (*Studies II–IV*) were updated, and all data sets (basic information, register- and questionnaire-based data sets) were rigorously modified and combined. All *Studies I–IV* utilized the basic information data set, which was combined with the other data sets. Furthermore, *Study I* was based on questionnaire data, whereas *Studies II–IV* were based on both registries and questionnaires. The present dissertation was based on longitudinal cohort studies, and the study designs are presented in Figure 1 (*Studies I–IV*).

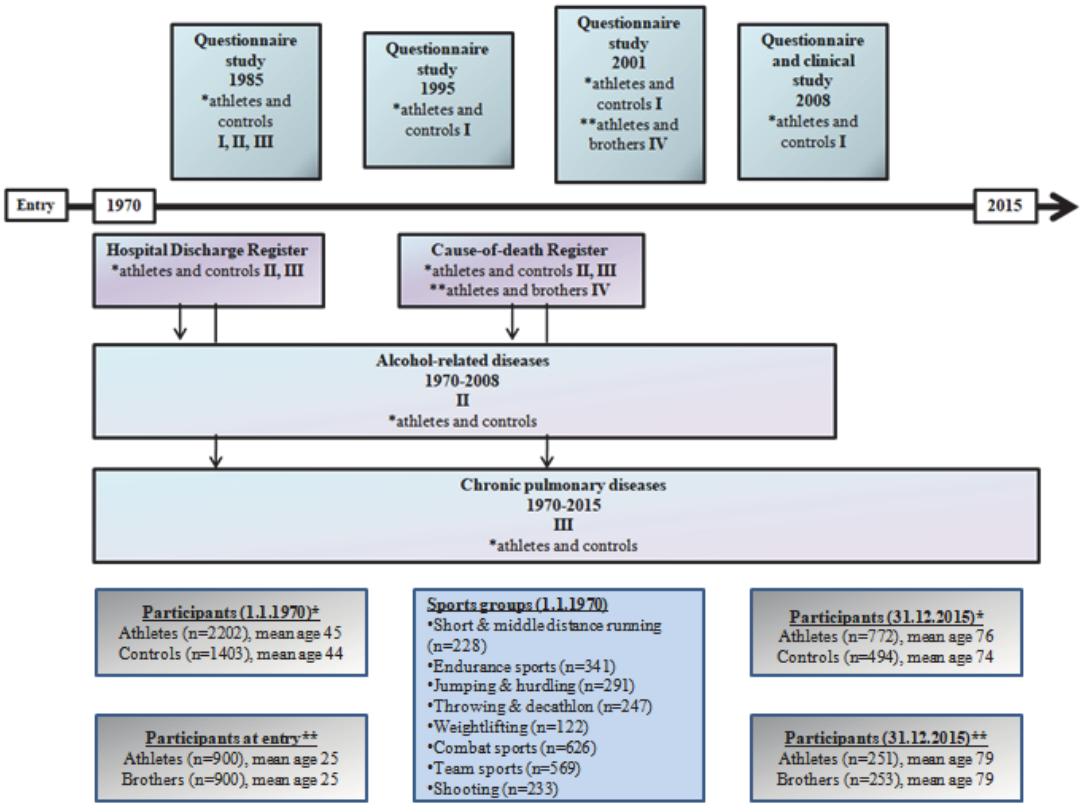


FIGURE 1 Study designs of *Studies I–IV* during the years.

4.2.2 Registries

The register-based follow-up of hospitalizations started on January 1, 1970 (*Studies II* and *III*), and ended on December 31, 2008 (*Study II*) and 2015 (*Study III*). Using the National Hospital Discharge Register, participants who had hospital admissions for alcohol-related diseases (*Study II*) or chronic pulmonary diseases (*Study III*) were identified from among patients admitted to Finnish hospitals for the primary treatment of alcohol-related diseases (*Study II*) or chronic pulmonary diseases (*Study III*), according to the ICD-codes (ICD-8, ICD-9, or ICD-10). Alcohol-related diseases, which were strongly related to alcohol use, were categorized into five main groups: *mental and behavioral disorders due to alcohol use*, *alcohol dependence syndrome*, *alcoholic liver disease*, *alcoholic pancreatic diseases,* and *other alcohol-related diseases* (*Study II*) (For more details, see Appendix 1). There are changes in the diagnostic category in the ICD-10, as COPD is defined as *other chronic obstructive pulmonary disease* (J 44.0), but earlier ICD-codes (ICD-8, ICD-9) do not include this specific code (*Study III*). Chronic pulmonary diseases were categorized into five main groups: *chronic bronchitis*, *emphysema*, *other obstructive pulmonary diseases*, *asthma,* and *bronchiectasia* (*Study III*) (For more details, see Appendix 3). The primary diagnosis was used to determine the reason for hospitalization, and the secondary diagnoses were ignored in the analysis. In Finland, the overall correspondence between individual hospital discharge records and their written

patient histories for different diagnoses has been reported to be 94.4 % (Keskimäki & Aro 1991).

Participants who died from alcohol-related diseases (*Study II*) or chronic pulmonary diseases (as the underlying cause of death) (*Study III*) or any reasons (*Study IV*) were identified from the National Death Register of Statistics Finland (cause of death alcohol-related diseases or accidental poisoning by alcohol, code 41, chronic bronchitis, emphysema and asthma, grouped according to Statistics Finland internal codes 33 and 34).

4.2.3 Questionnaires

Questionnaires eliciting information on the discontinuation of a sports career, sociodemographic factors (including occupational loading), PA, and health-related lifestyle habits, such as alcohol consumption and smoking, were sent to the surviving former athletes and controls in 1985 (*Studies I, II,* and *III*), 1995 (*Study I*), and 2001 (*Studies I* and *IV*). The Mini-Finland health survey's functioning of daily living questions was used to measure daily physical and psychological ability in questionnaire 2001 (*Study IV*). In 2008, an invitation to participate in a clinical study was sent to all the former athletes and controls who were still alive and had answered at least one of the previous questionnaires (*Study I*). The clinical study included a physical examination, laboratory tests, and questionnaires (Laine et al. 2014). Furthermore, in 2001, a postal questionnaire was sent to the surviving elite athletes and their brothers residing in Finland, eliciting information on health, lifestyle, and sociodemographic characteristics (*Study IV*).

4.2.4 The 1985, 1995, 2001, and 2008 questionnaire studies

Former elite athletes (n=1255, 82 % response rate in 1985) and controls (n=764, 76 % response rate in 1985) who answered the PA-, alcohol-, or smoking-related questions in any questionnaires were included in the statistical analysis (*Study I*). Furthermore, in 2001, there were 199 athletes who with their 199 age-matched brothers responded to the questionnaire, and we also did a sensitivity analysis for these athletes (n=199) and all their brothers (n=322) (*Study IV*). In 1985, a postal questionnaire was sent to the surviving cohort members (total n=2528, athletes n=1518 and controls n=1010) (Kujala et al. 1996). The response rate to the 1985 questionnaire was 90 % (n=1364) for the athletes and 77 % (n=777) for the controls (*Study II*). Former elite athletes (n=1326) and controls (n=755) who answered the alcohol-related questions were included in the statistical analysis (*Study II*). The response rate was 52.5 % (n=137) among those participants alive in 1985 who had hospital admissions at any time for any alcohol-related disease or death, and among this subgroup, the response rate was higher among the former athletes, at 58.7 % (n=91), than among the controls, at 43.4 % (n=46) (*Study II*). Correspondingly, the response rate was 67.2 % (n=119) among those participants alive in 1985 who had hospital admissions at any time for any chronic pulmonary disease or death, and among this subgroup, the response rate was higher

among the former athletes, at 71.1 % (n=59), than among the controls, at 63.8 % (n=60) (*Study III*).

4.2.5 Variables based on the questionnaires

Physical activity (PA) (*Studies I, II, III,* and *IV*) The volume of PA (metabolic equivalent MET-hours/day or MET-hours/week) was based on responses to three structured questions on intensity, duration, and frequency of activity using a previously validated method (*Studies I–IV*) (Kujala et al. 1998, Waller, Kaprio & Kujala 2008). The participants were classified into tertiles of PA (*Studies II, III,* and *IV*). Sedentary time was assessed using a continuous variable called sitting time (hours/day) (*Study IV*). Engagement in competitive sports or leisure-time sports was dichotomized (*Study II*). The term *discontinued sports* means that based on questions in the 1985 questionnaire (*Study II*), the athlete had retired from competitive sports.

Smoking (*Studies I, II, III,* and *IV*) Based on a detailed smoking history, smoking status was defined as never, former, current, or occasional smokers (*Studies I–IV*). Those who had never smoked more than 100 lifetime cigarettes were defined as never smokers. Former smokers had smoked more than 100 cigarettes in their lifetime, smoked regularly, but had not smoked during the last month. Current smokers were regular (daily or almost daily) smokers at the time of the data collection (Kaprio & Koskenvuo 1988). Occasional smokers were men who smoked no more than 2 cigarettes in a week (Paavola, Vartiainen & Puska 2001) or had last smoked 2–30 days ago (Luoto, Uutela & Puska 2000). In *Study IV*, among the current or former smokers, nicotine dependence was assessed using the Heaviness of Smoking Index (HSI), that is, 2 items of the Fagerström Test for Nicotine Dependence (FTND): the number of CPD and the time to the first cigarette in the morning (Haddock et al. 1999). The HSI sum score ranges from 0–6, reflecting the degree of physical dependence, and accounts for about 80 % of the variance in the FTND (*Study IV*) (Haddock et al. 1999, Bäckmand et al. 2010). In *Study III*, smoking exposure was determined by the pack-years in 1985. It was calculated by multiplying the number of packs of cigarettes smoked daily by the number of years the person has smoked. Furthermore, the number of cigarettes per day (CPD) was calculated based on all questionnaires (*Study I*).

Use of alcohol (*Studies I, II, III,* and *IV*) Alcohol consumption was assessed using questions on beer, wine, and spirits consumption, blackouts, and HDOs. Alcohol consumption based on quantity–frequency measures of beverage use was converted into grams of pure alcohol per month as previously reported (*Studies I–IV*) (Romanov et al. 1987). The responses formed three categories: abstainers and light (3 or fewer drinks per week), moderate (more than 3 but no more than 14 drinks per week), and heavy users (on average more than 2 drinks a day) (*Study II*) (Järvenpää et al. 2005). The Substance Abuse and Mental Health Services Administration (SAMHSA) defines an HDO as drinking 5 or more alcoholic drinks (>60 grams of pure alcohol) on a single occasion on at least 1 day in the

past 30 days (SAMHSA 2004).The responses formed two categories: no HDO and at least one HDO (*Studies I* and *II*). Blackouts were defined by the frequency of alcohol-related loss of consciousness or temporary amnesia during the past 12 months (Hamin, Sungwon & Dai 2009). Three response categories were formed for analysis: no blackouts in the past year, one, or 2 or more (*Study II*).

Socioeconomic status (SES) (*Studies I, II, III,* and *IV*) To adjust for occupational loading, the participants were classified into five socioeconomic groups based on occupation: upper white collars, lower white collars, skilled (blue) collars, unskilled workers, and farmers (Aromaa et al. 1989), according to the occupation in which they had practiced the longest. This classification also reflects the participants' SESs. The socioeconomic group distribution of the athletes differed from that of the controls ($p<0.001$, χ^2 -test). The occupational data were collected partly from the Central Population Registry of Finland and partly from questionnaires, asking for the occupation in which the participants had been active the longest.

Marital status (*Study I*) The participants' marital statuses were classified into 6 categories in the 1985 questionnaire study: unmarried, married, remarried, cohabitated, divorced, and widowed.

Working status (*Study I*) The participants' working statuses were classified into 4 categories in the 1985 questionnaire study: employed (salaried or self-employed), retired for old age, retired on a disability pension, and unemployed.

Reasons for ending the athletic sports career (*Study I*) Reasons for ending the athletic sports career were categorized into six groups based on questions in the 1985 questionnaire: enthusiasm had ended, sports injury, work or studies, age, disease, and other reasons.

A history of physician-diagnosed chronic diseases (*Studies III* and *IV*) A history of physician-diagnosed chronic diseases, such as asthma, emphysema, and CB, was asked for use as dichotomous variables (*Study III*). Self-reported current symptoms and a history of physician-diagnosed chronic diseases, such as any chronic disease, hypertension with medication, angina pectoris, myocardial infarct, diabetes, kidney disease, gastric ulcer, asthma, and osteoarthritis, were grouped as dichotomous variables. A history of any sports injury and Achilles tendinitis—or total rupture—was queried (*Study IV*).

Self-rated health (SRH) (*Study IV*) SRH was measured by asking the participants to rate their current health status on a previously validated 5-point Likert scale: 1) very good, 2) quite good, 3) average, 4) quite poor, and 5) poor (Idler & Benyamini 1997).

Activities of Daily Living (ADL) (*Study IV*) Activities of daily living (ADL) and instrumental activities of daily living (IADL) were assessed by a total of nine

items (Aromaa et al. 1989). These national survey questions were modified from the classification of functional capacity (Katz et al. 1963, Lawton & Brody 1969).

Self-rated mood (*Study IV*) Self-rated mood was defined by two partial scales (6 depression items and 6 anxiety items) of the short stress symptom survey extracted from the Brief Symptom Inventory-53 (BSI-53) (Derogatis & Melisaratos 1983).

Body mass index (BMI) (*Study IV*) BMI was self-reported as weight/height^2 (kg/m^2).

Coffee use (*Study I*) Coffee use (cups per day) was based on the 1985, 1995, and 2008 questionnaires.

A summary of the main variables due to PA, alcohol use, and smoking in *Studies I–IV* is presented in Table 3. Selection of the main variables to the present dissertation was based on literature and previous studies. The applied questions on PA (Waller, Kaprio & Kujala 2008), smoking (Vartiainen et al. 2002), and the use of alcohol (Poikolainen 1985) have been previously validated (*Studies I–IV*).

TABLE 3 Summary of the main variables due to physical activity, alcohol use, and smoking in *Studies I–IV*.

	Study I	*Study II*	*Study III*	*Study IV*
Alcohol use				
g/month	x	x	x	x
HDO*	x	x		
Blackouts		x		
Physical activity				
MET-hours/day OR MET-hours/week	x	x	x	x
MET-tertiles		x	x	x
Sitting time (sedentary time)				x
Smoking				
Smoking status	x	x	x	x
HSI*				x
Pack-years			x	
CPD*	x			

*HDO=heavy drinking occasion, HSI=heaviness of smoking index, CPD=cigarettes per day.

4.3 Ethics of the study

The present dissertation was conducted according to good clinical and scientific practice and the Declaration of Helsinki. The authors declare that the results of this dissertation are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation. Approval for the register data collection was given by the Ministry of Social Affairs and Health in Finland and Statistics Finland (*Studies II–IV*). Correspondingly, approval for the questionnaire data collection was given by the Ethics Committee of the Hospital District of Helsinki and Uusimaa and the Ministry of Social Affairs and Health in Finland (*Studies I–IV*). All the participants gave informed consent by returning the questionnaires, which were accompanied by a cover letter explaining the purpose of the study (*Studies I–IV*).

4.4 Statistical methods

In all *Studies I–IV*, p-values <0.05 were considered statistically significant. Statistical analyses were performed using SPSS statistical software (version 22.0/24.0/26.0 for Windows; SPSS Inc., Chicago, IL) (*Studies I–IV)*, Stata 14.0 (Stata Corp, College Station, Texas, USA) (*Studies II–IV),* and the Mplus statistical software package (version 8.2 for Windows; Mplus Corp, California) (*Study I)*. The descriptive data was presented as a mean and standard deviation (SD) or 95 % confidence intervals (CI) if distributed normally; otherwise, the results were shown as a median and range. The differences in the distributions of the categorical variables were examined using cross-tabulations with the Chi-square (χ^2) test. The questionnaire data was analyzed using a nonparametric Kruskal–Wallis test and using the Dunn–Bonferroni approach for post hoc testing in pairwise comparisons for more than two groups ($p<0.05$), because some of the variables were not normally distributed, and the variances were not equal. The Mann–Whitney-U test was used to compare the differences between the sports groups and controls ($p<0.05$). The homogeneity of variances was assessed using Levene's test and the normality using Kolmogorov–Smirnov's test ($p<0.05$) (*Studies I–IV*).

Study I The follow-up started on January 1, 1985, and ended on January 1, 2008. A bivariate cross-lagged path model was used to investigate the direction and magnitude of the associations between PA and alcohol consumption at four time points (the 1985, 1995, 2001, and 2008 questionnaire studies). In the specified bivariate cross-lagged model, all stability paths, cross-lagged paths, and covariances between PA and alcohol consumption were freely estimated at four time points. Furthermore, in the model, a specific latent factor was specified for each observed variable to estimate the error variances of the observed variables. To permit model identification, the error variances of the first and second measurement and the error variances of the third and fourth measurements were set as

equal. The full cross-lagged path model is shown in Figure 7. This model was not used to examine the associations between PA and smoking because neither CPD nor the categorical smoking status variable were appropriate to this model.

Latent profile analysis (LPA) was used to find simultaneous developmental profiles in PA and alcohol and smoking behaviors measured in 1985, 1995, 2001, and 2008. Profiles in the latent classes were specified to differ in the mean PA and alcohol consumption values, whereas smoking behavior was specified as categorical (three categories), allowing the proportion of the three categories to differ between profiles. The variances of the two continuous variables were allowed to differ between latent profiles. The estimation method was a full-information maximum likelihood with robust standard error estimates (MLR estimator in Mplus). The large number of missing values was supposed to be missing at random (MAR). To evaluate the number of latent profiles, the Bayesian information criteria and the adjusted Lo–Mendell–Rubin likelihood ratio test (AdjLMR) were used.

To evaluate the classification quality, the average posterior probabilities (AvePP) for the cases of most likely latent profile membership were used. In the LPA model, this AvePP should be at least 0.70 to be able to interpret the solution (Nagin 2005). After deciding on a certain number of latent profiles, the differences in the auxiliary variables (age, SES, marital and working status, coffee use, CPD, and HDO) between the latent profiles were tested using the Bose–Chaudhuri–Hocquenghem (BCH) method (Asparouhov & Muthén 2014). In the first stage, BCH weights were saved as variables, and in the second stage, it was possible to analyze the differences between the latent profiles as well as the changes across the years within the profiles. Furthermore, it was also possible to test whether the changes across the years were different between the latent profiles. These weights were used when analyzing the differences between the latent profiles and the auxiliary variables. The advantage of using this method is that the latent profile solutions are invariant when testing the auxiliary variables. Because the data consisted of former athletes and controls, this dummy-coded variable was used to control the difference between the auxiliary variables.

Study II The follow-up of hospitalizations and deaths started on January 1, 1970, and continued until the end of 2008 or until the date of emigration, death, or hospitalization due to the first alcohol-related disease event, whichever came first. The primary event of incident alcohol-related disease was defined as the first recorded hospital episode or as death from any alcohol-related disease. A Cox proportional hazards model was used to calculate the age-adjusted hazard ratios (HRs) with their 95 % CIs for alcohol-related disease or death between the former athletes and controls. A post hoc analysis accounting for the number of comparisons was used to compare the statistical differences between the specific sports groups. Participants still alive at the end of the follow-up and those who died from any other cause were censored. The Cox regression assumptions were tested using Schoenfeld residuals (ph-test in Stata) and also by plotting.

Study III The follow-up of hospitalizations and deaths started on January 1, 1970, and continued until the end of 2015 or until the date of emigration, death, or hospitalization due to the first chronic pulmonary disease event, whichever came first. The primary event of incident chronic pulmonary disease was defined as the first recorded hospital episode or as death from any chronic pulmonary disease. A Cox proportional hazards model was used to calculate the age- and SES-adjusted HRs with their 95 % CIs for the incidence of chronic pulmonary disease between the former athletes and controls. Time-invariant covariates were included in the analysis: age at entry and sports groups. A post hoc analysis accounting for the number of comparisons was used to compare the statistical differences between the specific sports groups. Participants still alive at the end of the follow-up, those who emigrated, and those who died from any other cause were censored. The Cox regression assumptions were tested using Schoenfeld residuals (ph-test in Stata) and also by plotting.

Study IV The follow-up of the all-cause mortality of each athlete and brother started from the time when the athlete was an elite athlete and continued until the date of death (outcome event) or emigration (censoring event) or the end of the follow-up on December 31, 2015. A paired Cox proportional hazards model was used to calculate the age-adjusted HRs with 95 % CIs for the all-cause mortality of the former athletes relative to their brothers. A post hoc analysis considering the number of comparisons was used to compare the statistical differences between the specific sports groups. The mortality analysis for family clustering in the subgroup analysis based on all the 2001 questionnaire respondents was also adjusted. The Cox regression assumptions were tested using Schoenfeld residuals (ph-test in Stata) and by plotting log-log plots.

The 2001 questionnaire data was mainly analyzed using a nonparametric Wilcoxon signed rank test (a matched-pair analysis) for the ordinal or continuous variables and McNemar's test (a matched-pair analysis) for the nominal variables to analyze the differences between each athlete and the age-matched brother.

5 RESULTS

5.1 Participant characteristics in *Studies I, II, III*, and *IV*

Various data sets were used in the present dissertation (see Figure 1). *Studies I, II,* and *III* consisted of male former athletes and matched controls, and *Study IV* also included the former athletes' age-matched brothers. In *Studies I, III,* and *IV,* the former athletes were categorized into three groups according to the type of training needed to achieve optimal results: endurance, mixed, and power sports (Åstrand & Rodahl 1986), whereas for the purpose of *Study II* and the literature review, the former male athletes were classified into more specific sports groups, and shooters were also included (for more details, see Chapter 4.1 in this dissertation).

In *Studies I, II,* and *III*, the former athletes were more physically active and smoked less compared to the controls. *Study IV* showed that the former athletes were more physically active, smoked less, and their SRH was higher compared to their age-matched brothers. Correspondingly, the brothers' lifestyle habits were healthier than the controls' (*Study IV*). A summary of the participants (N and mean age (years) in *Studies I–IV* is presented in Table 4.

TABLE 4 Summary of the participants (N and mean age (years)) in *Studies I–IV**.

	Study I 1970-2008		**Study II 1970-2008**		**Study III 1970-2015**		**Study IV the time when the athlete was an elite athlete-2015**	
In the beginning of the follow-up	Former athletes (n=1276), mean age 57.3 years	Controls (n=777), mean age 55.6 years	Former athletes (n=2202), mean age 46.0 years	Controls (n=1403), mean age 43.7 years	Former athletes (n=2078), mean age 45.5 years	Controls (n=1453), mean age 44.3 years	Former athletes (n=900), mean age 24.6 years	Brothers (n=900), mean age 25.1 years
In the end of the follow-up	Former athletes (n=747), mean age 73.8 years	Controls (n=436), mean age 72.8 years	Former athletes (n=885), mean age 74.7 years	Controls (n=529), mean age 73.6 years	Former athletes (n=772), mean age 75.8 years	Controls (n=494), mean age 73.8 years	Former athletes (n=251), mean age 79.3 years	Brothers (n=253), mean age 79.1 years

*For more details, see Table 1 in *Studies I-IV*.

5.2 Physical activity, alcohol use, and smoking among former athletes, their brothers, and controls (*Studies I, II, III*, and *IV*)

5.2.1 Physical activity, use of alcohol and smoking among former athletes and controls (*Studies I, II,* and *III*)

Physical activity (PA) The former athletes were more physically active than the controls at the four time points (1985, 1995, 2001, and 2008), and the mean MET-hours/week was significantly higher among the former athletes than the controls during the 23-year follow-up ($p<0.0001$) (*Study I)* (for more details, see Table A4.1 in Appendix 4). Furthermore, 16.0 % (n=200) of the former athletes and 12.1 % (n=33) of the controls participated in competitive sports in 1985 ($p<0.001$) (*Study II)*. The mean MET-hours/week among the former athletes and controls at the four time-points are presented in Figure 2.

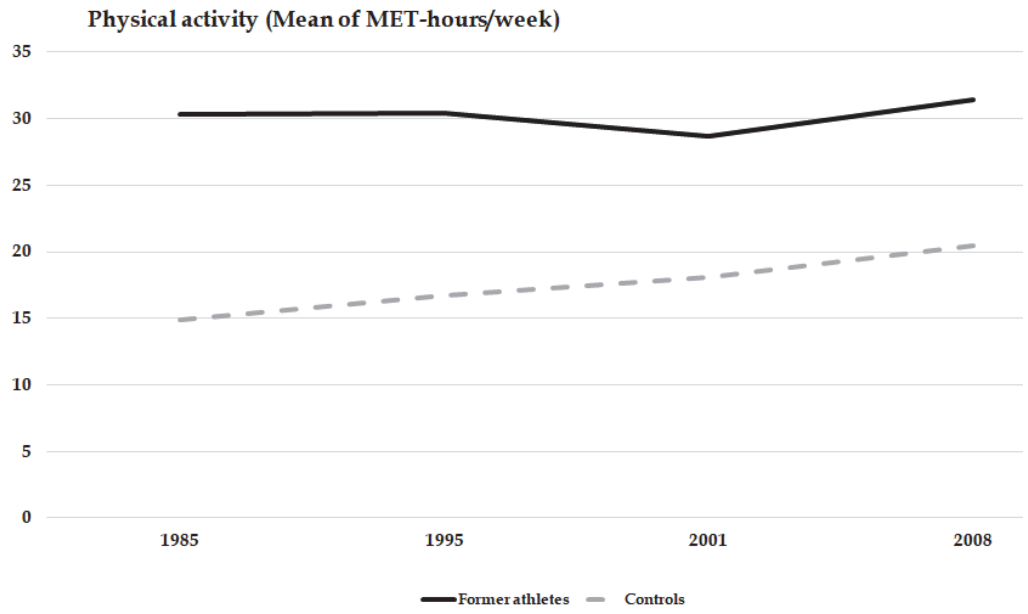


FIGURE 2 Mean MET-hours/week among former athletes and controls at the four time-points.

Alcohol consumption In the 1985 questionnaire, the former athletes consumed more alcohol (425 g/month) compared to the controls (398 g/month) ($p<0.05$), but no differences were seen in the other questionnaires (*Studies I* and *II)* (Figure 3). The proportion having HDOs did not differ between former athletes and controls ($p>0.05$). In 1985, the team sports athletes consumed significantly more alcohol ($p<0.05$), especially beer ($p<0.01$), and had significantly more HDOs than

the controls (p<0.001) (*Study II)*. Consequently, the total alcohol and beer consumption (g/month) was higher in the team sports athletes compared to all the other sports groups (p<0.05) (*Study II)*.

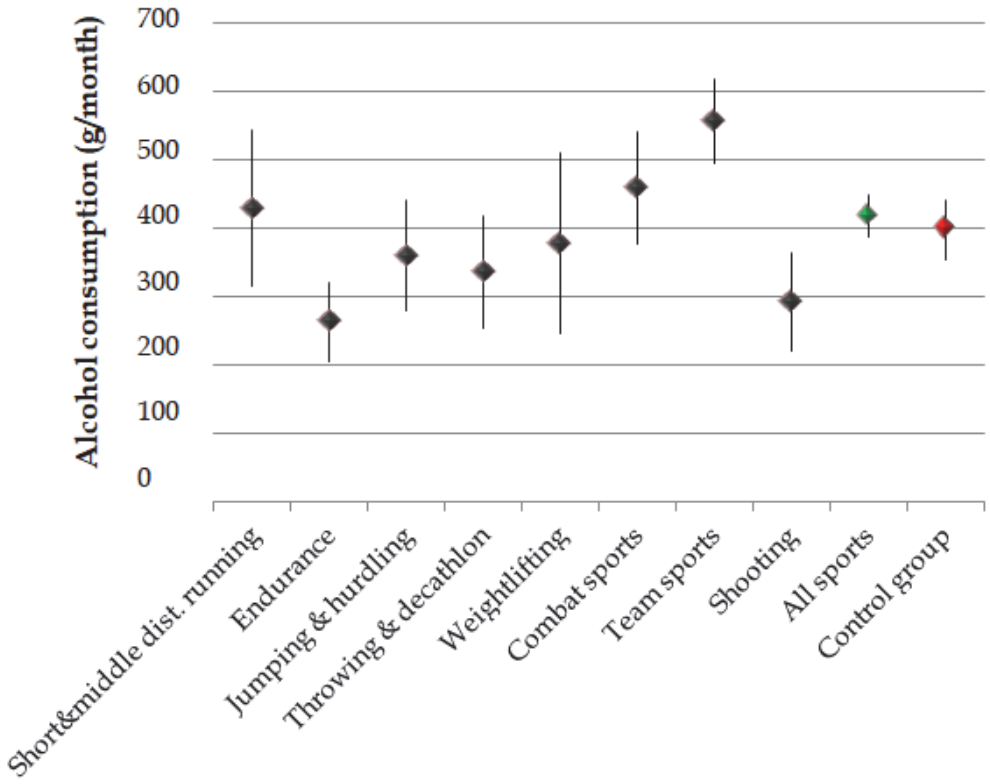


FIGURE 3 Mean total alcohol consumption (95 % CI) among former athletes and controls in 1985. (Figure as originally published in Kontro et al (2017)).

There was no significant difference in the total alcohol consumption between the competitive athletes and those athletes who had discontinued competitive sports. However, alcohol consumption was increased among athletes who were not engaged in leisure-time sports after an active athletic career (p<0.05) (*Study II)*. Correspondingly, the use of alcohol and proportions having HDOs was higher among the former athletes if their athletic sports career had ended suddenly by sports injury or if their enthusiasm had ended compared to age or disease (p<0.05) (*Study I)*. Furthermore, alcohol consumption (g/month) was the most common among the current smokers, while the never-smokers used the least alcohol in all the questionnaires. The mean total alcohol use (g/month) among the former athletes and controls at the four time-points is shown in Figure 4.

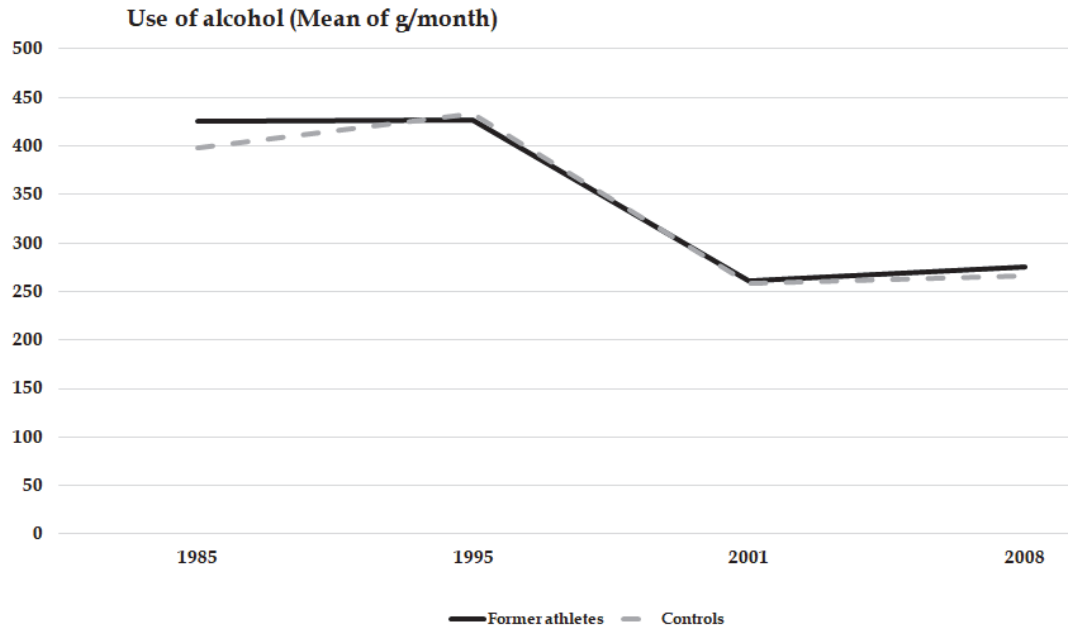


FIGURE 4 Mean total alcohol consumption among former athletes and controls at the four time-points.

Smoking The controls smoked more than the former athletes in all the surveys ($p<0.0001$). The mean CPD was higher among the controls compared to the former athletes at the four timepoints ($p<0.001$) (*Study I)* (Figure 5).

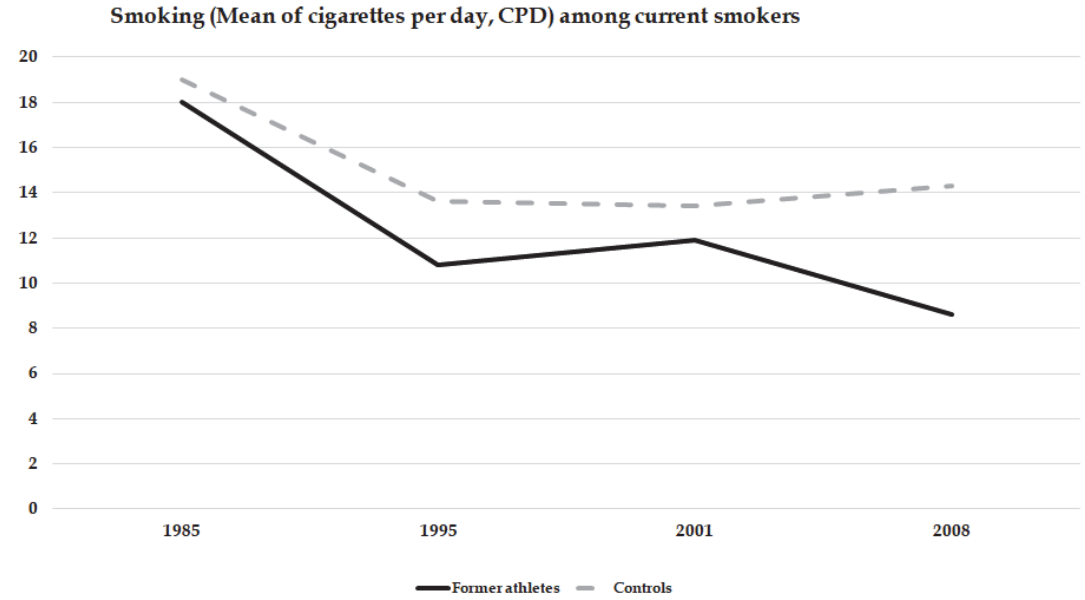


FIGURE 5 Mean CPD among former athletes and controls at the four timepoints.

In 1985, 31 % of the controls and 21 % of the former athletes were current smokers, while in 2008, these numbers were 9 % and 5 %, respectively (*Study I)* (Figure 6). Current smoking was more common in team sports and weightlifting than in other sports ($p<0.05$) (*Study II)*. In the 1985 questionnaire, the mean pack-years was higher among controls, at 17.0 (95 % CI 15.7-18.3), than former athletes, at 9.6 (95 % CI 8.7-10.5), who had ever smoked during their lifetime ($p<0.001$) (*Study III)*. However, the reasons for quitting an active sports career among the former athletes were not associated with smoking status ($p=0.21$) (*Study I)*.

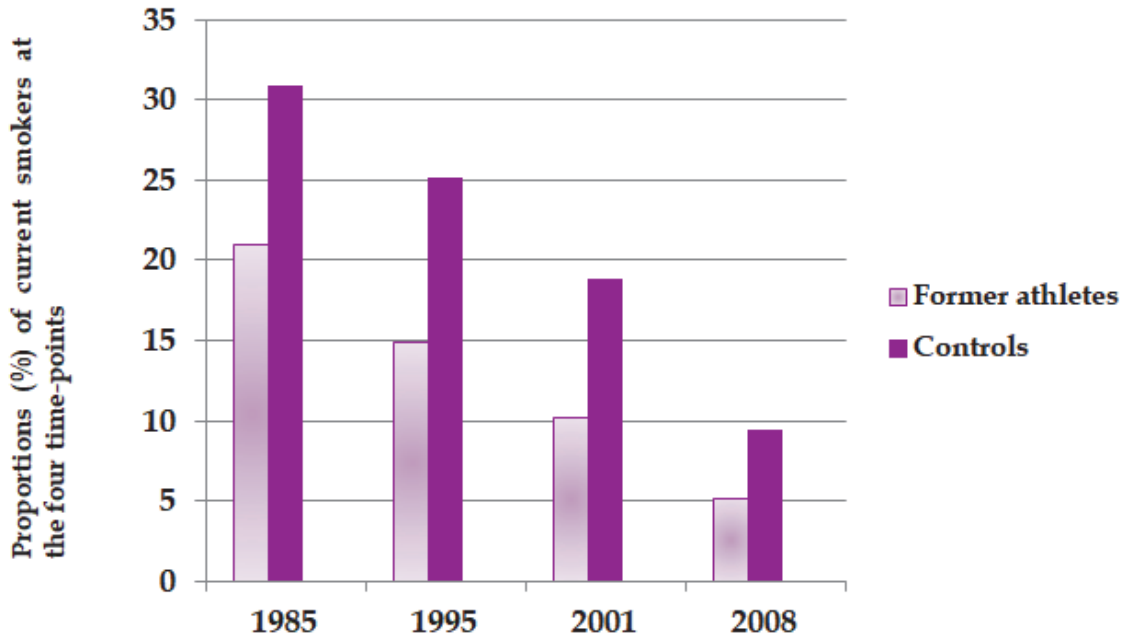
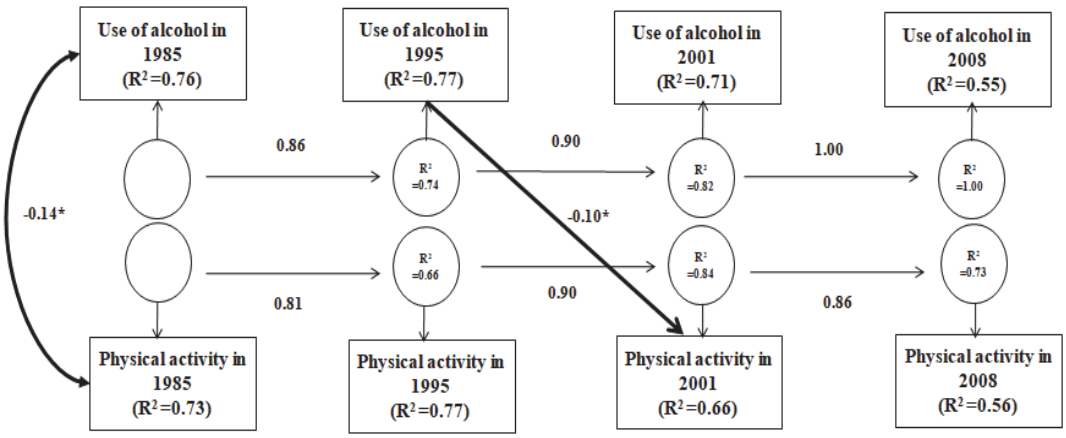


FIGURE 6 Proportions (%) of current smokers among former athletes and controls at the four time-points.

PA, alcohol use, and smoking The specific bivariate cross-lagged model fitted well to the data (for more details, see Table 2 in *Study I*). Among all participants, there was only one statistically significant cross-lagged regression coefficient: -0.10 (beta) from alcohol use in 1995 to PA in 2001 ($p<0.001$). Correspondingly, the correlation of -0.14 between alcohol use and PA was statistically significant only in 1985 ($p<0.001$). There was no interaction between athletic status and the use of alcohol ($\chi^2(12) = 14.55$, $p = 0.267$) (*Study I*). The full cross-lagged path model is shown in Figure 7.



*Only statistically significant cross-lagged paths are shown, $p<0.05$

FIGURE 7 A bivariate cross-lagged model for physical activity and alcohol use among all participants. (Figure as originally published in Kontro et al (2020)).

According to the LPA, the five latent profile solution was decided upon (Figure 8). The mean PA and alcohol use values (standardized using whole data) and the estimated smoking status proportions showed that the longitudinal profiles were very stable across the measurements. Profile 1 (total 14.0 % (n=317)) included participants who were physically inactive, used more alcohol than average, and did not smoke. Profile 2 (21.7 % (n=494)) included participants who were physically inactive, used average amounts of alcohol, but smoked more than average. Profile 3 (30.8 % (n=700)) included participants who were physically inactive, used less alcohol, and were average smokers. Profile 4 (15.4 % (n=351)) included participants who were highly physically active, used less alcohol, and smoked less than average. Profile 5 (18.1 % (n=413)) included participants who were highly physically active and used alcohol and smoked more than average (*Study I)*.

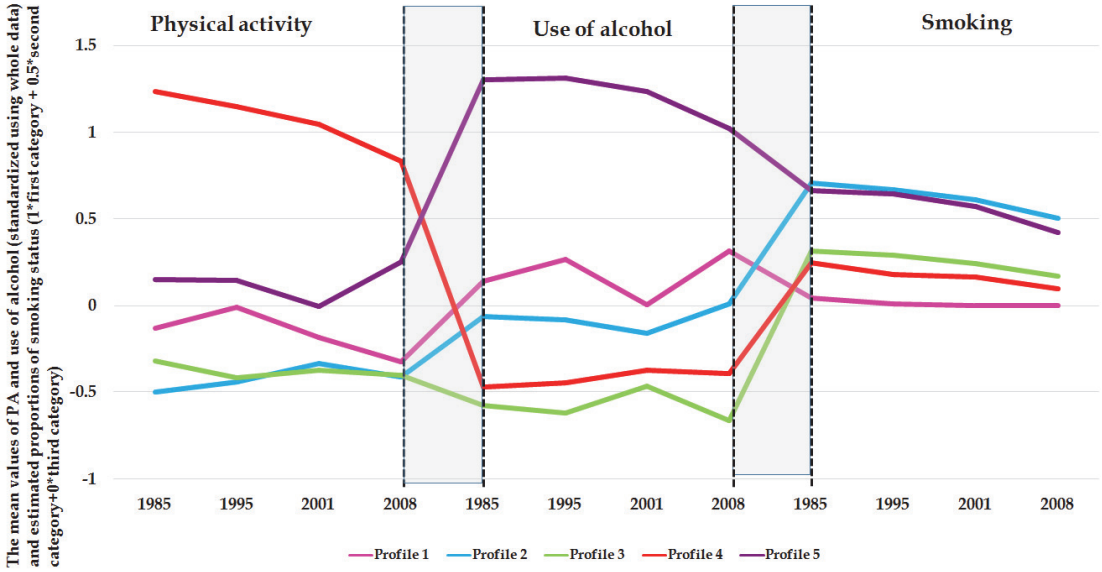


FIGURE 8 Characteristics of the 5 profiles from the LPA. (Figure as originally published without colors in Kontro et al (2020)).

Figure 8 shows that during the follow-up, PA decreased in profile 1, while the use of alcohol both increased and decreased, and smoking decreased ($p<0.05$). In profile 2, PA and the use of alcohol increased, but smoking decreased over time ($p<0.05$). In profile 3, PA, the use of alcohol, and smoking decreased ($p<0.05$). In profile 4, PA decreased, the use of alcohol increased, and smoking decreased over time ($p<0.05$). In profile 5, there was very little change in PA and the use of alcohol between surveys, but smoking decreased ($p<0.05$) (*Study I*).

However, neither the mean values of PA and alcohol use nor the proportion of smoking statistically significantly differed between the former athletes and controls in any profile. However, there was a statistically significant difference between the former athletes and controls in the probability of belonging to profiles 1-4: profile 1 ($p=0.002$), profile 2 ($p<0.001$), profile 3 ($p=0.002$), profile 4 ($p<0.001$), but not profile 5 ($p=0.31$) (*Study I*). Figure 9 presents the probability of belonging to profiles 1 to 5 among the former athletes and controls.

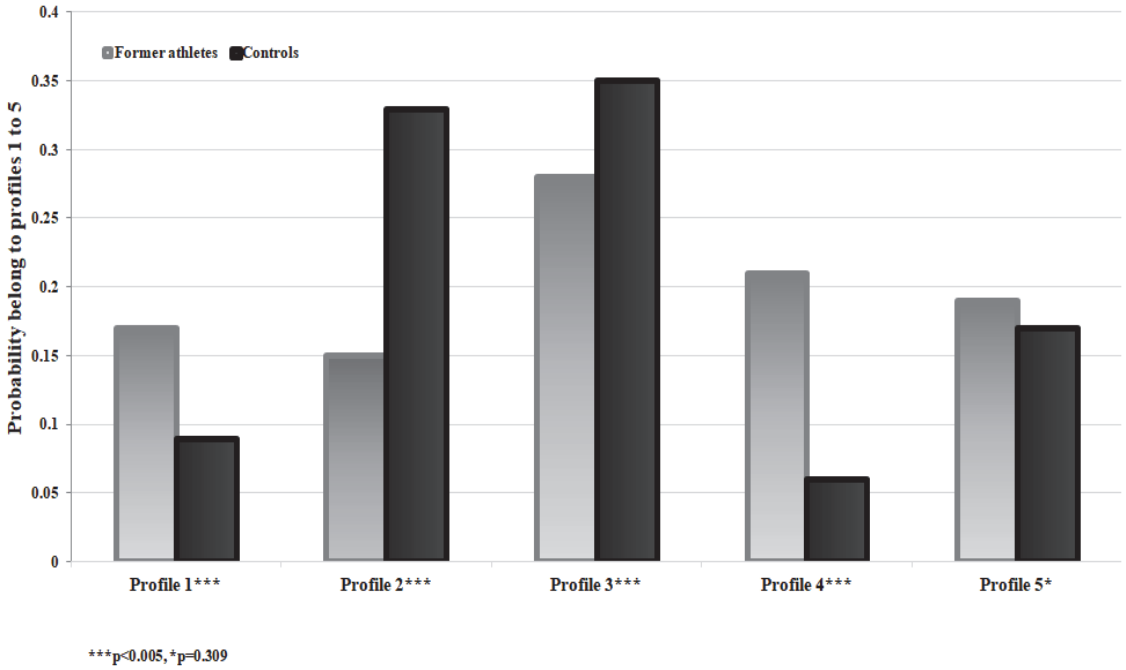


FIGURE 9 Probability of belonging to profiles 1 to 5 among former athletes and controls (Figure as originally published in Kontro et al (2020)).

5.2.2 Physical activity, alcohol use, and smoking among the former athletes and their brothers *(Study IV)*

In 2001, the former athletes (n=199) were more physically active than their age-matched brothers (n=199) (4.4 vs. 3.2 MET-hours/day) ($p<0.05$). Correspondingly, the former athletes smoked less than their age-matched brothers ($p<0.001$). The former athletes also self-reported their health as very good much more frequently (20.6 %) than their age-matched brothers (8.6 %) ($p<0.05$). However, the former athletes had more self-reported physician-diagnosed sports injuries and Achilles tendinitis than their age-matched brothers ($p<0.05$). The mean anxiety score was higher among the brothers than among the former athletes ($p<0.05$), and the brothers reported feeling more restless and excited than the former athletes ($p<0.05$).

Finally, a sensitivity analysis based on the former elite athletes (n=199), all their brothers (n=322), and the population control (n=416) was conducted, and the results were consistent with those of the age-matched pair analysis in the 2001 (for more details, see Supplementary Tables 6 and 7 in *Study IV*). The proportions (%) of current smokers, mean MET-hours/day, and alcohol use (g/month) among the former athletes, their brothers, and the controls in 2001 are presented in Figures 10a–c.

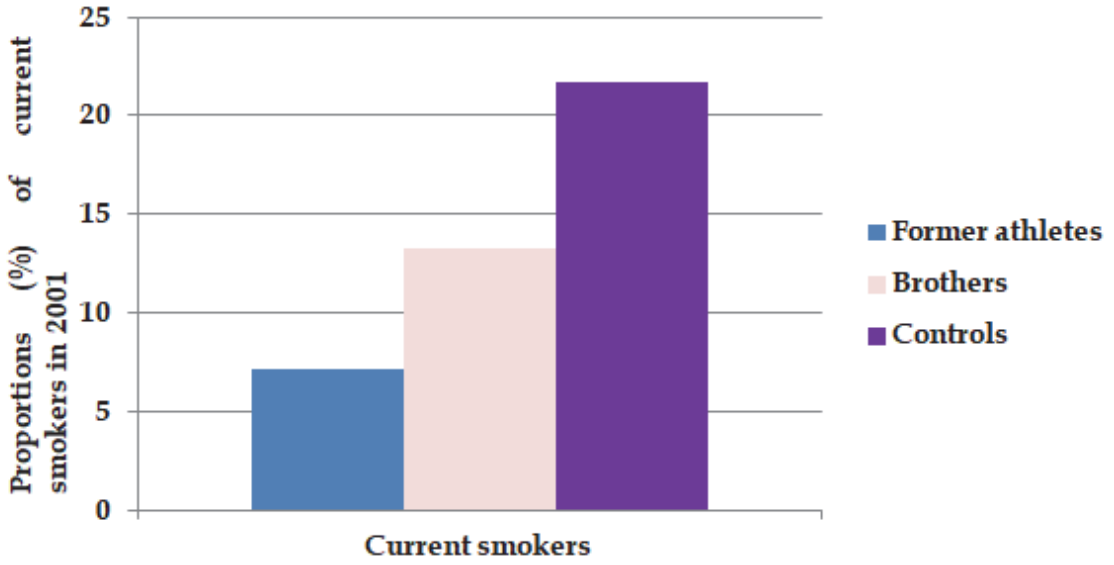


FIGURE 10a Proportions (%) of current smokers among former athletes, their brothers, and controls in 2001.

Figure 10a shows that there were more current smokers among the controls (21.7 % (n=70)) compared to the former athletes (7.1 % (n=14)) and their brothers (13.3 % (n=42)) in 2001 ($p<0.001$). Furthermore, Figure 10b shows that the controls tended to use more alcohol than the former athletes and their brothers in 2001. Correspondingly, Figure 10c shows that the controls were less physically active than the former athletes and their brothers ($p<0.05$).

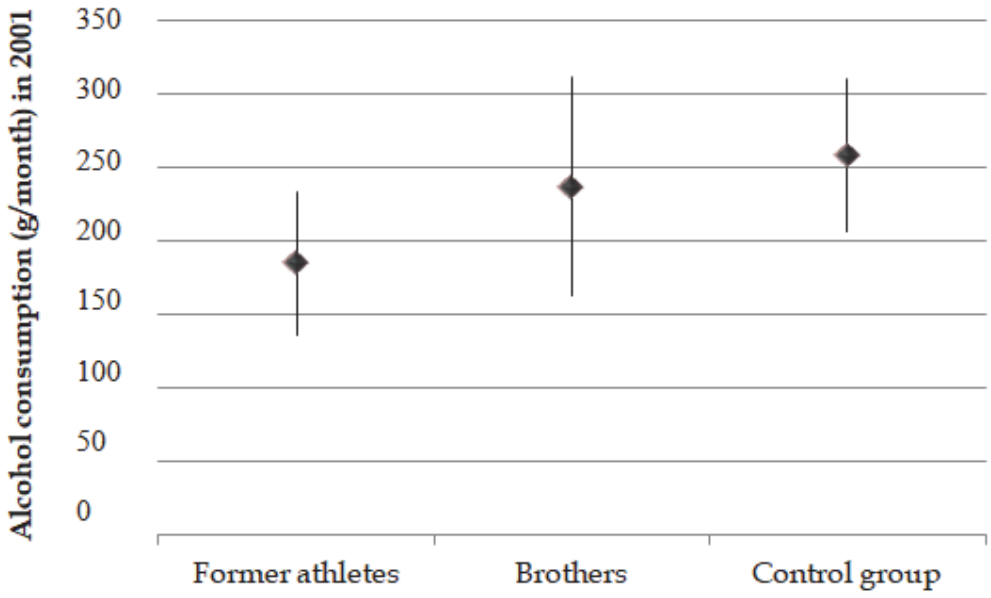


FIGURE 10b Mean total alcohol consumption (95 % CI) among former athletes, their brothers, and controls in 2001.

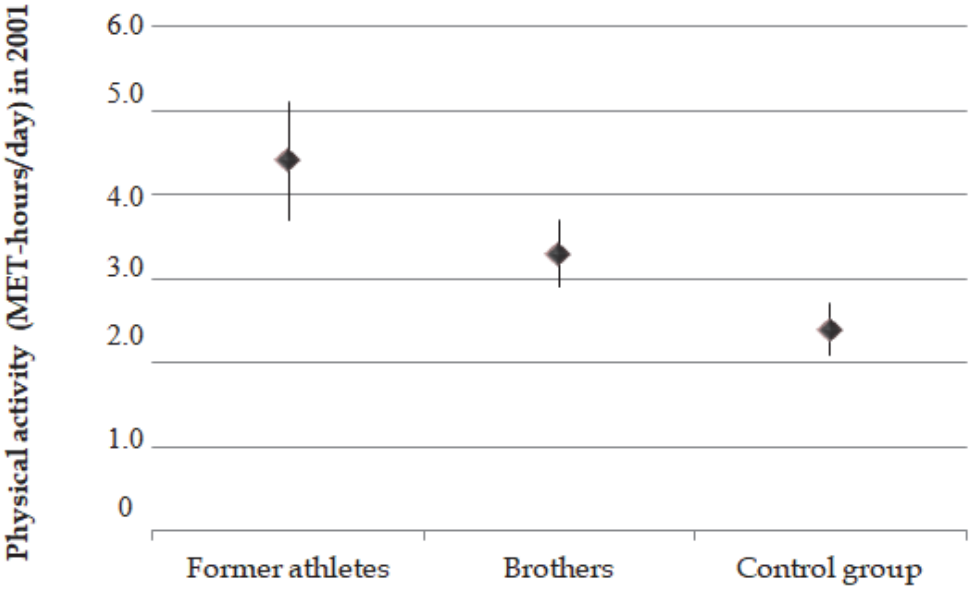


FIGURE 10c Mean MET-hours/day (95 % CI) among former athletes, their brothers, and controls in 2001.

5.3 Alcohol-related morbidity and mortality among former athletes and controls (*Study II*)

Altogether, 6.2 % (n=136) of the former athletes and 7.1 % (n=99) of the controls were admitted to the hospital for any alcohol-related disease during the 39-year follow-up period.The most common reasons for admissions were mental and behavioral disorders due to alcohol use (athletes 2.4 % (n=53), controls 2.1 % (n=34)), alcohol dependence syndrome (athletes 3.9 % (n=85), controls 4.5 % (n=72)), and alcoholic liver disease (athletes 0.8 % (n=18), controls 0.7 % (n=10)) (Figure 11). Other reasons were alcoholic pancreatitis (athletes 0.4 % (n=8), controls 0.4 % (n=5)) and other alcohol-related diseases (athletes 0.3 % (n=6), controls 0.1 % (n=1)) (For more details, see Table 2 in *Study II)*.

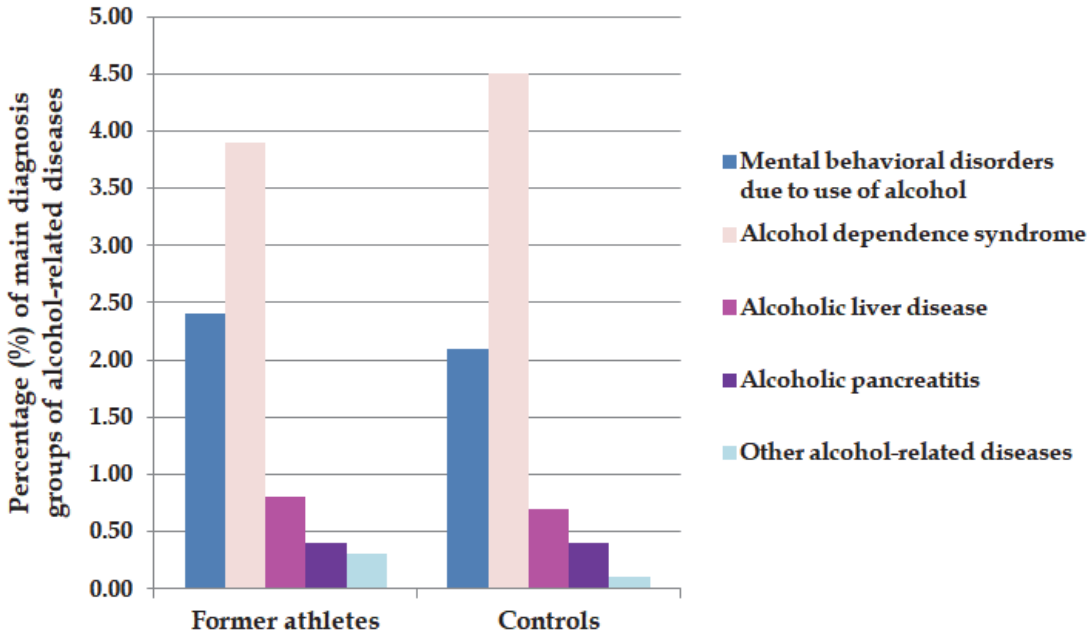


FIGURE 11 Percentage (%) of main diagnosis groups of alcohol-related diseases among former athletes and controls.

The age-adjusted HRs for admissions to hospital for any alcohol-related disease or death during the follow-up in the former athletes was 0.93 (95 % CI 0.73–1.20, p=0.59) compared to the controls, although the former athletes consumed more alcohol than the controls in 1985 (Figure 12). The results for the risk of alcohol-related disease or death persisted after an adjustment for SES (for more details, see Supplemental Table 5 in *Study II)*. In a secondary analysis, compared to weightlifters (the group with the highest risk), the statistically significant HR for admissions to hospital for any alcohol-related disease or death was 0.42 (95 % CI 0.19–0.93) for endurance sports athletes, 0.39 (95 % CI 0.17–0.86) for jumpers and hurdlers, and 0.32 (95 % CI 0.11–0.91) for shooters. Accordingly, compared to combat sports athletes, the HR was 0.46 (95 % CI 0.25–0.85) for endurance sports athletes, 0.42 (95 % CI 0.22–0.79) for jumpers and hurdlers, and 0.35 (95 % CI 0.14–0.87) for shooters.

Finally, the risk of any alcohol-related disease or death was higher among those who did not respond to the 1985 questionnaire study compared to the respondents, both among all participants (HR=2.34, 95 % CI 1.72–3.18, p<0.001), among the former athletes (HR=1.74, 95 % CI 1.11–2.73, p=0.016), and among the controls (HR=3.14, 95 % CI 2.02–4.87, p<0.001).

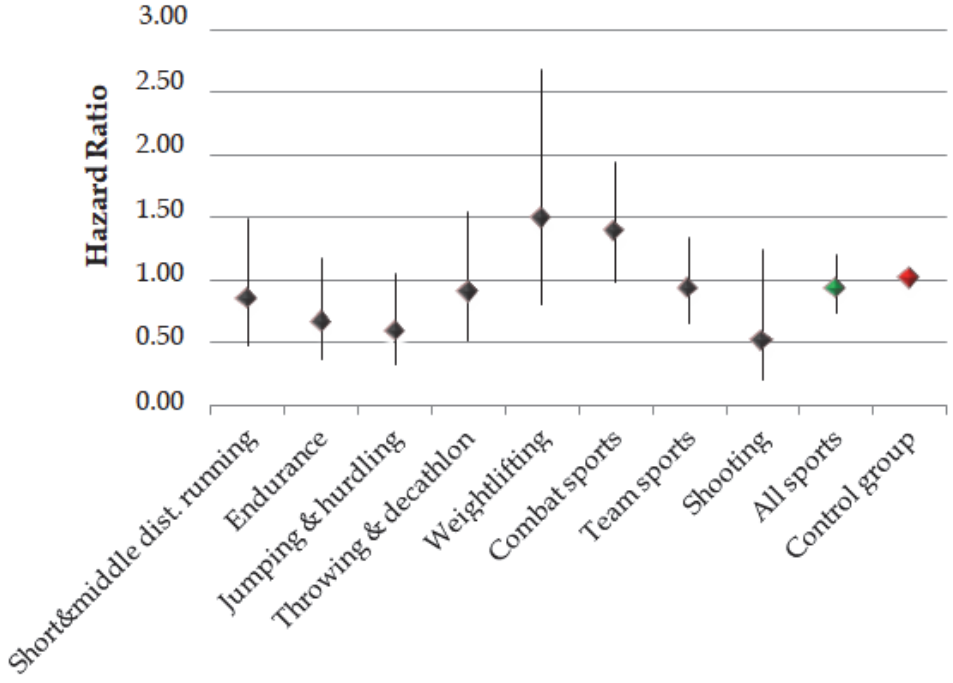


FIGURE 12 Age-adjusted hazard ratios (HRs) (95 % CI) for alcohol-related diseases or deaths among former athletes compared to controls. (Figure as originally published in Kontro et al (2017)).

Figure 13 and Table 5 show that there were the most alcohol-related diseases or deaths (n=62) in profile 5 and the least in profile 1 ($p<0.001$) (*Studies I* and *II*). Profile 5 (18.1 % (n=413)) included participants who were highly physically active, used alcohol, and smoked more than average, whereas profile 1 (14.0 % (n=317)) included participants who were physically inactive, used more alcohol than average, and did not smoke (*Study I*).

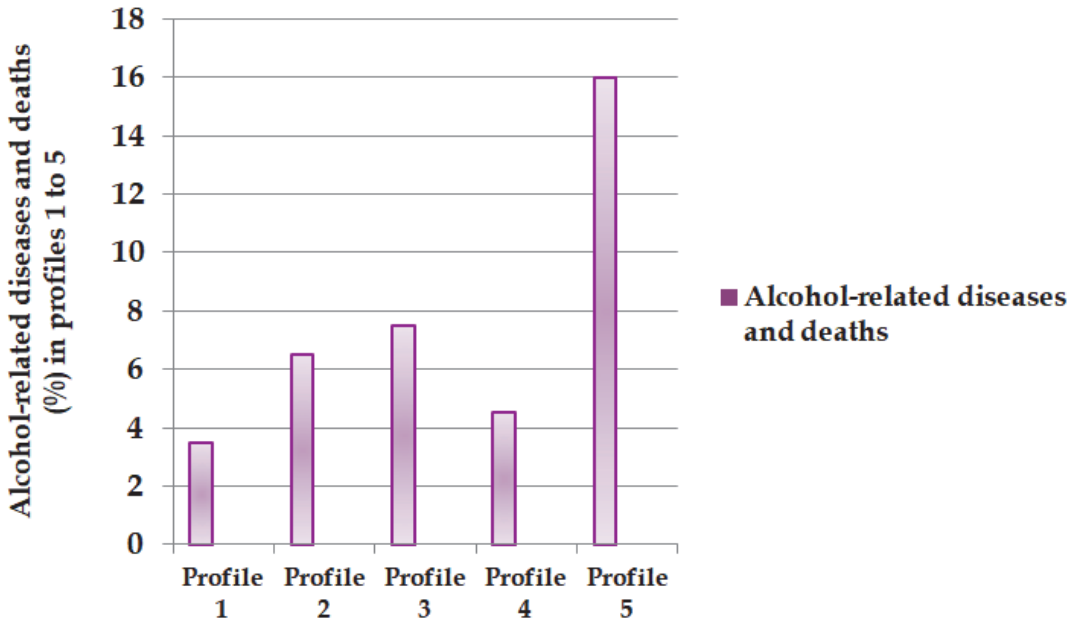


FIGURE 13 Alcohol-related diseases and deaths (%) in profiles 1 to 5.

TABLE 5 Alcohol-related diseases or deaths % (n)* in profiles 1 to 5.

	Profile 1	Profile 2	Profile3	Profile 4	Profile 5
No alcohol-related disease or death % (n)	96.5 (766)	93.5 (446)	92.5 (295)	95.5 (295)	84.0 (326)
Alcohol-related disease or death % (n)	3.5 (28)	6.5 (31)	7.5 (23)	4.5 (14)	16.0 (62)

*All participants, $p<0.001$ for group differences in alcohol-related diseases or deaths using a χ^2 -test.

5.4 Chronic pulmonary morbidity and mortality among former athletes and controls (*Study III*)

Altogether, 4.0 % (n=83) of the former athletes and 6.5 % (n=94) of the controls were admitted to the hospital for any chronic pulmonary disease during the 45-year follow-up period. However, 20 former athletes and 22 controls died of chronic pulmonary disease without being hospitalized at least once. The most common reasons for admissions were asthma (athletes 1.7 % (n=35), controls 2.5 % (n=37)) and chronic bronchitis (athletes 0.5 % (n=10), controls 1.4 % (n=20)) (Figure 14). Other reasons were emphysema (athletes 0.05 % (n=1), controls 0.07 % (n=1)), other obstructive chronic pulmonary disease (athletes 0.6% (n=14), controls 0.7 % (n=10)), and bronchiectasia (athletes 0.1 % (n=3), controls 0.1 % (n=2)) (For more details, see Table 2 in *Study III)*. Proportions (% (n)) of the main diagnosis groups of chronic pulmonary diseases among former athletes (n=83) and controls (n=94) who had hospital admissions are presented in Figure 14. The proportion of chronic bronchitis was larger among the controls (29 % (n=20)) compared to the former athletes (16 % (n=10)).

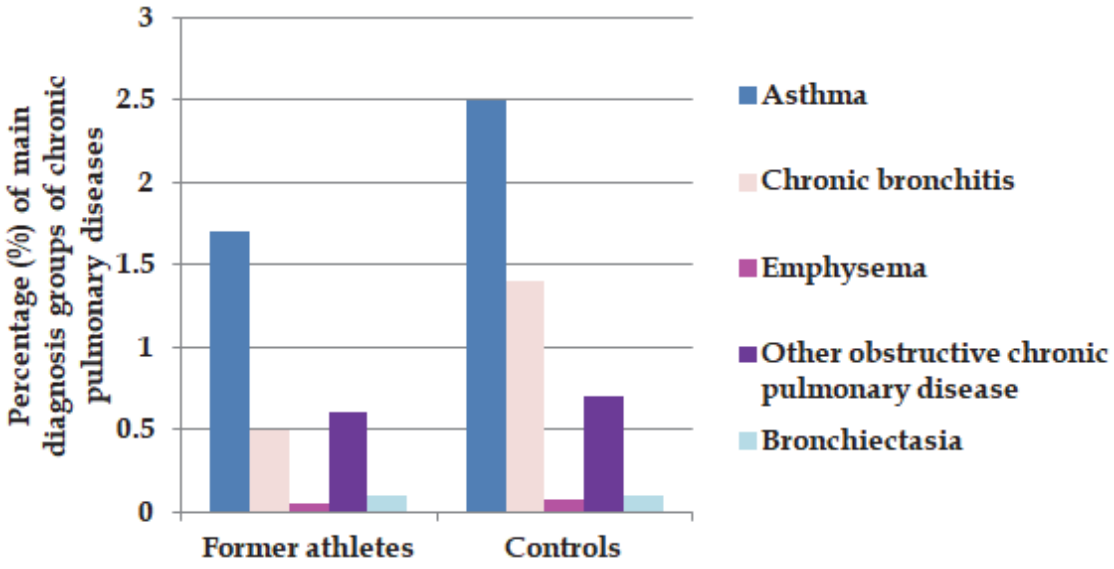


FIGURE 14 Percentage (%) of main diagnosis groups of chronic pulmonary diseases among former athletes and controls.

The age-adjusted HR for admissions to hospital for any chronic pulmonary disease or death in the former athletes was 0.61 (95 % CI 0.46–0.83, p=0.001) compared to the controls and, furthermore, the controls smoked more than the former athletes (Figure 15). In an additional analysis by sports category, compared to the controls, statistically significant HRs were 0.54 (95 % CI 0.31–0.96) for endurance sports athletes, 0.61 (95 % CI 0.42–0.89) for mixed sports athletes, and 0.66 (95 % CI 0.44–0.98) for power sports athletes (for more details, see Table 3 in *Study III)*.

Among all the participants, no differences in HRs were observed between those who did not respond to the 1985 questionnaire study and the respondents (HR=1.10, 95 % CI 0.78–1.53, p=0.60), among the former athletes (HR=1.01, 95 % CI 0.61–1.66, p=0.98), and among the controls (HR=1.34, 95 % CI 0.84–2.13, p=0.21). The age- and pack-year-adjusted HRs from the time of the 1985 questionnaire until the end of the follow-up in the former athletes was 0.59 (95 % CI 0.37–0.93, p=0.024) compared to the controls. Furthermore, the age-adjusted HR in the current smokers was 4.89 (95 % CI 1.98–11.78, p=0.001) compared to the never-smokers, and there was no evidence of a smoking status by athlete status interaction on disease risk (p=0.25).

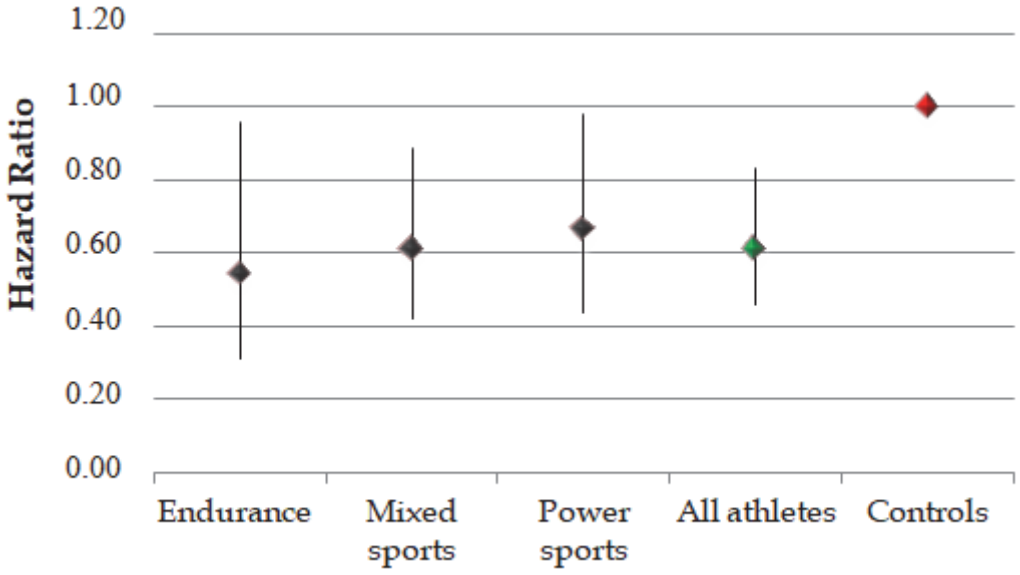


FIGURE 15 Age-adjusted hazard ratios (HRs) (95 % CI) for chronic pulmonary diseases or deaths among former athletes compared to controls.

Figure 16 and Table 6 show that there were the most chronic pulmonary diseases or deaths in profiles 1 (n=56) and 2 (n=47) and the least in profile 4 (n=4) (p<0.001) (*Studies I* and *III*). Profile 1 (total 14.0 % (n=317)) included participants who were physically inactive, used more alcohol than average, and did not smoke (*Study I)*. Correspondingly, profile 2 (21.7 % (n=494)) included participants who were physically inactive, used average amounts of alcohol, but smoked more than average (*Study I)*, whereas profile 4 (15.4 % (n=351)) included participants who were highly physically active, used less alcohol, and smoked less than average (*Study I)*.

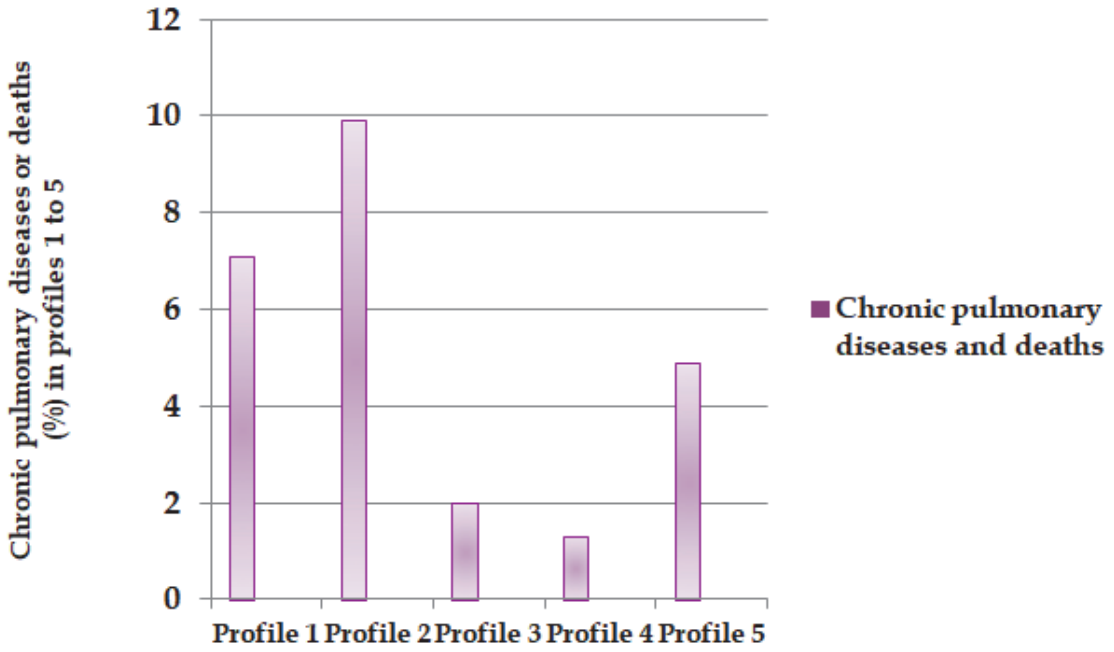


FIGURE 16 Chronic pulmonary diseases or deaths (%) in profiles 1 to 5.

TABLE 6 Chronic pulmonary diseases or deaths % (n)* in profiles 1 to 5.

	Profile 1	Profile 2	Profile3	Profile 4	Profile 5
No chronic pulmonary disease or death % (n)	92.9 (738)	90.1 (430)	98.0 (300)	98.7 (305)	95.1 (369)
Chronic pulmonary disease or death % (n)	7.1 (56)	9.9 (47)	2.0 (6)	1.3 (4)	4.9 (19)

*All participants, $p<0.001$ for group differences in chronic pulmonary diseases or deaths using a χ^2 -test.

5.5 Mortality among former athletes and their brothers (*Study IV*)

The final sample for the mortality analysis included 900 former elite athletes and their 900 age-matched brothers. The mean age at inclusion was 24.8 years, and the mean year of inclusion was 1943, while the median follow-up time was 52.0 years (ranging from 0.1–83.4 years). During the total follow-up period, 1296 deaths occurred; 72.1 % (n=649) of the athletes and 71.9 % (n=647) of the brothers died. The median age at death among the former athletes (75.9 years, 95 % CI

75.1–76.7) was higher than among the brothers (73.0 years, 95 % CI 72.0–74.0). The median age at death among endurance (79.9 years, 95 % CI 79.1–80.7) and mixed sports athletes (75.9 years, 95 % CI 75.1–76.7) was statistically significantly higher than among power sports athletes (74.1 years, 95 % CI 73.3–75.0).

The age-adjusted HRs for all-cause mortality in the former athletes was 0.75 (95 % CI 0.65–0.87, p<0.001) compared to their age-matched brothers (Figure 17). In a subgroup analysis, compared to their age-matched brothers, the HR for all-cause mortality was 0.61 (95 % CI 0.45–0.82, p=0.001) for endurance sports athletes, 0.85 (95 % CI 0.65–1.10, p=0.220) for mixed sports athletes, and 0.78 (95 % CI 0.62–0.98, p=0.033) for power sports athletes (For more details, see Supplementary Table 3. in *Study IV)*.

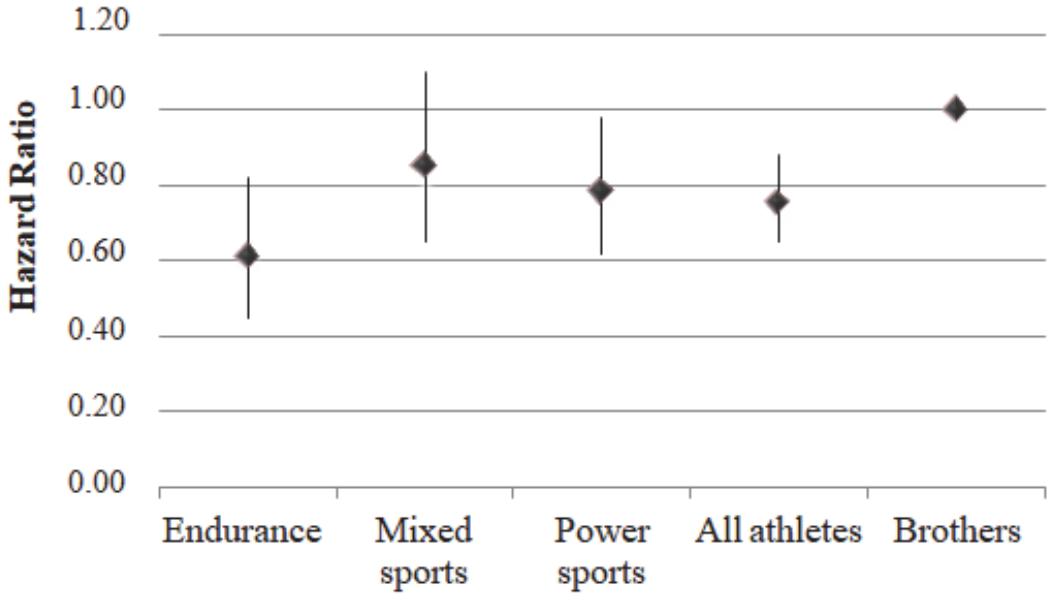


FIGURE 17 Age-adjusted hazard rations (HRs) for all-cause deaths from the time of entry to December 31, 2015, among former athletes (n=900) compared to age-matched brothers (n=900).

It was found that in the individual-based nonpairwise analysis the assumptions of the Cox regression model were violated, suggesting a lower mortality of athletes at younger ages but less so at older ages. Therefore, the follow-up time was divided into two periods: age at entry to median age at death and median age at death to December 31, 2015. The age-adjusted pairwise HRs for all-cause mortality in the former athletes was 0.68 (95 % CI 0.57–0.82, p<0.001) compared to their age-matched brothers in the first follow-up period and 0.74 (95 % CI 0.57–0.98, p=0.03) in the second period (for more details, see Supplementary Tables 4a-d in *Study IV)*. The results of the sensitivity analysis were consistent with those of the age-matched pair analysis, but there was no difference in mortality during the later period.

6 DISCUSSION

Regular PA plays an important role in the prevention and treatment of many chronic diseases and in the reduction of disabilities, especially among elderly people (Heikkinen et al. 1993, King et al. 2000, Heikkinen 2005, Paterson & Warburton 2010). As stated earlier, the health statuses of former Finnish athletes have been previously widely studied (Sarna et al. 1993, Fogelholm, Kaprio & Sarna 1994, Kujala et al. 2003, Bäckmand et al. 2010, Kettunen et al. 2015), but little is known about how former elite athletic careers in different sports groups are associated with health-related behaviors, such as alcohol consumption, smoking, and PA, in later life. It is not known whether a decrease in PA is associated with binge drinking among former athletes. Therefore, the present dissertation revealed novel data on the associations between PA and other health-related behaviors in middle-aged and aging men (*Study I*).

The impact of a history of competitive sports on later alcohol use and the occurrence of alcohol-related diseases is poorly known. Previous studies have mainly focused on cross-sectional associations of alcohol consumption among current athletes, but much fewer investigators have examined alcohol use in longitudinal settings among former athletes. So, the present dissertation investigated how a history of elite-level sports was associated with alcohol consumption in middle-age and with alcohol-related morbidity and mortality (*Study II*). The results of *Study II* showed that the former athletes reported higher alcohol consumption than the controls, but there was no difference in the risk of alcohol-related diseases or deaths between the former athletes and controls.

The impact of a history of competitive sports on later smoking behavior and the occurrence of chronic pulmonary diseases is scarcely studied. The findings of the present dissertation revealed that the risk of any chronic pulmonary disease was lower among the former athletes than the controls while also considering smoking status and pack-years (*Study III*).

There is conflicting evidence on the associations between participation in vigorous sports, health habits, familial factors, and subsequent mortality. The one purpose of the present dissertation was to focus on investigating all-cause mortality and health-related behavior among former elite athletes and their brothers

(*Study IV*). This unique *Study IV* revealed new novel data on the associations between PA, familial factors, health-related behavior, and mortality. The findings of *Study IV* showed that the elite athletes survived 2–3 years longer than their brothers, but there was no difference in the risk of all-cause mortality at older ages. Furthermore, the former elite athletes' overall SRH and health-related lifestyle habits were better than those of their brothers. Correspondingly, health-related habits among the brothers were better compared to the population control (*Study IV*).

6.1 Comparisons with other studies

First, the definition of "*athlete*" in the literature as compared to the present dissertation should be discussed. There are several criteria or types for the classification of athlete in the literature: given sports or modality, position or characteristics, sex, age, nature of participation (recreational or competitive), and type of predominant metabolic pathway for energy production or muscle action (endurance versus strength) (Araújo & Scharhag 2016). Furthermore, competitive and elite athletism is defined as being determined and aiming for international success and ethically sustainable individual and group sports (Robinson & France 2011, OKM 2014). Correspondingly, the AHA, the ACC, and the ESC defined competitive athletes as individuals, either amateur or professional (Pelliccia et al. 2005), who are engaged in official sports competitions (Maron et al. 2004, Pelliccia et al. 2005, Maron, Zipes & Kovacs 2015) in an organized team or individual sport (Maron et al. 2004, Maron, Zipes & Kovacs 2015). These definitions are in line with those in the previous studies of the same cohort, as well as in the present dissertation, former elite athletes have been defined as men who had represented Finland at least once at the Olympic Games, European or World championships, or international contests between two or three countries between 1920 and 1965 (Sarna et al. 1993).

Generally it is good to notice that there were some limitations in previous studies, such as alcohol consumption was self-reported, data were collected on athletes from a single university, studies were focused on limited age and sports groups and levels of athletic participation (not represented all the athletes and limited the generalizability), and data were cross-sectional in nature (limited the causal implications) (Martens et al. 2011, Dams-O'Connor, Martin & Martens 2007, Simon & Docherty 2017). Furthermore, selectional bias might be occurred (Simon & Docherty 2017) and sample size was varied in different studies.

6.1.1 Physical activity, alcohol use, and smoking among former athletes and controls (*Study I*)

There is a lack of studies focused on PA, alcohol consumption, and smoking among former athletes several decades after their peak sporting performance. Competitive sports are often associated with high levels of PA (Sorenson et al.

2015), although the evidence on former competitive athletes' long-term PA behavior is conflicting. Some studies have observed that former athletes are less physically active than controls (Reifsteck, Gill & Brooks 2013, Sorenson et al. 2015). The present dissertation found that former athletes were more physically active after their athletic careers than their controls, which has also been previously reported (Sarna et al. 1993, Fogelholm, Kaprio & Sarna 1994, Sarna et al. 1997, Kujala et al. 1998, Bäckmand et al. 2006, Bäckmand et al. 2010, Laine et al. 2014). Correspondingly, the present dissertation found that former athletes were more physically active than their age-matched brothers who were more physically active than population controls.

The findings of this dissertation support the hypothesis that former athletes smoke less than controls. In line with the present findings, the previous studies on this cohort (Sarna et al. 1993, Fogelholm, Kaprio & Sarna 1994, Bäckman et al. 2010) and other studies have shown that current athletes smoke less than non-athletes (Wechsler et al. 1997, Peretti-Watel et al. 2003, Alaranta et al. 2004, Martinsen & Sundgot-Borgen 2014). Additionally, the present dissertation revealed that former athletes smoked less than their brothers, while brothers smoked less than population control.

The findings of this dissertation were partly consistent with those of other studies that have shown that current athletes consumed more alcohol than non-athletes (O'Brien & Lyons 2000, Nelson & Wecshler 2001, Martens et al. 2006a). In the present dissertation, this was found only in the 1985 questionnaire study but not later. So, it seems that engagement in sports is not replaced with alcohol use among former athletes after an active sports career if it did not end suddenly by sports injury. However, it was found that alcohol consumption after a top-sports career was greater if participation in leisure-time sports was discontinued. It has been recognized that sports career termination causes significant changes in an athlete's personal and social life (Wylleman, Alfermann & Lavallee 2004). Furthermore, adaptation to forced retirement from athletics will be challenging if an individual has a high athletic identity, and the active sports career ends unexpectedly or suddenly (Alfermann, Stambulova & Zemaityte 2004).

Finally, five latent profiles were found in the present dissertation, and there were significant differences between the former athletes and controls in the probabilities of belonging to four of them. The former athletes were more likely than the controls to belong to a profile in which individuals were more physically active, used less alcohol, and smoked less. Changes in the profile means were seen. First, in three profiles, PA decreased, while it increased in only one profile during the follow-up, reflecting the overall decrease in PA with age (Caspersen, Pereira & Curran 2000, Craig et al. 2004, Sun, Norman & While 2013). Second, alcohol use both increased and decreased in two profiles and increased in two profiles during the follow-up, reflecting the overall increased alcohol consumption in Finland in the 1980s, 1990s, and early 2000s. Third, smoking decreased in all the profiles during the follow-up, reflecting the strong decrease of smoking among Finnish men during the same time (THL 2018b).

6.1.2 Alcohol use, alcohol-related diseases, and deaths among former athletes and controls (*Study II*)

There is a paucity of studies focused on the risk of alcohol-related diseases or alcohol consumption among former athletes several decades after their peak sporting performance. However, the findings of the present dissertation, based on the 1985 questionnaire study, were consistent with those of other studies that have shown that current athletes consumed more alcohol than nonathletes (Leichliter et al. 1998, Garry & Morrissey 2000, O'Brien & Lyons 2000, Nelson & Welcher 2001, Martens et al. 2006a, Dietze, Fitzgerald & Jenkinson 2008, O'Brien & Kypri 2008, Wichstrøm & Wichstrøm 2009, O'Farrell et al. 2010, O'Brien et al. 2011, Bedendo et al. 2013, Sønderlund et al. 2014), and alcohol consumption was more common among team sports athletes than other sports (Brenner & Swanik 2007, Partington et al. 2013). The present dissertation showed that combat and team sports athletes consumed alcohol the most, while jumpers and hurdlers and shooters and endurance sports athletes consumed less alcohol than other groups. Endurance athletes do not appear to engage in excessive and risky alcohol use (Gmel, Kuendig & Daeppen 2009, Martens et al. 2006b).

Presumably, most alcohol-related risk factors among the general population, such as sociodemographic and familial factors, are also applicable to athletes (Martens et al. 2006a). There is limited evidence on why athletes use more alcohol compared to nonathletes (Yusko et al. 2008, Weaver et al. 2013), but there are some motivational factors behind athletes and their reasons for engaging in risky alcohol consumption behaviors (Martens et al. 2010). Sports-induced anxiety or pressure, the competitive nature of athletes, peer- or teammate-induced influence, and cultural relations between athletes and alcohol might be related to risky alcohol consumption (Martens et al. 2006a, Dams-O'Connor, Martin & Marten 2007). Furthermore, it is generally known that alcohol, especially beer, distributors support sports, and commercials for alcohol are commonly used (Martens et al. 2006a). Externalizing behaviors may associate with a higher alcohol consumption and alcohol dependence (Grant et al. 2004, Barr & Dick 2019). Studies have suggested that team sports athletes are more extroverted and neurotic than endurance sports athletes (Allen, Greenlees & Jones 2013), whereas contact sports athletes have high scores on the antisocial and histrionic scales (Sohrabi, Atashak & Aliloo 2011). Bäckmand et al. (2001) previously reported that Finnish former power, combat, and team sports athletes were more extroverted compared to controls. Additionally, shooters and endurance sport athletes had lower neuroticism scores than controls (Bäckmand et al. 2001). Finally, further studies will be needed to investigate the associations between personality of athletes and alcohol consumption and other factors, such as socioeconomic status, education, and social environment.

The same factors, especially personal and psychological factors, may be related to heavy drinking during and after an active athletic career. Studies have found that retirement can lead to an increased use of alcohol among older people, who may use alcohol to counter boredom and combat loneliness (Menninger 2002, Klimstra & Mahgoub 2010, Blazer & Wu 2011). It has been recognized that

sports career termination causes significant changes in an athlete's personal and social life (Wylleman, Alfermann & Lavallee 2004). If an active sports career ends unexpectedly, and an individual has a high athletic identity, adaptation to forced athletic retirement will be challenging (Alfermann, Stambulova & Zemaityte 2004) and might lead to alcohol-related problems (Chambers 2002).

Although overall, the former athletes reported higher alcohol consumption than the controls in the 1985 questionnaire study, the risk of alcohol-related diseases was not increased in the former athletes group. The response rates of those participants who had alcohol-related diseases were lower among the controls compared to the athletes, and heavy alcohol users underestimate their use of alcohol (Poikolainen 1985). It may be that former athletes are more receptive to medical advice, reduce alcohol consumption at the first signs of harmful consumption and, therefore, are hospitalized less often; studies on alcohol-related disease biomarkers would be needed. Furthermore, it could be speculated whether PA protects against some of the harmful consequences of alcohol consumption, such as abdominal adiposity and liver fat accumulation (Leskinen et al. 2009). For example, it has been found that better CRF is associated with the decreased risk of fatty liver (Pälve et al. 2017). However, some studies have found that alcohol-related harms are more common among college athletes than nonathletes (Nelson & Wechsler 2001, Dams-O'Connor, Martin & Marten 2007, Yusko et al. 2008). Correspondingly, athletes had a higher mean Alcohol Use Disorders Identification Test (AUDIT) score than nonathletes (Weiss & Rundell 2011). This was consistent with Partington et al.'s (2013) findings; additionally, they found that team sports athletes seemed to have a higher mean AUDIT score than individual sports athletes.

Finally, it seems that engagement in sports is not replaced with either binge drinking or alcohol dependence among former athletes after an active sports career. Former athletes' overall better health habits (such as high PA and less tobacco use) and the subsequent direct or indirect biological factors are likely to explain the observation that there was no difference in the risk of alcohol-related diseases among former elite athletes compared to controls. This was found despite the greater use of alcohol among former elite athletes than among controls.

6.1.3 Chronic pulmonary diseases and deaths among former athletes and controls (*Study III*)

There is a lack of studies focused on the risk of chronic pulmonary diseases among former athletes several decades after their peak sporting performance. Kujala et al. (1996) reported that the lifetime occurrence of asthma or other pulmonary diseases was not increased in former elite athletes in later life. The results of the present dissertation show that the risk of any chronic pulmonary disease or death was lower among former athletes than controls. Accordingly, the risk was lower among endurance, mixed, and power sports athletes compared to controls. However, some studies have shown that the prevalence of asthma is increased in elite athletes (Turcotte et al. 2003, Helenius, Lumme & Haahtela 2005).

Correspondingly, epidemiologic studies suggest that self-reported and physician-diagnosed asthma are twice as common in Finnish athletes (Alaranta et al. 2004) and elite Norwegian athletes (Nystad, Harris & Borgen 2000) than in control populations. The higher prevalence of asthma reported in athletes may be a result of overdiagnosis, particularly because a diagnosis of asthma is often made based on the individual's history alone (Parsons et al. 2006). Furthermore, respiratory symptoms may occur when exercising at high intensities, which are common in athletes but uncommon in nonathletes, although if nonathletes also exercise at high intensities, respiratory symptoms would probably occur more commonly.

6.1.4 Mortality and health-related habits among former athletes and their brothers (*Study IV*)

It is debated whether high-intensity exercise is beneficial for reducing mortality risk in later life (Blair et al. 1992, Blair & Connelly 1996). Most studies have focused on professional athletes and used the general population as a control group. The findings from these studies differ; some studies showed a lower mortality in athletes than in their nonathletic counterparts from the general population (Karvonen et al. 1974, Menotti et al. 1990, Sarna et al. 1993, Kujala et al. 2001, Lee & Skerrett 2001, Gajewski & Poznanska 2008, Teramoto & Bungum 2010, Garatachea, Santos-Lozano & Sanchis-Gomar 2014, Kettunen et al. 2015), whereas other studies did not find a survival benefit (Schnohr 1971, Beaglehole & Stewart 1983, Waterbor et al. 1988, Belli & Vanacore 2005, Kuss, Kluttig & Greiser 2011, DeKosky, Jaffee & Bauer 2018, Nguyen, Zafonte & Chen 2019).

The present dissertation extends the previous findings that former athletes, especially former endurance sports athletes, have a higher longevity and lower mortality than the general population (Sarna et al. 1993, Kettunen et al. 2015). In general, runners have a 25 % – 40 % reduced risk of premature mortality and live approximately 3 years longer than nonrunners (Lee et al 2017). Although a high lifelong exercise volume among surviving athletes is associated with coronary artery calcifications, the atherosclerotic plaques of the most active athletes have a more benign composition (Aengevaeren et al. 2017). The evidence is mostly based on observational follow-up studies, one limitation of which is the difficulty of controlling for genetic or other selection bias. There is no irrefutable evidence on a causal relationship between PA in adulthood and mortality. Based on previous studies on twins and former elite athletes (Kujala et al. 1998, Kujala et al. 2003a) and both animal and human findings, it could be proposed that some of the association between high PA (Karvinen et al. 2015, Lavie et al. 2015, Harber et al. 2017) and low mortality is explained by genetic or other familial factors. Despite this, the study found a lower mortality in former athletes, especially at a younger age, compared to their brothers, knowing the limitation that some brothers may have had health concerns at the beginning of the follow-up.

It was found that there was no difference in anthropometry (BMI) between former athletes and brothers. It has been observed that if formerly vigorously physically active individuals reduced their PA, there was a higher risk for body

fat accumulation, which led to a waist gain similar to that associated with being persistently inactive (Rottensteiner et al. 2014). Furthermore, the present dissertation showed that former athletes were more physically active than their brothers in 2001. The findings of the present dissertation support the previous studies on the same cohort that have shown that former athletes were more physically active than controls (Sarna et al. 1993, Fogelholm, Kaprio & Sarna 1994, Sarna et al. 1997, Kujala et al. 1998, Bäckmand et al. 2006, Bäckmand et al. 2010, Laine et al. 2014). Additionally, this study found that the brothers were more physically active and engaged in more vigorous activities than the population control, suggesting a familial contribution to PA that probably originated prior to the elite athlete's career. It is well known that a physically active lifestyle is partly embraced at young adulthood (Telama et al. 2005, Hirvensalo & Lintunen 2011). In particular, those who participated in competitive sports during adolescence were more physically active in later life, too (Kujala et al. 1996). Previous studies have found many factors affecting participation in life-course PA, such as individual characteristics (age, gender, and socioeconomic and health status) and the social environment (Kuh & Cooper 1992, Tammelin et al. 2003, Cleland et al. 2009, Hirvensalo & Lintunen 2011). Furthermore, genetic factors significantly affect the level of PA (Kaprio et al. 1981, Kujala, Kaprio & Koskenvuo 2002).

Although the brothers smoked more than the former athletes, there was no difference in alcohol use. Current athletes (Wechsler et al. 1997, Peretti-Watel et al. 2003, Alaranta et al. 2004, Martinsen & Sundgot-Borgen 2014) and former athletes (Sarna et al. 1993, Fogelholm, Kaprio & Sarna 1994, Sarna et al. 1997, Bäckmand et al. 2006, Bäckman et al. 2010) are known to smoke less than controls, and in the present dissertation, it was found that the brothers also smoked less than the population control. It has been found that persistent physical inactivity in adolescence relates to adult smoking, even after familial factors are considered (Kujala, Kaprio & Rose 2007).

The present dissertation found that there were better SRH and fewer chronic diseases among the former athletes in accordance with a healthy life expectancy. However, the brothers had fewer chronic diseases compared to population based controls. There was no difference in disability, possibly because those with the highest disability may not be able to respond to a questionnaire. As discussed earlier, both the athletes and brothers were physically active, which could partly explain why no differences were found in functioning. Observational studies have found that regular PA is associated with better functioning and well-being, especially at an older age (King et al. 2000, Paterson & Warburton 2010), and a lower morbidity (Kujala et al. 2003a, Gajewski & Poznanska 2008).

6.2 Strengths and limitations of the study

The large study population, long follow-up time, and the use of the valid register data covering all the participants were the strengths of the present dissertation. Because the former elite athletes were a genetically and psychologically selected

group, their health-related behavior at an older age may be healthier than that of the general population. According to Kujala et al. (1996), individuals who have participated in sports competitions at a younger age were more physically active in later life, too. Self-reported data on health-related behaviors include known limitations, but the applied questions on PA (Waller, Kaprio & Kujala 2008), smoking (Vartiainen et al. 2002), and the use of alcohol (Poikolainen 1985) have been previously validated (*Studies I–IV)*. Although heavy alcohol users may underestimate their alcohol use (Poikolainen 1985), self-reported alcohol-use data are generally valid in ranking persons as light, moderate, or heavy users (Miller et al. 2002). In *Study II*, the questionnaire response rates were lower in individuals with alcohol-related hospitalizations, but the risk for bias in comparing the athlete group to the nonathlete group was low. Furthermore, the response rate was lower among the controls compared to the former elite athletes. In addition, the format of the alcohol-related questions was optimal for avoiding misclassification by study groups. Although there may be some variation in assigning alcohol-related diagnoses by physicians, this is unlikely to be related to a history of elite athleticism and, hence, is not a source of bias (*Study II)*.

Although the questionnaire response rates were lower in individuals with chronic obstructive pulmonary disease–related hospitalizations, the risk for bias in comparing the athlete group to the nonathlete group was low in *Study III*. Furthermore, there may have been a selection bias at the beginning of the present study, as persons with suboptimal ventilatory function as young adults may have been incapable of becoming top athletes in endurance sports, thus reducing the prevalence of later chronic pulmonary disease. It could also be discussed whether training in adolescence and young adulthood promoted the development of lung function, yielding greater lung health in later life. Furthermore, it is good to notice that the classification and definition of *COPD* has changed during the decades and is likely to change in the future (*Study III)*.

Although the questions on PA (Waller, Kaprio & Kujala 2008), smoking (Vartiainen et al. 2002), and alcohol use (Poikolainen 1985) have been previously validated, in *Study IV*, the self-reported data on health-related behaviors is only based on an age-matched respondents subgroup from 2001. There was no exact data on either the level of PA across the lifespan or on health at younger ages among the brothers. Partly insufficient data on SES or the causes of death among the brothers could be also considered a limitation. However, each athlete and their brother(s) are from the same family, and any differences in SES may be caused by the athletic career (*Study IV)*.

Finally, the former elite athletes were genetically and psychologically a selective group. Additionally, the controls were fit enough for military participation which considerably limits the generalizability of the findings to general population, but it increases the comparability between former athletes and controls. Because former athletes were a selected group, and they competed at toplevel before 1965, it is not known exactly how well the results can be generalized to

today's elite athletes, to athletes who had competed at a lower level, to non-athletes, to women, or to today's athletes from different sports groups or cultures, such as NFL players (Selden et al. 2009, Helzberg et al. 2010) or US basketball players (Harmon et al. 2016), who seem to have high prevalences of clinically significant cardiac abnormalities (*Studies I–IV*). Additionally, there has been a lot of discussion on traumatic brain injury (TBI) as a risk factor for the development of dementia among athletes in later life, but a general association between TBI and dementia is poorly defined (Barr 2020). Furthermore, it is generally known that compared with elite athletes in the past, today's elite athletes have more progressive training methods, better equipment, techniques, and specializations, and more specific coaches with better knowledge of biomechanics, nutrition, and psychological factors.

6.3 Implications and future directions

Alcohol- and smoking-related health problems are global issues, and alcohol-related problems have become more common among athletes and nonathletes, especially among older people. Therefore, prevention is the keyword. The present dissertation aimed to investigate how a history of elite-level sports was associated with later alcohol consumption or smoking and alcohol- or smoking-related morbidity or mortality. This research revealed new information on health-related habits and the risk of alcohol- or smoking-related morbidity and mortality among former athletes and their age-matched controls. The results may motivate coaches, sports clubs, and public health officials to develop models to prevent such dangers as excessive alcohol consumption among risk sports during career termination. Mostly due to the apparent association with a healthier lifestyle, PA engagement in particular results in a lower prevalence of the risk factors associated with major chronic diseases. These research findings can be considered a stimulus among athletes and nonathletes for engaging in sports at an older age.

There is a lack of studies focused on assessing the effect of interventions implemented in sports settings on alcohol consumption and alcohol-related harms (Kingsland et al. 2016). Correspondingly, there is a paucity of studies focused on the risk of chronic pulmonary diseases among former athletes several decades after their peak sporting performance. In particular, genome-wide association studies (GWASs) have been identifying genetic variants in the development of lung function in early life and the later progression to asthma and COPD but, at most, they account for a modest fraction of the variability in lung function. Whether PA modifies the association between genetic liability to disease and to manifest disease is still largely unknown; PA does attenuate the association of the genetic variants linked to obesity and BMI (Rask-Andersen et al. 2017). There are still challenges to understanding the effects of genetic variants on health and disease, and how they contribute opportunities for therapeutic intervention (Hall, Hall & Sayers 2019).

A deeper understanding of the relationships between alcohol use, smoking and sports is necessary to determine the true consequences of alcohol and tobacco on health and well-being among current and former athletes. Interventions should be developed on how to avoid excessive alcohol consumption among risk sports during career termination. Furthermore, the results of the present dissertation were based on men who were former athletes 50 or more years ago. So, further studies will be needed to investigate the associations between PA, familial factors, health-related behavior, and mortality among today's athletes, non-athletes, and women. Traditional and genetically informative designs are needed to investigate how elite performance relates to future health outcomes.

7 MAIN FINDINGS AND CONCLUSIONS

The present dissertation was based on the long-term follow-up *Studies I–IV*, which found unique data on health-related habits and the risk of alcohol- or smoking-related morbidity and mortality among former athletes. First, new information on the associations between PA and health-related behavior in middle-aged and aging men was revealed (*Study I*). Second, new findings on the risk of alcohol-related diseases and alcohol consumption among former athletes were shown (*Study II)*. Third, new data on the long-term risk of chronic obstructive pulmonary diseases and morbidity and mortality among former athletes was found (*Study III)*. Fourth, novel data on the associations between health-related behavior and mortality among individuals from the same family, who share both genes and childhood environment, was revealed (*Study IV*).

7.1 Main findings

Study I The former athletes were more physically active and smoked less than the controls at the four time points from 1985 to 2008. However, the former athletes consumed significantly more alcohol compared to the controls, but only in the 1985 questionnaire study. The use of alcohol was higher especially if the athletic sports career had ended suddenly by sports injury.

Five latent profiles were found, and there was a statistically significant difference between the former athletes and controls in their probabilities of belonging to four of them. The former athletes were more likely that the controls to belong to a profile in which individuals were more physically active, used less alcohol, and smoked less. Generally PA and smoking decreased, but alcohol consumption could also increase in profiles during the follow-up.

Study II There was no significant difference in the risk of any alcohol-related disease or death between all the former athletes and controls. However, the risk was

higher among both combat sports athletes and weightlifters compared to endurance sports athletes and shooters or jumpers and hurdlers. In 1985, the former athletes consumed more alcohol than the controls. Consequently, team sports athletes consumed significantly more alcohol compared to other athletes and controls. Athletes who were not engaged in leisure-time sports after their active sports career consumed more alcohol than those who were engaged in leisure-time sports or physical activities. Finally, current and former smokers consumed more alcohol than non-smokers.

Study III The risk of any chronic pulmonary disease was lower among the former athletes than the controls, also considering smoking status and pack-years. Furthermore, the risk was lower among endurance, mixed, or power sports athletes compared to controls. The former athletes were more physically active, were less often smokers, had started smoking later, and smoked less than the controls. The former athletes also reported less physician-diagnosed emphysema than the controls in the 1985 survey.

Study IV The former elite athletes survived 2–3 years longer than their brothers, with the largest difference seen for endurance athletes and none for power athletes. However, there was no difference in the risk of all-cause mortality between the athletes and all their brothers at an older age. Overall, the former elite athletes' SRH and health-related lifestyle habits were better than those of their brothers. Nevertheless, there were no significant differences in mobility or in physical or psychosocial functioning of daily living between the former athletes and their brothers who responded to the 2001 questionnaire. Finally, the health-related habits among the brothers were better compared to the population control.

7.2 Conclusions

Study I The former athletes have distinct profiles from nonathletes with respect to PA, alcohol use, and smoking. However, PA did not predict later alcohol consumption at any time point. Although alcohol consumption may increase in individuals whose athletic career has ended suddenly by sports injury, overall PA and alcohol use affected each other's development only modestly over a 23-year follow-up.

Study II The former athletes reported a higher alcohol consumption than the controls, but there was no difference in the risk of alcohol-related diseases or deaths between the former athletes and controls. The risk varied between different sports groups, being the highest in combat sports athletes and weightlifters. The total alcohol use and HDOs seemed to be significantly more common in team sports than other sports. It is important to note that the risk of excessive alcohol consumption may increase in individuals who do not continue leisure-time sports after elite-level sports career termination.

Study III In conclusion, the ability to compete at the highest level of sports in young adulthood associates with a reduced risk of pulmonary disease in later life.

Study IV The former elite athletes have a lower premature mortality than their brothers and, correspondingly, better SRH and health-related lifestyle habits. Former endurance sports athletes and their brothers had a reduced mortality compared to power sports athletes and their brothers. In conclusion, the findings support the previous findings for a role of genetic or childhood family factors in determining high aerobic fitness and reduced mortality.

YHTEENVETO (FINNISH SUMMARY)

Entisten suomalaisten mieshuippu-urheilijoiden fyysisen aktiivisuuden, alkoholinkäytön ja tupakoinnin väliset yhteydet sekä niihin liittyvät sairaudet sekä kuolleisuus.

Tausta: Vähäinen liikunta, tupakointi ja haitallinen alkoholinkäyttö ovat globaaleja ongelmia. Viime vuosina addiktiot ovat lisääntyneet nyky-yhteiskunnassa aiheuttaen inhimillistä kärsimystä yksilötasolla ja suuria kustannuksia väestötasolla. Tutkimustiedon mukaan nykyurheilijat käyttävät verrokkeja enemmän alkoholia ja mediassa keskustellaan paljon entisten huippu-urheilijoiden alkoholinkäytöstä. Toisaalta tutkimusnäyttö siitä, miten urheilu-uran loppuminen vaikuttaa alkoholinkäyttöön on vähäistä. Kilpaurheilu voi iän myötä loppua suunnitellusti tai yllättäen urheiluvammojen vuoksi.

Tavoitteet: Tutkimuksen tarkoituksena oli tutkia entisten huippu-urheilijoiden alkoholinkäytön, fyysisen aktiivisuuden ja tupakoinnin välisiä yhteyksiä sekä niihin liittyviä sairauksia ja kuolleisuutta. Tutkimus sisälsi neljä osatyötä, joilla oli omat päätavoitteensa (Osatyöt I-IV). Ensimmäisen osatyön (I) tavoitteena oli tutkia fyysisen aktiivisuuden, alkoholinkäytön ja tupakoinnin välisiä yhteyksiä pitkittäisseurannassa. Toisen osatyön (II) tavoitteena oli selvittää entisten huippu-urheilijoiden ja heidän verrokkiensa alkoholinkäyttöä sekä siihen liittyvää sairastuvuutta ja kuolleisuutta. Kolmannen osatyön (III) tavoitteena oli tutkia obstruktiivisten keuhkosairauksien riskiä ja niihin liittyvää kuolleisuutta entisillä huippu-urheilijoilla ja verrokeilla myöhemmällä iällä. Neljännen osatyön (IV) tavoitteena oli selvittää, eroavatko entisten huippu-urheilijoiden ja heidän veljiensä kuolleisuus tai elintavat, kuten alkoholinkäyttö, tupakointi ja fyysinen aktiivisuus, toisistaan.

Menetelmät: Aineistoa on kerätty kyselytutkimuksina vuosina 1985, 1995, 2001 ja 2008 sekä lisäksi kliinisillä tutkimuksilla vuosina 1992 ja 2008. Kyselytutkimuksista otettiin analyyseihin mukaan alkoholinkäyttöön, fyysiseen aktiivisuuteen, kilpa- ja kuntourheiluaktiviteettiin, urheilu-uran lopettamisen syihin sekä tupakointistatukseen liittyviä muuttujia, joita vertailtiin urheilijoiden ja verrokkien tai veljien välillä sekä eri urheilulajiryhmittäin (I-IV). Urheilijoiden veljien data on kerätty sukututkimuksella ja rekistereistä, veljet ovat myös vastanneet vuoden 2001 kyselytutkimukseen (IV). Kyselytutkimusten lisäksi aineistonkeruumetodina käytettiin kansallista sairaalapoistorekisteriä ja tilastokeskuksen kuolleisuusrekisteriä, jotka tarjoavat mahdollisuuden alkoholi- ja keuhkosairauksiin liittyvään sairaalahoidon ja kuolleisuuden tutkimiseen (II-III). Tutkimusaineisto analysoitiin SPSS-, Stata- ja Mplus-tilastoanalyysiohjelmilla. Aineiston pääanalyyseinä käytettiin muun muassa latenttiprofiilianalyysiä (LPA) ja kaksimuuttujaista polkumallia (I), Coxin regressiomallia (II-IV) ja Wilcoxon matched pair -analyysiä (IV).

Tulokset: Entiset huippu-urheilijat olivat fyysisesti aktiivisempia ja tupakoivat vähemmän kuin verrokit kaikissa kyselytutkimuksissa, mutta alkoholinkulutuksessa ei ollut eroa ryhmien välillä lukuun ottamatta vuoden 1985 kyselyä (I). Vuoden 1985 kyselyssä entiset huippu-urheilijat käyttivät enemmän alkoholia kuin verrokit (I-II). Alkoholinkulutus oli runsasta etenkin niillä urheilijoilla, joiden

urheilu-ura oli päättynyt yllättäen loukkaantumisen vuoksi tai jotka eivät olleet fyysisesti aktiivisia urheilu-uran jälkeen (I-II). Kaksimuuttujainen polkumalli osoitti, että poikittaiset ja pitkittäiset yhteydet alkoholinkulutuksen ja fyysisen aktiivisuuden välillä olivat pääosin heikkoja (I). Aineistosta löydettiin viisi profiilia, joista neljään kuulumisen todennäköisyys erosi entisten huippu-urheilijoiden ja verrokkien välillä (I). Entiset huippu-urheilijat kuuluivat verrokkeja todennäköisemmin profiiliin, jossa oli eniten fyysisesti aktiivisia, vähän tupakoivia ja vähän alkoholia käyttäviä tutkittavia (I). Toisaalta verrokit kuuluivat urheilijoita todennäköisemmin profiiliin, jossa oli fyysisesti vähemmän aktiivisia, tupakoivia ja alkoholia keskimääräistä enemmän käyttäviä henkilöitä (I). Seurannan aikana tupakointi väheni kaikissa profiileissa ja fyysinen aktiivisuus väheni kolmessa profiilissa, kun taas alkolinkäyttö myös lisääntyi joissakin profiileissa (I). Entisten huippu-urheilijoiden ja verrokkien alkoholisairauksien tai -kuolemien riski ei eronnut toisistaan, vaikka urheilijat käyttivät enemmän alkoholia kuin verrokit vuoden 1985 kyselyssä (II). Suurin alkoholisairauksien riski oli kontaktilajiurheilijoilla ja painonnostajilla, kun taas kestävyysurheilijoilla riski oli pienin (II). Lisäksi tutkimuksessa havaittiin, että urheilijoiden keuhkosairauksien riski oli verrokkeja alhaisempi (III). Entiset huippu-urheilijat elivät 2-3 vuotta pidempään kuin heidän veljensä, mutta eroa ei havaittu myöhemmällä iällä (IV). Entiset huippu-urheilijat olivat myös fyysisesti aktiivisempia, tupakoivat vähemmän ja arvioivat terveytensä paremmaksi veljiinsä verrattuna (IV).

Johtopäätökset: Fyysinen aktiivisuus ei näyttänyt ennustavan entisten huippu-urheilijoiden tai verrokkien alkoholinkäyttöä 23 vuoden seurannan aikana. Toisaalta aineistosta löydettiin viisi profiilia, joissa seurannan aikana fyysinen aktiivisuus ja tupakointi vähenivät, mutta alkoholinkulutus saattoi myös kasvaa. Entisten huippu-urheilijoiden alkoholinkulutus kasvoi, jos urheilu-ura päättyi yllättäen loukkaantumiseen tai he eivät olleet fyysisesti aktiivisia urheilu-uran jälkeen. Toisaalta loukkaantuminen on voinut myös olla uran loppupuolella lisääntyneen alkoholinkäytön seurausta johtaen uran päättymiseen. Entiset huippu-urheilijat raportoivat käyttävänsä enemmän alkoholia, mutta alkoholisairauksien riski ei eronnut verrokeista. Entiset urheilijat tupakoivat vähemmän kuin verrokit. Kyky kilpaurheilla varhaisaikuisuudessa näyttäisi olevan yhteydessä alhaisempaan kroonisten keuhkosairauksien riskiin myöhemmällä iällä. Entisten huippu-urheilijoiden ja veljien väliset geneettiset eroavaisuudet, kestävyysurheilijoiden aerobinen harjoittelu ja terveellisemmät elintavat saattavat olla yhteydessä alhaisempaan kuolleisuuteen.

Tämä tutkimus on merkittävä etenkin näinä COVID-19-pandemian aikoina, jolloin terveellisten elintapojen merkitys eri sairauksien preventiossa ja niiden hoidossa korostuu. Tulokset voivat motivoida valmentajia, urheilujärjestöjä, terveydenhuollon ammattilaisia ja päättäjiä kehittämään malleja, joilla ennaltaehkäistään alkoholin riskikulutusta urheilu-uran päättyessä tai sen jälkeen. Tutkimus voi kannustaa etenkin ikääntyviä sitoutumaan fyysisesti aktiivisempaan elämäntapaan urheilijoiden ja muun väestön keskuudessa. Lisäksi tämä ajankohtainen tutkimus voi toimia katalyyttinä jatkotutkimuksille, sillä tässä aihepiirissä riittää vielä paljon selvitettävää.

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APPENDICES

Appendix 1

TABLE A1.1. International Classification of Diseases (ICD) codes and main diagnostic categories for alcohol-related diseases. (Table originally published as Supplementary Table in *Study II* in 2017)

Diagnostic categories	ICD8 1969–1986	ICD9 1987–1995	ICD10 1996–	No. of participants in diagnostic categories (N)*
Mental and behavioral disorders due to the use of alcohol (except alcohol dependence F10.2)	**291 Alcoholic psychosis 291.00–291.30, 291.98–291.99**	**291 Alcoholic psychoses 291A–2914A, 2918A**	**F10 Mental and behavioral disorders due to the use of alcohol F10.0–F10.9 (except F10.2)**	**87**
Alcohol dependence syndrome	**303 Alcoholism 303.00–303.20, 303.98–303.99**	**303 Alcohol dependence syndrome 303, 3039X, 980.01, 980.98**	**F10.2Alcohol dependence syndrome F10.20–10.29**	**157**
Alcoholic liver disease	**571.00 Alcoholic cirrhosis of liver 571.01 Alcoholic fatty liver**	**571 Chronic liver disease and cirrhosis 5710A,5711A,5712A, 5713X**	**K70 Alcoholic liver disease K70.0–70.4, 70.9**	**28**
Diseases of the pancreas		**577 Diseases of the pancreas Acute Pancreatitis 5770D–F Chronic Pancreatitis 5771C–D**	**K 86 Other diseases of the pancreas K86.00 Alcohol-induced acute pancreatitis K86.01Recidivans K86.08 Alcohol-induced chronic pancreatitis**	**13**
Other alcohol-related diseases		**3573 Alcoholic polyneuropathy 4255 Alcoholic cardiomyopathy 5353 Alcoholic gastritis**	**G31.2Degeneration of the nervous system due to alcohol G40.51 Epileptic seizures related to alcohol G62.1 Alcoholic polyneuropathy G72.1 Alcoholic myopathy I42.6 Alcoholic cardiomyopathy K29.2 Alcoholic gastritis**	**7**

*One person could have more than one diagnosis if there are several admissions for alcohol-related diseases.

Appendix 2

Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria

Classification of severity of airflow limitation in COPD:

Airflow limitation is categorized into 4 stages by the GOLD system.GOLD stages 1–4 in patients with FEV1/FVC <0.70 (diagnostic for COPD) are presented in Table A2.1:

TABLE A2.1 GOLD stages 1–4.

GOLD stages	**Severity of airflow limitation in COPD**
GOLD 1	(1) mild: FEV1≥ 80 % predicted
GOLD 2	(2) moderate: 50 % ≤FEV1 <80 % predicted
GOLD 3	(3) severe: 30 % ≤FEV1 <50 % predicted
GOLD 4	(4) very severe: FEV1 <30 % predicted

Reference: Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2018. A global strategy for the diagnosis, management, and prevention of COPD.

Appendix 3

TABLE A3.1. International Classification of Diseases (ICD) codes and main diagnostic categories for chronic pulmonary diseases. (Table originally published as Supplementary Table in *Study III* in 2019)

Diagnostic categories	ICD8 1969–1986	ICD9 1987–1995	ICD10 1996–	No. of cases in diagnostic categories (N)*
Chronic bronchitis	490 Chronic bronchitis 490.01, 491.02, 491.04, 491.09	491 Chronic bronchitis (NUD) 4910A, 4911A, 4912A, 4912B, 4919X	J40 Bronchitis, not specified acute or chronic J41 Simple and mucopurulent chronic bronchitis J41.0, J41.1, J41.8 J42 Unspecified chronic bronchitis	30
Emphysema	492 Emphysema 492.01, 492.02, 492.09	492 Emphysema 4920A, 4928A, 4928X	J43 Emphysema J43.0–43.2, J43.8–J43.9	2
Other obstructive pulmonary disease			J44 Other obstructive pulmonary disease J44.0–J44.1, J44.8–J44.9	24
Asthma	493 Asthma 493.00, 493.01, 493.02, 493.08, 493.09	493 Asthma 4930A, 4930B, 4931A, 4931B, 4939A, 4939B, 4939C, 4939X	J45 Asthma J45.0–J45.1, J45.8–J45.9 J46 Status asthmaticus	72
Bronchiectasia	518.99Bronchiectasis	494 Bronchiectasis	J47 Bronchiectasis	5

*One person could have more than one diagnosis if there are several admissions for obstructive pulmonary diseases.

Appendix 4

TABLE A4.1. Characteristics of participants who answered physical activity-, alcohol- or smoking-related questions in 1985, 1995, 2001, or 2008. (Table originally published as Supplementary Table S1 in *Study I* in 2020)

	Former athletes	**Controls**	**P-value**
Age 85 (years) Mean (95% CI)	57.8 (56.2–57.4)	55.0 (54.3–55.8)	0.001[b]
SES 85 %(n)			p <0.0001[a]
Executive	25.4 (506)	9.5 (123)	
White collar	37.5 (748)	22.0 (286)	
Blue collar	30.0 (598)	39.7 (516)	
Unskilled worker	2.3 (46)	9.9 (129)	
Farmer	4.7 (94)	18.5 (240)	
Marital status 85 %(n)			p <0.0001[a]
Unmarried	3.7 (47)	7.9 (61)	
Married	76.4 (975)	74.5 (578)	
Remarried	6.0 (76)	4.3 (33)	
Cohabitated	2.3 (29)	3.7 (29)	
Divorced	6.9 (88)	6.2 (48)	
Widowed	4.8 (61)	3.5 (27)	
Working status 85 %(n)			p <0.0001[a]
Employed	61.8 (783)	59.3 (455)	
Retired for old age	25.6 (325)	20.9 (160)	
Retired for disability	11.0 (139)	16.0 (123)	
Unemployed	1.7 (21)	2.2 (44)	
PA 85 Mean (95% CI)	30.3 (28.4–32.3)	14.9 (13.2–16.6)	p < 0.0001[b]
PA 95 Mean (95% CI)	30.4 (28.2–32.6)	16.7 (14.9–8.5)	p < 0.0001[b]
PA 01 Mean (95% CI)	28.7 (26.5–31.0)	18.1 (16.1–20.2)	p < 0.0001[b]
PA 08 Mean (95% CI)	31.4 (28.5–34.2)	20.5 (17.5–23.6)	p < 0.0001[b]
Use of alcohol 85 (g/month) Mean (95% CI)	425.3 (393.5–457.2)	397.5 (354.5–440.5)	0.007[b]
Heavy drinking occasion (HDO) %(n)	32.9 (399)	31.8 (234)	0.601[a]
Use of alcohol 95 (g/month) Mean (95% CI)	427.4 (391.2–463.7)	433.9 (384.6–483.1)	0.831[b]
Use of alcohol 01 (g/month) Mean (95% CI)	261.6 (224.8–298.3)	258.7 (207.1–310.3)	0.191[b]
Use of alcohol 08 (g/month) Mean (95% CI)	275.5 (239.3–311.7)	266.1 (216.2–316.1)	0.486[b]
Smoking status 85 %(n)			p <0.0001[a]
Current smoker	21.0 (260)	30.9 (231)	
Former smoker	29.1 (360)	40.8 (305)	
Never-smoker	49.9 (617)	28.3 (212)	

CPD Mean (95% CI)	16.9 (16.1–17.7)	18.4 (17.7–19.2)	p < 0.0001[b]
Smoking status 95 %(n)			p <0.0001[a]
Current smoker	14.9 (140)	25.2 (144)	
Former smoker	32.4 (304)	44.1 (252)	
Never-smoker	52.7 (495)	30.7 (176)	
CPD Mean (95% CI)	1.6 (1.3–1.9)	3.4 (2.9–4.0)	p < 0.0001[b]
Smoking status 01 %(n)			p <0.0001[a]
Current smoker	10.3 (80)	18.9 (76)	
Former smoker	32.0 (248)	44.7 (180)	
Never-smoker	57.7 (448)	36.5 (147)	
CPD Mean (95% CI)	5.7 (5.0–6.4)	9.7 (8.7–10.7)	p < 0.0001[b]
Smoking status 08 %(n)			p <0.0001[a]
Current smoker	5.2 (22)	9.4 (22)	
Former smoker	23.8 (100)	46.2 (108)	
Never-smoker	71.0 (299)	44.4 (104)	
CPD Mean (95% CI)	0.4 (0.2–0.6)	1.3 (0.7–1.9)	p < 0.0001[b]
Cof-fee(cups/day) 85 Mean (95% CI)*	4.4 (4.2–4.5)	5.2 (4.9–5.4)	p < 0.0001[b]
Cof-fee(cups/day) 95 Mean (95% CI)	3.8 (3.6–3.9)	4.1 (3.8–4.3)	0.081 [b]
Cof-fee(cups/day) 08 Mean (95% CI)*	3.1 (2.9–3.3)	3.6 (3.3–3.9)	0.005 [b]

SES=Socioeconomic status
PA=Physical activity
HDO=Heavy drinking occasion
CPD=Cigarettes per day

* p<0.05 for the statistical differences between former athletes and controls using a) the Chi-square test for the categorical variables and b) the Mann–Whitney-U test for the continuous variables.

ORIGINAL PUBLICATIONS

I

PHYSICAL ACTIVITY, USE OF ALCOHOL AND SMOKING IN MIDDLE-AGED AND AGING MEN. A LONGITUDINAL STUDY AMONG FINNISH MALE FORMER ATHLETES AND CONTROLS

by

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Physical activity, use of alcohol and smoking in middle-aged and aging men. A longitudinal study among Finnish male former athletes and controls.

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ABSTRACT

Objective: It is not known whether decrease in physical activity (PA) is associated with binge drinking among former athletes. The purpose of this study was to investigate the reciprocal associations between PA and use of alcohol among former athletes and controls at four time points. Furthermore, we examined whether there were longitudinal latent profiles related to use of alcohol, smoking and PA during the follow-up.

Methods: Finnish male former elite athletes (n=1633) and matched controls (n=1099) questionnaire-reported their PA, alcohol consumption and smoking at four time points in 1985, 1995, 2001 and 2008.

Results: Former athletes were more physically active and smoked less than controls, but in all profiles smoking decreased during the follow-up. Former athletes consumed alcohol significantly more compared to controls in 1985, especially if their athletic career had ended suddenly by sports injury. At other time points, no differences were seen. Five latent profiles were found, and there were significant differences between former athletes and controls in the probabilities to belong to four of them. PA decreased in four of five profiles, while alcohol consumption decreased or increased in some profiles. But PA did not predict later alcohol consumption at any time point. Cross-lagged path model indicated that the mutual associations of alcohol use and PA were weak at most.

Conclusions: Although risk of excessive alcohol consumption may increase in individuals, whose athletic career has ended suddenly by sports injury, overall PA and alcohol affected each other's development only modestly among former athletes and controls during the 23-year follow-up.

Key words: ALCOHOL, COHORT STUDY, FORMER ATHLETE(S), PHYSICAL ACTIVITY, SMOKING.

INTRODUCTION

There is a widespread scientific and public health policy consensus that behavioral factors such as low physical activity (PA), cigarette smoking, and alcohol drinking are major contributors to morbidity and mortality.[1-3] PA exerts several positive health effects on adult chronic diseases, on psychological health and functionality.[4]

Cross-sectional and longitudinal studies have found that habitual physical inactivity, compared to continuous PA during late adolescence, predicts higher prevalence of smoking during young adulthood even after familial factors are accounted for.[5-6] Correspondingly, people who drink are much more likely to smoke,[7-8] and dependence on alcohol and tobacco is also correlated.[9]

Studies have found that current and former athletes smoke less than non-athletes.[10-14] Correspondingly, sports participation is identified in some studies as a protective factor against drinking in young individuals.[15] But athletic participation has been associated in other studies with excessive alcohol consumption,[16-18] alcohol-related problems[19] and alcohol dependence.[20] There is a paucity of scientific data on alcohol consumption among former athletes, because most studies are only focused on drinking habits among younger and current athletes.[15-20] Correspondingly, it is not known whether decrease in PA is associated with increased alcohol use and binge drinking among former athletes in later life. Furthermore, the longitudinal course of drinking among individuals with variable degrees of athletic participation is less studied.[16]

Studies have found that retirement can lead to increased binge drinking among older people, who may use alcohol to combat loneliness and counter boredom.[21-23] It has been detected that if an active sports career ends unexpectedly and an individual has a high athletic identity, adaptation to forced athletic retirement will be challenging [24] and it might lead to alcohol-related problems.[25]

The first aim of this study was to examine if there was difference in use of alcohol or smoking between Finnish former athletes from a well-studied cohort having different reasons for quitting active sports career. Correspondingly, it is not known whether changes in PA are associated with changes in the overall consumption and pattern of use of alcohol among former athletes. The second purpose of this study was to investigate the associations between PA and use of alcohol among former elite athletes and controls at four time points spanning 23 years from middle-age onwards after an active sports career. Furthermore, we examined whether the characteristics of longitudinal latent profiles related to use of alcohol, smoking and PA during the follow-up. So, this unique longitudinal study revealed novel data on the associations between PA and other health-related behaviors in middle-aged and aging men.

METHODS

Participants

An original cohort of former elite athletes (n=2657) was formed by identifying men who had represented Finland between 1920 and 1965 at least once at the Olympic Games, European or World championships, or international contests between two or three countries.[14,26] A control cohort (n=1712) was selected from Finnish men who at the age of 20 years had been identified healthy in the medical inspection for enlisting in ordinary military service (class A1, which means fully fit for ordinary military service). The control cohort was formed by matching the same age groups and area of residence with the former elite athletes.[14,26] After first finding the athlete in the register, the selection of each control subject was done. The control subject was chosen nearest the A1 conscript listed to the athlete. This procedure was carried out in the years 1978-1979, when 85.3 % of the athletes had been identified.[26]

Participants who had died before the follow-up started in January 1, 1985 were excluded from this study. Thus, the final study population was 2732 men (1633 former athletes, 1099 controls) shown in Table 1 and

Figure 1. The athletes were divided into three groups according to the type of training needed to achieve optimal results:[27] endurance (n=287), mixed (n=769) and power sports (n=577).

Participants were not involved in setting the research question, the outcome measures or study design. Before taking part in the study all the participants gave informed consent by returning the questionnaires, which were accompanied by a cover letter explaining the purpose of the study. This study was conducted according to good clinical and scientific practice and the Declaration of Helsinki. Ethical approval for questionnaire data collection was given by The ethics committee of the Hospital District of Helsinki and Uusimaa, and the Ministry of Social Affairs and Health in Finland.

The 1985, 1995, 2001 and 2008 questionnaire studies

Questionnaires eliciting information on reasons for ending sports career, socio-demographic factors, and health-related lifestyle habits, were sent to the surviving cohort members in the years 1985, 1995 and 2001 (Figure 1). In 2008, an invitation to participate in a clinical study was sent to all former athletes and controls who were still alive and had answered at least one of the previous questionnaires. The clinical study included a physical examination, laboratory tests and questionnaires.[28] Former elite athletes (n=1255, 82% response rate in 1985) and controls (n=764, 76% response rate in 1985), who answered the physical activity-, alcohol- or smoking-related questions in any questionnaires, were included in the statistical analysis. Because the analyses were made by using the full information maximum likelihood method, there was no need to use imputation. The missing values are supposes to be missing at random (MAR).

Questionnaire-based covariates

The volume of physical activity (MET-hours/week, MET, metabolic equivalent) in questionnaires was computed from the responses to three structured questions on intensity, duration and frequency of activity using a previously validated method.[29]

Alcohol consumption based on quantity-frequency measures of beverage use, asked separately for beer, wine and spirits, was converted into grams of pure alcohol per month as previously reported[14,30] in each survey. In the 1985 questionnaire study alcohol consumption was assessed by heavy drinking occasions (HDO). HDO is defined as drinking 5 or more alcoholic drinks (>60 grams of pure alcohol) on a single occasion on at least 1 day in the past 30 days.[31] Responses formed two categories: no HDO and at least one HDO.[14] The smoking status of the participants was classified into three categories from responses to a detailed smoking history: never, former, current (including occasional) smokers (for more details see Table S1, Supplemental Digital Content 1). Furthermore, cigarettes per day (CPD) for former and current smokers was calculated based on all questionnaires. Use of coffee (cups per day) was based on responses from 1985, 1995 and 2008 questionnaires.

Marital status of the participants was classified into 6 categories in the questionnaire study of 1985: unmarried, married, remarried, cohabitated, divorced, widowed. The working status of the participants was classified into 4 categories in the questionnaire study 1985: employed (salaried or self-employed), retired for old age, retired on a disability pension and unemployed. The participants were classified into five social class groups: executives and professionals, lower white collar workers, skilled (blue) collar workers, unskilled workers and farmers [32] according to the occupation in which they had practiced the longest (for the distributions see Table S2, Supplemental Digital Content 2). This classification also reflects the occupational loading and socioeconomic status of the participants. The social group distribution of athletes differed from that of controls ($p<0.001$, χ^2 -test). Occupational data were collected partly from the Central

Population Registry of Finland and partly from the 1985 questionnaire, asking for the occupation in which they had been active the longest.

Reasons for ending the athletic sports career was categorized into six groups based on questions in the 1985 questionnaire: enthusiasm had ended, sports injury, work or studies, age, disease and other reasons.

Statistical analysis

The descriptive data are presented as the mean and standard deviation (SD) or 95% confidence intervals (CI) if distributed normally; otherwise the descriptive data are shown as the median and range. The differences in the distributions of the categorical variables were examined using cross-tabulations with the Chi-square (χ^2) -test.[14]

The questionnaire data was analyzed using non-parametric Kruskall-Wallis-test and using the Dunn-Bonferroni approach for post hoc testing in pairwise comparisons for more than two groups ($p<0.05$), because some of the variables were not normally distributed and variances were not equal. The Mann-Whitney-U-test was used to compare differences between sports groups and controls ($p<0.05$). Homogeneity of variances were assessed using Levene`s test and normality using Kolmogorov-Smirnov`s test ($p<0.05$).[14]

A bivariate cross-lagged path model was used to investigate the direction and magnitude of the associations between PA and alcohol consumption at four time points. The full cross-lagged path model and its specifications are shown in Supplemental Digital Content 4 (Figure S4). A bivariate cross-lagged path model was not used to examine associations between PA and smoking, because neither CPD nor categorical smoking status variable were not appropriate to this model.

Latent profile analysis (LPA) was used to find simultaneous developmental profiles in PA, alcohol and smoking behaviors measured at 1985, 1995, 2001 and 2008. Profiles in the latent classes were specified to differ in the mean values of PA and alcohol consumption whereas smoking behavior was specified as categorical (three categories) allowing the proportion of three categories differ between profiles. Variances of the two continuous variables were allowed to differ between latent profiles. The estimation method was full information maximum likelihood with robust standard errors estimates (MPLUS).

Missing values were supposed to be missing at random (MAR). Total sample size was 2275 in which covariance coverage in year 1985 was between 0.85 - 0.86, in year 1995 0.56 - 0.67, in year 2001 0.48 - 0.52 and in year 2008 0.23 - 0.29. For details of the analysis method and use of covariates, see Supplemental Digital Contents 5 and 6.

P-values <0.05 were considered statistically significant. Statistical analyses were performed using SPSS statistical software (versions 24.0 and 26.0 for Windows; SPSS Inc., Chicago, IL), and Mplus statistical software package (version 8.2 for Windows; Mplus Corp, California).

RESULTS

The former athletes (n=1276) were 56.8 (SD 11.0) years old on average in 1985, compared to 55.0 (SD 10.3) years among controls (n=777 respondents). Former athletes self-reported reasons for quitting athletic career in the 1985 questionnaire: 22.6 % (n=231) enthusiasm had ended, 20.2 % (n=207) sports injury, 9.9 % (n=101) work or studies, 32.1 % (n=328) age, 1.8 % (n=18) disease and 13.5 % (n=138) other reasons. In 2008 the mean age of the former athletes (n=747) was 73.8 (SD 7.0) years and controls 72.8 (SD 6.3) years (N=436) (for more details, see Table 1).

Survey descriptives in 1985, 1995, 2001 and 2008

Former athletes were more physically active than controls at the four time points, also mean of MET-hours/week was significantly higher among former athletes (30.3 MET-hours/week in 1985 and 31.4 MET-hours/week in 2008) than controls (14.9 MET-hours/week in 1985 and 20.5 MET-hours/week in 2008) during the 23-year follow-up ($p<0.0001$) (For more details see Table S1, Supplemental Digital Content 1).

In 1985 former athletes consumed more alcohol (425 g/month) compared to controls (398 g/month) ($p<0.05$), but the proportion having HDOs did not differ between former athletes and controls ($p=0.60$) (For more details see Table S1, Supplemental Digital Content 1). The use of alcohol was higher among former athletes if their athletic sports career had ended suddenly by sports injury (524 g/month) or enthusiasm had ended (536 g/month) compared to age (478 g/month) or disease (406 g/month) ($p<0.05$). Correspondingly, the proportion having HDOs differed between reasons for athletic career ending ($p<0.001$): HDOs were more common among former athletes if their athletic sports career had ended suddenly by sports injury (35.8 %) or enthusiasm had ended (43.5 %) compared to age (12.0 %) or disease (28.4 %).

Reasons for quitting active sports career among former athletes were not associated with smoking status ($p=0.21$). Controls smoked more than former athletes at all surveys ($p<0.0001$). In 1985 31 % of controls and 21% of former athletes were current smokers, while 9 % of controls and 5 % of former athletes were current smokers in 2008 (For more details see Table S1, Supplemental Digital Content 1). Finally, current and ex-smokers consumed more alcohol than non-smokers in 1985 ($p<0.05$).

A bivariate cross-lagged path model

The specific bivariate cross-lagged model fitted well to the data (Model fit: Chi^2 (d.f.=9)=10.29, p=.328, RMSEA=0.007, CFI=0.999, TLI=0.997 and SRMR=0.018 (see Figure S4)). R-squares (R^2) and standardized regression coefficients for stability of PA and use of alcohol between consecutive time points are given in Supplemental Table for all participants, former athletes and controls (see Table S3, Supplemental Digital Content 3). R^2- values for use of alcohol in 1995, 2001 and 2008: 0.74, 0.82 and 1.00. R^2- values for PA in 1995, 2001 and 2008: 0.66, 0.84 and 0.73 (PA 2008) (for more details, see Table S3 and Figure S4). Among all participants there was only one statistically significant cross-lagged regression coefficient -0.10 (beta) from use of alcohol in 1995 to PA in 2001 (p<0.001). Correspondingly, the correlation of -0.14 between use of alcohol and PA was statistically significant only in 1985 (p<0.001) (see Figure S4, Supplemental Digital Content 4). There was no interaction between athletic status and the use of alcohol ($\chi^2(12) = 14.55, p = 0.267$).

Latent profile analysis (LPA)

According to the Adjusted Lo-Mendell-Rubin likelihood ratio test (AdjLMR) tests the five latent profile solution fitted to the data best. At least five latent profiles were supported by the BIC (see Table S5, Supplemental Digital Content 5). Even if the BIC value decreased to the six latent profile solution the mean profiles for two latent classes were very similar. So, we decided upon the five latent profile solution. In the Figure 2 the mean values of PA and use of alcohol (standardized using whole data) and estimated proportions of smoking status showed that the longitudinal profiles were very stable across measurements. Confidence intervals for key estimates are presented in Supplementary Tables S6b (standardized) and S6c (distributions).

Profile 1 (total 14.0 % (n=317)) included participants who were physically inactive, used more alcohol than average and did not smoke. Profile 2 (21.7 % (n=494)) included participants who were physically inactive, used average amounts of alcohol, but smoked more than average. Profile 3 (30.8 % (n=700)) included participants who were physically inactive, used less alcohol and were average smokers. Profile 4 (15.4 % (n=351)) included participants who were highly physically active, used less alcohol and smoked less than average. Profile 5 (18.1 % (n=413)) included participants who were highly physically active, used alcohol and smoked more than average (Figure 2).

During follow-up, PA decreased in profile 1, while the use of alcohol both increased and decreased, and smoking decreased ($p<0.05$). In profile 2 PA and the use of alcohol increased, but smoking decreased over time ($p<0.05$). In profile 3 PA, the use of alcohol and smoking decreased ($p<0.05$). In profile 4 PA decreased, the use of alcohol increased and smoking decreased over time ($p<0.05$). In profile 5 there was very little change in PA and the use of alcohol between surveys, but smoking decreased ($p<0.05$) (for more details, see Figure 2 and Supplemental Digital Contents 6 and 7, tables S6b-c and description).

Profile 4 differed from other profiles in PA at every timepoint ($p<0.05$), but profiles 1 and 3 did not differ from each other. There was a significant difference in the use of alcohol between all profiles at every timepoint ($p<0.05$), except profiles 1 and 2 did not differ from each other at baseline (1985) (Figure 2). Furthermore, there were statistically significant differences in the proportion of current smokers between profiles at all timepoints, but profiles 3 and 4 as well as profiles 2 and 5 did not differ from each other (for more details, see Figure 2 and Supplemental Digital Contents 6 and 7, tables S6b-c and description).

Average Latent Class Posterior Probabilities (AvePP) showed a clear distinction of latent profiles, which were 0.90, 0.87, 0.83, 0.90 and 0.94 for latent profiles 1-5. Probability of former athletes (62.0 % (n=1410)) belong to profile 1 was 0.17, profile 2 was 0.15, profile 3 was 0.28, profile 4 was 0.21 and profile 5 was 0.19, whereas probability of controls (38.0% (n=864)) belong to profile 1 was 0.09, profile 2 was 0.33,

profile 3 was 0.35, profile 4 was 0.06 and profile 5 was 0.17 (Figure 3). There was statistically significant difference between former athletes and controls in probability belong to profiles 1-4: profile 1 ($p=0.002$), profile 2 ($p<0.001$), profile 3 ($p=0.002$), profile 4 ($p<0.001$), but not profile 5 ($p=0.31$). Neither mean values of PA and use of alcohol nor proportion of smoking did not differ statistically significantly between former athletes and controls in any profile.

Profile distributions of covariates are presented in tables and figures in supplemental digital contents 6 and 8.

DISCUSSION

Main findings

Former athletes were more physically active and smoked less than controls at all four time points. In contrast, former athletes consumed significantly more alcohol compared to controls only in the 1985 questionnaire study, but not later. Among all participants there was only one statistically significant cross-lagged regression coefficient from use of alcohol in 1995 to PA in 2001, and correspondingly the correlation between use of alcohol and PA was statistically significant only in 1985. The use of alcohol was higher among former athletes especially if their athletic sports career had ended suddenly by sports injury or enthusiasm had ended compared to age or ill-health as reasons for career termination.

We found five latent profiles, and there was a statistically significant difference between former athletes and controls in probabilities of belonging to profiles 1-4, but not profile 5. Within profiles, PA, use of alcohol, and the proportion of smokers did not differ between former athletes and controls in any profile. This indicates that the latent class profiles captured distinct longitudinal lifestyle characteristics of ageing

middle-aged men. Former elite athletes differ from healthy controls in the probability of belonging to a specific profile. Former athletes belonged more likely than controls to profile in which individuals were more physically active, used less alcohol and smoked less. Changes in profile means were seen: generally PA and smoking decreased but alcohol consumption could also increase during the follow-up.

Strengths and limitations of our study

This long-term follow-up study revealed new information on the associations between PA and health-related behavior in middle-aged and aging men. The large study population and long follow-up time were strengths of this study. Self-reported data on health-related behaviors include known limitations, but our questions on PA,[29] smoking[33] and the use of alcohol[34] have been previously validated. Because these former athletes competed at top-level before 1965, we cannot predict whether these results can be generalized to today's elite athletes or athletes who had competed in lower level, to non-athletes or to women. Additionally, the controls were fit enough for military participation which considerably limits the generalisability of this study findings to general population, but it increases the comparability between former athletes and controls. Furthermore, it is generally known that today's elite athletes have more progressive training methods, better equipment, techniques, specializations and more specific coaches with better knowledge of such as biomechanics, nutrition and psychological factors compared with elite athletes in the past.

Comparisons with other studies

Binge drinking and alcohol-related harms are generally known among current athletes.[16-18] But there is a paucity of evidence on the longitudinal course of drinking among current[16] and former athletes and how vigorous PA is associated with alcohol consumption and smoking among former athletes several decades after their peak sporting performance.

Current athletes smoke less than non-athletes as we have shown in previous studies on this cohort[10,13-14] but also by other investigators.[11-12] Furthermore, our findings were partly consistent with other studies that have shown that active athletes consume more alcohol than non-athletes.[16-18] Former athletes consumed more alcohol than controls in the 1985 questionnaire study, but there were no differences later.

Of note is that use of alcohol was higher among those former athletes especially if their athletic career had ended suddenly by sports injury. The role of unplanned change in PA with later increase in alcohol use is consistent with our previous observation that alcohol consumption was greater if participation in leisure-time sports was discontinued after athletic career.[14] Other studies have also observed that if an active sports career ends unexpectedly and an individual has a high athletic identity, adaptation to athletic retirement will be challenging [24] and it might lead to alcohol-related problems.[25] According to Chambers (2002) alcohol-related problems were associated with disability and adaption to life after athletic career among former ice-hockey players.[25] Some studies suggest that sports-induced pressure, peer- or teammate-induced influence, and competitive nature of athletes, might be related to heavy use of alcohol among young athletes.[16,35] Additionally, most alcohol-related risk factors, such as familial and sociodemographic factors, among the general population may also be applicable to current and former athletes.[16] It has been indicated that different factors can predispose to alcohol-related problems in different ages.[36] Studies have found that retirement can lead to increased binge drinking among older people, who may use alcohol to combat loneliness and counter boredom.[21-23]

Finally, we found that PA did not predict later alcohol consumption at any time point. However, findings of the latent profile analysis reflected the overall decrease in PA with age which is consistent with other studies.[37-39] Correspondingly, smoking decreased in all profiles during the follow-up, reflecting the strong decrease of smoking among Finnish men in the same time. However, the use of alcohol both increased and

311 decreased during the follow-up, reflecting the overall increased consumption of alcohol in Finland in the
312 1980s, 1990s and early 2000s.
313
314 **Future directions**
315
316 There is less studies focused on assessing the effect of interventions implemented in sports settings on
317 alcohol consumption.[40] Additionally, further studies will be needed to examine the associations between PA,
318 alcohol consumption and smoking among today's athletes, non-athletes or women. A deeper understanding
319 of the relationships between PA, the use of alcohol and smoking is necessary to determine the true
320 consequences of alcohol and tobacco on health and well-being among current and former athletes.
321
322 **CONCLUSIONS**
323
324 Former athletes have distinct profiles from non-athletes with respect to PA, alcohol and smoking. However,
325 PA did not predict later alcohol consumption at any time point. Although alcohol consumption may increase
326 in individuals, whose athletic career has ended suddenly by sports injury, overall PA and alcohol affected
327 each other's development only modestly over a 23 year follow-up.
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Contributors

SS, JK and UMK collected the data. TKK and AT analyzed the data. TKK drafted the manuscript. All authors contributed to study design, and the revision of the manuscript, and accepted the final version. The authors apologize for not being able to cite all the noteworthy work in this area because of constraints on space.

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Competing interest

None.

Ethical approval

This study was conducted according to good clinical and scientific practice and the Declaration of Helsinki. The authors declare that the results of this study are presented clearly, honestly, and without fabrication, falsification or inappropriate data manipulation. Approval for questionnaire data collection was given by The ethics committee of the Hospital District of Helsinki and Uusimaa, and the Ministry of Social Affairs and Health in Finland. All the participants gave informed consent by returning the questionnaires, which were accompanied by a cover letter explaining the purpose of the study.

Data sharing

The former athletes are well known persons in Finnish society; hence the data cannot be openly shared. Researchers are encouraged to contact the authors and we will make every effort to accommodate additional analyses.

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List of Tables

List of Figures

Table 1. Number of participants alive in 1985, 1995, 2001 and 2008.

Sports groups	Participants alive in 1985		Questionnaire responders in 1985		Participants alive in 1995		Questionnaire responders in 1995		Participants alive in 2001		Questionnaire responders In 2001		Participants alive in 2008		Questionnaire responders in 2008	
	(N)	Mean age (SD)	(N)	Mean age (SD)	(N)	Mean age (SD)	(N)	Mean age (SD)	(N)	Mean age (SD)	(N)	Mean age (SD)	(N)	Mean age (SD)	(N)	Mean age (SD)
1. Endurance	287*	61.0 (11.4)	226	60.6 (11.1)	219	68.0 (9.4)	170	67.9 (9.1)	177	72.0 (8.2)	132	71.6 (8.4)	134	76.9 (7.0)	121	77.1 (6.7)
2. Mixed sports	769†	55.2 (11.1)	607	54.9 (10.8)	633	62.4 (9.1)	467	62.6 (9.0)	561	67.3 (8.3)	415	67.1 (8.0)	449	72.5 (7.1)	396	72.6 (6.9)
3. Power sports	577‡	58.3 (11.4)	443	57.5 (10.7)	413	64.1 (8.8)	300	64.2 (8.3)	341	68.6 (7.8)	234	68.5 (7.7)	257	73.8 (6.8)	230	74.1 (6.8)
All athletes	1633	57.3 (11.5)	1276	56.8 (11.0)	1265	63.9 (9.3)	937	64.1 (9.0)	1079	68.5 (8.3)	785	68.3 (8.1)	840	73.6 (7.2)	747	73.8 (7.0)
Controls	1099	55.6 (10.6)	777	55.0 (10.3)	832	62.5 (8.7)	576	62.2 (8.1)	683	67.1 (7.6)	416	66.9 (7.0)	529	72.6 (6.5)	436	72.8 (6.3)
Total	**2732**	**56.4 (11.1)**	**2053**	**56.1 (10.8)**	**2097**	**63.4 (9.1)**	**1513**	**63.3 (8.7)**	**1762**	**67.9 (8.0)**	**1201**	**67.9 (7.8)**	**1369**	**73.2 (6.9)**	**1183**	**73.4 (6.8)**

Data are numbers in 1985.
*Long distance running 128, middle distance running 66, cross-country skiing 93.
†Soccer 199, ice hockey 144, basketball 80, high jump 39, pole vault 43, long jump 26, triple jump 30, hurdling 74, short distance running 99, decathlon 35.
‡Weightlifting 91, boxing 177, wrestling 182, shotput 29, discus 29, javelin 41, hammer 28.

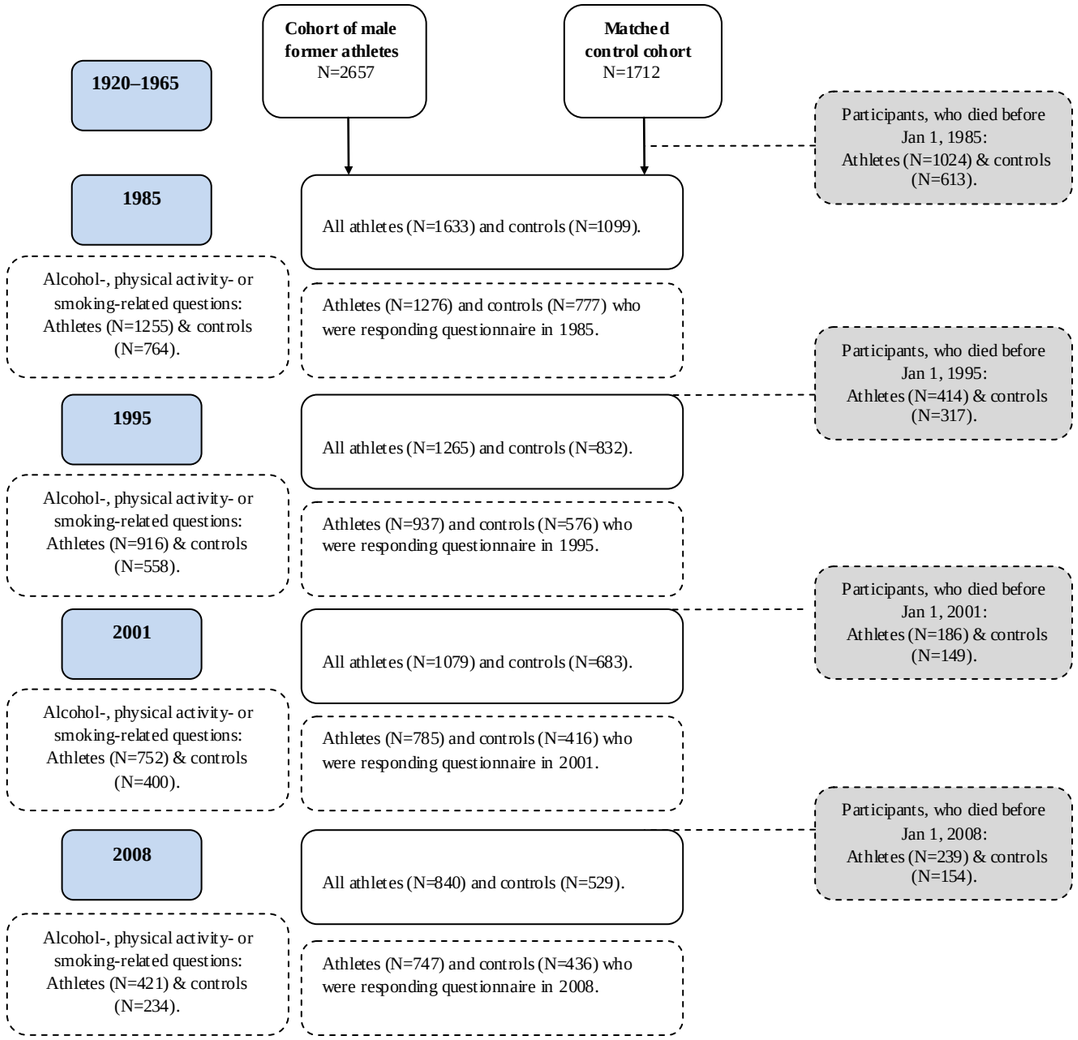


Figure 1. Study profile.

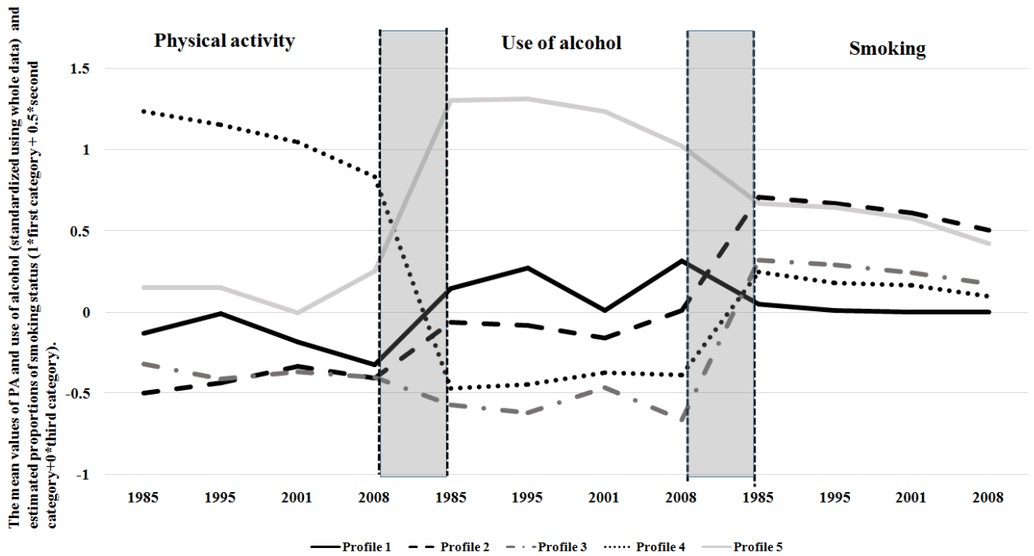
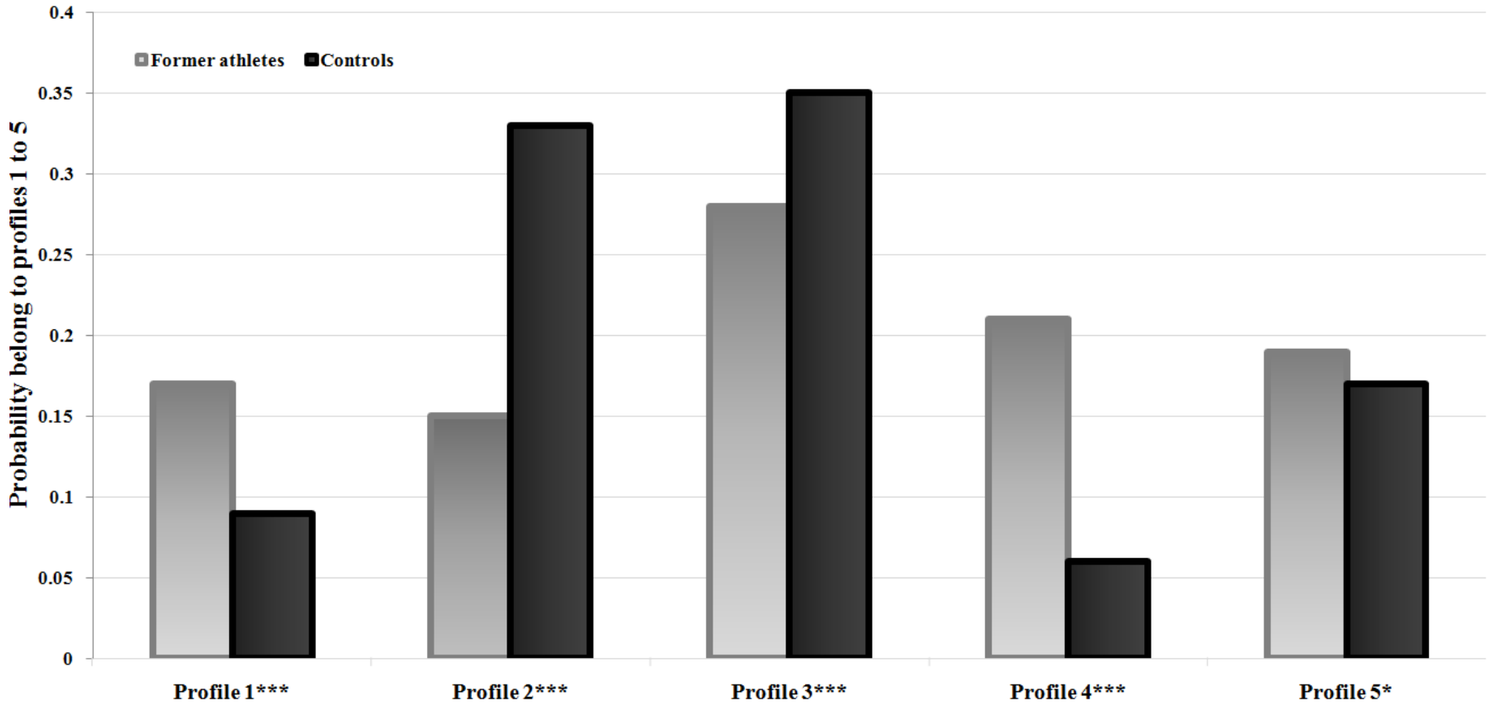


Figure 2.Characteristics of the 5 profiles from LPA among all participants.



*****p<0.005, *p=0.309 (P-values for the probability distributions across 5 latent classes separately for athletes and controls)**

Figure 3. Probability of former athletes and controls to belong to profiles 1 to 5.

II

USE OF ALCOHOL AND ALCOHOL-RELATED MORBIDITY IN FINNISH FORMER ELITE ATHLETES

by

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Use of Alcohol and Alcohol-related Morbidity in Finnish Former Elite Athletes

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ABSTRACT

Introduction: The impact of a history of competitive sports on later use of alcohol and occurrence of alcohol-related diseases is poorly known. We investigated how a history of elite level sports was associated with alcohol consumption in middle-age and with alcohol-related morbidity and mortality. **Methods:** Occurrence of alcohol-related diseases and deaths were followed using national registers from 1970 to 2008 among Finnish male former elite athletes (n=2202) and matched controls (n=1403) alive in 1970 (mean age 45.1 years). Hazard ratios (HRs) were calculated by Cox proportional hazards model. In 1985 surviving participants questionnaire-reported their alcohol consumption and engagement in physical activity/sports. **Results:** The risk of any alcohol-related diseases or deaths did not differ between former athletes and controls (HR 0.93; 95% CI 0.73-1.20, p=0.59), although, the risk was higher among both combat sports athletes and weightlifters compared to endurance sports athletes, shooters or jumpers & hurdlers ($p<0.05$). In 1985 athletes (417 grams/month; 95% CI 386-447) consumed more alcohol than controls (397grams/month; 95% CI 355-441) ($p<0.05$). Consumption was lower among endurance sports athletes than among controls ($p<0.05$). Team sports athletes consumed more alcohol ($p<0.05$), especially beer ($p<0.01$), compared to other athletes and controls. Athletes no longer engaged in leisure-time sports consumed more alcohol than those who continued to be physically active ($p<0.05$). **Conclusions:** Overall former athletes reported higher alcohol consumption than controls. There was no difference in alcohol-related morbidity, but the risk varied between different sports groups. Alcohol consumption after top-sports career was greater if participation in leisure-time sports was discontinued. Key words: ALCOHOL, CHRONIC DISEASE, COHORT STUDY, FORMER ATHLETE(S), PHYSICAL ACTIVITY.

INTRODUCTION

The excessive use of alcohol is harmful globally and alcohol consumption is the world`s third largest risk factor for diseases (20,39). The increased mortality related to heavy alcohol consumption is associated with cirrhosis, pancreatitis, certain cancers, stroke, accidents and external causes of deaths (8,20). Use of alcohol is associated with an increasingly higher risk of sport injuries compared to other injuries (28), especially among women (11). Furthermore, studies have shown addictions, anxiety, depression, amnesia and insomnia to be associated with heavy alcohol consumption (5-6,33).

Some studies have emphasized sports participation as a possible protective factor against excessive alcohol and other substance use in young individuals (14,22), while many studies indicate that athletic participation is associated with more excessive alcohol consumption (9,23,25,27-28) and alcohol dependence (29). Athletes have reported more binge drinking episodes than non-athletes (27) and alcohol-related problems are more common among college athletes than non-athletes (7,27). Unfortunately, many studies are only focused on drinking habits among college or university athletes (4,7,21,23,27,29). An essential limitation of these cross-sectional studies is that they cannot differentiate the longitudinal course of risky drinking among individuals with variable degrees of athletic participation (23). Some studies have associated team sports athletes with higher levels of alcohol consumption compared to individual sports athletes (3,29), but there is no consistent evidence across different sports. Furthermore, team sports athletes seemed to have a higher mean AUDIT (the Alcohol Use Disorders Identification Test) score than those who took part in individual sports (29).

Study on retirement from elite sports has recognized that sports career termination causes significant changes in athlete`s personal and social life, particularly high athletic identity has contributed to more problems in the adaptation process of retirement (1). Senior year students, who ended their participation in intercollegiate athletics, consumed less alcohol than consistent athletes (4). We know very little about how a history of competitive sports is related to later use of alcohol and occurrence of alcohol-related diseases.

Our study had three aims, the first of which was to examine how former competitive sports career was associated with the risk of alcohol-related diseases or deaths and with alcohol consumption among former athletes followed for several decades. Secondly, we compared the risk of alcohol-related diseases or deaths and alcohol consumption between different sports groups. Thirdly, we studied how different factors, such as physical activity, engagement to competitive or leisure-time sports and smoking status, were associated with alcohol consumption among former athletes after their active athletic career.

METHODS

Participants

An original cohort of former elite athletes (n=2657) was formed by identifying men who had represented Finland between 1920 and 1965 at least once at the Olympic Games, European or World championships, or international contests between two or three countries (32). A control cohort (n=1712) was selected from Finnish men who at the age of 20 years had been identified

healthy in the medical inspection for enlisting in ordinary military service. The control cohort was formed by matching the same age groups and area of residence with the former elite athletes (32).

Participants who had died before the register-based follow-up started in January 1, 1970 were excluded from this study. Thus the final study population (n=3605) consisted of 2202 former male athletes and 1403 matched control participants (Table 1 and Figure 1). For the purpose of this study the former male athletes were classified according to sports and their specific physical loading types into the following groups: short & middle distance running (n=202), endurance sports (n=280, including long distance running and cross country skiing), jumping & hurdling (n=251, including high jump, pole vault, long jump and triple jump), throwing & decathlon (n=211, including javelin, discus, shotput and hammer), weightlifting (n=111), combat sports (n=487, including wrestling and boxing), team sports (n=488, including soccer, ice hockey and basketball) and shooting (n=171).

To adjust for occupational loading, the participants were classified into five occupational groups: executives, white collars, blue collars, unskilled workers and farmers (10) according to the occupation in which they had practiced the longest [for classification see Table, Supplemental Digital Content 1, Socioeconomic status % (n) among former athletes and controls, http://links.lww.com/MSS/A793]. This classification also reflects the socioeconomic status of the participants. The occupational group distribution of athletes differed from that of controls ($p<0.001$, χ^2 -test). Occupational data were collected partly from the Central Population Registry of Finland and partly from questionnaires.

The register-based follow-up of hospitalizations started in January 1, 1970 and ended in December 31, 2008. Participants who had hospital admissions for alcohol-related diseases were identified from the National Hospital Discharge Register according to ICD-codes (ICD-8, ICD-9 or ICD-10). Alcohol-related diseases, which were strongly related to alcohol use, were categorized into five main groups: mental and behavioral disorders due to use of alcohol, alcohol dependence syndrome, alcoholic liver disease, alcoholic pancreatic diseases and other alcohol-related diseases [for all details see Table, Supplemental Digital Content 2, International Classification of Diseases (ICD) codes and main diagnostic categories, http://links.lww.com/MSS/A794]. The primary diagnosis was used to determine the reason for hospitalization, and the secondary diagnoses were ignored in the analysis. The overall correspondence in Finland between individual hospital discharge records and their written patient histories for different diagnoses has been reported to be 94.4% (16). Participants who had an alcohol-related death were identified from the National Death Register of Statistics Finland (cause of death alcohol-related diseases or accidental poisoning by alcohol, code 41; http://www.stat.fi/til/ksyyt/2005/ksyyt_2005_2006-10-31_luo_002.html).

Questionnaire study 1985

In 1985 a postal questionnaire eliciting information on discontinuation of sports career, socio-demographic factors (including occupational loading), physical activity, and health-related lifestyle habits, such as alcohol consumption and smoking, was sent to the surviving cohort members (total n=2528, athletes n=1518 and controls n=1010) (18). The response rate was 90% (n=1364) for athletes and 77% (n=777) for controls. Former elite athletes (n=1326) and controls

(n=755), who answered the alcohol-related questions, were included in the statistical analysis. In epidemiological research those who are the heaviest alcohol users tend to have lower response rates to questionnaire studies. As hospital admission data covered all participants in this study, we were able to investigate such selection effects. The response rate was 52.5% (n=137) among those participants alive in 1985 who had admissions to hospital at any time for any alcohol-related disease or death, and among this subgroup the response rate was higher among former athletes 58.7% (n=91) than among controls 43.4% (n=46).

The volume of physical activity (MET-hours/week) in 1985 was computed from the responses to three structured questions, using a previously validated method (37). MET-hours/week was categorized into tertiles of physical activity: MET-hours/week ≤6.0 (less active), 6.0< MET-hours/week ≤22.5 (moderate active) and MET-hours/week >22.5 (highly active). Engagement in competitive sports or leisure-time sports was dichotomized. The term "discontinued sports" means that the athlete had retired from competitive sports based on questions in the 1985 questionnaire (for more details see the items given in Supplemental Digital Content 3, Alcohol-related questions and sports career termination questions in the 1985 questionnaire, http://links.lww.com/MSS/A795).

Alcohol consumption was assessed by questions on beer, wine and spirits consumption, blackouts and heavy drinking occasions (HDO) (for the actual questions and response items, see Supplemental Digital Content 3, Alcohol-related questions and sports career termination questions in the 1985 questionnaire, http://links.lww.com/MSS/A795). Alcohol consumption based on quantity-frequency measures of beverage use was converted into grams of pure alcohol

per month as previously reported (31). Responses formed three categories: abstainers and light (3 or fewer drinks per week), moderate (more than 3 but no more than 14 drinks per week) and heavy users (on average more than 2 drinks a day) (15). The Substance Abuse and Mental Health Services Administration (SAMHSA) defines heavy drinking occasions (HDO), as drinking 5 or more alcoholic drinks (>60 grams of pure alcohol) on a single occasion on at least 1 day in the past 30 days (35). Responses formed two categories: no HDO and at least one HDO. Blackouts were defined by the frequency of alcohol-related loss of consciousness or temporary amnesia during the past 12 months (13). Three response categories were formed for analysis: no blackouts in the past year, one, or 2 or more.

The tobacco smoking status of the participants was classified into four categories from responses to a detailed smoking history: never, ex-, current or occasional smokers. Never smokers were men who had smoked no more than 5 to 10 packs of cigarettes (or equivalent of other tobacco product) throughout their lifetime. Ex-smokers were participants who have smoked greater than 100 cigarettes in their lifetime but have not smoked during the last month. Participants were classified as current smokers according to whether they were smoking daily or almost daily at the time. Occasional smokers were men who had smoked no more than 2 cigarettes in a week or last smoked 2-30 days ago [for more details see Table, Supplemental Digital Content 4, Smoking status % (n) among former athletes and controls, http://links.lww.com/MSS/A796].

Ethical Approval

This study was conducted according to good clinical and scientific practice and the Declaration of Helsinki. All the participants gave informed consent by returning the questionnaires, which were accompanied by a cover letter explaining the purpose of the study.

Statistical analysis

The descriptive data are presented as the mean and standard deviation (SD) or 95% confidence intervals (CI) if distributed normally; otherwise the results are shown as the median and range. The differences in the distributions of the categorical variables were examined using cross-tabulations with the Chi-square (χ^2) -test.

The follow-up of alcohol-related hospitalization started on January 1, 1970 and continued until the end of 2008, or until the date of hospitalization due to first alcohol-related disease, emigration or date of death, whichever date came first. The event was based on the first recorded hospital episode of any alcohol-related disease or alcohol-related death [for more details see Table, Supplemental Digital Content 5, Age- and SES-adjusted hazard ratios (HRs) for admissions to hospital for any alcohol-related diseases or deaths during January 1, 1970 to December 31, 2008 among former athletes compared to controls and mean (SD) age at first admission, http://links.lww.com/MSS/A797].

A Cox proportional hazards model was used to calculate age-adjusted hazard ratios (HRs) with their 95% CIs for alcohol-related disease or death between former athletes and controls. Post hoc analysis taking into account the number of comparisons was used to compare statistical differences between specific sports groups. Participants still alive at the end of follow up, and those who died from any other cause, were censored. The Cox regression assumptions were tested by using Schoenfeld residuals (ph-test in Stata) and also by plotting.

The 1985 questionnaire data was analyzed using non-parametric Kruskal-Wallis-test and using the Dunn-Bonferroni approach for post hoc testing in pairwise comparisons for more than two groups ($p<0.05$), because some of the variables were not normally distributed and variances were not equal. The Mann-Whitney-U-test was used to compare differences between sports groups and controls ($p<0.05$). Homogeneity of variances were assessed using Levene`s test and normality using Kolmogorov-Smirnov`s test ($p<0.05$).

P-values <0.05 were considered statistically significant. Statistical analyses were performed using SPSS statistical software (version 22.0 for Windows; SPSS Inc., Chicago, IL) and Stata 14.0 (Stata Corp, College Station, Texas, USA).

RESULTS

Altogether, 6.2% (n=136) of former athletes and 7.1% (n=99) of controls were admitted to hospital for any alcohol-related disease during the 39-year follow-up period. The most common reasons for admissions were Mental and behavioral disorders due to use of alcohol (athletes 2.4% (n=53), controls 2.1% (n=34)), alcohol dependence syndrome (athletes 3.9% (n=85),

controls 4.5% (n=72)) and alcoholic liver disease (athletes 0.8% (n=18), controls 0.7% (n=10)) (Table 2). The median total number of days in hospital was 12.0 (range 1-1442) in all former athletes and 17.0 (1-1365) in controls. The mean age at the first admission of any alcohol-related disease was 57.2 years (SD 14.7) for the former athletes and 51.5 years (SD 13.0) for the controls.

The age-adjusted hazard ratios (HRs) for admissions to hospital for any alcohol-related disease or death in former athletes was 0.93 (95% CI 0.73-1.20, p=0.59) compared to controls (Figure 2) [for more details see Table, Supplemental Digital Content 4, Smoking status % (n) among former athletes and controls, http://links.lww.com/MSS/A796]. The result persisted after adjustment for SES. No statistically significant SES differences were observed between former athletes and controls, and SES was not a significant covariate in this Cox regression model (p=0.26). Respectively, no statistically significant differences were observed between former athletes and controls in the analysis of different main diagnose groups separately.

In a secondary analysis, compared to weightlifters (the group with the highest risk) statistically significant HR for admissions to hospital for any alcohol-related disease or death was 0.42 (95% CI 0.19 - 0.93) for endurance sports athletes, 0.39 (95% CI 0.17 - 0.86) for jumpers & hurdles and 0.32 (95% CI 0.11 - 0.91) for shooters. Accordingly, compared to combat sports athletes the HR was 0.46 (95% CI 0.25 - 0.85) for endurance sports athletes, 0.42 (95% CI 0.22 - 0.79) for jumpers & hurdles and 0.35 (95% CI 0.14 - 0.87) for shooters.

Furthermore, the risk of any alcohol-related disease or death was higher among those who did not respond to 1985 questionnaire study compared to respondents, both among all participants (HR=2.34, 95% CI 1.72-3.18, p<0.001), among former athletes (HR=1.74, 95% CI 1.11-2.73, p=0.016) and among controls (HR=3.14, 95% CI 2.02-4.87, p<0.001).

Compared to controls, former athletes consumed significantly more beer (p<0.01), and wine (p<0.001), and their total alcohol consumption (g/month) was significantly higher (p<0.05). Furthermore, team sports athletes consumed significantly more alcohol (p<0.05), especially beer (p<0.01), and had significantly more heavy drinking occasions (HDO) than controls (p<0.001) (Table 3).

Total alcohol and beer consumption (g/month) was higher in team sports athletes compared to all other sports groups (p<0.05) (Figure 3). Accordingly, spirits consumption was significantly higher in team sports athletes compared to shooters (p<0.01), endurance sports athletes (p<0.01), jumpers & hurdles (p<0.01) and throwers & decathlonists (p<0.01). Wine consumption was significantly lower in both endurance sports athletes and combat sports athletes compared to jumpers & hurdles (p<0.01), short & middle distance runners (p<0.001), shooters (p<0.001) and team sports athletes (p<0.01).

Mean MET-hours/week in 1985 were significantly higher among former athletes than controls (p<0.001). One sixth (16.0%, n=200) of former athletes and 12.1% (n=33) of controls participated in competitive sports in 1985 (p<0.001) [for more details see Table, Supplemental Digital Content 6, Characteristic of participants who were responding alcohol-related questions

in 1985 questionnaire: overall physical activity (MET-hours/week), current engagement in competitive sports and smoking status, http://links.lww.com/MSS/A798].There was no significant difference in total alcohol consumption between competitive athletes and those athletes who had discontinued competitive sports, but latter consumed significantly more beer ($p<0.05$). Athletes, who had discontinued competitive sports and after that were not engaged in leisure-time sports, consumed more alcohol than those who were engaged in leisure-time sports or physical activities after active sports career ($p<0.05$).

Former athletes smoked less than controls ($p<0.001$). Among controls 27.7% and former athletes 48.5% were never smokers; 60.8% endurance sports athletes and 55.7% throwers and decathlonists were never smokers. On the other hand, current smoking was more common in team sports and weightlifting than other sports [For more details see Tables, Supplemental Digital Content 4, Smoking status % (n) among former athletes and controls, http://links.lww.com/MSS/A796; and Supplemental Digital Content 6, Characteristic of participants who were responding alcohol-related questions in 1985 questionnaire: overall physical activity (MET-hours/week), current engagement in competitive sports and smoking status, http://links.lww.com/MSS/A798]. Alcohol consumption (g/month) was the most common among current smokers, while never smokers used least alcohol. Current smokers consumed more alcohol than ex- and never smokers ($p<0.001$), and occasional smokers ($p=0.029$).

DISCUSSION

Principal findings

There was no significant difference in the risk of any alcohol-related disease or death between all former athletes and controls. However, the risk was higher among both combat sports athletes and weightlifters compared to endurance sports athletes, shooters or jumpers & hurdlers. Former athletes consumed more alcohol than controls, but consumption was lower among endurance sports athletes than controls. Team sports athletes consumed significantly more alcohol, especially beer, compared to other athletes and controls. Athletes who were not engaged in leisure-time sports after their active sports career consumed more alcohol than those who were engaged in leisure-time sports or physical activities.

Strengths and limitations of our study

This long-term follow-up study revealed new information on the risk of alcohol-related diseases and alcohol consumption among former athletes. The use of the valid register data covering all participants was the strength of this study. Though there may be some variation of assigning alcohol-related diagnoses by physicians, this is unlikely to be related to a history of elite athleticism, and hence not a source of bias. Self-reported data on health-related behaviors include known limitations. The self-reported smoking status has shown to be valid and reliable (36). Though heavy alcohol users may underestimate their use of alcohol (30), self-report alcohol-use data are generally valid in ranking persons as light, moderate or heavy users (26). The response

rate was lower among controls compared to former elite athletes. Although questionnaire response rates were lower in individuals with alcohol-related hospitalizations, the risk for bias in comparing the athlete group to non-athlete group was low. Also, the format of alcohol-related questions was optimal to avoid misclassification by study groups (for the actual questions and response items see Supplemental Digital Content 3, Alcohol-related questions and sports career termination questions in the 1985 questionnaire, http://links.lww.com/MSS/A795).

Furthermore, former athletes are a selected group and they competed at top-level before 1965 and we do not know exactly how well the results can be generalized to today's elite athletes or athletes who had competed in lower level or non-athletes or women.

Comparisons with other studies

There is lack of studies focused on the risk of alcohol-related diseases or alcohol consumption among former athletes several decades after their peak sporting performance. But our findings were consistent with other studies that have shown current athletes consumed more alcohol than non-athletes (9,23,25,27-28), and alcohol consumption was more common among team sports athletes than other sports (3,29). Our study showed that combat and team sports athletes consumed alcohol most, while jumpers & hurdlers, shooters and endurance sports athletes consumed less alcohol than other groups. Endurance athletes do not appear to engage in an excessive and risky alcohol use (11,25).

The same factors, especially personal and psychological factors, may be related to heavy drinking during and after an active athletic career. Furthermore, it has been observed that if an individual has a high athletic identity and their active sports career ends unexpectedly, adaptation to athletic retirement will be challenging (1) and it might predispose to alcohol-related problems. Presumably most alcohol-related risk factors, such as sociodemographic and familial factors, among the general population are also applicable to athletes (23). There is limited evidence on why athletes use more alcohol than non-athletes (38,40), but there are some motivational factors behind athletes and their reasons for engaging in risky alcohol consumption behaviors (24). Sports-induced anxiety or pressure, competitive nature of athletes, peer- or teammate-induced influence, and cultural relations between athletes and alcohol, might be related to risky alcohol consumption (7,23). Furthermore, it is generally known that alcohol, especially beer, distributors support sports and commercials for alcohol are commonly used (23). Some personality traits, such as histrionism, antisocial behavior may associate with higher alcohol consumption and alcohol dependence (12), but there is no consistent evidence. Studies have suggested that team sports athletes are more extraverted and neurotic than endurance sports athletes (2), whereas contact sports athletes have got high scores in the antisocial and histrionic scales (34).

Although, overall former athletes reported higher alcohol consumption than controls in our study, the risk of alcohol-related diseases was not increased in the former athletes group. The response rates of those participants, who had alcohol-related diseases, were lower among controls than athletes, and heavy alcohol users underestimate their use of alcohol (30). It may be that former athletes are more receptive to medical advice and reduce alcohol consumption at the

first signs of harmful consumption and therefore are hospitalized less often; studies on alcohol-related disease biomarkers would be needed. Furthermore, it could be speculated, whether physical activity protects against some of the harmful consequences of alcohol consumption, such as abdominal adiposity and liver fat accumulation (19).

Finally, it seems that engagement to sports does not become replaced with either binge drinking or alcohol dependence among former athletes after an active sports career. Former athletes' overall better health habits (such as less tobacco use), and the directly or indirectly subsequent biological factors are likely to explain our observation that there was no difference in the risk of alcohol-related diseases among former elite athletes compared to controls. This was found despite the greater use of alcohol among former elite athletes than among controls.

Future directions

There is lack of studies focused on assessing the effect of interventions implemented in sports settings on the use of alcohol and alcohol-related harms (17). A deeper understanding of the relationships between alcohol consumption and the risk of alcohol-related diseases and sports is necessary to determine the true consequences of alcohol on health and well-being in athletes. Models should be developed how to avoid an excessive alcohol consumption among risk sports during career termination.

CONCLUSIONS

Former athletes reported higher alcohol consumption than controls, but there was no difference in the risk of alcohol-related diseases or deaths between former athletes and controls. The risk varied between different sports groups being highest in combat sports athletes and weightlifters. Total alcohol and heavy drinking occasions seemed to be significantly more common in team sports than other sports. It is important to notice that the risk of excessive alcohol consumption may increase in individuals, who do not continue leisure-time sports after elite level sports career termination. Finally, current and ex-smokers consumed more alcohol than non-smokers.

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The authors declare that the results of this study are presented clearly, honestly, and without fabrication, falsification or inappropriate data manipulation. The results of the present study do not constitute endorsement by the American College of Sports Medicine.

SS, JK and UMK collected the data. TKK and UMK analyzed the data. TKK drafted the manuscript. All authors contributed to study design, and the revision of the manuscript, and accepted the final version. The authors apologize for not being able to site all the noteworthy work in this area because of constraints on space.

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List of Figures

Figure 1

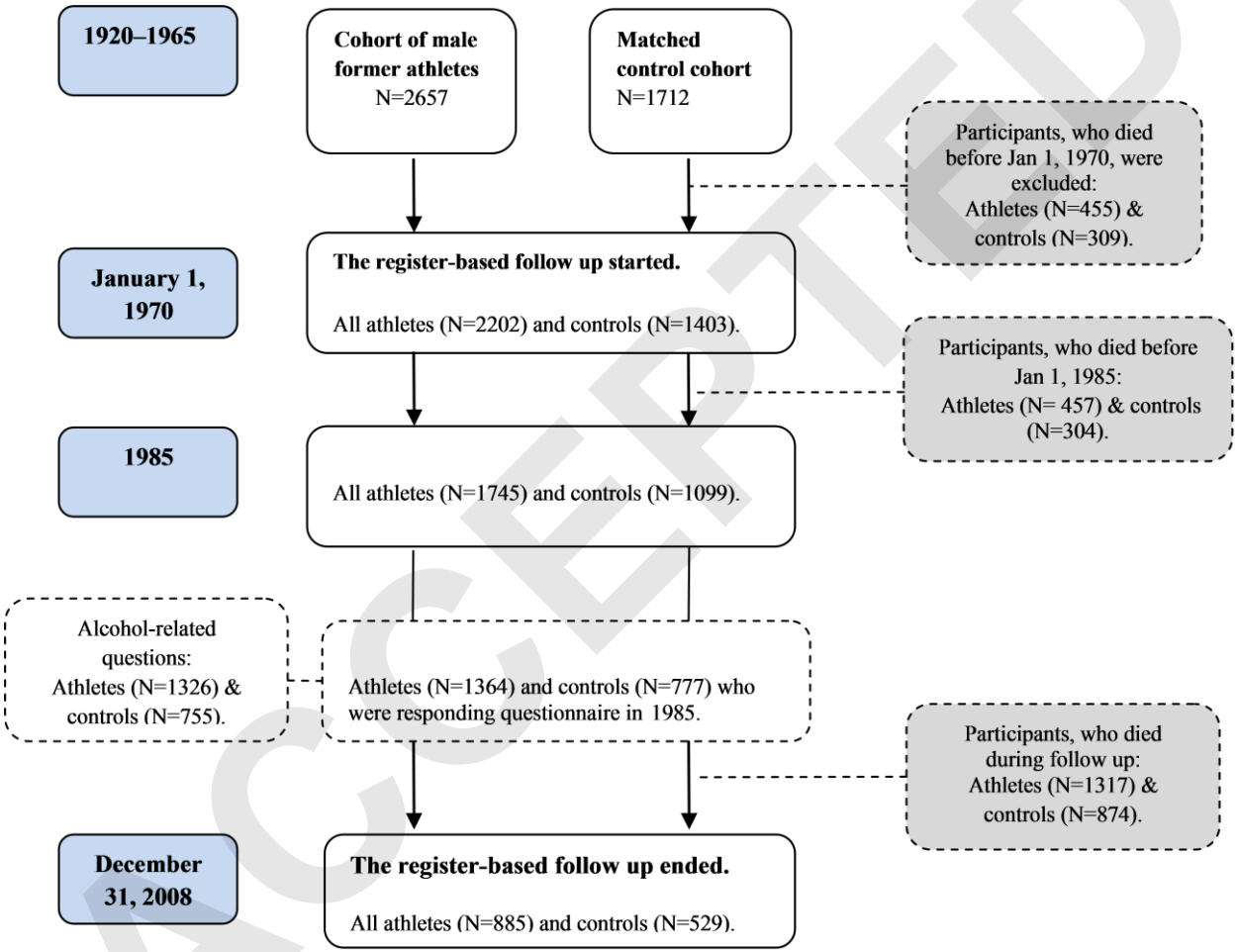

Figure 2

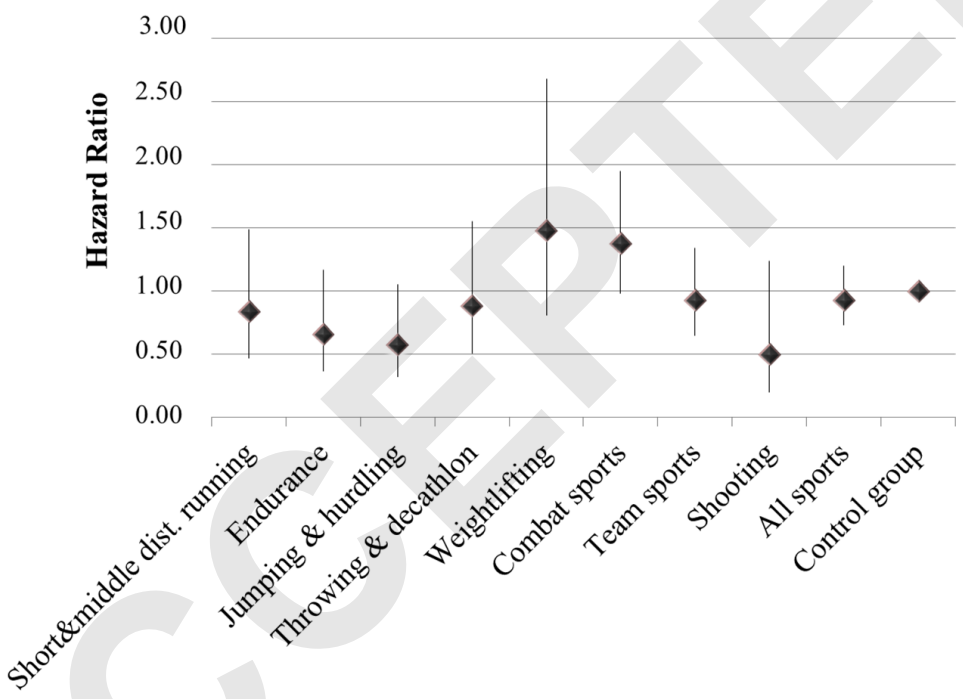

Figure 3

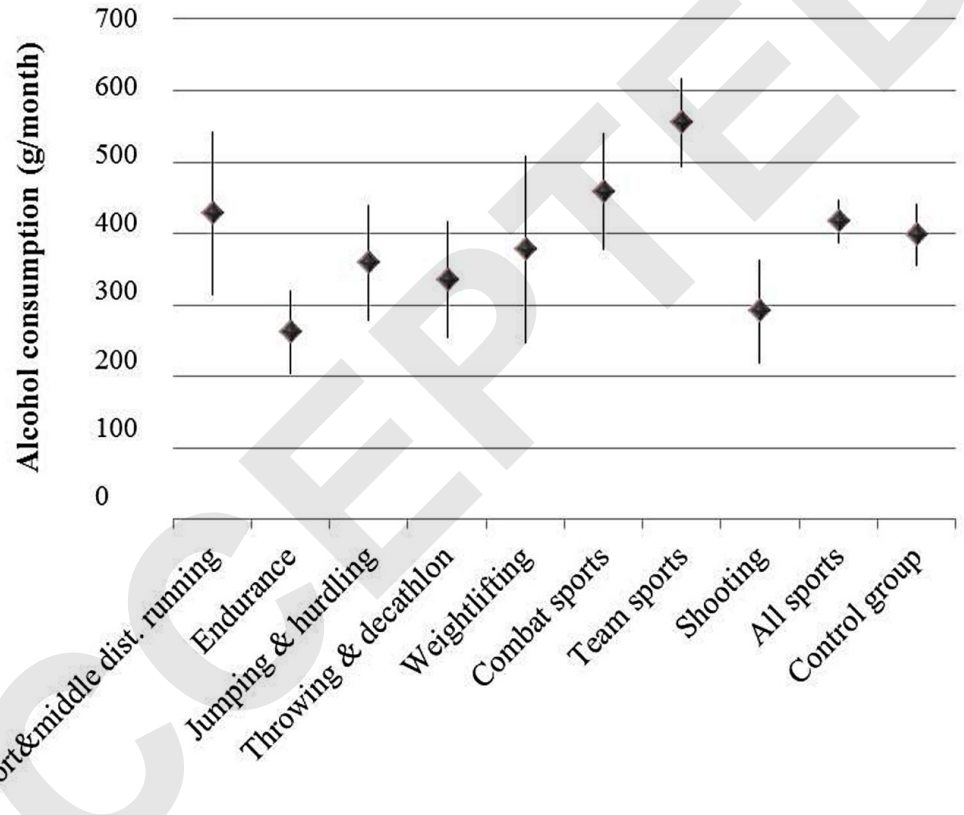

Table Socioeconomic status % (n) among former athletes and controls.

Socioeconomic status (SES)*	Short & middle distance running	Endurance	Jumping & hurdling	Throwing & decathlon	Weightlifting	Combat sports	Team sports	Shooting	All athletes	Controls
1. Executive	6.2 (17)	46.7 (91)	50.2 (124)	29.3 (61)	8.1 (9)	7.8 (37)	34.7 (166)	52.1 (88)	27.4 (593)	9.5 (123)
2. White collar	49.6 (137)	30.8 (60)	34.0 (84)	39.9 (83)	34.2 (38)	30.2 (144)	42.2 (202)	36.1 (61)	37.4 (809)	22.0 (286)
3. Blue collar	25.5 (69)	17.4 (34)	12.6 (31)	19.2 (40)	54.1 (60)	53.0 (253)	22.8 (109)	8.9 (15)	28.3 (611)	39.7 (516)
4. Unskilled worker	4.7 (13)	0.5 (1)	0.4 (1)	2.4 (5)	2.7 (3)	4.4 (21)	0.4 (2)	0	2.1 (46)	9.9 (129)
5. Farmer	14.5 (40)	4.0 (8)	2.4 (6)	8.2 (17)	0.9 (1)	4.6 (22)	0	2.4 (4)	4.5 (98)	18.5 (240)
6. Other (unknown)	0	0.5 (1)	0.4 (1)	1.0 (2)	0	0	0	0.6 (1)	0.2 (5)	0.4 (5)

*All participants, there are 146 missing values, $p<0.001$ for group differences in socioeconomic status by χ^2 -test.

Table International Classification of Diseases (ICD) codes and main diagnostic categories.

Diagnostic categories	ICD8 1969-1986	ICD9 1987-1995	ICD10 1996-	No of participants in diagnostic categories (N)*
Mental and behavioural disorders due to use of alcohol (except alcohol dependence F10.2)	291 Alcoholic psychosis 291.00-291.30, 291.98-291.99	291 Alcoholic psychoses 291A-2914A, 2918A	F10 Mental and behavioural disorders due to use of alcohol F10.0-F10.9 (except F10.2)	87
Alcohol dependence syndrome	303 Alcoholism 303.00-303.20, 303.98-303.99	303 Alcohol dependence syndrome 303, 3039X, 980.01, 980.98	F10.2 Alcohol dependence syndrome F10.20-10.29	157
Alcoholic liver disease	571.00 Alcoholic cirrhosis of liver 571.01 Alcoholic fatty liver	571 Chronic liver disease and cirrhosis 5710A,5711A,5712A 5713X	K70 Alcoholic liver disease K70.0-70.4, 70.9	28
Diseases of pancreas		577 Diseases of pancreas Acute Pancreatitis 5770D-F Chronic Pancreatitis 5771C-D	K 86 Other diseases of pancreas K86.00 Alcohol-induced acute pancreatitis K86.01 Recidivans K86.08 Alcohol-induced chronic pancreatitis	13
Other alcohol-related diseases		3573 Alcoholic polyneuropathy 4255 Alcoholic cardiomyopathy 5353 Alcoholic gastritis	G31.2 Degeneration of nervous system due to alcohol G40.51 Epileptic seizures related to alcohol G62.1 Alcoholic polyneuropathy G72.1 Alcoholic myopathy I42.6 Alcoholic cardiomyopathy K29.2 Alcoholic gastritis	7

*One person could have more than one diagnosis if there are several admissions for alcohol-related diseases.

Table Smoking status % (n) among former athletes and controls.

Smoking status*	Short & middle distance running	Endurance	Jumping & hurdling	Throwing & decathlon	Weightlifting	Combat sports	Team sports	Shooting	All athletes	Controls
1. Never smoker	53.6 (67)	60.8 (96)	51.6 (83)	55.7 (68)	46.9 (30)	47.2 (126)	40.9 (132)	36.1 (30)	48.5 (632)	27.7 (205)
2. Ex-smoker	33.6 (42)	19.6 (31)	32.9 (53)	25.4 (31)	29.7 (19)	31.5 (84)	31.0 (100)	54.2 (45)	31.1 (405)	41.4 (307)
3. Occasional smoker	3.2 (4)	8.2 (13)	4.3 (7)	4.9 (6)	0	3.0 (8)	5.9 (19)	2.4 (2)	4.5 (59)	2.6 (19)
4. Current smoker	9.6 (12)	11.4 (18)	11.2 (18)	13.9 (17)	23.4 (15)	18.4 (49)	22.3 (72)	7.2 (6)	15.9 (207)	28.3 (210)

* $p<0.001$ for group differences in smoking status by χ^2 -test.

The tobacco smoking status of the subjects was classified into four categories from responses to a detailed smoking history: never, ex-, current or occasional smokers. Never smokers were men who had smoked no more than 5 to 10 packs of cigarettes (or equivalent of other tobacco product) throughout their lifetime. Ex-smokers were participants who have smoked greater than 100 cigarettes in their lifetime but have not smoked during the last month. Participants were classified as current smokers according to whether they were smoking daily or almost daily at the time (1). Occasional smokers were men who had smoked no more than 2 cigarettes in a week (3) or last smoked 2-30 days ago (2).

References:

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2 Luoto R, Uutela A , Puska P. Occasional smoking increases total and cardiovascular mortality among men. *Nicotine Tob Res* 2000;2:133-9.
3. Paavola M, Vartiainen E, Puska P. Smoking cessation between teenage years and adulthood. *Health Educ Res* 2001;16:49-57.

Table Age- and SES-adjusted hazard ratios (HRs) for admissions to hospital for any alcohol-related diseases or deaths during January 1, 1970 to December 31, 2008 among former athletes compared to controls and mean (SD) age at first admission.

Sports groups	N of participants with alcohol-related disease or death	Mean age (SD) at first admission	Age-adjusted HR	95.0% Confidence Interval		P-value*	SES-adjusted HR	95.0% Confidence Interval		P-value*
				Lower	Upper			Lower	Upper	
Short & middle distance running	13	51.3 (11.7)	0.839	0.471	1.492	0.549	0.919	0.508	1.660	0.779
Endurance	13	62.4 (15.4)	0.655	0.366	1.170	0.152	0.673	0.375	1.206	0.183
Jumping & hurdling	12	58.7 (18.8)	0.579	0.319	1.052	0.073	0.631	0.340	1.170	0.144
Throwing & decathlon	14	60.8 (20.1)	0.888	0.509	1.551	0.677	0.933	0.529	1.644	0.810
Weightlifting	12	56.9 (14.3)	1.475	0.812	2.682	0.202	1.504	0.823	2.746	0.184
Combat sports	46	54.6 (13.1)	1.380	0.975	1.953	0.069	1.369	0.956	1.960	0.086
Team sports	40	56.6 (12.5)	0.931	0.647	1.341	0.702	1.006	0.681	1.486	0.977
Shooting	5	73.7 (15.7)	0.501	0.204	1.235	0.133	0.544	0.218	1.357	0.192
All athletes	155	57.2 (14.7)	0.934	0.729	1.197	0.591	1.001	0.765	1.310	0.995
Controls	106	51.5 (13.0)	1.000	-	-	-	1.000	-	-	-
Total	261	54.8 (14.3)	-	-	-	-	-	-	-	-

*p-values for statistical differences between sports groups and controls by Cox regression analysis.

Table Characteristic of participants who were responding alcohol-related questions in 1985 questionnaire: overall physical activity (MET-hours/week), current engagement in competitive sports and smoking status.

Sports groups	Mean MET-hours/ week (95% CI)*	Physical Activity tertiles† % (n)***			Currently engaged in competitive sports % (n)***	Smoking status‡ % (n)***	
		1	2	3		current smoker	never smoker
Short & middle distance running	28.5 (22.2-34.7)	25.4 (32)	34.9 (44)	39.7 (50)	12.9 (16)	9.6 (12)	53.6 (67)
Endurance	36.1 (29.6-42.6)	23.2 (38)	32.3 (53)	44.5 (73)	18.8 (29)	11.4 (18)	60.8 (96)
Jumping & hurdling	27.9 (23.1-32.6)	28.7 (47)	33.5 (55)	37.8 (62)	12.1 (19)	11.2 (18)	51.6 (83)
Throwing & decathlon	37.3 (29.0-45.7)	26.0 (32)	31.7 (39)	42.3 (52)	23.3 (27)	13.9 (17)	55.7 (68)
Weightlifting	26.9 (18.6-35.2)	34.8 (23)	28.8 (19)	36.4 (24)	6.5 (4)	23.4 (15)	46.9 (30)
Combat sports	28.2 (24.0-32.5)	31.4 (85)	30.3 (82)	38.4 (104)	7.4 (19)	18.4 (49)	47.2 (126)
Team sports	28.9 (25.7-32.1)	22.3 (73)	33.3 (109)	44.3 (145)	22.0 (69)	22.3 (72)	40.9 (132)
Shooting	19.3 (14.9-23.8)	37.2 (32)	37.2 (32)	25.6 (22)	25.0 (17)	7.2 (6)	36.1 (30)
All athletes	29.5 (27.7-31.4)	27.3 (362)	32.6 (433)	40.1 (532)	16.0 (200)	15.9 (207)	48.5 (632)
Controls	14.5 (12.8-16.1)	53.1 (401)	29.0 (219)	17.9 (135)	12.1 (33)	28.3 (210)	27.7 (205)

* $p<0.001$for statistical differences between sports groups and controls by Mann-Whitney-U-test.
† Overall physical activity (MET-hours/week): 1=MET-hours/week ≤6.0 (less active), 2= 6.0< MET-hours/week ≤22.5 (moderate active), 3= MET-hours/week >22.5 (highly active).
‡ Smoking status, see more details in Supplementary table 3.
*** $p<0.001$ for sports group differences in physical activity tertiles, currently engaged in competitive sports or smoking status by χ^2 -test.

Table 1. Number of participants at entry to study and still alive in January 1, 1970, 1985 and December 31, 2008.

Sports groups	Participants at entry	Participants in 1970		Participants in 1985		Questionnaire responders in 1985		Participants in 2008	
	(N)	(N)	Mean age (SD)	(N)	Mean age (SD)	(N)	Mean age (SD)	(N)	Mean age (SD)
1. Short & middle distance running	228	202	45.2 (13.1)	165	57.1 (11.5)	128	57.4(11.3)	84	73.8 (6.9)
2. Endurance	341	280*	50.0 (12.9)	221	61.9 (11.2)	175	61.6 (11.0)	98	79.0 (7.0)
3. Jumping & hurdling	291	251†	44.8 (13.1)	212	57.3 (11.9)	169	57.1 (11.6)	118	74.7 (7.9)
4. Throwing & decathlon	247	211‡	46.4 (14.3)	162	57.0 (11.8)	125	56.4 (11.3)	85	74.4 (7.0)
5.Weightlifting	122	111	43,9 (10.1)	91	57.2 (9.2)	67	57.0 (8.7)	35	76.4 (6.9)
6.Combat sports	626	487§	47.0 (13.4)	359	58.5 (11.7)	277	57.9 (10.8)	161	74.8 (6.8)
7. Team sports	569	488¶	41.0 (12.6)	423	53.4 (10.4)	334	53.0 (9.8)	262	73.0 (6.6)
8. Shooting	233	171	54.6 (14.2)	112	63.9 (13.1)	89	64.2 (12.5)	42	75.8 (8.4)
All athletes	2657	2202	46.0 (13.6)	1745	57.6 (11.7)	1364	55.0 (10.3)	885	74.7 (7.2)
Controls	1712	1403	43.7 (12.6)	1099	55.3 (10.6)	777	57.3 (11.3)	529	73.6 (6.5)
Total	**4369**	**3605**	**45.1 (13.2)**	**2844**	**56.7 (11.3)**	**2141**	**56.5(11.0)**	**1414**	**74.3 (7.0)**

Data are numbers in 1970.
* Long distance running 162, cross country skiing 118.
† High jump 45, pole vault 53, long jump 34, triple jump 34, hurdling 85.
‡ Shot put 38, discus 35, javelin 56, hammer 37, decathlon 45.
§ Boxing 234, wrestling 254.
¶ Soccer 250, ice hockey 154, basketball 84.

Table 2. Number of participants in alcohol-related main diagnosis groups, total days in hospital, median of days in hospital, mean of exposure time and total days in hospital/total exposure time among former athletes and controls.

Sports groups	Mental & behavioural disorders due to use of alcohol*	Alcohol dependence syndrome*	Alcoholic liver disease*	Alcoholic pancreatitis*	Other alcohol-related diseases*	Any alcohol-related disease*	Alcohol-related death (no alcohol-related hospitali-zation before)	Any alcohol-related disease or death	Total days in hospital for any alcohol-related disease	Median of days in hospital for any alcohol-related disease (range)	Mean of exposure time, years (95% CI)	Total days in hospital / Total exposure years (days / 100 exposure years)†
Short & middle distance running	7	7	1	0	0	12	1	13	701	38.5 (1-227)	27.3 (25.5-29.1)	13
Endurance	4	8	1	0	2	11	2	13	1136	11.0 (1-1001)	26.9 (25.5-28.3)	15
Jumping & hurdling	4	7	0	0	0	9	3	12	224	8.0 (1-84)	29.4 (27.9-30.8)	3
Throwing & decathlon	7	6	4	0	1	13	1	14	1783	17.0 (2-1276)	26.4 (24.6-28.2)	32
Weightlifting	3	9	3	0	0	12	0	12	276	8.0 (1-89)	26.7 (24.4-29.0)	9
Combat sports	15	26	3	3	0	40	6	46	805	11.5 (1-98)	24.5 (23.3-25.7)	7
Team sports	12	21	4	5	2	34	6	40	2410	13.5 (1-1442)	30.1 (29.0-31.2)	16
Shooting	1	1	2	0	1	5	0	5	393	7.0 (3-336)	22.5 (20.5-24.5)	10
All athletes	53	85	18	8	6	136	19	155	7782	12.0 (1-1442)	27.0 (26.5-27.5)	13
Controls	34	72	10	5	1	99	7	106	4914	17.0 (1-1365)	26.4 (25.7-27.0)	13
Total*	87	157	28	13	7	235	26	261	12642	14.0 (1-1442)	26.7 (26.3-27.2)	13

*Number of participants who have any alcohol-related disease. One participant may have more than one alcohol-related main diagnosis if there are more than 1 hospital admissions.

†$p<0.001$ for statistical differences between specific sports groups and controls by Mann-Whitney-U-test.

Table 3. Mean (95% CI) of alcohol consumption (beer, wine, spirits & total alcohol, g/month), alcohol-induced blackouts and heavy drinking occasions among former athletes and controls.

Sports groups	Beer g/month (95% CI)	Wine g/month (95% CI)	Spirits g/month (95% CI)	Total alcohol g/month (95% CI)	Type of alcohol drinker % (n)† 1	2	3	Alcohol-induced blackouts % (n)‡ 1	2	3	Heavy drinking occasions (HDO) % (n)§
Short & middle distance running	163.7 (97.5-229.9)	75.2 (53.3-97.0)***	189.6 (120.8-258.4)	428.5 (314.1-542.9)	42.9 (54)	43.7 (55)	13.5 (17)	89.3 (109)	8.2 (10)	2.5 (3)	22.6 (28)
Endurance	76.1 (54.3-97.9)	36.6 (19.6-53.5)	150.0 (107.4-192.5)	262.6 (205.0-320.3)*	56.1 (92)	36.6 (60)	7.3 (12)	87.8 (129)	8.8 (13)	3.4 (5)	21.9 (34)
Jumping & hurdling	97.8 (71.1-124.5)	110.9 (71.9-149.9)***	150.5 (105.9-195.1)	359.2 (278.8-439.5)	45.7 (75)	38.4 (63)	15.9 (26)	93.6 (146)	4.5 (7)	1.9 (3)	23.6 (38)
Throwing & decathlon	108.9 (68.8-148.9)	60.3 (38.1-82.5)	166.1 (116.9-215.4)	335.3 (254.2-416.4)	49.6 (61)	36.6 (45)	13.8 (17)	93.0 (107)	5.2 (6)	1.7 (4)	26.7 (32)
Weightlifting	118.2 (67.0-169.3)	54.3 (24.1-84.4)	204.9 (108.1-301.6)	377.3 (246.3-508.2)	48.5 (32)	31.8 (21)	19.7 (13)	90.0 (54)	3.3 (2)	6.7 (4)	30.6 (19)
Combat sports	119.2 (91.6-146.9)	83.6 (48.9-118.3)	255.9 (204.5-307.3)	458.7 (377.4-540.0)	43.2 (117)	38.0 (103)	18.8 (51)	88.5 (223)	6.3 (16)	5.2 (13)	32.8 (87)
Team sports	229.4 (193.0-265.9)***	114.9 (91.4-138.4)***	210.3 (179.0-241.7)***	554.7 (492.9-616.6)***	25.4 (83)	48.0 (157)	26.6 (87)	87.7 (265)	9.3 (28)	3.0 (9)	49.7 (161)
Shooting	79.8 (53.7-105.9)	87.3 (56.3-118.3)***	124.2 (81.1-167.2)*	291.3 (219.4-363.2)	51.2 (44)	33.7 (29)	15.1 (13)	97.6 (81)	2.4 (2)	0 (0)	14.0 (12)
All athletes	139.1 (124.9-153.2)**	84.7 (73.5-96.0)***	192.9 (174.9-210.8)	416.6 (386.4-446.8)*	42.0 (558)	40.2 (533)	17.8 (236)	90.1 (1114)	6.8 (84)	3.2 (39)	31.7 (411)
Controls	132.6 (111.5-153.7)	69.0 (54.1-83.8)	195.9 (173.9-218.0)	397.5 (354.5-440.5)	44.5 (336)	38.9 (294)	16.6 (125)	87.7 (615)	7.4 (52)	4.9 (34)	31.8 (234)

*p<0.05 **p<0.01 ***p<0.001 for statistical differences between sports groups and controls by Mann-Whitney-U-test.
†1=abstainers or light user, 2=moderate user, 3=heavy user, p<0.001, χ^2 -test.
‡1=no blackouts, 2=one blackout, 3=2 or more blackouts.
§p<0.001 for sports group differences in HDO by χ^2 -test.

List of Supplemental Digital Content

- **Supplemental Digital Content 1:** Table, Socioeconomic status % (n) among former athletes and controls.
- **Supplemental Digital Content 2:** Table, International Classification of Diseases (ICD) codes and main diagnostic categories.
- **Supplemental Digital Content 3**: Alcohol-related questions and sports career termination questions in the 1985 questionnaire.
- **Supplemental Digital Content 4:** Table, Smoking status % (n) among former athletes and controls.
- **Supplemental Digital Content 5:** Table, Age- and SES-adjusted hazard ratios (HRs) for admissions to hospital for any alcohol-related diseases or deaths during January 1, 1970 to December 31, 2008 among former athletes compared to controls and mean (SD) age at first admission.
- **Supplemental Digital Content 6:** Table, Characteristic of participants who were responding alcohol-related questions in 1985 questionnaire: overall physical activity (MET-hours/week), current engagement in competitive sports and smoking status.

Table Socioeconomic status % (n) among former athletes and controls.

Socioeconomic status (SES)*	Short & middle distance running	Endurance	Jumping & hurdling	Throwing & decathlon	Weightlifting	Combat sports	Team sports	Shooting	All athletes	Controls
1. Executive	6.2 (17)	46.7 (91)	50.2 (124)	29.3 (61)	8.1 (9)	7.8 (37)	34.7 (166)	52.1 (88)	27.4 (593)	9.5 (123)
2. White collar	49.6 (137)	30.8 (60)	34.0 (84)	39.9 (83)	34.2 (38)	30.2 (144)	42.2 (202)	36.1 (61)	37.4 (809)	22.0 (286)
3. Blue collar	25.5 (69)	17.4 (34)	12.6 (31)	19.2 (40)	54.1 (60)	53.0 (253)	22.8 (109)	8.9 (15)	28.3 (611)	39.7 (516)
4. Unskilled worker	4.7 (13)	0.5 (1)	0.4 (1)	2.4 (5)	2.7 (3)	4.4 (21)	0.4 (2)	0	2.1 (46)	9.9 (129)
5. Farmer	14.5 (40)	4.0 (8)	2.4 (6)	8.2 (17)	0.9 (1)	4.6 (22)	0	2.4 (4)	4.5 (98)	18.5 (240)
6. Other (unknown)	0	0.5 (1)	0.4 (1)	1.0 (2)	0	0	0	0.6 (1)	0.2 (5)	0.4 (5)

*All participants, there are 146 missing values, $p<0.001$ for group differences in socioeconomic status by χ^2 -test.

Table International Classification of Diseases (ICD) codes and main diagnostic categories.

Diagnostic categories	ICD8 1969-1986	ICD9 1987-1995	ICD10 1996-	No of participants in diagnostic categories (N)*
Mental and behavioural disorders due to use of alcohol (except alcohol dependence F10.2)	**291 Alcoholic psychosis 291.00-291.30, 291.98-291.99**	**291 Alcoholic psychoses 291A-2914A, 2918A**	**F10 Mental and behavioural disorders due to use of alcohol F10.0-F10.9 (except F10.2)**	**87**
Alcohol dependence syndrome	**303 Alcoholism 303.00-303.20, 303.98-303.99**	**303 Alcohol dependence syndrome 303, 3039X, 980.01, 980.98**	**F10.2 Alcohol dependence syndrome F10.20-10.29**	**157**
Alcoholic liver disease	**571.00 Alcoholic cirrhosis of liver 571.01 Alcoholic fatty liver**	**571 Chronic liver disease and cirrhosis 5710A,5711A,5712A 5713X**	**K70 Alcoholic liver disease K70.0-70.4, 70.9**	**28**
Diseases of pancreas		**577 Diseases of pancreas Acute Pancreatitis 5770D-F Chronic Pancreatitis 5771C-D**	**K 86 Other diseases of pancreas K86.00 Alcohol-induced acute pancreatitis K86.01 Recidivans K86.08 Alcohol-induced chronic pancreatitis**	**13**
Other alcohol-related diseases		**3573 Alcoholic polyneuropathy 4255 Alcoholic cardiomyopathy 5353 Alcoholic gastritis**	**G31.2 Degeneration of nervous system due to alcohol G40.51 Epileptic seizures related to alcohol G62.1 Alcoholic polyneuropathy G72.1 Alcoholic myopathy I42.6 Alcoholic cardiomyopathy K29.2 Alcoholic gastritis**	**7**

*One person could have more than one diagnosis if there are several admissions for alcohol-related diseases.

Supplemental Digital Content 3: Alcohol-related questions and sports career termination questions in questionnaire 1985.

Use of alcohol

1. **How much of the following alcoholic beverages do you drink on average?**

Beer

1) Never

2) Less than a bottle a week

3) 1-4 bottles on a week

4) 5-12 bottles on a week

5) 13-24 bottles a week

6) 25-47 bottles a week

7) More than 48 bottles a week

Wine or other mild alcoholic beverages

1) Never
2) Less than a glass a week
3) A glass to 4 glasses a week
4) 1-2.5 bottles a week
5) 3-4.5 bottles a week
6) 5-9 bottles a week
7) More than 10 bottles a week

Hard liquor

1) Never
2) Less than a half bottle per month
3) A half bottle to a bottle and a half per month
4) 2-3.5 bottles a month
5) 4-9 bottles a month
6) 10-19 bottles a month
7) More than 20 bottles a month

2. **How often do you use alcohol? Which of the following alternatives best describes your use of beer, wine and hard liquor?**

	Never	On less than two days a month	On 3-8 days a month	On 9-16 days a month	Over than 16 days a month
Beer	1	2	3	4	5
Wine	1	2	3	4	5
Liquor	1	2	3	4	5

3. **Does it happen that at least once a month and on the same occasion you drink more than five bottles of beer or more than bottle of wine or more than half a bottle of hard liquor?**

   1=No

   2=Yes

4. **How often have you passed out while using alcohol during the last year?**

   0=Not once

   1=Once

   2=Two - three times

   3=Four - six times

   4=Seven times or more

***Notice: Continuous variables (wine, beer, spirits and total alcohol consumption, g/month) based on quantity-frequency measures of beverage use was converted into grams of pure alcohol per month as previously reported (Romanov et al 1987). One drink includes 12 g of pure alcohol; it means 33 cl beer, 12 cl wine or 4 cl spirits.

Alcohol (g/month): 12 g/drink x Number of drinks /month

Reference: Romanov K, Rose RJ, Kaprio J, Koskenvuo M, Langinvainio H, Sarna S. Self-reported alcohol use: a longitudinal study of 12954 adults. Alcohol Alcohol Suppl. 1987;1:619-23.

Sports career termination

1. **Why did you retired from competitive sports?**
   1) Lack of enthusiasm
   2) Injury/injuries
   3) Work or studying
   4) Family reasons
   5) Other reasons, which_____

2. **Have you continued exercising after active sports career?**
   1) No

2) Yes
    a. Did you have a break of exercising after active sports career
        i. No
        ii. Yes → _____years

3. Are you still into competitive sports?

1) No

2) Yes → Write in the first line sport which you mostly do:

1._____

2._____

3._____

Table Smoking status % (n) among former athletes and controls.

Smoking status*	Short & middle distance running	Endurance	Jumping & hurdling	Throwing & decathlon	Weightlifting	Combat sports	Team sports	Shooting	All athletes	Controls
1. Never smoker	53.6 (67)	60.8 (96)	51.6 (83)	55.7 (68)	46.9 (30)	47.2 (126)	40.9 (132)	36.1 (30)	48.5 (632)	27.7 (205)
2. Ex-smoker	33.6 (42)	19.6 (31)	32.9 (53)	25.4 (31)	29.7 (19)	31.5 (84)	31.0 (100)	54.2 (45)	31.1 (405)	41.4 (307)
3. Occasional smoker	3.2 (4)	8.2 (13)	4.3 (7)	4.9 (6)	0	3.0 (8)	5.9 (19)	2.4 (2)	4.5 (59)	2.6 (19)
4. Current smoker	9.6 (12)	11.4 (18)	11.2 (18)	13.9 (17)	23.4 (15)	18.4 (49)	22.3 (72)	7.2 (6)	15.9 (207)	28.3 (210)

* $p<0.001$ for group differences in smoking status by χ^2 -test.

The tobacco smoking status of the subjects was classified into four categories from responses to a detailed smoking history: never, ex-, current or occasional smokers. Never smokers were men who had smoked no more than 5 to 10 packs of cigarettes (or equivalent of other tobacco product) throughout their lifetime. Ex-smokers were participants who have smoked greater than 100 cigarettes in their lifetime but have not smoked during the last month. Participants were classified as current smokers according to whether they were smoking daily or almost daily at the time (1). Occasional smokers were men who had smoked no more than 2 cigarettes in a week (3) or last smoked 2-30 days ago (2).

References:

1. Kaprio J, Koskenvuo MA. prospective study of psychological and socioeconomic characteristics, health behaviour and morbidity in cigarette smokers prior to quitting compared to persistent smokers and non-smokers. *J Clin Epidemiol*.1988;41:139-50.
2 Luoto R, Uutela A , Puska P. Occasional smoking increases total and cardiovascular mortality among men. *Nicotine Tob Res* 2000;2:133-9.
3. Paavola M, Vartiainen E, Puska P. Smoking cessation between teenage years and adulthood. *Health Educ Res* 2001;16:49-57.

Table Age- and SES-adjusted hazard ratios (HRs) for admissions to hospital for any alcohol-related diseases or deaths during January 1, 1970 to December 31, 2008 among former athletes compared to controls and mean (SD) age at first admission.

Sports groups	N of participants with alcohol-related disease or death	Mean age (SD) at first admission	Age-adjusted HR	95.0% Confidence Interval		P-value*	SES-adjusted HR	95.0% Confidence Interval		P-value*
				Lower	Upper			Lower	Upper	
Short & middle distance running	13	51.3 (11.7)	0.839	0.471	1.492	0.549	0.919	0.508	1.660	0.779
Endurance	13	62.4 (15.4)	0.655	0.366	1.170	0.152	0.673	0.375	1.206	0.183
Jumping & hurdling	12	58.7 (18.8)	0.579	0.319	1.052	0.073	0.631	0.340	1.170	0.144
Throwing & decathlon	14	60.8 (20.1)	0.888	0.509	1.551	0.677	0.933	0.529	1.644	0.810
Weightlifting	12	56.9 (14.3)	1.475	0.812	2.682	0.202	1.504	0.823	2.746	0.184
Combat sports	46	54.6 (13.1)	1.380	0.975	1.953	0.069	1.369	0.956	1.960	0.086
Team sports	40	56.6 (12.5)	0.931	0.647	1.341	0.702	1.006	0.681	1.486	0.977
Shooting	5	73.7 (15.7)	0.501	0.204	1.235	0.133	0.544	0.218	1.357	0.192
All athletes	155	57.2 (14.7)	0.934	0.729	1.197	0.591	1.001	0.765	1.310	0.995
Controls	106	51.5 (13.0)	1.000	-	-	-	1.000	-	-	-
Total	261	54.8 (14.3)	-	-	-	-	-	-	-	-

*p-values for statistical differences between sports groups and controls by Cox regression analysis.

Table Characteristic of participants who were responding alcohol-related questions in 1985 questionnaire: overall physical activity (MET-hours/week), current engagement in competitive sports and smoking status.

Sports groups	Mean MET-hours/ week (95% CI)*	Physical Activity tertiles† % (n)***			Currently engaged in competitive sports % (n)***	Smoking status‡ % (n)***	
		1	2	3		current smoker	never smoker
Short & middle distance running	28.5 (22.2-34.7)	25.4 (32)	34.9 (44)	39.7 (50)	12.9 (16)	9.6 (12)	53.6 (67)
Endurance	36.1 (29.6-42.6)	23.2 (38)	32.3 (53)	44.5 (73)	18.8 (29)	11.4 (18)	60.8 (96)
Jumping & hurdling	27.9 (23.1-32.6)	28.7 (47)	33.5 (55)	37.8 (62)	12.1 (19)	11.2 (18)	51.6 (83)
Throwing & decathlon	37.3 (29.0-45.7)	26.0 (32)	31.7 (39)	42.3 (52)	23.3 (27)	13.9 (17)	55.7 (68)
Weightlifting	26.9 (18.6-35.2)	34.8 (23)	28.8 (19)	36.4 (24)	6.5 (4)	23.4 (15)	46.9 (30)
Combat sports	28.2 (24.0-32.5)	31.4 (85)	30.3 (82)	38.4 (104)	7.4 (19)	18.4 (49)	47.2 (126)
Team sports	28.9 (25.7-32.1)	22.3 (73)	33.3 (109)	44.3 (145)	22.0 (69)	22.3 (72)	40.9 (132)
Shooting	19.3 (14.9-23.8)	37.2 (32)	37.2 (32)	25.6 (22)	25.0 (17)	7.2 (6)	36.1 (30)
All athletes	29.5 (27.7-31.4)	27.3 (362)	32.6 (433)	40.1 (532)	16.0 (200)	15.9 (207)	48.5 (632)
Controls	14.5 (12.8-16.1)	53.1 (401)	29.0 (219)	17.9 (135)	12.1 (33)	28.3 (210)	27.7 (205)

* $p<0.001$for statistical differences between sports groups and controls by Mann-Whitney-U-test.
† Overall physical activity (MET-hours/week): 1=MET-hours/week ≤6.0 (less active), 2= 6.0< MET-hours/week ≤22.5 (moderate active), 3= MET-hours/week >22.5 (highly active).
‡ Smoking status, see more details in Supplementary table 3.
*** $p<0.001$ for sports group differences in physical activity tertiles, currently engaged in competitive sports or smoking status by χ^2 -test.

III

THE DIFFERENCE IN RISK OF CHRONIC PULMONARY DISEASE MORBIDITY AND MORTALITY BETWEEN FORMER ELITE ATHLETES AND ORDINARY MEN IN FINLAND

by

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The difference in risk of chronic pulmonary disease morbidity and mortality between former elite athletes and ordinary men in Finland.

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Key words: SMOKING, OBSTRUCTIVE PULMONARY DISEASE, ASTHMA, EMPHYSEMA, CHRONIC BRONCHITIS, COPD, CHRONIC DISEASE, COHORT STUDY, FORMER ATHLETE(S), PHYSICAL ACTIVITY.

Word count: 3193

ABSTRACT

Introduction: The impact of a history of competitive sports on later smoking behavior and occurrence of chronic pulmonary diseases is poorly known. We investigated how a history of elite level sports predicted later pulmonary disease morbidity and mortality.

Methods: Chronic pulmonary disease incidence were assessed from national hospital and cause-of-death registers from 1970 to 2015 among Finnish male former elite athletes (n=2078) and matched controls (n=1453) alive in 1970 (mean age 45.0 years). Hazard ratios (HRs) were calculated by Cox proportional hazards model. In 1985, cohort members reported on their smoking habits, engagement in physical activity/sports and physician-diagnosed chronic diseases.

Results: The risk of any chronic pulmonary disease or death was lower among former athletes than controls (age-adjusted HR 0.61; 95% CI 0.46-0.83). The risk was significantly lower among endurance (HR 0.54), mixed (HR 0.61), and power sports athletes (HR 0.66) compared to controls. The age- and smoking pack-year-adjusted HRs of incident diseases from the time of the 1985 questionnaire until end of follow-up in former athletes was 0.58 (95% CI 0.37-0.93) compared to controls. In 1985 athletes smoked less and their cumulative smoking quantity was lower than that of controls. Former athletes were more physically active and self-reported less physician-diagnosed emphysema.

Conclusions: The risk of any chronic pulmonary disease was lower among former athletes than controls even after considering smoking status and cumulative smoking quantity. Ability to compete at the highest level of sports in young adulthood associates with a reduced risk of pulmonary disease in later life.

Key words: SMOKING, OBSTRUCTIVE PULMONARY DISEASE, ASTHMA, EMPHYSEMA, CHRONIC BRONCHITIS, COPD, CHRONIC DISEASE, COHORT STUDY, FORMER ATHLETE(S), PHYSICAL ACTIVITY.

INTRODUCTION

There is a widespread scientific and public health policy consensus that behavioral factors such as cigarette smoking, hazardous alcohol drinking and physical inactivity are major contributors to morbidity and mortality.[1-2] The healthcare expenditure on treating smoking attributable diseases is 5.7% of global health expenditure, a heavy economic burden that occurs particularly in Europe and North America.[3]

Smoking is associated with less leisure-time physical activity in cross-sectional and longitudinal studies.[4-6] Longitudinally habitual physical inactivity during late adolescence as compared to continuous physical activity through adolescence, predicts higher prevalence of smoking during young adulthood even after familial, including genetic factors are considered.[7] Studies have shown that current athletes smoke less than non-athletes.[8-10] Elite athletes also smoke less after concluding their competitive career.[11-13]

Cigarette smoke exposure, either directly or indirectly, has been highly correlated with the development of chronic obstructive pulmonary disease (COPD) and mortality[14] and smoking is well-established as the main cause of COPD. COPD is a heterogeneous collection of diseases with differing causes, pathogenic mechanisms, and physiological effects.[15] Conventionally COPD is subdivided into several entities, including emphysema and chronic bronchitis.[16] COPD was the third leading cause of death in the United States in 2013.[17]

Asthma is also a heterogenic and multifactorial disease which has different symptoms.[18] The Lancet Commission argued that asthma is not an adequate name, underlying treatable traits should be recognized, and comorbidities, lifestyle and environmental factors should be taken into account.[18] In the clinical setting, patients who present with a combination of COPD and asthma related traits are quite common.[19-21] However, there is also evidence that inflammatory processes differ in asthma and COPD and findings

suggest that there is no strong common genetic component in asthma and COPD,[22] but a genetic correlation between COPD and asthma has found.[23]

Asthma, airway hyperresponsiveness (AHR), and exercise-induced bronchoconstriction (EIB) appear to be more common in elite athletes than less-trained controls.[24-25] Correspondingly, epidemiologic studies suggest that self-reported and physician-diagnosed asthma are twice as common in Finnish athletes[26] and elite Norwegian athletes[27] than in randomly selected age-matched and sex-matched control populations. The higher prevalence of asthma reported in athletes may be a result of overdiagnosis, particularly because a diagnosis of asthma is often made on the basis of the history alone[28] and athletes often experience the symptoms during intensive exercise only.

Kujala et al (1996) reported that the lifetime occurrence of asthma or other pulmonary diseases was not increased in former elite athletes, and exercise alone, even in a cold environment, was not associated with a greater prevalence of asthma in later life.[29] Cohort studies have also revealed that physical activity decreases the number of exacerbations and short-acting bronchodilator use among COPD patients,[30] whereas physical inactivity may increase COPD patient`s perception of dyspnea.[31]

Prevention of chronic pulmonary diseases should be a major public health goal.[15,18] Furthermore, we know very little about the long-lasting effects of a history of competitive sports activity during young adulthood. Peak performance during competitive sports requires excellent lung function, but is inferred pulmonary health in young adulthood related to occurrence of chronic pulmonary disease in later life? In our study we especially focused on hospitalizations and deaths for selected chronic pulmonary diseases, with several decades longer follow-up time compared to our previous study of the same cohort.[29] We had three aims, the first of which was to examine how former competitive sports career and smoking was associated with the incidence and mortality of any chronic pulmonary diseases from 1970 and the time of the 1985 questionnaire until end of follow-up. Secondly, we compared these risks between different sports groups.

Thirdly, we studied how different factors, such as physical activity, were associated with smoking among former athletes after their active athletic career.

METHODS

Participants and participants involvement

An original cohort of former elite athletes (n=2657) was formed by identifying men who had represented Finland between 1920 and 1965 at least once at the Olympic Games, European or World championships, or international contests between two or three countries.[32] A control cohort (n=1712) was selected from Finnish men who at the age of 20 years had been identified healthy in the medical inspection for enlisting in ordinary military service (class A1, which means fully fit for ordinary military service). The control cohort was formed by matching the same age groups and area of residence with the former elite athletes. After first finding the athlete in the register, the selection of each control subject was done. The control subject was chosen nearest the A1 conscript listed to the athlete. This procedure was carried out in the years 1978-1979, when 85.3 % of the athletes had been identified.[32]

Participants who had died before the register-based follow-up started in January 1, 1970 were excluded from this study. Thus, the final study population (n=3531) consisted of 2078 former male athletes and 1453 matched control participants (Table 1 and Figure 1). Male former elite athletes are divided into three groups according to the type of training needed to achieve optimal results[33]: endurance (n=359), mixed (n=924) and power sports (n=795).

To adjust for occupational loading, the participants were classified into five socioeconomic groups based on occupation: upper white collar, lower white collars, skilled (blue) collars, unskilled workers and farmers[34] according to the occupation in which they had practiced the longest (for classification see Table,

Supplemental Digital Content 1). This classification also reflects the socioeconomic status (SES) of the participants. The socioeconomic group distribution of athletes differed from that of controls ($p<0.001$, χ^2 - test). Occupational data were collected partly from the Central Population Registry of Finland and partly from questionnaires, asking for the occupation in which they had been active the longest.

The register-based follow-up of hospitalizations started in January 1, 1970 and ended in December 31, 2015. Participants who had hospital admissions for chronic pulmonary diseases were identified from the National Hospital Discharge Register according to ICD-codes (ICD-8, ICD-9 or ICD-10). There are changes in diagnostic category, in ICD-10 COPD is defined as other chronic obstructive pulmonary disease (J 44.0), but earlier ICD-codes (ICD-8, ICD-9) do not include this specific code. Chronic pulmonary diseases were categorized into five main groups: chronic bronchitis, emphysema, other obstructive pulmonary diseases, asthma and bronchiectasia (for all details see Table, Supplemental Digital Content 2). The primary diagnosis was used to determine the reason for hospitalization, and the secondary diagnoses were ignored in the analysis. Participants who died from chronic pulmonary diseases (as the underlying cause of death) were identified from the National Death Register of Statistics Finland (cause of death chronic bronchitis, emphysema and asthma, grouped according to Statistics Finland internal codes 33 and 34; http://www.stat.fi/til/ksyyt/2005/ksyyt_2005_2006-10-31_luo_002.html).

Participants were not involved in setting the research question, the outcome measures or study design. Before taking part in the study all the participants gave informed consent by returning the questionnaires, which were accompanied by a cover letter explaining the purpose of the study. This study was conducted according to good clinical and scientific practice and the Declaration of Helsinki. Approval for register data collection was given by the Ministry of Social Affairs and Health in Finland, National Institute for Health and Welfare, and Statistics Finland.

Questionnaire study 1985

In 1985 a postal questionnaire eliciting information on discontinuation of sports career, socio-demographic factors (including occupational loading), health-related lifestyle habits, such as smoking and physical activity, and physician-diagnosed chronic diseases, was sent to the surviving cohort members (total n=2528, athletes n=1518 and controls n=1010).[29] Former elite athletes (n=1248, 82% response rate) and controls (n=759, 76%), who answered the smoking-related questions, were included in the statistical analysis. The response rate was 67.2% (n=119) among those participants alive in 1985 who had admissions to hospital at any time for any chronic pulmonary disease or death, and among this subgroup the response rate was higher among former athletes 71.1% (n=59) than among controls 63.8% (n=60).

The volume of physical activity (MET-hours/day, MET, metabolic equivalent) in 1985 was computed from the responses to three structured questions, using a previously validated method.[35] MET-hours/week was categorized into tertiles of physical activity: 1=MET-hours/day ≤0.9 (less active), 2= 0.9< MET-hours/day ≤3.2 (moderate active), 3= MET-hours/day >3.2 (highly active). Engagement in competitive sports was dichotomized. The term "discontinued sports" means that the athlete had retired from competitive sports based on questions in the 1985 questionnaire.

Smoking exposure was determined by the pack-years in 1985. It was calculated by multiplying the number of packs of cigarettes smoked daily by the number of years the person has smoked (https://www.cancer.gov/publications/dictionaries/cancer-terms/def/pack-year).

The smoking status of the participants was classified into five categories from responses to a detailed smoking history: never, former, current or occasional smokers and other (no exact information about smoking). Never smokers were men who had smoked no more than 5 to 10 packs of cigarettes (or equivalent of other tobacco product) throughout their lifetime. Former smokers were participants who had

smoked regularly and more than 5-10 packs of cigarettes in their lifetime but have quit. Participants were classified as current, daily smokers if they smoked regularly, i.e. daily or almost daily at the time. Occasional smokers were men who have smoked greater than 5 to 10 packs of cigarettes in their lifetime but were not regular smokers (for more details see Table, Supplemental Digital Content 3). For some analyses, we combined daily and occasional smokers as current smokers.

A history of physician-diagnosed chronic diseases was asked as dichotomous variables, such as asthma, emphysema and chronic bronchitis.

Statistical analysis

The descriptive data are presented as the mean and standard deviation (SD) or 95% confidence intervals (CI) if distributed normally; otherwise the descriptive data are shown as the median and range. The differences in the distributions of the categorical variables were examined using cross-tabulations with the Chi-square (χ^2) -test.

The follow-up of hospitalization and deaths started on January 1, 1970 and continued until the end of 2015, or until the date of hospitalization due to first chronic pulmonary disease event, emigration or date of death, whichever date came first. The primary event of incident chronic pulmonary disease was defined as the first recorded hospital episode or of death from any chronic pulmonary disease. Furthermore, incidence and mortality of any chronic pulmonary diseases were examined from the time of the 1985 questionnaire until end of follow-up.

A Cox proportional hazards model was used to calculate age-, SES- and smoking pack-year -adjusted hazard ratios (HRs) with their 95% CIs for incidence of chronic pulmonary disease between former athletes and controls. Time-invariant covariates were included to the analysis: age at entry and sports groups. Post

hoc analysis taking into account the number of comparisons was used to compare statistical differences between specific sports groups. Participants still alive at the end of follow up, those who emigrated, and those who died from any other cause, were censored. The Cox regression assumptions were tested by using Schoenfeld residuals (ph-test in Stata) and also by plotting.

The 1985 questionnaire data was analyzed using non-parametric Kruskal-Wallis-test and using the Dunn-Bonferroni approach for post hoc testing in pairwise comparisons for more than two groups ($p<0.05$), because some of the variables were not normally distributed and variances were not equal. The Mann-Whitney-U-test was used to compare differences between sports groups and controls ($p<0.05$). Homogeneity of variances were assessed using Levene`s test and normality using Kolmogorov-Smirnov`s test ($p<0.05$).

P-values <0.05 were considered statistically significant. Statistical analyses were performed using SPSS statistical software (version 24.0 for Windows; SPSS Inc., Chicago, IL) and Stata 14.0 (Stata Corp, College Station, Texas, USA).

RESULTS

The mean age of the athletes at the start of follow-up in 1970 was 45.4 years, and 44.3 years among controls. Altogether, 4.0% (n=83) of former athletes and 6.5% (n=94) of controls were admitted to hospital for any chronic pulmonary disease during the 45-year follow-up period. The most common reasons for admissions were asthma (athletes 1.7% (n=35), controls 2.5% (n=37)) and chronic bronchitis (athletes 0.5% (n=10), controls 1.4% (n=20)) (Table 2). Total days in hospital were 1693 among former athletes and 8761 among controls, mean of total days in hospital/total exposure years (days/years) was 3.5 (95% CI 0.2-6.9) among former athletes and 7.2 (95% CI 1.7-12.7) among controls (Table 2). The median total number of days in hospital was 14 (range 2-225) in all former athletes and 17 (1-6141) in controls; however, 20 former

athletes and 22 controls died of chronic pulmonary disease without being hospitalized at least once (Table 2). The mean age at the first admission of any chronic pulmonary disease was 68.2 (13.7) years for the former athletes and 66.9 (12.2) years for the controls (Table 2).

The age-adjusted hazard ratios (HRs) for incidence of overall hospitalization or death due to chronic pulmonary disease in former athletes was 0.61 (95% CI 0.46-0.83, p=0.001) compared to controls (Table 3). Correspondingly, age- and SES-adjusted hazard ratios (HRs) for incidence of overall hospitalization or death due to chronic pulmonary disease in former athletes was 0.62 (95% CI 0.45-0.86, p=0.004) compared to controls (Table 3). However, no statistically significant differences were observed between former athletes and controls in the analysis of different main diagnosis groups separately (Table 3).

In an additional analysis by sports category, compared to controls statistically significant age-adjusted HRs were 0.54 (95% CI 0.31 - 0.96) for endurance sports athletes, 0.61 (95% CI 0.42 - 0.89) for mixed sports athletes and 0.66 (95% CI 0.44 - 0.98) for power sports athletes (Table 3). Correspondingly, the age- and SES-adjusted risk of chronic pulmonary diseases was higher among controls compared to endurance and power sports athletes ($p<0.05$) (Table 3).

No differences in incidence were observed between those who did not respond to 1985 questionnaire study and respondents, among all participants (HR=1.10, 95% CI 0.78-1.53, p=0.60), among former athletes (HR=1.01, 95% CI 0.61-1.66, p=0.98) and among controls (HR=1.34, 95% CI 0.84-2.13, p=0.21).

The 1985 questionnaire data

Former athletes smoked less than controls ($p<0.001$). Among controls 27.8% were current smokers compared with 16.1% among the former athletes, with 10.1% of endurance, 17.0% of mixed and 17.8% of power sports athletes being current smokers (For more details see Table, Supplemental Digital Content 3).

Mean pack-years was higher among those controls 17.0 (95% CI 15.7-18.3) than former athletes 9.6 (95% CI 8.7-10.5), who have ever smoked during their lifetime ($p<0.001$).

The age- and pack-year-adjusted HRs of incident diseases from the time of the 1985 questionnaire until end of follow-up in former athletes was 0.59 (95% CI 0.37-0.93, $p=0.024$) compared to controls; those with incident disease prior to 1985 or reported physician diagnoses of asthma, emphysema and chronic bronchitis were excluded from this analysis (For more details see Table, Supplemental Digital Content 4). The age-adjusted HR of incident pulmonary disease in current smokers was 4.89 (95% CI 1.98-11.78, $p=0.001$) compared to never smokers, and there was no evidence of a smoking status by athlete status interaction on disease risk ($p=0.25$) (For more details see Table, Supplemental Digital Content 5).

Mean MET-hours/week in 1985 were significantly higher among former athletes than controls ($p<0.001$). One sixth (15.6%, n=189) of former athletes and 12.5% (n=35) of controls participated in competitive sports in 1985 ($p<0.001$). Athletes self-reported less physician-diagnosed emphysema than controls ($p<0.05$) (for more details see Table, Supplemental Digital Content 6).

DISCUSSION

There is lack of studies focused on the risk of chronic pulmonary diseases among former athletes several decades after their peak sporting performance. Our study showed that the risk of any chronic pulmonary disease or death was lower among former athletes than controls. Accordingly, the risk was lower among endurance, mixed or power sports athletes compared to controls. Some studies have shown that the prevalence of asthma is increased in elite athletes.[24-25] Correspondingly, epidemiologic studies suggest that self-reported and physician-diagnosed asthma are twice as common in Finnish athletes[26] and elite Norwegian athletes[27] than control populations. The higher prevalence of asthma reported in athletes may be a result of overdiagnosis, particularly because a diagnosis of asthma is often made on the basis of the history

alone.[28] Furthermore, respiratory symptoms may occur when exercising at high intensities which is common in athletes but uncommon in non-athletes. Although if non-athletes also exercise at high intensities, respiratory symptoms would probably occur more commonly. Former athletes smoked less than controls, as we have also reported earlier.[11-13] Our findings were consistent with other studies that have shown current athletes smoke less than non-athletes.[8-10]

This long-term follow-up study revealed new information on the long-term risk of chronic obstructive pulmonary diseases morbidity and mortality among former athletes. This study extended a previous study on the same cohort[29] which was based on questionnaire data and a nationwide reimbursable medication register. The use of the valid register data covering all participants was the strength of this study, data was specific but not sensitive concerning mild symptoms. Self-reported data on health-related behaviors include known limitations, but the self-reported smoking status has shown to be valid and reliable reflecting the social acceptance of smoking at that time.[36] Although questionnaire response rates were lower in individuals with chronic obstructive pulmonary diseases-related hospitalizations, the risk for bias in comparing the athlete group to non-athlete group was low.

Furthermore, there may have been selection bias at the beginning of our study as persons with suboptimal ventilatory function as young adults may have been incapable of becoming top athletes in endurance sports, thus reducing the prevalence of later chronic pulmonary disease. Furthermore, there is lack of consensus whether vigorous physical activity in a cold environment, which is common in Finland for several months of the year, predisposes to chronic pulmonary diseases. Some studies have found that persons who routinely perform strenuous exercise in cold conditions have a high prevalence of chronic airway inflammation and hyperreactivity,[37] also cold weather exercise can lead to asthma-like airway disease.[38]

Because former athletes are a selected group and they competed at top-level before 1965, we do not know exactly how well the results can be generalized to today's elite athletes or athletes who had competed in

lower level, to non-athletes or to women. Thus, we cannot conclude whether the decreased risk of pulmonary disease is due to selection of persons with extraordinary lung function to start with, and hence high reserve of that function in later life, or does training in adolescence and young adulthood promote development of lung function, yielding greater lung health in later life. Studies have suggested that interactions between genetic factors and early environmental exposures patterns may explain the early development of chronic obstructive lung diseases.[39] Particularly genome-wide association studies (GWASs) have been identifying genetic variants in the development of lung function in early life and later progression to asthma and COPD, but they account for at most a modest fraction of variability in lung function. There are still challenges to understand effects of genetic variants on health and disease and how they contribute opportunities for therapeutic intervention.[40]

Conclusions

The risk of any chronic pulmonary disease was lower among former athletes than controls even after considering smoking status and cumulative smoking quantity. Former athletes were more physically active, were less often smokers, had started smoking later and smoked less than controls. Furthermore, former athletes reported less physician-diagnosed emphysema than controls. In conclusion, ability to compete at the highest level of sports in young adulthood associates with a reduced risk of pulmonary disease in later life.

Contributors

SS, JK and UMK collected the data. TKK and UMK analyzed the data. TKK drafted the manuscript. All authors contributed to study design, and the revision of the manuscript, and accepted the final version. The authors apologize for not being able to cite all the noteworthy work in this area because of constraints on space.

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Competing interest

None.

Ethical approval

This study was conducted according to good clinical and scientific practice and the Declaration of Helsinki. The authors declare that the results of this study are presented clearly, honestly, and without fabrication, falsification or inappropriate data manipulation. Approval for register data collection was given by the Ministry of Social Affairs and Health in Finland, and Statistics Finland. All the participants gave informed consent by returning the questionnaires, which were accompanied by a cover letter explaining the purpose of the study.

Data sharing

The former athletes are well known persons in Finnish society; hence the data cannot be openly shared. Researchers are encouraged to contact the authors and we will make every effort to accommodate additional analyses.

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Table 1. Number of participants at entry to study and still alive in January 1, 1970, 1985 and December 31, 2015.

Sports groups	Participants at entry	Participants alive in 1970		Participants alive in 1985		Questionnaire responders in 1985		Participants alive in 2015	
	(N)	(N)	Mean age (SD)	(N)	Mean age (SD)	(N)	Mean age (SD)	(N)	Mean age (SD)
1. Endurance	437	359[*]	48.9 (13.0)	287	64.2 (13.0)	226	60.6 (11.1)	110	78.2 (11.3)
2. Mixed sports	1046	924[†]	43.1 (13.2)	785	58.3 (13.2)	607	54.9 (10.8)	411	76.1 (10.8)
3. Power sports	941	795[‡]	46.5 (13.2)	607	61.8 (13.2)	443	57.5 (10.7)	251	74.3 (12.4)
All athletes	2424	2078	45.4 (13.3)	1679	60.6 (13.3)	1276	56.8 (11.0)	772	75.8 (11.6)
Controls	1712	1453	44.3 (13.0)	1149	59.6 (13.0)	777	55.0 (10.3)	494	73.8 (13.4)
Total	**4136**	**3531**	**45.0 (13.2)**	**2828**	**60.2 (13.2)**	**2053**	**56.1 (10.8)**	**1266**	**75.0 (12.4)**

Data are numbers in 1970.

[*]Long distance running 162, middle distance running 79, cross country skiing 118.

[†]Soccer 262, ice hockey 154, basketball 84, high jump 46, pole vault 54, long jump 35, triple jump 34, hurdling 85, short distance running 123, decathlon 45.

[‡]Weightlifting 115, boxing 253, wrestling 261, shot put 38, discus 35, javelin 56, hammer 37.

Table 2. Number of participants in chronic pulmonary diseases main diagnosis groups, total days in hospital, median of days in hospital, mean of exposure time and mean of total days in hospital/total exposure time among former athletes and controls.

Sports groups	Asthma*	Chronic bronchitis*	Emphysema*	Other obstructive chronic pulmonary disease*	Bronchi-ectasia*	Any chronic pulmo-nary disease*	Chronic pulmonary disease-related death (no hospitali-zation before)	Any chronic pulmonary disease-related disease or death	Total days in hospital for any chronic pulmonary disease during follow-up	Median of days in hospital for chronic pulmonary disease (range)	Mean of exposure years (95% CI)	Mean of total days in hospital / Total exposure years (days/ years)
Endurance	6	1	1	3	2	13	1	14	302	14.0 (5-61)	29.3 (27.8-30.8)	9.8 (9.1-28.7)
Mixed sports	14	7	0	6	0	27	11	38	458	10.0 (2-82)	33.1 (32.2-34.1)	2.1 (0.4-4.7)
Power sports	15	2	0	5	1	23	8	31	933	19.0 (3-225)	28.3 (27.2-29.3)	2.2 (0.7-3.7)
All athletes	35	10	1	14	3	63	20	83	1693	14.0 (2-225)	30.6 (30.0-31.2)	3.5 (0.2-6.9)
Controls	37	20	1	10	2	70	24	94	8761	17.0 (1-6141)	29.5 (28.7-30.2)	7.2 (1.7-12.7)
Total	72	30	2	24	5	133	44	177	10454	15.0 (1-6141)	30.1 (29.6-30.6)	-

*Number of participants who have any chronic pulmonary disease. One participant may have more than one main diagnosis if there are more than 1 hospital admissions.

Table 3. Age- and socioeconomic status (SES) -adjusted hazard ratios (HRs) for incidence of chronic pulmonary disease from January 1, 1970 to December 31, 2015 among former athletes compared to controls and mean (SD) age at first admission.

			Age -adjusted				Age- and SES-adjusted			
Sports groups	**N of participants with incident disease**	**Mean age (SD) at disease onset**	**HR**	**95.0% Confidence Interval**		**P-value***	**HR**	**95.0% Confidence Interval**		**P-value***
				Lower	**Upper**			**Lower**	**Upper**	
Endurance (n=359)	14	69.1 (17.4)	0.544	0.310	0.955	0.034	0.472	0.266	0.837	0.010
Mixed sports (n=924)	38	68.2 (12.1)	0.612	0.419	0.894	0.011	0.715	0.470	1.089	0.118
Power sports (n=795)	31	67.8 (14.0)	0.655	0.436	0.984	0.041	0.631	0.414	0.961	0.032
All athletes (n=2078)	83	68.2 (13.7)	0.614	0.457	0.826	0.001	0.621	0.450	0.859	0.004
Controls (n=1453)	94	66.9 (12.2)	1.000	-	-	-	1.000	-	-	-
Total (n=3531)	177	67.6 (13.0)	-	-	-	-	-	-	-	-

*p-values for statistical differences between sports groups and controls from Cox regression analysis.

IV

MORTALITY AND HEALTH-RELATED HABITS IN 900 FINNISH FORMER ELITE ATHLETES AND THEIR BROTHERS

by

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Mortality and health-related habits in Finnish former elite athletes and their brothers.

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Key words: ALCOHOL, CHRONIC DISEASE, COHORT STUDY, FORMER ATHLETE, MORTALITY, PHYSICAL ACTIVITY, SMOKING.

Word count: 2983 (with text modifications and new references 3085)

ABSTRACT

Background: There is conflicting evidence on the associations between participation in vigorous sports, health habits, familial factors and subsequent mortality. We investigated all-cause mortality and health-related behavior among former elite athletes and their brothers.

Methods: The mortality of Finnish male former elite athletes, who had represented Finland between 1920 and 1965 (n=900) and their age-matched brothers (n=900) was followed from the time when athlete started an elite athlete career until December 31, 2015. The age-adjusted Hazard ratios (HRs) were calculated by a paired Cox proportional hazards model. In 2001 surviving participants (n=199 athletes and n=199 age-matched brothers) reported their self-rated health, physical activity, alcohol consumption and smoking habits in the questionnaire.

Results: During the total follow-up period 1296 deaths (72% of the cohort) occurred. The age-adjusted HRs for all-cause mortality in former athletes was 0.75 (95% CI 0.65 to 0.87, $p<0.001$) compared to their age-matched brothers. Median age at death was 79.9 years for endurance, 75.9 years for mixed sports, and 72.2 years for power sports athletes, and 77.5, 73.7 and 72.2 years for their age-matched brothers respectively. In 2001, compared to their brothers, former athletes smoked less ($p<0.001$), were more physically active ($p<0.05$), and rated their health more often as very good ($p<0.05$).

Conclusions: Former elite athletes are more physically active, smoke less, have better self-rated health and live longer than their brothers. Genetic differences between athletes and brothers, aerobic training for endurance elite sports and a healthier lifestyle may all contribute to reduced mortality.

Key words: ALCOHOL, CHRONIC DISEASE, COHORT STUDY, FORMER ATHLETE, MORTALITY, PHYSICAL ACTIVITY, SMOKING.

INTRODUCTION

Observational studies show that certain lifestyle habits, such as hazardous alcohol drinking, cigarette smoking, and low physical activity are important predictors of morbidity and mortality.[1] Furthermore, self-rated health (SRH) is a predictor of mortality,[2] and physical activity (PA) is related to better SRH.[3-4]

Regular PA is associated with better functioning and well-being especially at older age,[5-6] a lower risk for morbidity,[7-8] and all-cause mortality.[9-12] Former endurance and mixed sports athletes have lower mortality than the general population,[13] but previous studies report inconsistent results for power sports athletes.[14] However, very intense patterns of exercise may increase risk for cardiovascular morbidity and mortality compared to light to moderate amounts of exercise.[9,15]

There is no irrefutable evidence on a causal relationship between physical activity in adulthood and mortality, either in animal experiments, or randomized controlled intervention studies with healthy individuals.[16] Cardiorespiratory fitness (CRF) is as a strong, independent predictor of all-cause and disease-specific mortality[17] and high level of CRF is associated with a lower risk of all-cause and cardiovascular disease (CVD) mortality, in both rats[18] and humans.[19,20] It has previously been widely reported that former athletes have lower morbidity and mortality compared to general population,[12] which may be due to their genetic background, increased PA levels or other better health habits.[7-8,13] Former athletes are more physically active and smoke less than controls,[12] while former[21-22] or current athletes consume more alcohol than non-athletes.[23]

In contrast to our earlier studies on a cohort of former elite athletes before,[11,13] but we use brothers as control rather than unrelated healthy men. We selected brothers as controls because they are genetically related and generally share the same family environment in childhood. Thus, they may serve to control for experiences and exposures that cannot be assessed when using unrelated controls. The aim of this study was to investigate whether all-cause mortality and the development of behavioral and biological risk factors differ between athletes and their brothers in later life. This unique study reveals novel data on the associations between PA, familial factors, health-related behaviour, and mortality.

METHODS

Participants

An original cohort of former elite athletes (n=2657) was formed by identifying men who had represented Finland between 1920 and 1965 at least once at the Olympic Games, European or World championships, or inter-country competitions.[12] The athlete's brothers' (n=2674) data have been collected from local parish registry data in early 1980s.

We excluded 219 athletes and 1774 brothers due to missing or uncertain information on date of birth, date of death or death before 1936. We also excluded brothers who had died before the time when the athlete started an elite athlete career. After including only athletes who had brothers with complete data, the final study population (n=2755) consisted of 900 former male athletes and their 1855 brothers (Table 1 and Figure 1). Former elite athletes (n=900) and their individually matched brothers who were closest in age (n=900) were included in the primary mortality analysis (paired Cox regression model). The former male athletes were

classified according to sports and sports-specific aerobic fitness characteristics into the following three groups:[12] endurance sports (n=217), mixed sports (n=307) and power sports (n=376) (individual sports are given in Table 1 and Figure 1). We also did a sensitivity analysis based on former elite athletes (n=900) and all of their brothers (n=1855) (Figure 1).

Socioeconomic status was defined by five occupation-based social class groups: upper white collar (executive and professionals), lower white collar workers (clerical work and equivalent), skilled workers, unskilled workers and farmers.[24] Data were collected partly from the Central Population Registry of Finland and partly from questionnaires based on the occupation in which they had practiced the longest. The occupational group distribution of athletes differed from that of brothers ($p<0.001$, χ^2 -test) (Supplementary Table 1).

The data consisted of time and cause of death until December 31, 2015. The main outcome variable was total all-cause mortality, and it was analyzed by using Cox regression model. Secondly, the distributions of cause-specific deaths were analyzed by using cross-tabulations. Mortality data were collected from the National Death Register of Statistics Finland (causes of death; http://www.stat.fi/til/ksyyt/2005/ksyyt_2005_2006-10-31_luo_002.html).

Comparison of athletes and brothers: the questionnaire study 2001

In 2001 a postal questionnaire was sent to surviving elite athletes and brothers resident in Finland eliciting information on health, lifestyle and sociodemographic characteristics. In 2001, 460 of 900 former athletes and 392 of their 900 age-matched brothers were alive; there were 199 athletes who with their 199 age-matched brothers responded to the questionnaire. We also did sensitivity analysis for the athletes (n=199) and all of their brothers (n=322)

(Figure 1). Furthermore, we used population controls (n=416), who were the unrelated controls in the present cohort.[25]

Questionnaire-based covariates

Body mass index (BMI) was calculated as self-reported weight/height^2 (kg/m^2). The volume of PA (MET-hours/day, MET, metabolic equivalent) was based on responses to three structured questions on intensity, duration and frequency of activity using a previously validated method.[10,26] The participants were classified into tertiles of PA. Sedentary time was assessed by continuous variable called as sitting time (hours/day).

Based on a detailed smoking history, we define smoking status as never, former, current or occasional smokers. Those who have never smoked more than 100 cigarettes lifetime were defined as never smokers. Former smokers had smoked more than 100 cigarettes in their lifetime, who had smoked regularly, but had not smoked during the last month. Current smokers were regular (daily or almost daily) smokers at the time of data collection.[27] Occasional smokers were men who smoked no more than 2 cigarettes in a week[28] or had last smoked 2-30 days ago.[29] Among current or ex-smokers, nicotine dependence was assessed using the Heaviness of Smoking Index (HSI), i.e. 2 items of the Fagerström Test for Nicotine Dependence (FTND): the number of cigarettes per day and the time to the first cigarette in the morning.[30] The HSI sum score ranges from 0 – 6, reflecting the degree of physical dependence, and accounts for about 80 % of the variance in the FTND.[25,30]

Alcohol use was assessed by questions on total alcohol consumption and heavy drinking occasions (HDO). Alcohol consumption based on quantity-frequency measures of beverage

use was converted into grams of pure alcohol per month as previously reported.[31] Drinkers were classified as abstainers and light (3 or fewer drinks per week), moderate (more than 3 but not more than 14 drinks per week) and heavy drinkers (on average more than 2 drinks a day).[32] Heavy drinking occasions (HDO) were asked by question on the frequency of drinking 5 or more alcoholic drinks on a single occasion in the past 30 days.[33] A HDO variable had three categories: no HDO, HDO 1-2 times a month and HDO at least weekly.

Self-rated health (SRH) was measured by asking the participants to rate their own health status at this moment on a previously validated[2] 5-point Likert scale: 1) very good, 2) quite good, 3) average, 4) quite poor and 5) poor. Self-reported current symptoms and a history of physician-diagnosed chronic diseases were grouped as dichotomous variables, such as any chronic disease, hypertension with medication, angina pectoris, myocardial infarct, diabetes, kidney disease, gastric ulcer, asthma and osteoarthritis. A history of any sports injury- and Achilles tendinitis- or total rupture – was queried.

Activities of Daily Living (ADL) and Instrumental Activities of Daily Living (IADL) were assessed by a total of nine items.[34] These national survey questions were modified from classification of functional capacity.[35-36]

Self-rated mood was defined by two partial scales (6 depression items and 6 anxiety items) of the short stress symptom survey extracted from the Brief Symptom Inventory-53 (BSI-53).[37]

Statistical analysis

The descriptive data are presented as the mean and standard deviation (SD) or 95% confidence intervals (CI) if distributed normally; otherwise the results are shown as the median. The differences in the distributions of the categorical variables among different sports groups and brothers were examined using cross-tabulations with the Chi-square (χ^2) -test.

The follow-up of all-cause mortality of each athlete and brother started from the time when the athlete was an elite athlete, and continued until the date of death (outcome event), date of emigration (censoring event) or end of follow-up on December 31, 2015. A paired Cox proportional hazards model was used to calculate age-adjusted hazard ratios (HRs) with 95% CIs for all-cause mortality of former athletes relative to their brothers. Post hoc analysis taking into account the number of comparisons was used to compare statistical differences between specific sports groups. We also adjusted the mortality analysis for family clustering in the subgroup analysis based on all questionnaire 2001 respondents. The Cox regression assumptions were tested by using Schoenfeld residuals (ph-test in Stata) and by plotting "log-log plots".

The 2001 questionnaire data was mainly analyzed using non-parametric Wilcoxon`s signed rank test (a matched-pair analysis) for ordinal or continuous variables and McNemar`s test (a matched-pair analysis) for nominal variables to analyze the differences between each athlete and the age-matched brother. The Chi-square (χ^2) -test and The Mann-Whitney-U-test were used to compare differences between athletes and all their brothers, because some of the variables were not normally distributed and variances were not equal. Homogeneity of variances were assessed using Levene`s test and normality using Kolmogorov-Smirnov`s test ($p<0.05$).

P-values <0.05 were considered statistically significant. Statistical analyses were performed using SPSS statistical software (version 24.0 for Windows; SPSS Inc., Chicago, IL) and Stata 14.0 (Stata Corp, College Station, Texas, USA).

RESULTS

Mortality data

The final sample for mortality analysis included 900 former elite athletes and their 900 age-matched brothers. Mean age at inclusion was 24.8 years and mean year of inclusion was 1943, and median follow-up time was 52.0 years (ranging from 0.1 to 83.4 years). Study group characteristics are shown in Table 1. During the total follow-up period 1296 deaths occurred; 72.1% (n=649) of the athletes and 71.9% (n=647) of the brothers died. Natural causes accounted for death in 92.2% (n=1085) of the participants. The most common specific cause of death was ischemic heart disease (IHD), which occurred in 35.5% (n=418) of all men (34.3% (n=219) athletes, 36.9% (n=199) brothers) (Supplementary Table 2).

Median age at death among the former athletes (75.9 years, 95% CI 75.1 to 76.7) was statistically significantly higher than among brothers (73.0 years, 95% CI 72.0 to 74.0). Median age at death among endurance (79.9 years, 95%CI 79.1 to 80.7) and mixed sports athletes (75.9 years, 95% CI 75.1 to 76.7) was statistically significantly higher than among power sports athletes (74.1 years, 95% CI 73.3 to 75.0) (Table 2).

The age-adjusted hazard ratios (HRs) for all-cause mortality in former athletes was 0.75 (95% CI 0.65 to 0.87, p<0.001) compared to their age-matched brothers (Table 2). In a

subgroup analysis, compared to their age-matched brothers HR for all-cause mortality was 0.61 (95% CI 0.45 to 0.82, p=0.001) for endurance sports athletes, 0.85 (95% CI 0.65 to 1.10, p=0.220) for mixed sports athletes and 0.78 (95% CI 0.62 to 0.98, p=0.033) for power sports athletes (Table 2). In the sensitivity analysis the age-adjusted HRs for all-cause mortality in former athletes (n=900) was 0.76 (95% CI 0.67 to 0.86, p<0.001) compared to all their brothers (n=1855) (Supplementary Table 3).

We found that in the individual-based non-pairwise analysis the assumptions of Cox regression model were violated suggesting a lower mortality of athletes at younger ages but less so at older ages. We divided the follow up time into two periods: age at entry to median age at death and median age at death to December 31, 2015. The age-adjusted pairwise HRs for all-cause mortality in former athletes was 0.68 (95% CI 0.57 to 0.82, p<0.001) compared to their age-matched brothers in the first follow-up period and 0.74 (95% CI 0.57 to 0.98, p=0.03) in the second period. The results of the sensitivity analysis were consistent with the results of age-matched pair analysis, but there was no difference in mortality during the later period (Supplementary Table 4).

We also calculated adjusted HRs for all-cause mortality for all questionnaire 2001 respondents until 2015. There were no differences in HRs between groups (Supplementary Table 5).

Questionnaire data

There were no statistically significant differences in anthropometric data between former athletes and brothers (Table 3). Mean MET-hours/month in 2001 were significantly higher

among former athletes than their age-matched brothers (4.4. *vs.* 3.2 MET-hours/day) ($p<0.05$). More athletes (11.2%) than brothers (2.1%) participated in vigorous PA ($p<0.05$). The athletes sat less than brothers ($p<0.001$) (Table 3).

Former athletes smoked less than their age-matched brothers ($p<0.001$), and among smokers the athletes were less nicotine-dependent. No significant differences in alcohol use were observed (Table 3). Former athletes self-reported their health as very good much more frequently (20.6%) than their brothers (8.6%) ($p<0.05$). There were no statistically significant differences in specific chronic diseases, such as hypertension, heart disease, diabetes or asthma between former athletes and their brothers. However, brothers reported more chronic disease than former athletes ($p<0.05$). Former athletes had more self-reported physician-diagnosed sports injuries and Achilles tendinitis than their brothers ($p<0.05$) (Table 4). There were no differences in mobility, physical or psychosocial functioning of daily living between former athletes and their brothers, except travelling by public transport. Mean of anxiety score was higher among brothers than among former athletes ($p<0.05$), and brothers felt themselves more restless and excited than former athletes ($p<0.05$) (Table 4).

We also did sensitivity analysis based on former elite athletes (n=199), all of their brothers (n=322) and population control (n=416) (Supplementary Tables 6 and 7). The results were consistent with the results of age-matched pair analysis.

DISCUSSION

Principal findings

Former elite athletes survived 2-3 years longer than their brothers, with the largest difference seen for endurance athletes and none for power athletes. However, there was no difference in the risk of all-cause mortality between athletes and all of their brothers at older age. Overall SRH and health-related lifestyle habits of former elite athletes were better than those of their brothers. However, there were no significant differences in mobility, physical or psychosocial functioning of daily living between former athletes and their brothers who responded to the 2001 questionnaire. The brothers showed evidence for worse mood than athletes.

Strengths and limitations of our study

This unique long-term follow-up study revealed novel data on associations between health-related behaviour, and mortality among individuals from the same family, who share both genes and childhood environment. Valid death register data covering all participants was the strength of this study. Although our questions on PA,[26] smoking[38] use of alcohol[39] have been previously validated, self-reported data on health-related behaviors is only based on an age-matched respondents subgroup from 2001. We have exact data on neither level of PA across the lifespan nor health at younger ages among brothers. A partly insufficient data of SES or causes of death among brothers could be considered as a limitation. However, athletes and their brothers are from same family and differences in SES may be caused by athletic career. We do not exactly know how well the results can be generalized to non-athletes, women or today`s athletes from different sports groups or cultures, such as NFL players[40-41] or U.S basketball players[42] who seem to have high prevalence of clinically significant cardiac abnormalities.

Comparisons with other studies

Observational studies in general have found that regular PA is associated with a lower risk for all-cause mortality,[9-11] but high amounts of exercise among poorly trained individuals may increase risk for CVD morbidity and mortality compared to light to moderate amounts of exercise.[9,15] Studies of elite athletes, i.e, among well-trained sportsmen do not support that. The pattern of training and frequency of high-intensity exercise needs to be taken into account.

Our study extends previous findings that former athletes, especially former endurance sports athletes, have higher longevity and lower mortality than the general population.[12-13] In general, runners have a 25%-40% reduced risk of premature mortality and live approximately 3 years longer than non-runners.[43] Although high lifelong exercise volume among surviving athletes is associated with coronary artery calcifications, the atherosclerotic plaques of the most active athletes have a more benign composition.[44] The evidence is mostly based on observational follow-up studies, one limitation of which is the difficulty of controlling for genetic or other selection bias. There is no irrefutable evidence on a causal relationship between PA in adulthood and mortality. Based on previous studies on twins and former elite athletes[8,10] and both animal and human findings, it could be proposed that some of the association between high PA[16-17,19] and low mortality is explained by familial or genetic factors. Despite this, we see a lower mortality in former athletes, especially at younger age compared to their brothers knowing the limitation that some of the brothers may have had health concerns at the beginning of our follow-up.

Our findings support previous studies that have shown athletes were more physically active than controls.[12,22] But we also observed that brothers were more physically active and went in

for more vigorous activities than population controls (Supplementary Table 6), suggesting a familial contribution to PA that probably originated prior to the elite athletes career.

Though brothers smoked more than former athletes, there was no difference in alcohol use. Former athletes are known to smoke less than controls,[12,22] and we find that brothers also smoked less than population (Supplementary Table 6). It has been found that persistent physical inactivity in adolescence relates to adult smoking, even after familial factors are taken into account.[45]

There were better SRH and less chronic diseases among former athletes in accordance with healthy life expectancy. However, brothers had less chronic diseases than population controls (Supplementary Table 7). There was no difference in disability, possibly because those with highest disability may not be able to respond to questionnaire. As earlier discussed both athletes and brothers were physically active, which could partly explain that no differences were found in functioning. Observational studies have found that regular PA is associated with better functioning and well-being especially at older age,[5-6] and a lower morbidity.[7-8]

Future directions

Our results are based on men who were former athletes 50 or more years ago. So, it is important to investigate the associations between PA, familial factors, health-related behaviour, and mortality among today's athletes, non-athletes or women. Traditional and genetically informative designs are needed to investigate how elite performance related to future health outcomes.

CONCLUSIONS

Former elite athletes have lower premature mortality than their brothers, and correspondingly better SRH and health-related lifestyle habits. Former endurance sports athletes and their brothers had reduced mortality compared to power sports athletes and their brothers. In conclusion, our findings support previous findings for a role of genetic or childhood family factors in determining high aerobic fitness and reduced mortality.

What are the new findings?

- Former elite athletes survived 2-3 years longer than their brothers.
- There was no difference in the risk of all-cause mortality between athletes and all of their brothers at older age.
- Overall self-rated health and health-related lifestyle habits of former elite athletes were better than those of their brothers.

Contributors

SS, JK and UMK collected the data. TKK and UMK analyzed the data. TKK drafted the manuscript. All authors contributed to study design, and the revision of the manuscript, and accepted the final version.

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Competing interest

None.

Ethical approval

This study was conducted according to good clinical and scientific practice and the Declaration of Helsinki. The authors declare that the results of this study are presented

clearly, honestly, and without fabrication, falsification or inappropriate data manipulation. Approval for register data collection was given by the Ministry of Social Affairs and Health in Finland, and Statistics Finland. All the participants gave informed consent by returning the questionnaires, which were accompanied by a cover letter explaining the purpose of the study.

Data sharing

The former athletes are well known persons in Finnish society; hence the data cannot be openly shared. Researchers are encouraged to contact the authors and we will make every effort to accommodate additional analyses.

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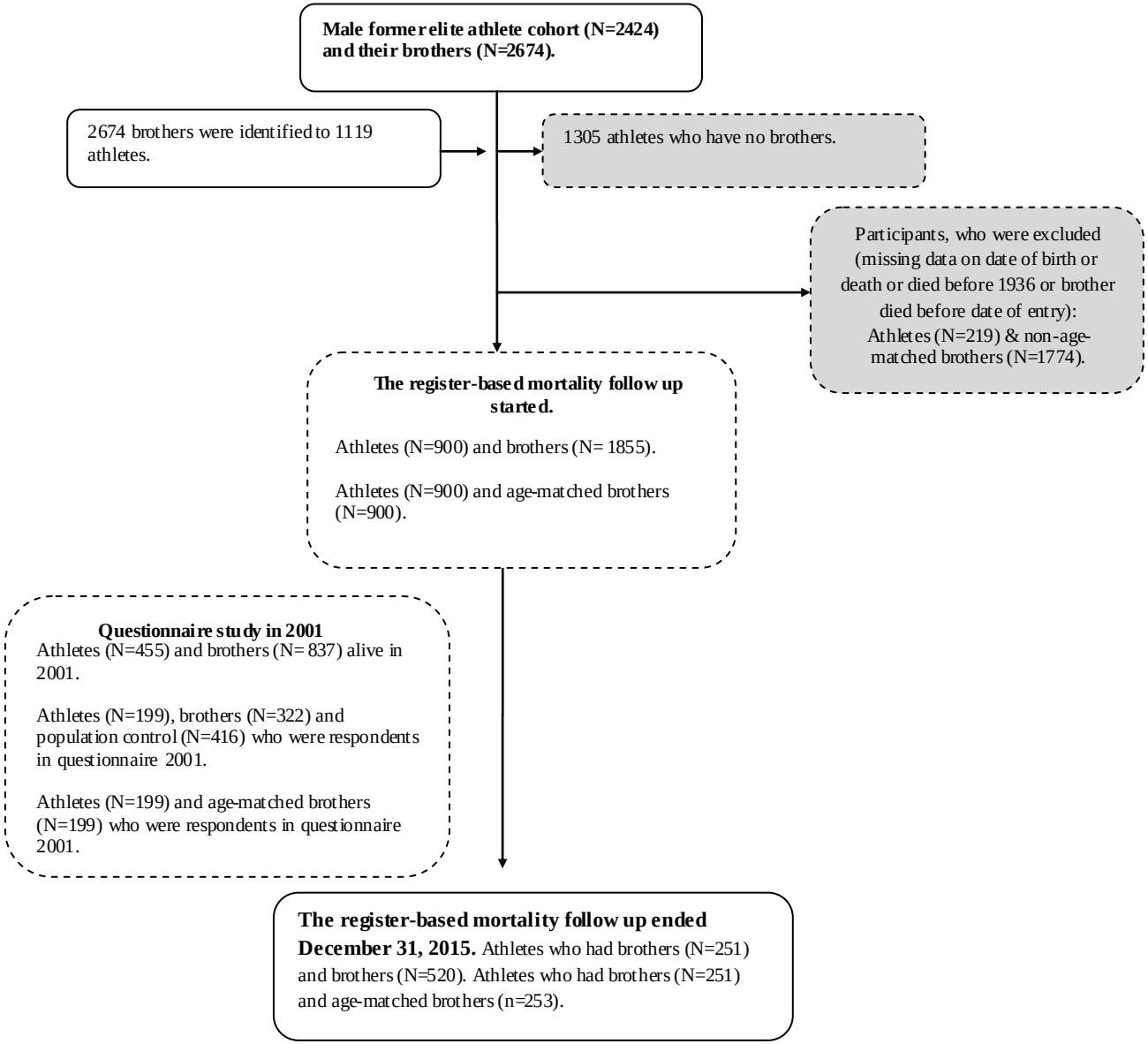

Figure 1. Study profile.

Table 1. Number of participants at entry to study and still alive in 2001, and December 31, 2015.

Sports groups	Participants at entry	Participants at entry included in statistical analysis		Participants alive in 2001		Questionnaire responders in 2001 included in statistical analysis		Participants alive in 2015	
	N	N	Mean age (SD)	N	Mean age (SD)	N	Mean age (SD)	N	Mean age (SD)
1. Endurance	341	217*	27.0 (4.1)	104	71.8 (8.4)	44	69.1 (7.4)	54	81.6 (5.8)
2. Mixed sports	941	307†	23.0 (3.0)	177	67.4 (8.7)	80	66.0 (8.1)	106	78.0 (5.8)
3. Power sports	1142	376‡	24.5 (3.7)	174	68.1 (7.3)	75	67.0 (6.6)	91	79.5 (5.5)
All athletes	2424	900	24.6 (3.9)	455	68.7 (8.3)	199	67.1 (7.5)	251	79.3 (5.8)
Brothers	2673	900	25.1 (6.6)	393	67.4 (9.6)	199	65.9 (8.6)	253	79.1 (8.0)
Total	**5097**	**1800**	**24.8 (5.4)**	**848**	**68.1 (9.0)**	**398**	**66.6 (8.0)**	**504**	**79.2 (7.0)**

Data are numbers at entry included statistical analysis.

* Long distance running 95, middle distance running 45, cross-country skiing 77.

† Soccer 75, ice hockey 33, basketball 22, high jump 17, pole vault 23, long jump 14, triple jump 15, hurdling 36, short-distance running 46, decathlon 26.

‡ Weight-lifting 43, Boxing 95, wrestling 155, shot-put 18, discus 15, javelin 32, hammer 19.

Table 2. Age-adjusted hazard ratios (HRs) for all-cause deaths from time of entry to December 31, 2015 among former athletes (n=900) compared to age-matched brothers (n=900), mean (SD), median age at entry, median and mean age at death.

Sports groups	Mean age (SD) at entry	Median age at entry	Median age at death (95% CI)	Mean age at death (95% CI)	Age-adjusted HR (95% CI)	P-value*
Endurance (n=217)	27.0(4.1)	26.0	79.9(79.1-80.7)	77.5(75.7-79.3)	0.607(0.448-0.823)	0.001
Mixed sports (n=307)	23.0(3.0)	23.0	75.9(75.1-76.7)	73.3(72.0-75.4)	0.848(0.652-1.104)	0.220
Power sports (n=376)	24.5(3.7)	24.0	74.1(73.3-75.0)	72.2(70.7-73.3)	0.779(0.619-0.980)	0.033
All athletes (n=900)	24.6(3.9)	24.0	75.9(75.1-76.7)	74.0(73.0-75.0)	0.753(0.648-0.875)	<0.001
Brothers (n=900)	25.1(6.6)	25.0	73.0(72.0-74.0)	69.9(68.7-71.0)	1.000	-

*p-values for statistical differences between sports groups and brothers by a paired Cox regression analysis.

Table 3. Demographic and lifestyle characteristics (physical activity, alcohol use and smoking) among former athletes (n=199) and brothers (n=199).

	Former athletes (N=199)	**Brothers (N=199)**	**P-value**
Age (mean (SD)), years*	67.1(7.5)	65.9(8.6)	0.007[a]
Marital status % (n)*			0.035[b]
Single	1.0(2)	4.5(9)	
Married	69.7(138)	77.4(154)	
Remarried	6.6(13)	3.5(7)	
Cohabiting	8.1(16)	3.5(7)	
Divorced	8.1(16)	4.5(9)	
Widowed	6.6(13)	6.5(13)	
Anthropometric data			
Height, m, mean (95% CI)	175.9(174.8-177.0)	175.1(174.2-176.0)	0.13[a]
Weight, kg, mean(95% CI)	81.4(79.6-83.3)	81.4(79.7-83.1)	0.94[a]
Mean BMI, kg/m^2 (95% CI)	26.2(25.8-26.7)	26.5(26.0-27.0)	0.41[a]
Physical activity			
Mean MET-hours/day (95% CI)*	4.4(3.7-5.1)	3.2(2.7-3.6)	0.012[a]
Physical Activity tertiles[†] % (n)			0.734[a]
Less active	34.7(69)	37.7(75)	
Moderately active	38.2(76)	29.1(58)	
Highly active	27.1(54)	33.2(66)	
Types of physical activity %(n)			
No physical activity	3.6(7)	3.1(6)	0.001[a]
Walking	45.2(89)	63.4(127)	
Walking and jogging	32.0(63)	20.6(40)	
Jogging	8.1(16)	10.8(21)	
Brisk running	11.2 (22)	2.1 (4)	
Sedentary lifestyle			
Mean sitting time hours/day (95% CI)*	5.3(4.9-5.7)	6.3(5.8-6.7)	<0.001[a]
Smoking			
Smoking status,% (n)*			2.57e^{-04}[a]
Current smoker	7.1(14)	13.9(27)	
Occasional smoker	1.0 (2)	2.1(4)	
Ex-smoker	25.9(51)	39.2(76)	
Never smoker	66.0(130)	44.8(87)	
Heaviness of smoking index (HSI, range 0-6), mean (SD)*			0.004[a]
among current / ex-smokers	1.5(1.6)/ 1.4(1.8)	2.0(1.7)/ 1.3(1.5)	

Alcohol			
Total alcohol consumption g/month (mean, 95% CI)	184.9(136.2-233.6)	239.3(137.0-341.6)	0.58[a]
Type of alcohol drinker %(n)			0.062[a]
Abstainer or light drinker	67.3(134)	60.3(120)	
Moderate drinker	21.1(42)	25.6(51)	
Heavy drinker	11.6(23)	14.1(28)	
Heavy drinking occasions (HDO) % (n)	65.8(129)	65.3(126)	0.70[b]
At least once a week	10.2(20)	16.1(31)	
1-2 times a month	16.3(32)	18.1(35)	
No HDO	73.5(144)	65.8(127)	

* $p<0.05$ for statistical differences between athletes and age-matched brothers by [a)] Wilcoxon`s signed rank -test or [b)] McNemar`s test.

[†] Overall physical activity (MET-hours/day, MET, metabolic equivalent): MET-hours/day $\leq$ 2.3 are less active, 2.3 < MET-hours/day $\leq$ 4.5 are moderately active and MET-hours/day > 4.5 are highly active.

Table 4. Self-rated health, diseases, functioning of daily living, mobility, self-rated mood and working status among former athletes (n=199) and brothers (n=199).

	Former athletes (n=199)	**Brothers (n=199)**	**P-value**
Self-rated health (SRH) % (n)*			3.3e^{-04}[a]
Very good	20.6(41)	8.6(17)	
Quite good	48.7(97)	48.0(95)	
Average	26.1(52)	34.8(69)	
Quite poor or poor	4.5(9)	8.6(17)	
Diseases % (n)			
Self-reported any chronic disease*	43.2(83)	52.8(102)	0.039[b]
Physician-diagnosed chronic disease			
Hypertension with medication	33.7(67)	40.1(79)	0.12[b]
Angina pectoris	17.2(25)	17.4(26)	0.13[b]
Myocardial infarct	10.6(21)	11.1(22)	0.86[b]
Diabetes	3.5(7)	6.1(12)	0.16[b]
Kidney disease	4.0(5)	2.7(9)	0.74[b]
Ulcer	14.8(20)	17.9(26)	0.84[b]
Asthma	10.4(14)	14.7(21)	0.59[b]
Osteoarthritis	43.5(70)	37.0(57)	0.32[b]
Sports injuries % (n)			
Any sports injury*	26.3(52)	16.3(32)	0.021[b]
Achilles tendinitis*	21.1(37)	6.1(10)	0.003[b]
Achilles total rupture	4.9(8)	1.9(3)	0.21[b]
Mobility % (n)			0.26[a]
No restrictions	84.3(161)	79.4(154)	
No ancillaries	14.7(28)	19.1(37)	
With ancillaries	1.8(2)	1.5(3)	
Functioning of daily living[†]			
Physical (score range 5-20), mean (95% CI)	5.5(5.3-5.8)	5.8(5.5-6.1)	0.211[a]
Physical, good functionality %(n)	98.0(195)	97.5(194)	0.74[b]
Physical, poor functionality %(n)	2.0(4)	2.5(5)	
Psychosocial (score range 4-16), mean (95% CI)	5.2(4.9-5.2)	5.3(5.0-5.5)	0.74[a]
Psychosocial, good functionality% (n)	98.5(196)	98.5(194)	1.000[b]
Psychosocial, poor functionality% (n)	1.5(3)	1.5(3)	
Self-rated mood (BSI-53)[‡]			
Depression, mean (95% CI)	2.1(1.7-2.5)	2.6(2.1-3.1)	0.28[a]
Depression, % (n) with high score (≥8)	6.5(13)	8.5(17)	0.43[b]
Anxiety, mean (95% CI)*	2.1(1.7-2.4)	2.7(2.3-3.1)	0.048[a]
Anxiety, %(n), with high score (≥7)	7.7(15)	10.1(20)	0.59[b]

Working status % (n)			0.67[a]
Employed	15.6(31)	19.6(39)	
On old age pension	57.8(115)	50.8(101)	
On disability pension	13.1(26)	18.6(37)	
Unemployed	4.0(8)	3.0(6)	
Other	9.5(19)	8.0(16)	

* $p<0.05$ for statistical differences between athletes and age-matched brothers by [a)] Wilcoxon`s signed rank -test or [b)] McNemar`s test.

[†]Functioning of daily living (Activities of daily living scales (Mini-Finland Health Survey)):
Physical Two groups: 0= good functioning, score 1 or 2, 1= poor functioning, score 3 or 4 on any item
Psychological Two groups: 0= good functioning, score 1 or 2, 1= poor functioning, score 3 or 4 on any item

[‡]Self-rated mood (Shortened anxiety and depression scales of BSI-53):
Depression Two groups: 0= no depressive, score ≤7.99, 1= depressive, score ≥8
Anxiety Two groups: 0= no anxious, score≤6.99, 1= anxious, score ≥7

Self-rated mood:

The participants were asked to rate their mood at this moment on a 5-point Likert scale of 0-4: never (0), seldom (1), sometimes (2), often (3), and very often (4). So, the range of the sum scores was 0–24. The highest decile of the outcome variable (depression or anxiety) was considered a*n abnormal outcome*, with others classified as not affected for the purposes of analyzing presence of a possible mood disorder. However, the scales are not diagnostic of clinical disease.