MOBILITY OF BODY FAT DISCORDANT OLDER FEMALE TWINS

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ABSTRACT

Background and Purpose of this Study

Obesity is a major health problem around the world. It causes high public health care costs, and several health problems. Physical function enables independence of older people and obesity worsens the age-related decline in physical function, which can lead to frailty and loss of independence. The aim of this study is to find out if the amount of body fat explains physical function difference and development in walking difficulty in three years time.

Material and Methods

This study is part of the Finnish Twin Study on Aging (FITSA). The selection criteria for study subjects included BMI and body fat (%) difference between the twin pairs. The other twin had to be normal weight (BMI 19-26) and the other one over weight (BMI 27 or more). The required body fat (%) difference was 10%. 15 MZ and 28 DZ twins met these criteria. In cross-sectional analysis the comprised groups were normal weight and obese twins. 10 meters and 6-minutes walking tests were used to assess the physical functioning. In the prospective longitudinal analysis the subjects were divided into four categories based on their walking habits and 2km walking abilities. Results were analyzed by using mean, SD, subject numbers and percentages. P-values are based on T-test with 95% confidence level.

Results

The two weight groups had no statistically significant difference in body composition, diseases, physical activity or in walking tests. Among MZ twins the weight group difference in 10m walking time was 0.2s (p=0.657) and in DZ twins 0.3s (p=0.265). In 6min walking test the difference between the MZ twin groups was 8.1m (p=0.754) and between the DZ twin groups 18.8m (p=0.325). The development in walking difficulty was stronger among subjects who had done some adaptation in walking habits in the baseline or had some or more problems in walking. No statistically significant difference between the two weight groups was found in the development of walking difficulty.

Conclusion

This study does not support the general belief that obesity has effect on physical functioning. The weaknesses of this study were that the subjects were relatively in good health and the amount of subjects was quite small. The strength of this study was the powerful study setting that was made possible because of the twin study subjects and objective laboratory measurements used in physical functioning tests. The study should be repeated with less selected and larger group of study subjects, where the twins are more discordant in body fat.

Key Words: Twin Study, Obesity, Body Fat, Physical Functioning, Walking

Fyysinen toimintakyky kehon rasvaprsentin suhteen erilaisilla ikääntyneillä kaksossiskopareilla.

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39 sivua

TIIVISTELMÄ

Tausta ja tarkoitus

Lihavuus on merkittävä terveysongelma maailmassa. Se aiheuttaa korkeita kustannuksia terveydenhuollossa ja monia terveysongelmia. Fyysinen aktiivisuus takaa ikääntyneille ihmisille itsenäisen elämän ja lihavuus lisää iän myötä esiintyvää fyysisen toimintakyvyn heikentymistä, minkä seurauksena riippuvuus toisista ihmisistä saattaa lisääntyä. Tämän tutkimuksen tarkoituksena on selvittää kehon rasvan määrän vaikutusta fyysisen toimintakyvyn eroihin ja kävelykyvyn kehitykseen kolmen vuoden seuranta-aikana.

Aineisto ja mentelmät

Tämä tutkimus on osana FITSA-tutkimusta. Tutkittavien valintaperusteina oli BMI ja kehon rasvaprosenttierot. Toisen kaksonen oli normaalipainoinen (BMI 19-26) ja toinen lihava (BMI 27 tai enemmän). Rasvaprosenttieron piti olla 10%. 15 MZ ja 28 DZ kaksosparia valittiin tutkittaviksi. Poikkileikkausanalyysissa vertailturyhminä toimi normaalipainoisten ja lihavien ryhmät. Kävelytesteinä käytettiin 10 metrin ja 6 minuutin testejä. Prospektiivisessa pitkittäistutkimusanalyysissä tutkittavat jaettiin neljään ryhmään perusten heidän kävelytottumuksiinsa ja 2 km kävelykykyynsä. Tulokset analysoitiin keskiarvoilla, keskihajonnalla, tutkittavien lukumäärillä ja prosenttiosuuksilla. P-arvot perustuvat T-testiin, jossa käytettiin 95% luotamusväliä.

Tulokset

Vertailtavilla painoryhmillä ei esiintynyt tilastollisesti merkitsevää eroa kehon koostumuksessa, sairauksissa, fyysisessä aktiivisuudessa tai kävelytesteissä. Monotsygooteilla 10 metrin kävelytestissä ryhmien välinen kävelyaikaero oli 0.2s (p=0.657) ja ditsygootella se oli 0.3s (p=0.265). 6 minuutin kävelytestissä matkaerot MZ-ryhmillä oli 8.1m (p=0.754) ja DZ-ryhmillä se oli 18.8m (p=0.325). Kävelykyky heikkeni enemmän niillä tutkittavilla, joiden kävelykyky ja tottumukset olivat jo alkumittauksissa heikempia. Tilastollista merkitsevyyseroa ei esiintynyt eri painoryhmien välillä.

Johtopäätökset

Tämän tutkimuksen tulos ei tue yleistä uskomusta siitä, että lihavuudella olisi vaikutus fyysiseen toimintakykyyn. Tutkimuksen heikkoudet olivat, että tutkittavat olivat liian hyväkuntoisia ja heidän määränsä oli pieni. Tutkimuksen vahvuuksia oli vahva tutkimusasetelma joka mahdollistettiin tutkittavilla kaksosilla ja käytetyillä objektiivisilla fyysisen toimintakyvyn laboratoriomittauksilla. Tutkimusasetelma tulisi toistaa monipuolisemmalla ja suurilukuisemmalla tutkittavien joukolla, jotka olisivat enemmän erilaisia rasvaprosentin suhteen verrattuna toteutetun tutkimuksen tutkittaviin.

Asiasanat: kaksostutkimus, lihavuus, rasvaprosentti, toimintakyky, kävely

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1 INTRODUCTION

More and more people are gaining extra weight and obesity is more common around the world. Obesity has increased since 1980's in all age groups. In Finland at the beginning of this millennium, every fifth employee was obese, and the majority of obese people were in the age group of 55 to 64 years. Obesity decreases towards the retirement age, more in men than in women. This is explained by the higher death rate due to the obesity related diseases in men. (Rissanen & Fogelholm 2006.)

Obesity is not only the problem of individuals; it causes high public health care costs every year. It is calculated that 2-7% of the health care expenses in Finland are related to obesity. It also increases the number of early retirements, because of the related diseases. This all could be prevented by effective prevention and care. (Rissanen & Fogelholm 2006.)

The major reason for the increased obesity is lowered level of physical activity. Also the amount of fat that is in the consumed food is one of the explaining factors. The phenomenon has expanded to all social-economic classes; obesity used to be the problem of wealthy people, but nowadays it is facing also the lower economic classes. When we talk about obesity and social-economic class differences, we have to remember that obesity can be either the reason or consequence for the social-economic class status of individual. (Rissanen & Fogelholm 2006.)

Physical functioning plays a major role in independent and disability free life in older people. Walking ability is an essential in physical functioning and is one of the factors of successful aging that is the ultimate goal for all the individuals. Obesity has important functional implications in older persons also because it worsens the agerelated decline in physical function, which can lead to frailty and loss of independence. In fact, obesity may be the single greatest cause of disability in older people. (Launer et al. 1994, Coakley et al. 1998, Himes 2000, Davison et al. 2002.) Changing lifelong habits of nutrition and exercise in older people with short life expectancies may be difficult to introduce and cause distress and anxiety. Such issues should also be considered before establishing rational clinical and public

health policies that aim to improve mortality and morbidity and well-being among older people. (Andrews 2001.)

Twin study is one way to study obesity and its heritability in older people. The classical twin study includes monozygotic (MZ) and dizygotic (DZ) twins. It is a powerful design for estimating genetic effects. This is because twins are always matched with age and therefore age-dependent influences of genes or environmental factors are the same for both twins. (Maes et al. 1997.) Also when studying twin pairs discordant for an exposure, such as body fat difference, it is possible to adjust for genetic factors and other familiar factors such as eating habits. Because of that this study setting allows us to make causal conclusions and it is comparable to randomly selected study sample.

In observational studies it is hard to determine if obesity is the cause of physical activity decline or visa versa. The research data in this study consisted community-living older MZ and DZ female twins that are in relatively good health. The aim of this study is to find out if the amount of body fat explains the difference in physical functioning and development in walking difficulties in three year follow-up time.

2 REVIEW OF THE LITERATURE

2.1 Obesity and Physical Functioning

2.1.1 Body Weight, Physical Activity and Physical Functioning

In older people, body weight, physical activity and physical functioning are closely related. In epidemiologic studies aging (Bijnen et al. 1998) and muscle strength (Skelton et al. 1994, Ferrucci et al. 1997) have been proven to be factors influencing the physical functioning and performance in older people. Other explaining factors are sociodemographic characteristics such as age, gender, race, and educational level (Caspersen & DiPietro 1991, DiPietro & Caspersen 1991). In the rodent studies age-related decline in physical activity is well-established phenomenon. Based on study done by Ingram (2000), locomotor activity was 50% lower in older mice compared to the younger mice. Regular exercise is associated with maintenance of physical performance and functional status (Nelson et al. 1994, Strawbridge et al. 1996, Huang et al. 1998) and visa versa (LaCroix et al. 1993, Huang et al. 1998). Identifying the predictors of functional limitations may promote health of older people and enable successful aging (Davison et al. 2002). This preventive work should be started early enough, because life-long physical fitness and exercise are the key issues of the prevention of chronic, degenerative diseases among older people (Spirduso 1994).

The connection between physical activity and obesity is hard to determine; obesity can be the cause of physical activity decline or visa versa. Physical activity increases the energy expenditure and as long as that is higher than energy intake, the result is loss of body weight. Physical activity seems to be the strongest influence on total-body fat mass after genetic factors (Samaras et al. 1999).

In studies of BMI's effect on physical activity, one research setting is genetically controlled studies of twins that are discordant in physical activity. These studies show that even a gradual body weight gain had effect on activity levels (Swallow et al. 1999). The connection was stronger for women than for men (Williamson et al. 1993); one longitudinal study on adults reported a decrease in physical activity related to

obesity in women, but no relationship between activity and weight was found in men (Klesges et al. 1992). In a twin study done by Samaras et al. (1999) it was found out that among all MZ twin pairs who were discordant for the total activity score (n=164 pairs), the twin with the higher activity level had significantly lower total-body fat mass (24.50±0.57kg and 26.01±0.68kg; p=0.013) and body fat percentage (38.1%±0.5% and 29.3%±0.6%; p<0.001) than the twin with lower activity level. Brownlow at al. (1996) proved that obesity is related to decreased physical activity among people in all ages, but it is less known how this is among the obese older people (age over 55) (DiPietro 1995).

The hypothesis that obese people have more problems related to physical activity compared to the leaner counterparts has been studied among humans and also in animal studies. In the study by Coakley et al. (1998) (n=69902) they found out that statistically significant lower functioning became apparent at higher levels of BMI (25-27.9). Level of physical functioning declined with higher BMI and the subjects who were extremely overweight had average 14-16% lower physical functioning level compared to the normal weight reference group.

Animal studies have been based on the hypothesis that body weight regulates the physical functioning and that aging rodents and humans have been observed to gain weight (Newby et al. 1990). Genetically obese and lean rodents provide a powerful model for studying age-weight-activity relationship because it is possible to have same age rodents that differ in weight in wide range. Ahima et al. (1999) reported that at 4–5 weeks old mice who gained weight by 25% showed no significant effect on locomotor activity. At 10 weeks these same study mice gained body weight by 80% and locomotion decreased by 50%. The same kind of setting is used in several other rodent studies and the results showed that obese rodents exhibit 51–70% less locomotor activity compared with lean mice, suggesting that body weight restricted physical functioning (Clark & Gay 1972, Borer & Kaplan 1977, Dauncey & Brown 1987).

Physical activity level was found to be related to the body weight in Australian study (Ball et al. 2001) (n=2298, aged 18-78 years). Compared with sedentary women, those with higher activity were 1.7 times (p=0.06), those moderately active were 2.3

times, and those highly active, 2.6 times more likely to have a BMI in the normal range. Among Finnish women Rissanen et al. (1991) showed that the ones who exercised regularly weighted 1,3kg less compared to the more sedentary subjects.

Self-reported activity studies have also given results proving the benefits of exercise: Samaras et al. (1999) reported that the physically active subjects had lower BMI (BMI 23.9 ± 0.2 and BMI 24.7 ± 0.2 , p=0.004), total body fat mass (23.4 ± 0.4 kg and 25.8±0.4kg; p<0.001), and central abdominal fat mass (1.40±0.04kg and 1.66±0.04kg; p<0.001) and also greater muscle mass strength (94.4±2.4W and 80.0±1.9W; p<0.001) compared to the reference, more sedentary subjects. This supports the earlier proven fact that benefits from exercising do not only limit to weight loss (Blair et al. 1989, Manson et al. 1991). Physical activity improves muscle strength and muscle mass (Roubenoff 2000) and has a key role in the management strategy for obesity; even without significant body composition modifications, physical activity improves the physical performances of obese subjects (Sartorio et al. 2001). It has also beneficial role maintaining the everyday tasks among older people. This was proven in the study by Voorrips et al. (1993) study (n=50 older women) where the subjects who were more active on occupational, home maintenance and leisure-time activities had significantly lower body weight and BMI, better flexibility of the hip and spine, and endurance on a walk test compared to the sedentary subjects.

2.1.2 Obesity Development and Measurement

Body composition changes are part of aging process. The amount of body fat mass increases and the amount of muscle mass decreases with age (Baumgartner et al. 1995, Gallagher et al. 1997). Older people have greater fat mass in the same BMI compared to the younger individuals, because fat replaces the muscle mass with age (Baumgartner et al. 1995) and the percentage of body fat is higher in older people, even if weight is not (Gallagher et al. 2000a). Sarcopenia is one of the major causes to decreased strength in older people (Newman et al. 2003). These observed changes may cause the onset and progressions of disability in old age (Baumgartner et al. 1995, Gallagher et al. 1997). Obesity is common in the population reaching retirement age, but the BMI level descends usually around 70-75 years of age (Räihä 2005). Based on the study in Finnish population, the increasing BMI levels in lifetime

peaks around 60 years and after that declines (Waaler 1988). The increase in mean BMI of population is not only caused by weight gain of older people, it is also due to the fact that current younger cohorts are heavier compared to the earlier ones (Schousboe et al. 2003). This increasing prevalence of obesity is caused by reductions in total energy expenditure brought by the sedentary behaviour patterns associated with technological and social changes (Prentice & Jebb 1995).

Obesity is a result of positive energy balance. The components of the balance are energy input (nutrition) and energy output (physical activity). When energy input equals energy output, body weight remains constant. However, imbalances resulting in a cumulative positive energy balance leads to weight gain and possibly obesity. (James 1992.) Most of the extra energy from food accumulates in body as fat and its energy content is 37,6 MJ/kg (9000 kcal/kg) (Uusitupa & Fogelholm 2005). The prevention and/or treatment of obesity will require a reduction in energy intake and increased energy expenditure (Tou & Wade 2002). The difference in body weight between two subjects is explained by the amount of body fat and muscle mass in the body. (Uusitupa & Fogelholm 2005.)

Obesity can be measured with several different ways. The main challenge with the measurement is the fact that it cannot be measured exactly and by using only one type of measurement. At least two different kinds of body composition features need to be measured in order to have realistic estimation of body composition. (Fogelholm 2006.)

The most common marker of obesity is body mass index (BMI) (Janssen et al. 2004, Wannamethee et al. 2004, Bigaard et al. 2005, Fogelholm 2006). BMI is developed by Belgian mathematician Adolphe Quetelet and is defined as the body weight divided by the square of the height (Body Mass Index 2006). Based on classification by WHO (1998), the BMI scale is divided into five segments: the underweight (BMI <18.5), the normal weight (BMI 18.5-24.9), the overweight (BMI 25.0-29.9), the obesity I (BMI 30.0-34.9) and the obesity II (BMI 35 or higher).

In clinical setting BMI is commonly used to asses the normality of body weight of individual patients (Bouchard 1991). Reasons for this are the easiness and guickness

of the measurement. BMI is an indicator of heaviness and only an indirect indicator of body fat (Garn et al. 1986). It does not differentiate the amount of fat and muscle mass from each other. (Fogelholm 2006.) Research reveals that the relationship between BMI and body fat may vary by age, gender and ethnic group (Gallagher et al. 2000b). This is relevant in gerontological studies, because of the changes in body composition and decline in height that appears with age (Fabsitz et al. 1994). The WHO's five segments classification is most suitable for 20-60 years old people (Fogelholm 2006).

Since the measurement of body composition is so complicated, there has been controversy as to whether BMI is an adequate marker of obesity-related problems and whether indicators such as waist circumference or body fat percentage should be used instead (Harris 2002, Janssen et al. 2000). The percentage of body fat is the most appropriate and used indicator of the body composition and therefore also for obesity (Bouchard 1991). It needs to be remembered that results given by different ways of measurements are not comparable with each other (Fogelholm & Uusitupa 2005).

Another efficient way to measure body composition is to measure waist circumference. This is important knowledge because the fat that accumulates around the waist area (visceral fat) and it has a major role in the development of metabolic disorders. According to the WHO guidelines the serious health risk is indicated 102cm in men and 88cm in women. The mild health risk levels are 94cm for men and 80cm for women. (Fogelholm 2006.) The recommended value for waist circumference is less than 90cm in men and less than 80cm in women (Uusitupa & Fogelholm 2005). There is a linear association between BMI and waist circumference, but for older people this should not be used as measurement of obesity, since the recommendation levels are only for people aged 60 years or younger (Fogelholm 2006).

The waist-hip ratio is more commonly used to estimate the type of obesity. It is calculated by dividing the waist circumference by the hip circumference. The higher number is related to the higher risk for health problems; increased health risks increase when the ratio is over 1.0 for men and 0.85 for women. There is no health

risk present if the ratio is for men 0.9 or less and for women 0.8 or less. (Uusitupa & Fogelholm 2005.)

The bioelectric impedance (BIA) is widely accepted way to assess body composition. It measures the body's capability to conduct electricity. The level rises when the amount of outer cell fluid increases. This method measures the amount of body fluids that the obese people have less in their body compared to the leaner counterparts. The measurement is quick and easy to perform. (Fogelholm & Uusitupa 2005.)

2.1.3 Effects of Obesity on Health

The link between BMI and different chronic conditions has been found in several studies. Excess weight is associated with increased incidence of stroke, dyslipidemia, osteoarthritis, some cancers (Burton et al. 1985) and also hypertension, type II diabetes, and cardiovascular diseases (Burton et al. 1985, Flegal et al. 2002).

Must et al. (1999) found out in their cross-sectional survey (n=16 884, 25 years and older) that the association between BMI and chronic diseases was present in all age groups in obese men and women. Among those in the most obese group, the prevalence ratio of chronic diseases for younger men was 18.1 (95% CI, 6.7-46.8) and 12.9 (95% CI, 5.7-28.1) for younger women compared to the normal weight reference group. Among older men and women, the association was more moderate but still substantially elevated, with the prevalence ratio of 3.4 (95% CI, 1.1-8.3) for the most obese men, and 5.8 (95% CI, 4.2-7.4) for the most obese women. In separate chronic condition analysis, there was steeply graded association for high blood pressure, high blood cholesterol levels and type II diabetes with obese subjects.

In the study by Ramsey et al. (2006) (n=7735 men aged 40–59 years) the odds of reporting fair/poor health increased with higher BMI, waist circumference, and fat mass index levels. The odds of cardiovascular disease increased with fat mass index and BMI levels (p<0.001). Subjects in the fourth and top fifth of fat mass index were 33% and 58% more likely to have cardiovascular diseases compared with those in the bottom fifth of fat mass index. These results suggest that increasing fat mass, BMI, and waist circumference are associated with physical disability, ill health

(cardiovascular diseases, diabetes, taking musculoskeletal medication, and reporting fair/poor health), and metabolic risk factors, that are, hypertension, low HDL-C, and insulin resistance (high homeostasis model assessment).

The association of body weight and mortality in older people has been described mostly by using U- and J-shaped curves. This means that individuals with extremely low and high BMI are at increased risk of death compared to the normal weight individuals. (Waaler 1988, Manson et al. 1995, Stevens et al. 1998, Seidell & Visscher 2000.) Stevens et al. (1998) observed that BMI increase had lower effect on mortality at older than at younger ages.

The relation between BMI and mortality (n=121700, 30-55 year old women) was examined by Manson et al. (1995). The main result was that the body weight is an important determinant of mortality among middle-aged women. The mortality did not increase until the BMI reached 27, which was explained by coronary heart disease and other cardiovascular diseases, as well as cancer. The weight in earlier life seemed to be a predictor of mortality in later life. Weight gain of 10kg or more since the age of 18 was associated with increased mortality in middle adulthood. This study did not find any U- or J-shaped curve between BMI and mortality, because the lowest mortality was among the omen who were underweight (those with BMI <19).

2.1.4 Disability

Various studies have shown that high and very low BMI and the amount of fat mass are increasing the risk of disability (Galanos et al. 1994, Launer et al. 1994, Dutta & Hadley 1995, Visser et al. 1998). There are limitation in activities of daily living (Launer et al. 1994), walking stairs and on flat surfaces (Visser et al. 1998); increased risk for pulmonary disease, diabetes and arthritis (Anderson & Felson 1998) and decreased mobility (Harris et al. 1989, Ma et al. 1998, Zamboni et al. 1999, Friedmann et al. 2001). Most of the studies are done by using middle aged study subjects and less attention has been given to older people (Elia 2001).

In a longitudinal study (3 years) done by Visser et al. (1998) (n=4809, aged 65-100 years) showed that fat mass was positively associated with disability (p<0.0001) and

had negative effect on the maintenance of muscle strength. The study subjects in the highest quintile of fat mass were three times more likely to be disabled than those in the lowest quintile. In the three year follow-up the risk ratio was the same as at the baseline. The odds ratio was 2.83 (95% CI: 1.80, 4.46) for women and 1.72 (95% CI: 1.03, 2.85) for men.

The reasons that obesity and underweight are related to disability are results of several different mechanisms. Obesity in adulthood may lead to disability because of one or more biological processes: skeletal stress (Hart & Spector 1993), protein glycation in connective tissue (Supiano et al. 1993, Pratley et al. 1995), or atherogenesis (Stevens et al. 1993). Some evidence indicates that obesity is associated with a greater risk for both lower-body (Felson et al. 1987, Felson 1996) and upper-body osteoarthritis (Carman et al. 1994) leading to disability.

Stewart & Brook (1983) (n=2756 families) found out that the amount of limitations in personal functioning increased as weight increased. Compared with 19% of normal-weight people, about 28% of moderately overweight-people (p<0.01) and 34% of obese people (p<0.01) had some limitations in personal functioning. This difference could be explained partly with high body weight associated problems such as diminished exercise tolerance and social or psychological disadvantages, which constitute a severe and mostly hidden burden of obesity.

Rissanen et al. (1990) studied the employees (aged 25-64 years) in Finland (n=12053 women, 19076 men) finding out that the risk of work disability increased linearly with increasing BMI in both women and men. The association for overweight women was 2.0 (1.8-2.3) and for men was 1.5 (1.3-1.7). These results confirmed that even modest overweight predicted significantly premature functional impairment of the cardiovascular and locomotor systems.

It has been proven that the association between BMI and disability is stronger among women (LaCroix et al. 1993), especially in older ages (Launer et al. 1994). Davison et al. (2002) showed (n=1391 men, 1526 women, aged 70 and older) that women in the highest quintile for percentage of body fat were twice (PR=1.82) as likely to the report functional limitations compared with women in the lowest quintile. For BMI

category, women in the underweight, overweight, and obese categories were significantly more likely to report functional limitations than women in the normal-weight category. In particular, women in the underweight and the obese II categories were twice as likely to report functional limitations (underweight PR=2.03; obese II PR=2.31). Among men, the ones in the highest quintile for fat mass were 1.5 (PR=1.62) times more likely to report functional limitations than men in the lowest fat quintile. These relations between body composition and functional limitations suggest that a high percentage of body fat and high BMI are related with greater functional limitations among women. Among men, the pattern is less clear.

Friedmann et al. (2001) examined further the gender differences in functional limitations. Based on the results women had higher rates of functional limitations within all BMI categories than men and the BMI value at which risk increased was lower for women than for men. Both men and women with BMI ≥40 had significantly increased risk of functional limitations. Among women, risk appeared to increase for those categorized as obesity I (ROR=1.27, 95% CI: 0.86–1.88) and obesity II (ROR=1.55, 95% CI: 0.94–2.55). For men the result was opposite, the risk of functional limitations was decreased for those categorized as overweight (ROR=0.64, 95% CI: 0.42–0.96) and obesity I (ROR=0.55, 95% CI: 0.32–0.94).

The possible explanations for the gender differences are that women are often overrepresented in the highest BMI categories (Jensen et al. 1997, Flegal et al. 1998) and women have in average lower levels of physical activity compared to men. In addition, women have more body fat at any given BMI or percentage of fat quintile than men. Finally, differences in findings for men and women may also reflect the fact that women tend to overreport, whereas men underreport functional limitations (Merril et al. 1997).

2.1.5 Muscle Mass and Strength

Studies of basic physiology suggest that the muscles of obese persons had specific metabolic and histological characteristics (Krotkiewski & Bjorntorp 1986, Lillioja et al. 1987, Marin et al. 1994, Kriketos et al. 1996, Tanner et al. 2002). Hulens et al. (2001) studied healthy obese women (BMI >30) and normal weight women aged 20-65

years (n=223) reporting that obese women had lower muscle strength than lean women had. Absolute maximal isometric handgrip strength data tended to be higher (no statistical significance, p=0.1) in lean women when compared to the obese women. This study suggests that overall muscle function is impaired in obese women compared to their normal weight women, even when the physical activity is controlled for. Muscle impairment in obese persons could be related to metabolic changes or to changes in muscle architectural components, such as fat infiltration.

There are several other studies that also suggest that muscle strength is affected in obesity. Samaras et al. (1999) studied the muscle strength in different physical activity levels and the results showed that the muscle strength was greatest in the highest tertile of physical activity (90.2±2.7W) compared to the lowest (76.5±2.6W) and middle (81.4±2.3W) tertiles (p=0.01). And since the physical activity decreases with higher BMI, the muscle strength also decreases with higher BMI.

Newman et al. (2003) studied 2623 men and women in a cross-sectional analysis to describe muscle strength in relationship to age and body composition. Age and higher body fat were inversely associated with arm strength. Although the differences associated with age were small, they translated to a 10% lower leg muscle strength in men and 11% lower leg muscle strength in women across the age 70 to 79 years.

Rolland et al. (2004) noted that there are important limitations in the studies that comparing muscle strength between obese and lean subjects. There are many factors other than BMI or fat mass that can influence the strength performance. Osteoarthritis, knee and hip pain, the use of many medications, and co-morbid diseases are very common in obese subjects and may induce lower strength performances. Their study among older subjects (mean age 80.4±3.9 years) showed that the obese women had significantly more fat mass and all the anthropometric measures (except height), fat-free mass, total muscle mass, leg muscle mass and arm muscle mass were significantly higher than normal-weight and lean women had. Except for handgrip strength, absolute strength measures were significantly higher in the obese women than in the lean women. The obese women reported significantly less recreational physical activity than did the lean women or those with normal BMI. This was found to be a significant determinant of muscle strength in the older women

irrespective of BMI, especially among obese women. This significant interaction suggests that for the active women, lower limb strength increased with increasing BMI. All in all it can be said that the present study did not find a trend toward decreasing muscle strength with increasing BMI.

2.2 Heritability in Physical Functioning and Obesity

2.2.1 Physical Functioning

There are factors such as individual differences and also environmental and agerelated decline that effect on the physical functioning of an individual. (Ingram 2000). The effect of heritability to physical functioning is studied very little. There are some studies that have proved that genes have some effects on the physical functioning. Perusse et al. (1989) studied this with 1610 subjects from 375 families from whom they collected a three-day activity record. The results of this study showed that there was a high correlation between the physical functioning between parents and their children. When they compared this within sex (father vs. sons, mothers vs. daughters) it reached significance level only between mothers and daughters (p<0.01). Overall this study showed that there was a genetic effect of 20-29% for level of habitual physical functioning. Based on Bouchard et al. (1988a) training response due to exercise was up to 77% influenced by the genotype.

2.2.2 Obesity

There are several factors that effect on the development of obesity. Some of it can be explained by the environmental factors and lifestyle choices, but most of it can be explained by heritability; different genes are responsible of the basis of obesity. However the possibility that genetic factors could explain all of the increasing prevalence of the obesity phenomenon is impossible (Hill & Peters 1998). Variation in excess weight- for height or body fat is caused by complex network including genes (Bouchard 1991, Hill & Peters 1998) and also balance in energy intake and energy expenditure (Hill & Peters 1998). That is why there are also several environmental factors that have an effect on the development of obesity. Bouchard (1991) proved in his study that behavioural and lifestyle factors had an effect on the development and maintenance of obesity. Also age, education, ethnicity, and chronic illness or disease, smoking history, and estrogen replacement therapy have previously been linked to percentage of body fat, muscle mass, and BMI (Kuczmarski 1989, Sobal & Stunkard 1989, Perrone et al. 1999). Obesity can be also caused by dysfunction of

mechanisms that control eating, feeling of hunger or fullness and usability of different energy yielding nutrients or differences in energy metabolism (Uusitupa 2005).

There are two different kinds of obesity explained by genes: monogenetic and polygenetic obesity. Monogenetic obesity is more common in rodents compared to humans. When that is found in humans, there is a very complex and heavy obesity present already in childhood. Polygenetic obesity means that there are several mutated genes or polymorphisms that causes obesity or exposes the body for it. (Uusitupa 2005.)

Energy metabolism covers approximately 70% of all energy expenditure. There are differences in it between individuals that cannot be explained by fat free mass, fat mass or by age or gender. It is known that low energy metabolism can be one of the exposure factors for obesity and because of that the research of the genes that effect on the energy metabolism is active today. (Uusitupa 2005.)

It is very important to study the genetics of obesity phenotypes, because they are associated with a high risk for various morbid conditions and mortality rate (Bouchard 1991). The research results have given wide variety of results on how much genotype explains obesity, but it is known that approximately 40–70% of obesity-related phenotypes such as BMI, skin fold thickness, fat mass, and leptin levels are heritable (Allison et al. 1996, Comuzzie et al. 1996).

The three most common settings to study the heritability of obesity are family, adoption and twin studies. In family studies the comparison is done between family members, in adoption studies between the adoptee and their biological parents versus their adoptive parents and in twin studies between the twin pairs. These three types of studies have given information about energy and energy yielding nutrient expenditure and also about eating habits. (Uusitupa 2005.) One of the largest and most published family studies is Quebec Family study (n=1698). Based on it, the estimated weight correlations were 0.23 for parent-offspring, 0.26 for siblings and 0.10 for spouses. (Bouchard et al. 1988b.)

The adoption study setting gives the change to study the environmental and genetic factors when adoptees are compared with their biological and adoptive parents and siblings. In most studies it is found out that the genetic factors overruled the environmental factors and very rarely there had been any correlation in weight between the adoptee and the adoptive parents. (Uusitupa 2005.) For example in Danish study done by Sorensen et al. (1992) they found out that the heritability correlations in weight in adoptees were lower with their adoptive parents (0.03-0.10) compared to the biological parents (0.16-0.17). The same kind of results came out it another Danish study done by Stundgard et al. (1986). They studied adult adoptees (n=540) trying to find out the relation of genetic factors and the biological family environment to obesity. With mothers and children this correlation was statistically significant (p<0.0001), but with fathers and children the correlation did not reach the significance level (p<0.02). When they compared the daughters with mothers, the correlation reached the significance level (p<0.0001), but with sons and mothers this connection was not present.

The twin studies are based on the fact that MZ twins have exactly the same genotype and DZ twins have 50% of the same genotype with each other. If the environment is the same for the twin pairs, the weight differences between MZ and DZ twins are due to the genetic differences. The differences in body composition between MZ twins are much less apparent than in DZ twins. (Uusitupa 2005.) The twin design in obesity studies has been used extensively when genetics of obesity has been studied. Most of the twin studies have found evidence for the genetic factors on obesity. (Maes et al. 1997.)

Study done by Fabsitz et al. (1994) used 121 MZ male twin pairs and 113 DZ male twin pairs aged 59-70 years old to find out if there is a genetic influence on the weight development with age. Based on the study subjects the majority of twins attained their maximum weight around age 60 years with a smaller proportion (25%) attaining their maximum weight around age 20. Genes estimated to account for 70% of the variation in maximum lifetime BMI and adult weight gain. These results indicated that the development and extent of obesity in adulthood are under significant genetic control (heritability correlation 0.71).

Research has proven that there are major differences between individuals in nutrient partitioning and also where and in which form this excess energy is been stored in the body. The type of person whose excess energy is been stored in the fat cells is more likely to gain extra weight compared to the individuals whose excess energy is stored as lean body mass such as muscle or in internal organs. (Uusitupa 2005.) Bouchard et al. (1990) studied the heritability of nutrient partitioning. In that study they overfed (extra 1000 kcal/day) MZ male twins (n=24) during a 100 day period. The data showed that there were considerable interpair differences in the adaptations to the excess calories: at least three times more variation in response between pairs than within pairs for the gains in body weight and fat mass was shown in this study. This shows that nutrient partitioning is one of the most important factors to explain the individuality in body mass gain.

3 RATIONALE FOR THE STUDY AND RESEARCH QUESTIONS

The relation of obesity and physical functioning has been the topic of several studies. Most of them have had younger study subjects, twin study setting has been used only in few of them and they have concentrated on the correlation between body weight and physical functioning level.

Heritability is a strong explaining factor to the individual differences of obesity and physical functioning. The genetic factors explain 60-70% of the individual differences in obesity and 20-50% of the differences in physical functioning. In this study the study subjects were MZ and DZ twins. This let us to adjust the genetic and familial factors between the study subjects. This means that the individual differences in the amount of body fat and physical functioning is explained by other factors than adjusted factors. With this strong study setting we are able to find out if there is a causal connection between body fat and physical functioning and in development of it in three years of time.

The research questions for this study are:

- 1) Do the different body weight groups have different health and physical functioning background?
- 2) Do the different body weight groups differ in walking speed in 10m walking test?
- 3) Do the different body weight groups differ in walking endurance in 6min walking test?
- 4) How does the physical functioning of different weight groups develop in three year follow-up?
- 5) Are there differences in these results between the MZ and DZ twin groups?

4 MATERIAL AND METHODS

4.1 Subjects

The study is part of the Finnish Twin Study on Aging (FITSA) that has been carried out in Jyväskylä, Finland. The participants were recruited from the Finnish Twin Cohort (Kaprio et al. 1978, Kaprio & Koskenvuo 2002) which comprises all same-sex twin pairs born before 1958 and both co-twins alive in 1975, making altogether 13 888 twin pairs. Four hundred and fourteen (414) twin pairs, aged 63-76 years received an invitation to participate in the FITSA study. The final sample consisted of 103 monozygotic (MZ) and 114 dizygotic (DZ) twin pairs. (Pajala et al. 2007). On presenting at the laboratory, participants provided written informed consent. (Pajala et al. 2005.)

There were two selection criteria for the study subjects in this study. These were (1) BMI and (2) the relation of body fat (%) between the twin sisters. Both of these criteria needed to be present in order to be selected into the research subject group.

The selection criteria "BMI" was divided into two categories: normal weight (BMI 19-26) and obese (BMI 27 or more). The selected twin pairs needed to represent oth of these BMI categories in order to be selected into this study. This means that the other sister needed to be normal weight and the other one obese.

The other selection criteria "the relation of body fat between the twin sisters" needed to be 10% or more. This number was calculated by dividing the difference of the body fat (%) of the twin sisters by the body fat (%) of the leaner sister. For example twin pair where the twins' body fat percentages were 15% and 25%. The difference of these was (25%-15%) 10. This result was divided by the leaner sister's body fat (%) = (10 / 15)*100=66%. This example twin pair would have been selected into the study sample, because it fitted into the requirements of the study subjects.

There were 21 MZ and 33 DZ twin pairs who fit into these two selection criteria. Altogether 11 pairs (MZ n=6, DZ n=5) of the initial study group were excluded from the analyses because data was missing for one of the sisters in both walking speed

and the endurance test, or because data was missing for both sisters in the walking tests. Finally the number of pairs in the analyses was 15 MZ and 28 DZ.

4.2 Physical Performance Tests

The two different physical performance tests were selected for this study: 10 meters walking speed test and 6-minute walking endurance test, because walking ability is one of the main indicators of independence and physical functioning in older people. Maximal walking speed over 10 meters was measured in the laboratory corridor. Participants were instructed to "walk as fast as possible, without compromising your safety." Three meters were allowed for acceleration. Timing was done using photocells. Participants wore walking shoes or sneakers, and use of a walking aid was allowed if needed. Maximal walking speed was tested twice, and the faster performance was documented as the result. During each test, an examiner walked behind the participant to ensure safety. (Pajala et al. 2005.)

Walking endurance was assessed using a validated 6-minute walking test (ATS statement 2002). The participants were requested to walk up and down a 50-meter indoor straight track for 6 minutes and to complete as many laps as possible. The standardized protocol and security conditions followed the American Thoracic Society Statement (ATS statement 2002). The distance covered by the end of the 6 minutes was recorded as the outcome. (Ortega-Alonso et al. 2006.)

The development of walking difficulty was measured by using the self-reported questionnaire from the baseline and from the three year follow-up study. The used questions for this analyze were (1) if they had problems in 2km walking and (2) if they had done any changes in their walking habits compared to the past. Based on these answers the group division was done.

4.3 Methods

All study subjects filled out a standardized questionnaire and took part in a clinical examination. Information on diseases, subjective health and functional statuses was gathered by the questionnaire. After that a physician ascertained disease status

during a clinical examination. Body height and weight were measured in the laboratory using calibrated scales. Body mass index (BMI) was calculated by dividing body weight by the squared body height in meters. (Pajala et al. 2005, Ortega-Alonso et al. 2006.) Other body composition measures were also measured in the laboratory. The body fat (kg) and lean body mass (LBM) were measured by bioimpedance. All the information that is based on the standardized questionnaire is self-reported information.

For the prospective longitudinal study setting the three year follow-up study results were used. The study subjects were divided into four different groups in order to analyze the development of walking difficulty in three year time. Information for this classification was taken from a questionnaire that study subjects filled out prior the baseline measurements and in three year follow-up study. The criteria for the division of the four groups were (1) no difficulty in 2km walking, no adaptation in walking habits, (2) no difficulty in 2km walking, some adaptation in walking habits, (3) some difficulty in 2km walking, (4) great deal of difficulty in 2km walking or unable to walk. For this analyze one MZ and three DZ twin pairs were disqualified, because of either death during the follow-up period or because the follow-up data was missing for one sister in used questionnaire. These disqualified study subjects were from all the physical activity groups. The final numbers for this analyze was 14 MZ and 25 DZ twin pairs.

4.4 Statistical Analysis

The statistical analysis was done using SPSS 14.0 program. Means and standard deviations (SD) were calculated from continuous variables and from grouping variables the subject numbers and percentages were calculated. T-test was used to describe the statistical significance (p-value) with 95% confidential level.

5 RESULTS

5.1 Descriptive Variables

Statistically significant difference (p≤0.05) appeared in several physical characteristics groups. Obviously the difference was in all groups in body mass (kg), body fat (kg and %) and BMI, because these characteristics formed part of the selection criteria for this study. Also statistically significant difference (p<0.001) was found in DZ twin groups in lean body mass (LBM kg) and waist-hip ratio. Subject characteristics and differences in body composition between normal weight and obese and also between MZ and DZ groups are presented in more detail in Table 1.

TABLE 1. Physical characteristics of 15 monozygotic and 28 dizygotic 63-74 years old twin pairs discordant in body fat, mean (SD)

	MZ normal mean (SD) n=15	MZ obese mean (SD) n=15	p-value*	DZ normal mean (SD) n=28	DZ obese mean (SD) n=28	p-value*
Age (years)	68.3 (3.2)	68.3 (3.2)	1.000	68.0 (3.0)	68.0 (3.0)	1.000
Body height (cm)	157.9 (8.7)	158.2 (8.1)	0.931	159.4 (7.2)	159.9 (5.5)	0.758
Body mass (kg)	61.6 (6.2)	72.0 (9.2)	0.001	60.3 (5.6)	76.1 (10.0)	0.000
LBM (kg)	44.5 (5.0)	46.6 (3.9)	0.206	43.1 (3.1)	47.4 (4.3)	0.000
Body Fat (kg)	17.2 (3.8)	25.5 (8.2)	0.001	17.0 (4.3)	28.4 (6.6)	0.000
Body Fat (%)	28.0 (5.2)	34.9 (6.6)	0.004	28.0 (5.3)	37.0 (3.8)	0.000
ВМІ	25.7 (3.9)	28.9 (4.2)	0.041	23.7 (2.2)	29.8 (3.7)	0.000
Waist-Hip Ratio	0.83 (0.05)	0.86 (0.07)	0.123	0.83 (0.06)	0.86 (0.05)	0.044

^{*} T-test based p-value

5.2 Physical Health and Activity

The results showed that there is no difference in exercising, self-reported health status or physical functioning between the two weight groups and also between MZ and DZ twins. The only statistically significant difference in physical health and

activity was found in weight loss in the past three years and also in arthrosis in lower joints. There were more subjects from obese groups (MZ and DZ) who had lost over 5kg body weight in the past three years. This proves that the subject who were obese in this study have been obese for a longer time and it is not a new phenomenon or recently happened body weight gain. Physical health and activity results are presented in Table 2 in more detail.

TABLE 2. Weight loss, physical exercise level, self-reported health and chronic diseases in 15 monozygotic and 28 dizygotic 63-74 years old twin pairs discordant in body fat.

	MZ normal n=15 n (%)	MZ obese n=15 n (%)	DZ normal n=28 n (%)	DZ obese n=28 n (%)
Over 5kg body weight lost (past 3 years)	2 (13)	4 (27)	1 (4)	12 (43)
Regular Exercising	14 (93)	12 (80)	25 (89)	25 (89)
Good or excellent physical functioning	14 (93)	12 (80)	27 (96)	27 (96)
Good or excellent health status	14 (93)	14 (93)	28 (100)	28 (100)
Daily Smoking	1 (7)	0 (0)	2 (7)	1 (4)
Cardiac Problems	1 (7)	5 (33)	11 (39)	8 (29)
Cardiac insufficiency symptoms in exercise	2 (13)	4 (27)	5 (18)	6 (21)
Lung Problems	0 (0)	1 (7)	3 (11)	6 (21)
Lung insufficiency symptoms in exercise	4 (27)	3 (20)	9 (32)	11 (39)
Ischaemic problems	1 (7)	2 (13)	0 (0)	1 (4)
Arthrosis in lower joints	3 (20)	6 (40)	5 (18)	9 (32)
Arthritis	0 (0)	0 (0)	0 (0)	0 (0)
Type II Diabetes	1 (7)	0 (0)	0 (0)	1 (4)

All study subjects were able to walk outside. Among the MZ twins there were no major differences in walking abilities or in physical activity: the two weight groups in MZ twins were quite similar. There were some differences between the weight groups of DZ twins. The most significant difference was shown in 2km walking and the frequency doing it. Only 18% of the normal weight DZ twins reported having

problems with 2km walking and 29% of the obese reported problems with 2km walking.

When the subjects reposted their physical activity in their lifetime, it was interesting to see that all subjects independent of the weight group have been equally active. Subjects in the obese groups have been as active or even more active as the normal weight subjects both in MZ and DZ twins.

5.3 Walking Tests

The study results show that the difference between the normal weight and obese study subjects in any of the walking test results were not statistically significant. Walking habit results, such as walking kilometres in week, is based on the self-reported standardized questionnaire that the study subjects filled out in the baseline study. The results shows that the normal weight subjects walk more compared to the obese counterparts both in MZ and DZ groups. In MZ twins the normal weight group walked 2.3km more than the obese group (p=0.424). In the DZ twins the normal weight subjects walked 2.5km more than the obese counterparts (p=0.385).

In the 10 meters walking test the walking time was slightly (0.2-0.3s) slower in both MZ and DZ obese groups compared to the normal weight groups. The difference between two weight groups was not statistically significant.

In the 6-minute walking endurance test the most unexpected results was the MZ twin walking distance result. The normal weight group walked 494.2 meters (SD 79.8) and the obese group 502.3 meters (SD 59.7, p=0.754). Even if the obese subjects walked more in 6 minutes, it cannot be said that their endurance is better compared to the normal weight group, since the difference was not statistically significant. In the DZ twins the normal weight subjects walked 552.4 meters (SD 68.7) and the obese weight group walked 533.6 meters (SD 72.8, p=0.325). Results from the walking tests are presented in Table 3 in more detail.

TABLE 3. Walking, 10m and 6min walking test results in 15 monozygotic and 28 dizygotic 63-74 years old twin pairs discordant in body fat, mean (SD)

	MZ normal mean (SD) n=15	MZ obese mean (SD) n=15	p-value*	DZ normal mean (SD) n=28	DZ obese mean (SD) n=28	p-value*
Walking (km/week)	12.1 (7.6)	9.9 (7.5)	0.424	15.4 (13.1)	12.9 (8.0)	0.385
10 meter test						
Walking time (s)	5.9 (1.1)	6.1 (0.9)	0.657	5.6 (1.1)	5.9 (1.3)	0.265
Heart rate after testing	87.5 (10.6)	88.9 (18.2)	0.799	91.1 (14.3)	93.4 (16.2)	0.578
6 minute test						
Heart rate before test	74.5 (12.2)	73.0 (14.3)	0.755	76.0 (11.3)	78.8 (14.8)	0.403
Walking distance after 1 min (m)	87.7 (11.3)	87.3 (11.2)	0.936	94.6 (11.5)	92.5 (13.0)	0.517
Heart rate after 1 min (times/min)	100.7 (12.7)	101.5 (14.6)	0.884	108.1 (18.2)	108.6 (14.6)	0.967
Walking distance after 2 min (m)	172.7 (23.1)	171.3 (21.7)	0.872	189.5 (26.1)	181.4 (25.5)	0.247
Heart rate after 2 min (times/min)	106.7 (14.2)	109.4 (16.5)	0.631	116.2 (18.9)	117.5 (16.2)	0.779
Walking distance after 3 min (m)	256.7 (34.6)	255.0 (31.3)	0.891	279.1 (35.1)	270.2 (37.4)	0.361
Heart rate after 3 min (times/min)	107.3 (17.3)	111.1 (15.9)	0.536	118.6 (20.3)	119.4 (17.6)	0.889
Walking distance after 4 min (m)	339.0 (46.1)	336.7 (40.4)	0.884	371.1 (46.7)	358.0 (49.1)	0.313
Heart rate after 4 min (times/min)	106.9 (16.6)	110.7 (15.6)	0.524	119.4 (20.5)	119.8 (18.0)	0.945
Walking distance after 5 min (m)	423.3 (58.1)	418.7 (49.8)	0.815	462.7 (58.8)	445.7 (60.7)	0.293
Heart rate after 5 min (times/min)	106.9 (17.5)	110.6 (15.4)	0.548	121.2 (23.1)	120.1 (18.0)	0.847
Walking distance after 6 min (m)	494.2 (79.8)	502.3 (59.7)	0.754	552.4 (68.7)	533.6 (72.8)	0.325
Heart rate after 6 min (times/min)	107.4 (17.6)	110.6 (14.6)	0.592	120.7 (20.6)	120.7 (18.7)	0.995

^{*} T-test based p-value

5.4 Development in Walking Difficulty

In MZ twins the most active group includes the study subjects who have no difficulty in 2km walking and has not done any adaptation in their walking habits. The group size remained unchanged in both normal weight and obese weight groups.

The other three groups had changes in their size during the follow-up. The second most active group (no difficulty in 2km walking, some adaptation in walking habit) decreased and the physical activity group status changes from better to worse groups. More detailed information of MZ physical activity changes is showed in more detail in Figure 1.

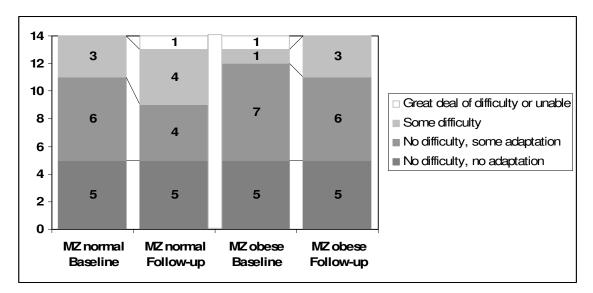


Figure 1. Physical activity groups in baseline and follow-up in monozygotic normal weigh and obese twins, n.

In the normal weight DZ twins group the changes in physical activity were both positive and negative. In the DZ obese group all the changes happened in the three least active groups; there were no changes in the most active group. More detailed information about the physical activity changes of DZ twins is found from Figure 2.

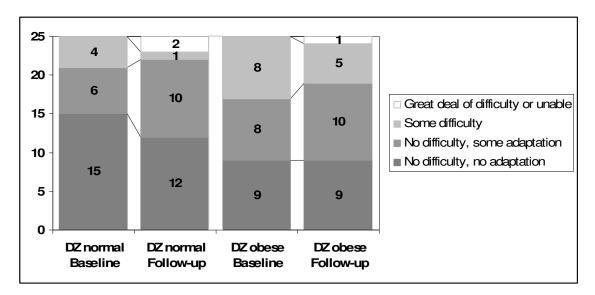


Figure 2. Physical activity groups in baseline and follow-up in dizygotic normal weight and obese twins, n.

These results showed that the ones that did not have any problems in the baseline study, their physical activity remained the same level in the follow-up time. Those who already had done some adaptation or had problems with walking at the baseline, their physical activity declined during the follow-up time (3 years). Those study subjects were in the end in the lower physical activity level groups.

6 DISCUSSION AND CONCLUSION

The purpose of this study was to investigate does the amount of body fat explain walking ability and development of walking difficulty over three years after adjusted for genetic and familial factors. This was done by selecting older female MZ and DZ female twin pairs as study subjects who were discordant in body weight. The results of this study show that the body fat does not have any effect on the walking ability or development of walking difficulty in the three years time when adjusted for genetic and familial factors.

The study subjects in the two weight groups in this study did not differ in terms of health and physical activity. The amount of diseases, self-reported health and physical activity levels and walking habits were similar in both weight groups. The only major difference between these weight groups was the history for weight lost. There were more obese study subjects who had lost weight during the past three years compared to the normal weight twins. This shows that the normal weight study subjects have been in normal weight for a longer time and it is not a new status for them, i.e. they have not lost weight recently and that the obese subjects have been obese for longer time.

The two walking tests (10m and 6min) were chosen as the physical functioning indicators, since walking abilities are required for independent living and decline in the ability is a precursor of disability (Verbrugge & Jette 1994) and also significant predictors of morbidity and mortality (Reuben et al. 1992) in older people.

For this study the 10% body fat intrapair difference was used to create the two different weight groups (normal weight and obese). Since this study setting did not give any statistical significance between the two weight group differences, it needs to be reviewed for the future analyses if the weight difference between the twin sisters was big enough. Maybe this was the reason why the possible differences were not shown in the results. In several animal studies it has been proven that overweight has effect on the walking ability (Clark & Gay 1972, Borer & Kaplan 1977, Dauncey & Brown 1987), but for example Ahima et al. (1999) proved in their study that only a minor weight gain did not make differences in locomotor activity. Major weight gain

(80% of body weight gain) only caused 50% changes in the physical functioning. It would be interesting to find out where the break point of the weight and physical functioning is; to find out the point when the obesity starts to effect negatively on the physical functioning.

It would be interesting to know why these twin sisters differ in the amount of body fat. Most of them had the similar physical activity and health background and they were all relatively in good health. Based on the information that the study data shows, the energy expenditure of the study subjects is known, but the energy intake of study subjects is missing. It would be interesting to know if the body fat difference could be explained by the nutritional habits. There has to be some environmental explanation for the body fat difference in MZ twins, since all the other factors (genetic and familial) were adjusted in this study setting. Among DZ twins there can be more explaining factors than just environmental ones, since their genetical background is only 50% the same.

Another questionable thing of setting of the study is if the chosen walking tests: do they describe the physical functioning well enough? The walking tests were used because walking ability is one of the most important factors that secure the independence of older people and it is one of the basic activities of daily living.

For the future studies it should be analyzed if this study sample was representative enough and if these study results could be generalized to the whole population. Twin studies are often criticized because the results from them could not be generalized because twins may not be representative of the population from which they are drawn. (Maes et al. 1997.) The sample size of this study was relatively small (n=96). It was divided into four groups based on zygosity and the weight group. In MZ twins these groups had only 15 study subjects and in DZ groups 28 subjects. Such a small sample size of study subjects was suitable for this study, since the created study setting was as strong as it was. The study subjects were adjusted by gender, age, familial factors and genotype (100% in MZ and 50% in DZ twins).

The study subjects were relatively in good health and this possibly had effect on the study results. Based on the Healthy 2000 health examination survey done by

National Public Health Institute (KTL 2004), 9% of the over 65 years old Finnish women had type II diabetes. In this study only 2% of the study subject had type II diabetes. The same situation was in arthrosis: in the whole population there is arthrosis in knees in 12% and in hip 18% of the people. In this study 27% had arthrosis in some lower joints.

The strength of this study was the powerful study setting that was made possible because of the twin study subjects and objective laboratory measurements used in physical functioning tests (walking tests). It is very unique possibility to use these study subjects, since they are extremely hard to be located and tested. Since the MZ twins' heritability is 100% the same, all the differences between them are explained by environmental factors because all the genetic and familial factors were adjusted. Since weight is strongly heritable element, it is hard to find body weight discordant MZ twins. Originally large study population enabled us to choose the most discordant pairs and enabled this study setting that was used.

The results of this study do not provide evidence for the hypothesis that body weight effects on the physical functioning of older people. The used study setting was very strong, which means that this study brings contradictory results disapproving the earlier assumptions that body weight would be the explaining factor for the differences in physical functioning. A future research should be made with less selected and larger group of twin study subjects.

REFERENCES

Ahima R, Bjorbaek C, Osei S, Flier J. Regulation of neuronal and glial proteins by leptin: implications for brain development. Endocrinology 1999;140:2755-2762.

Allison DB, Kaprio J, Korkeila M, Koskenvuo M, Neale MC, Hayakawa K. The heritability of body mass index among an international sample of monozygotic twins reared apart. Int J Obes 1996;20:501-506.

Anderson JJ, Felson DT. Factors associated with osteoarthritis of the knee in the first National Health and Nutrition Examination. Am J Epidemiol 1988;128:179-189.

Andrews GR. Promoting health and function in an ageing population. Br Med J 2001;322:728-729.

ATS statement: guidelines for the six-minute walk test. Am J Respir Crit Care Med 2002;166:111-117.

Ball K, Owen N, Salmon J, Bauman A, Gore CJ. Associations of physical activity with body weight and fat in men and women. Int J Obes 2001;25:914-919.

Baumgartner R, Heymsfield S, Roche A. Human body composition and the epidemiology of chronic disease. Obes Res 1995;3:73-95.

Bigaard J, Frederiksen K, Tjonneland A, Thomsen BL, Overvad K, Heitmann BL, Sorensen TI. Waist circumference and body composition in relation to all-cause mortality in middle-aged men and women. Int J Obes 2005;29:778-784.

Bijnen FC, Feskens EJ, Casperson CJ, Mosterd WL, Kromhout D. Age, period, and cohort effects on physical activity among elderly men during 10 years of follow-up; the Zutphen Elderly Study. J Gerontol Med Sci 1998;53:235-241.

Blair SN, Kohl HW III, Paffenbarger RS Jr, Clark DG, Cooper KH, Gibbons LW. Physical fitness and all-cause mortality: a prospective study of healthy men and women. JAMA 1989;262:2395-2401.

Body mass index. 2006. [WWW-document]. Referred 16.11.2006. http://en.wikipedia.org/wiki/Body_mass_index.

Borer K, Kaplan L. Exercise-induced growth in golden hamsters: effects of age, weight and activity levels. Physiol Behav 1977;18:23-34.

Bouchard C. Current understanding of the etiology of obesity: genetic and nongenetic factors. Am J Clin Nutr 1991;53:1561-1565.

Bouchard C, Boulay MR, Simoneau JA, Lortie G, Perusse L. Heredity and trainability of aerobic and anaerobic performances: an update. Sports Med 1988a;5:69-73.

Bouchard C, Perusse L, Leblanc C, Tremblay A, Theriault G. Inheritance of the amount and distribution of human body fat. Int J Obes 1988b;12:205-215.

Bouchard C, Tremblay A, Despres JP, Nadeau A, Lupien PJ, Theriault G, Dussault J, Moorjani S, Pinault S, Fournier G. The response to long-term overfeeding in identical twins. N Engl J Med 1990;322:1477-1482.

Brownlow B, Petro A, Feinglos M, Surwit R. The role of motor activity in diet-induced obesity in C57BL/mice. Physiol Behav 1996;60:37-41.

Burton BT, Foster WR, Hirsch J, VanItallie TB. Health implications of obesity: NIH consensus development conference. Int J Obes 1985;9:155-169.

Carman WJ, Sowers M, Hawthorne VM, Weissfeld LA. Obesity as a risk factor for osteoarthritis of the hand and wrist: a prospective study. Am J Epidemiol 1994;139:119-129.

Caspersen CJ, DiPietro L. National estimates of physical activity among older adults. Med Sci Sports Exerc 1991;23;S106.

Clark L, Gay P. Activity and body relationships in genetically obese animals. Biol Psychol 1972;4:247-250.

Coakley EH, Kawachi I, Manson JE, Speizer FE, Willet WC, Colditz GA. Lower levels of physical functioning are associated with higher body weight among middle-aged and older women. Int J Obes 1998;22:958-965.

Comuzzie A, Blangero J, Mahaney M, Haffner S, Mitchell B, Stern M, MacCluer MJ. Genetic and environmental correlations among hormone levels and measures of body fat accumulation and topography. J Clin Endocrinol Metab 1996;81:597-600.

Dauncey M, Brown D. Role of activity-induced thrmogenesis in twenty-four hour energy expenditure of lean and genetically obese (ob/ob) mice. Exp Physiol 1987;72:549-559.

Davison KK, Ford ES, Cogswell ME, Dietz WH. Percentage of body fat and body mass index are associated with mobility limitations in people aged 70 and older from NHANES III. J Am Geriatr Soc 2002;50:1802-1809.

DiPietro L. Physical activity, body weight, and adiposity: an epidemiologic perspective. Exerc Sport Sci Rev 1995;23:275-303.

DiPietro L, Caspersen CJ. National estimates pf physical activity among white and black Americans. Med Sci Sport Exerc 1991;23:S105.

Dutta C, Hadley EC. The significance of sarcopenia in old age. J Gerontol A Biol Sci Med Sci 1995;50:1-4.

Elia M. Obesity in the elderly. Obes Res 2001;9:S244-S248.

Fabsitz RR, Carmelli D, Hewitt JK. Evidence for independent genetic influences on obesity in middle age. Int J Obes 1992;16:657-666.

Fabsitz RR, Sholinsky P, Carmelli D. Genetic influences on adult weight gain and maximum body mass index in male twins. Am J Epidemiol 1994;140:711-720.

Felson DT. Weight and osteoarthritis. Am J Clin Nutr 1996;63:S430-S432.

Felson DT, Anderson JJ, Naimark A, Swift M, Castelli W, Meenan RF. Obesity and symptomatic knee osteoarthritis (OA): results from the Framingham Study. Arthritis Rheum 1987;30:S130.

Ferrucci L, Guralnik JM, Kasper J, Lamb SE, Simonsick EM, Corti MC, Bandeen-Roche K, Fried LP. Departures from linearity in the relationship between measures of muscular strength and physical performance of the lower extremities: the Women's Health and Aging Study. J Gerontol A Biol Med Sci Med 1997;52:275-285.

Flegal KM, Caroll MD, Kuczmarski RJ, Johnson CL. Overweight and obesity in the United States: prevalence and trends, 1960-1994. Int J Obes 1998;22:39-47.

Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults. 1999–2000. JAMA 2002;288:1723-1727.

Fogelholm M. Lihavuuden arviointi. In Mustajoki P, Fogelholm M, Rissanen A, Uusitupa M (edit) Lihavuus. 3rd edition. Kustannus Oy Duodecim: Hämeenlinna 2006:49-59.

Fogelholm M, Uusitupa M. Kehon koostumuksen arviointi. In Aro A, Mutanen M, Uusitupa M (edit) Ravitsemustiede. 2nd edition. Kustannus Oy Duodecim, Jyväskylä 2005:282-290.

Friedmann J, Elasy T, Jensen G. The relationship between body mass index and self-reported functional limitations among older adults: a gender difference. J Am Geriatr Soc 2001;49:398-403.

Galanos AN, Pieper CF, Cornoni-Huntley JC, Bales CW, Fillenbaum GG. Nutrition and function: is there a relationship between body mass index and the functional capabilities of community-dwelling elderly? J Am Geriatr Soc 1994;42:368-373.

Gallagher D, Visser M, De Meersman RE, Sepulveda D, Baumgartner RN, Pierson RN, Harris T, Heymsfield SB. Appendicular skeletal muscle mass: effects of age, gender, and ethnicity. J Appl Physiol 1997;83:229-239.

Gallagher D, Ruts E, Visser M, Heshka S, Baumgartner RN, Wang J, Pierson RN, Pi-Sunyer FX, Heymsfield SB. Weight stability masks sarcopenia in elderly men and women. Am J Physiol Endocrinol Metab 2000a;279:366-375.

Gallagher D, Heymsfield SB, Heo M, Jeb SA, Murgatroyd PR, Sakamoto Y. Healthy percentage body fat ranges: an approach for developing guidelines based on body mass index. Am J Clin Nutr 2000b;72:694-701.

Garn SM, Leonard WR, Hawthrone VM. Three limitations of the body mass index. Am J Clin Nutr 1986;44:996-997.

Harris TB. Body composition in studies of aging: new opportunities to better understand health risks associated with weight. Am J Epidemiol 2002;156:122-124.

Harris T, Kovar M, Suzman R, Kleinman JC, Feldman JJ. Longitudinal study of physical ability in the oldest old. Am J Public Health 1989;79:698-702.

Hart DJ, Spector TD. The relationship of obesity, fat distribution and osteoarthritis in women in the general population: the Chingford Study. J Rheumatol 1993;20:331-335.

Hill JO, Peters JC. Environmental contributions to the obesity epidemic. Science 1998;280;1371-1374.

Himes C. Obesity, disease, and functional limitation in later life. Demography 2000;37:73-82.

Huang Y, Macera CA, Blair SN, Brill PA, Kohl HW III, Kronenfeld JJ. Physical fitness, physical activity, and functional limitation in adults aged 40 and older. Med Sci Sports Exerc 1998;30:1430-1435.

Hulens M, Vansant G, Lysens R, Claessens AL, Muls E, Brumagne S. Study of differences in peripheral muscle strength of lean versus obese women: an allometric approach. Int J Obes 2001;25:676-681.

Ingram D. Age-related decline in physical activity: generalization to non-humans. Med Sci Sports Exerc 2000;32:1623-1629.

James W. Epidemiology of obesity. Int J Obes 1992;2:S23-S26.

Janssen I, Heymsfield S, Baumgartner R, Ross R. Estimation of skeletal muscle mass by electrical impedance analysis. J Appl Physiol 2000;89:465-471.

Janssen I, Katzmarzyk PT, Ross R. Waist circumference and not body mass index explains obesity-related health risk. Am J Clin Nutr 2004;79:379-384.

Jensen GL, Kita K, Fish J, Heydt D, Frey C. Nutrition risk screening characteristics of rural older persons: Relation to functional limitations and health care charges. Am J Clin Nutr 1997;66:819-828.

Kaprio J, Koskenvuo M. Genetic and environmental factors in complex diseases: the older Finnish twin cohort. Twin Res 2002;5:358-365.

Kaprio J, Sarna S, Koskenvuo M, Rantasalo I. The Finnish Twin Registry: formational and compilation, questionnaire study, zygosity determination procedures and research program. Prog Clin Biol Res. 1978;24B:179-184.

Klesges RC, Klesges LM, Haddock CK, Eck LH. Longitudinal analysis of the impact of dietary intake and physical activity on weight change in adults. Am J Clin Nutr 1992;55:818-822.

Kriketos AD, Pan DA, Lillioja S, Cooney GJ, Baur LA, Milner MR, Sutton JR, Jenkins AB, Bogardus C, Storlien LH. Interrelationships between muscle morphology, insulin action, and adiposity. Am J Physiol 1996;270:1332-1339.

Krotkiewski M, Bjorntorp P. Muscle tissue in obesity with different distribution of adipose tissue. Effects of physical training. Int J Obes 1986;10:331-341.

KTL. Health and functional capacity in Finland. Baseline results of the Health 2000 health examination survey. Publications of the National Public Health Institute. B12/2004.

Kuczmarski R. Need for body composition information in elderly subjects. Am J Clin Nutr 1989;50:1150-1157.

LaCroix AZ, Guralnik JM, Berkman LF, Wallace RB, Satterfield S. Maintaining mobility in late life. II. Smoking, alcohol consumption, physical activity, and body mass index. Am J Epidemiol 1993;137:858-869.

Launer LJ, Harris T, Rumpel C, Madans J. Body mass index, weight change, and risk of mobility disability in middle-aged and older women: the Epidemiologic Follow-up Study of NHANES I. JAMA 1994;271:1093-1098.

Lillioja S, Young AA, Culter CL, Ivy JL, Abbott WG, Zawadzki JK, Yki-Jarvinen H, Christin L, Secomb TW, Bogardus C. Skeletal muscle capillary density and fiber type are possible determinants of in vivo insulin resistance in man. J Clin Invest 1987;80:415-424.

Ma J, Markides KS, Perkowski LP, Stroup-Benham CA, Lichtenstein M, Goodin JS. Impact of selected medical conditions on self-reported lower extremity function in Mexican-American elderly. Ethn Dis 1998;8:52-59.

Maes HH, Neale MC, Eaves LJ. Genetic and environmental factors in relative body weight and human adiposity. Behav Genet 1997;27:325-351.

Manson JE, Rimm EB, Stampfer MJ, Golditz GA, Willett WC, Krolewski AS, Rosner B, Hennekens CH, Speizer FE. Physical activity and incidence of non-insulin dependent diabetes mellitus in women. Lancet 1991;338:774-778.

Manson JE, Willett WC, Stampfer MJ, Colditz GA, Hunter DJ, Hankinson SE, Hennekens CH, Speizer FE. Body weight and mortality among women. New Engl J Med 1995;333:677-685.

Marin P, Andersson B, Krotkiewski M, Bjorntorp P. Muscle fiber composition and capillary density in women and men with NIDDM. Diabetes Care 1994;17:382-386.

Merrill SS, Seeman TE, Kasl SV, Berkman LF. Gender differences in the comparison of self-reported disability and performance measures. J Gerontol A Biol Sci Med Sci 1997;52:19-26.

Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. JAMA 1999;282:1523-1529.

Nelson HD, Nevitt MC, Scott JC, Stone KL, Cummings SR. Smoking, alcohol, and neuromuscular and physical function of older women. JAMA 1994;272:1825-1831.

Newby F, Digirolam M, Cotsonis G, Kuner M. Model of spontaneous obesity in aging male Wistar rats. Am J Physiol Regul Integr Comp Physiol 1990;259:1117-1125.

Newman AB, Haggerty CL, Goodpaster B, Harris T, Kritchevsky S, Nevitt M, Miles TP, Visser M. Strength and muscle quality in a well-functioning cohort of older adults: the Health, Aging and Body Composition Study. J Am Geriatr Soc 2003;51:323-330.

Ortega-Alonso A, Pedersen NL, Kujala UM, Sipilä S, Törmäkangas T, Kaprio J, Koskesnvuo M, Rantanen T. A twin study on the heritability of walking ability among older women. J Gerontol 2006;61A:1082-1085.

Pajala S, Era P, Koskenvuo M, Kaprio J, Alen M, Tolvanen A, Tiainen K, Rantanen T. Contribution of genetic and environmental factors to individual differences in maximal walking speed with and without second task in older women. J Gerontol 2005;10:1299-1303.

Pajala S, Era P, Koskenvuo M, Kaprio J, Tolvanen A, Rantanen T. Genetic and environmental contribution to postural balance of older women in single and dual task situations. Neurobiol Aging 2007;28:947-954.

Perrone G, Liu Y, Capri O, Critelli C, Barillaro F, Galoppi P, Zihella L. Evaluation of the body composition and fat distribution in long-term users of hormone replacement. Gynecol Obstet Invest 1999;48:52-55.

Perusse L, Tremblay A, Leblanck C, Bouchard C. Genetic and environmental influences on level of habitual physical activity and exercise participation. Am J Epidemiol 1989;129:1012-1022.

Pratley RE, Coon PJ, Rogus EM, Goldberg AP. Effects of weight loss on norepinephrine and insulin levels in obese older men. Metabolism 1995;44:438-444.

Prentice AM, Jebb SA. Obesity in Britain: gluttony or sloth? BMJ 1995;311:437-439.

Räihä I. Vanhusten ravitsemus. In Aro A, Mutanen M, Uusitupa M (edit) Ravitsemustiede. 2nd edition. Kustannus Oy Duodecim, Jyväskylä 2005:312-324.

Ramsay SE, Whincup PH, Shaper AG, Wannamethee SG. The relations of body composition and adiposity measures to ill health and physical disability in elderly men. Am J Epidemiol 2006;164:459-469.

Reuben D, Rubenstein L, Hirsch SH. Value of functional status as a predictor of mortality: results of a prospective study. Am J Med 1992;93:663–669.

Rissanen A, Fogelholm M. Aikuisten lihavuus Suomessa ja muualla. In Mustajoki P, Fogelholm M, Rissanen A, Uusitupa M (edit) Lihavuus. 3rd edition. Kustannus Oy Duodecim: Hämeenlinna 2006:14-22.

Rissanen A, Heliövaara M, Knekt P, Reunanen A, Aromaa A, Maatela J. Risk of disability and mortality due to overweight in a Finnish population. BMJ 1990;301:835-837.

Rissanen A, Heliövaara M, Knekt P, Reunanen A, Aromaa A. Determinants of weight gain and overweight in adult Finns. Eur J Clin Nutr 1991;45:419-430.

Rolland Y, Lauwers-Cances V, Pahor M, Fillaux J, Grandjean H, Vellas B. Muscle strength in obese elderly women: effect of recreational physical activity in a cross-sectional study. Am J Clin Nutr 2004;79:552-557.

Roubenoff R. Sarcopenia and its implications for the elderly. Eur J Clin Nutr 2000;54:S40-S47.

Samaras K, Kelly PJ, Chiano MN, Spector TD, Campbell LV. Genetic and environmental influences on total-body and central abdominal fat: the effect of physical activity in female twins. Ann Intern Med 1999;130:873-882.

Sartorio A, Lafortuna CL, Conte G, Faglia G, Narici MV. Changes in motor control and muscle performance after a short-term body mass reduction program in obese subjects. J Endocrinol Invest 2001;24:393-398.

Schousboe K, Willemsen G, Kyvik KO, Mortensen J, Boomsma DI, Cornes BK, Davis CJ, Fagnani C, Hjelmborg J, Kaprio J, De Lange M, Luciano M, Martin NG, Pedersen N, Pietilainen KH, Rissanen A, Saarni S, Sorensen TI, Van Baal GC, Harris JR. Sex differences in heritability of BMI: a comparative study of results from twin studies in eight countries. Twin Res 2003;6:409-421.

Seidell JC, Visscher TLS. Body weight and weight change and their health implication for the elderly. Eur J Clin Nutr 2000;54:S33-S39.

Skelton DA, Greig CA, Davies JM, Young A. Strength, power and related functional ability of healthy people aged 65-89 years. Age Ageing 1994;23:371-377.

Sobal J, Stunkard A. Socioeconomic status and obesity: a review of the literature. Psychol Bull 1989;105:260-275.

Sorensen TIA, Holst C, Stunkard AJ. Childhood body mass index – Genetic and familial environmental influences assessed in a longitudinal study. Int J Obes 1992;16:705-714.

Spirduso WW. Physical activity and aging: Retrospections and visions for the future. J Aging Phys Activity 1994;2:233-242.

Stevens J, Gautam SP, Keil JE. Body mass index and fat patterning as correlates of lipids and hypertension in an elderly, biracial population. J Gerontol A Biol Sci Med Sci 1993;48:249-254.

Stevens J, Cai J, Pamuk ER, Williamson DF, Thun MJ, Wood JL. The effect of age on the association between body-mass index and mortality. N Engl J Med 1998;338;1-7.

Stewart A, Brook R. Effects of being overweight. Am J Public Health 1983;73:171-178.

Strawbridge WJ, Cohen RD, Shema SJ, Kaplan GA. Successful aging: predictors and associated activities. Am J Epidemiol 1996;144:135-141.

Supiano MA, Hogikyan RV, Morrow LA, Ortiz-Alonso FJ, Herman WH, Galecki AT, Halter JB. Aging and insulin sensitivity: role of blood pressure and sympathetic nervous system activity. J Gerontol A Biol Sci Med Sci 1993;48:237-243.

Swallow J, Koteja P, Carter P, Garland T. Artificial selection for increased wheel-running activity in house mice results in decreased body mass at maturity. J Exp Biol 1999;202:2513-2520.

Tanner CJ, Barakat HA, Dohm GL, Pories WJ. MacDonald KG, Cunningham PR, Swanson MS, Houmard JA. Muscle fiber type is associated with obesity and weight loss. Am J Physiol 2002;282:1191-1196.

Tou JC, Wade CE. Determinants affecting physical activity levels in animal models. Exp Bio Med 2002;227:587-600.

Uusitupa M. Lihavuus. In Aro A, Mutanen M, Uusitupa M (edit) Ravitsemustiede. 2nd edition. Kustannus Oy Duodecim, Jyväskylä 2005:369-393.

Uusitupa M, Fogelholm M. Antropometriset mittaukset. In Aro A, Mutanen M, Uusitupa M (edit) Ravitsemustiede. 2nd edition. Kustannus Oy Duodecim, Jyväskylä 2005:276-281.

Verbrugge LM, Jette AM. The disablement process. Soc Sci Med 1994;38:1–14.

Visser M, Langlois J, Guralnik JM, Cauley JA, Kronmal RA, Robbins J, Williamson JD, Harris TB. High body fatness, but not low fat-free mass, predicts disability in older men and women: the Cardiovascular Health Study. Am J Clin Nutr 1998;68:584-590.

Voorrips LE, Lemmink KAMP, van Heuvelen MJG, Bult P, van Staveren WA. The physical condition of elderly women differing in habitual physical activity. Med Sci Sports Exerc 1993;25;1152-1157.

Waaler HT. Hazard of obesity—the Norwegian experience. Acta Med Scand Suppl 1988;723:17-21.

Wannamethee SG, Shaper AG, Whincup PH, Walker M. Overweight and obesity and the burden of disease and disability in elderly men. Int J Obes 2004;28:1374-1382.

WHO. Obesity. Preventing and Managing the Global Epidemic. Report of a WHO Consultation on Obesity. Geneva, Switzerland; 1998.

Williamson DF, Madans J, Anda RF, Kleinman JC, Kahn HS, Byers T. Recreational physical activity and ten-year weight change in a US national cohort. In J Obes 1993;17:279-286.

Zamboni M, Turcato E, Santana H, Maggi S, Harris TB, Pietrobelli A, Heymsfield SB, Micciolo R, Bosello O. The relationship between body composition and physical performance in older women. J Am Geriatr Soc 1999;47:1403-1408.