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NARRATIVE REVIEW
Does it matter if I am overweight? 1. Some biomechanical, physiological, and performance-related consequences.
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Abstract

Objectives: The objective of this article is to consider the impact of obesity upon body biomechanics, physiology, and various measures of human performance and ability. ***Methods.*** Information obtained from Ovid/Medline and Google Scholar through to September 2018 was supplemented by a search of the author's personal files. ***Results.*** An excessive accumulation of body fat has a wide range of negative consequences for biomechanical and physiological functions. A few potentially positive consequences of obesity have also been suggested, but most of these supposed benefits do not withstand close examination. The mechanical efficiency of movement is impaired, with a consequent reluctance to engage in physical activity. Agility, flexibility, and balance are adversely affected, with a reduction of functional capacity both at work and in leisure activities. Deposition of fat around the airways leads to sleep apnoea, predisposing to daytime sleepiness, and heat tolerance is reduced. Body fat may protect the hipbones in the event of a fall, but on the other hand a combination of relative muscle weakness with poorer agility and balance increase the risk of falls. Thermal protection is increased in cold climates, but fat offers a less flexible form of insulation than additional clothing (or external application of a layer of fat in an ultra-long distance swimmer). Brown fat plays a role in cold acclimation, but the brown fat response seems attenuated in those who are obese. The greater buoyancy of an obese person increases flotation, but this benefit is offset by a greater drag as the body rises out of the water. Even the energy reserves of fat have little practical value unless prolonged starvation is anticipated. ***Conclusions.*** There are few mechanical or physiological advantages to allowing an excessive accumulation of body fat. Given the extensive range of chronic illnesses provoked by obesity, physiological considerations certainly do not warrant ignoring the clinical imperative to maintain an optimal body mass. **Health & Fitness Journal of Canada 2018;11(2):15-52.**

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Introduction

Many people who are overweight or frankly obese show little concern about the current and/or future implications of this fat accumulation. Physicians recognize that it increases the risk of contracting a wide range of chronic diseases, but the objective of this three-part narrative review is to summarize current knowledge on all of the many negative consequences of obesity; such information should provide useful ammunition for practitioners who face the difficult task of motivating their clients to maintain and/or to reach an optimal body fat content. Specific issues examined in the first section of this review are the biomechanical, physiological and performance-related concomitants of obesity. Subsequent parts of the review will explore the psychosocial consequences, and will detail the influence of excessive fat accumulation upon overall mortality and

the risk of various chronic diseases.

Among the negative biomechanical and physiological consequences of fat accumulation (Table 1), we may note an increased energy cost of body displacement with loss of agility, and an increased risk of sleep apnoea. There are problems of thermoregulation in a hot climate. Subcutaneous fat may offer some mechanical protection against injury during falls, but other characteristics of those who are obese increase the risk of those who are heavy because of obesity, although in many situations, detailed calculations of total energy expenditures are complicated by an exchange of energy between the various body parts over a movement cycle.

We will look at generalized theoretical models of the relationship between a person's body mass and the energy cost of human movement, noting how expenditures can also be increased by a decrease in mechanical efficiency, and we

Table 1: Some biomechanical and physiological consequences of obesity.

- Reduced mechanical efficiency
- Increased energy cost of body displacement
- Reduced habitual physical activity
- Reduced functional capacity
- Reduced agility, flexibility, and balance
- Increased risk of sleep apnoea
- Negative and positive influences on the risk of bone fractures
- Increased thermal stress
- Insulation in a cold environment
- Greater buoyancy but adverse posture for swimmer
- Greater energy reserves for prolonged famine

falls. Subcutaneous fat also provides insulation in a cold climate, although the degree of protection cannot be varied with work-rate. Further, buoyancy is greater in a fat person, although this does not appear to enhance the performance of a swimmer. Finally, fat provides a large resource of nutrients, although this is rarely exploited except during a prolonged famine.

Reduced mechanical efficiency and Increased energy cost of body displacement.

Most types of physical activity involve displacement of all or part of a person's body mass, so the energy cost of such activities is necessarily increased for will consider empirical data linking body mass with the energy costs of industrial work, walking, running and cycling, finally noting implications for the individual's functional capacity.

Generalized theoretical models. A number of early investigators pointed to an increased energy cost of various types of body movement in those who were obese (Brown, 1966; Hanson, 1973; Malhotra et al., 1962; MceKee and Bollinger, 1960; Passmore and Durnin, 1955). Some authors suggested a direct linear relationship, of the type

$$E = b(M)$$

where E was the energy cost of a task, M was the individual's body mass, and b was a constant. Brown (1966) proposed a variation in this formula:

$$E = a + b (M)^n$$

where a was a second component, and n was an exponent ranging from 0.75-1.00. Gaetan Godin and Shephard (Godin and Shephard, 1973) suggested that in general the total energy cost E of a given task should be calculated from a two-term equation reflecting both resting metabolism and the added cost of physical activity:

$$E = a(M)^{0.75} + b(M)^n$$

The first term in the equation represented the impact of body mass on the energy cost of resting metabolism (roughly proportional to $[M]^{0.75}$, although with lean tissue having a much greater impact than an equivalent weight of fat). The second term was the added energy expenditure required for body displacement against the force of gravity, and here the mass exponent n rose from close to zero for a task such as cycling (where there was little vertical displacement of body tissue) to close to 1.0 for an activity such as running on a level surface.

Notice that the potential energy accumulated when moving uphill is also directly proportional to body mass; cyclists may regain much of this energy on moving downhill, but for the runner the controlled descent of a downhill gradient places a heavier mechanical burden on a heavy than on a lightweight individual.

These various early analyses all made the assumption that the adverse effects of obesity were due simply to the greater body mass of the individual. But in fact, in some activities such as walking the generalized relationship is modified by body size and limb length, with taller individuals spending less energy than those who are shorter. (Weyand et al., 2010). Moreover, for various reasons (discussed below) the mechanical efficiency of movement is commonly decreased by the accrual of body fat.

Decreased mechanical efficiency.
Mechanical efficiency reflects the relationship between the energy expended by the body and the physical work that is accomplished. It may be expressed in gross terms, or as a net value (after deducting resting energy expenditure). It is important to distinguish between these options, since the resting energy expenditure is often greater for an obese person than for someone with a normal body mass.

The direct effects of displacing a greater body mass are usually exacerbated by a decrease of mechanical efficiency in those who are obese, leading to a substantial increase in the energy cost of many daily activities (Table 2). The healthy human body has a *net* mechanical efficiency of 25-30% when performing simple physical tasks such as walking or cycling on level ground. Some early empirical studies found that although oxygen consumptions were greater in those who were obese, there were no apparent differences of mechanical efficiency between lean and obese individuals when they were operating a pulley system (McKee, 1960) or were walking relatively slowly on a treadmill (Turell et al., 1964). However, more recent observations have generally shown a decreased mechanical efficiency during walking, and sometimes also during cycling or cycle ergometry.

Physiological consequences of obesity

Table 2: Association between obesity and an increase in the energy cost of daily activities.

Author	Sample	Physical task	Mechanical efficiency	Comments
Ayub and Bar-Or (2003)	9 obese, 9 normal boys, aged 11-18 yr	Treadmill walking	Obese boys had 12% higher oxygen consumption at speed of 6 km/h	No loss of efficiency in obese at 4 or 5 km/h
Browning et al. (1985)	19 class II obese, 20 normal adults	Treadmill walking	Mechanical efficiency decreased in obese as walking speed increased to 6.3 km/h	
Chen et al. (2004)	60 men, 55 women	Walking and stepping on force platform	Mechanical efficiency unaffected by obesity at slow speeds, but at normal speed of walking 25% vs. 30%	Obese people normally walked at 2.2 km/h rather than 4.3 km/h as seen in normal peers
Davies et al. (1975)	17 obese girls and young adults	Cycle ergometry and treadmill walking	Higher than expected oxygen costs for given work output on both tasks	
Dempsey et al. (1966)	14 obese, 14 normal men	Cycle ergometry	Mechanical efficiency lower in obese subjects	
Huang et al. (2013)	16 obese, 16 normal children	Lap walking	Energy cost 72.7% greater in obese children, but net efficiency (J/kg/m] not abnormal	
Hulens et al. (2001)	225 obese, 81 normal women	Cycle ergometry	Mechanical efficiency (W/L oxygen) 15% lower in obese	
Jabbour et al. (2013)	464 pre-pubertal children	Cycle ergometry	Mechanical efficiency similar for normal, overweight and obese children	Resting energy consumption higher in obese children
LaFortuna et al. (2006)	9 obese women (BMI 40.4 kg/m²), 9 normal women	Cycle ergometry	Mechanical efficiency 21.7% vs. 24.8%	Effect seen over power outputs 40-100 W
LaFortuna et al. (2009)	10 obese, 10 normal pubertal girls	Cycle ergometry	Mechanical efficiency 21.5% vs. 24.2%	
LaRoche et al. (2015)	13 overweight/obese, 13 normal seniors	Treadmill walking	Energy cost 62% greater in overweight/obese group	
Maciejczyk et al. (2016)	22 young men (23% vs. 16% body fat)	Treadmill walking	Gross oxygen costs normal at 4.8 km/h, but 2 mL/kg/min higher in obese at 6 km/h	
McKee (1960)	19 obese, 25 normal adults	Operating pulley system by right arm	No inter-group difference of net efficiency	Obese group had higher resting energy consumption
Turell et al. (1964)	15 very obese, 12 normal adults	Treadmill walking	No inter-group difference in mechanical efficiency	Obese individuals had double the energy expenditure at given speed

Walking. Davies et al. (1975) found an increased oxygen cost of treadmill walking (i.e., a greater *gross* energy expenditure) in girls and young women who were obese, Nevertheless, much appears to depend on the degree of obesity and the speed of movement that is required. Thus, one more recent report found that obese individuals had a normal *net* mechanical efficiency when they were walking at the very slow speeds with which they were familiar (2.2 km/h), but efficiency became much lower than normal (25% vs. 30%) when the obese individuals attempted to walk at speeds (4.3 km/h) closer to those adopted by their slimmer peers (Chen et al., 2004).

Ayub and Bar-Or (2003) reported that in adolescents who were matched for total body mass, the net energy costs of treadmill walking at 4 and 5 km/h were independent of body composition, but at 6 km/h, costs were 12% higher in obese (36.6% fat) than in lean boys (9.1% fat). Likewise, Maciejczyk et al. (2016) noted that body composition had little effect upon the energy costs of young men when they were treadmill walking at a slow speed (4.8 km/h), but at a brisk walking speed (6.0 km/h) *gross* oxygen costs were some 2 mL/kg/min greater in individuals with a substantial body fat content (23.1%, compared with 16.3% in controls), even if total body mass was comparable with that of their leaner peers.

In the case of walking, the *net* mechanical efficiency of obese individuals tends to be reduced in part by adverse changes in the biomechanics of locomotion. Thus one study of older adults found that those who were obese had 11% lower ground reaction forces, an 8% slower stride rate, a 12% shorter stride, a 13% longer ground-foot contact time, and 21% longer double-support times, these various changes increasing the costs of body displacement (LaRoche et al., 2011; LaRoche et al., 2015). Obese adult women also had a shorter stride length and a slower gait (Pataky et al., 2014), and they expended a greater amount of energy in a medio-lateral direction, rather than in the development of forward motion (Ko et al., 2010; Peyrot et al., 1985); the extent of medio-lateral movement was significantly correlated with body fatness ($r^2 = 0.64$). Again, observations in children have demonstrated significant adverse changes in the gait pattern in those who were obese. Fat children walked at a 0.15 m/s slower walking speed, with a 10% shorter cadence, and a 31% longer double-support phase than those with a normal body build (Huang et al., 2013). Natel et (2006) also reported increased lateral hip movements in those who were obese, and an accumulation of fat in the extremities has been shown to increase the energy cost of leg movements (Myers and Steudel, 2005). A poor general level of physical fitness and a lack of practice of vigorous physical activity in those who are obese compounds all of these issues.

Table 3: Some factors contributing to reduced net mechanical efficiency in obese individuals.

- Poor overall physical fitness
- Slower speed of leg movement
- Shorter stride length
- Longer ground-foot contact time
- Longer double support time
- Increased lateral movement
- Increased weight of limbs

Cycle ergometry and cycling. The *net* mechanical efficiency of cycle ergometry and cycling also tends to be reduced in those who are grossly obese, probably

due to a combination of increased friction imposed by pendulous skin-folds, an increase of postural work needed to maintain the body in an appropriate posture on the bicycle or ergometer, and an increased physical effort in raising and lowering the legs repeatedly (Davies et al., 1975; Dempsey et al., 1966).

In one study, the *net* mechanical efficiency at power outputs ranging from 40 to 100 W was only 21.7% in obese young women, compared with 24.8% in those of normal body mass ((LaFortuna et al., 2006). Adolescent girls who were obese showed a similar adverse differential (LaFortuna et al., 2009).

Energy expenditures during industrial work. Because of the above factors, one might anticipate that in the occupational setting, obese people would expend a larger amount of energy than a person of normal body mass, but empirical data have not shown any strong relationship between such energy expenditures and the individual's BMI (Brown, 1966). John Brown collected empirical data on industrial employees; finding an added oxygen cost of 5 mL/kg/min in obese clerks, rising to a penalty of 12 mL/kg/min in "heavy" workers, but in some seated jobs such as press operator, energy expenditures were not correlated with body mass.

The most likely explanation of limited effects from obesity is that the obese worker moves more slowly and more infrequently than his or her peers of normal weight. Moreover, in many occupations there is an ever-diminishing requirement for body movement, irrespective of a person's body mass (Gutiérrez-Fisac et al., 2002). Finally, there is a tendency for obese individuals to seek sedentary out employment [as illustrated by the differing physical

characteristics of London bus drivers and conductors at their recruitment (Morris et al., 1956).

Energy costs of walking and running. As might be expected, the *gross* energy costs of walking and running are greater for heavier individuals. Hanson (1973) deliberately increased the weight of 4 volunteers by over-eating, and found that the energy cost of walking was increased in direct proportion to the increase of body mass. Another study found that at a fixed speed of treadmill walking, the energy cost of an elderly group per unit distance covered was 62% larger in those with a BMI greater than 25 kg/m^2 (301 J/m) than in those of normal body mass (186 J/m), due to an exacerbation of the effects of increased body mass by a mechanically inefficient gait, and the choice of what was a higher than preferred speed of walking for the obese (Delextrat et al., 2011; LaRoche et al., 2015). In children, also, the *net* metabolic cost of lap-walking was 66% higher in obese children relative to those of normal body mass, even when they were allowed to walk at a self-selected pace (Huang et al., 2013).

Browning et al. (1985) noted that on average, the *gross* energy cost of treadmill walking at speeds in the range 1.8-6.1 km/h was10% per kg greater in class II obese subjects; moreover, as walking speeds were increased, costs rose faster in the obese than in those of normal body mass.

Energy costs of cycling. Hanson (1973) found no increase in the *net* energy cost of cycle ergometry in 4 volunteers when they had increased their body mass 19% by over-eating. Jabbour et al. (2013) also noted that although the resting energy expenditure was increased in obesity,

there were no differences in the *net* energy costs of cycle ergometry between obese, overweight and normal weight children at power outputs over the range 25-125 W.

On the other hand, several reports have indicated a decreased mechanical efficiency of cycle ergometry in chronically obese adults., possibly linked to a poorer oxygen supply to the working muscles (Salvadori et al., 1999). LaFortuna et al. (2006) found a greater energy expenditure in grossly obese women at all power outputs from 40-120 W. Likewise, Hulens et al. (2001a,b) reported a greater oxygen consumption at a loading of 70 W. At this loading, the obese individuals had already reached 78% of their peak oxygen intake, compared with 69% in those of normal weight.

Reduced habitual physical activity.

There are clearly associations between low levels of habitual physical activity and the presence of overweight or obesity, although the direction of such relationships is less clear. Nevertheless, one may hypothesize that the greater energy cost of body movement (LaRoche et al., 2015) and problems of thermal regulation during hot weather combine with self-consciousness about physical appearance, poor agility and a lack of involvement in vigorous sports to create a vicious cycle of inadequate participation in voluntary physical activity and a growing level of obesity in the individuals concerned.

Studies of total energy expenditures of adults, using the doubly-labeled water technique, have shown rather similar daily averages of 1.8 times basal values at all levels of body mass index up to 40 kg/m^2, but in those with gross obesity (BMI > 40 kg/m^2), the average daily energy expenditure was substantially smaller, at only ~1.6 times basal (Prentice et al., 1996; Westerterp and Speakman, 2008). Prentice et al. (1996) also commented that in men with a BMI > 35kg/m^2.there was a decrease of active energy expenditures (periods of the day when metabolic rate was substantially above resting levels). Moreover, Bonomi et al. (2013) demonstrated that after a 13% reduction of body mass had been achieved by dieting, their subjects showed a 9% increase in daily physical activity, as assessed by accelerometer counts; however, because their body weights were now reduced, the active energy expenditure were not increased relative to values observed when they had been obese. Averaging over 6 reports, Garby et al. (1982) found a decrease in physical activity index of 0.0022 per kg increase in body mass relative to ideal values.

A systematic review of the literature for children and adolescents found that in 38 of 48 studies, the volume of habitual physical activity was negatively associated with obesity (Jimenez-Pavon et al., 2010).

Reduced functional capacity and physical fitness.

Another significant handicap imposed by obesity is a reduction in functional capacity and physical fitness. In most reports, an individual's maximal aerobic power is expressed as the maximal rate of oxygen transport (ml/min) per kg of body mass. Likewise, the maximal strength of individual muscle groups is commonly presented as Newtons of force per kg of body mass. Relative to a person with an ideal body mass of 75 kg, 20 kg of excess fat would thus have the effect of reducing the relative power of a given absolute oxygen transport and the relative force of

a given muscle group by a substantial 21%.

A middle-aged individual who is obese can to some extent compensate for this reduction of effective aerobic fitness by moving more slowly, but as old age advances, a critical threshold is reached where the residual functional capacity is no longer sufficient to undertake the basic activities of daily living without external help. The critical level is probably an aerobic power of around 14 mL/kg/min, (Paterson and Stathokostas, 2001; Shephard, 1997). A person who is sedentary and obese is likely to reach this point 10-20 years earlier than a person with a normal body mass, with a correspondingly adverse impact upon quality-adjusted life expectancy.

Decreased agility, flexibility and balance

A priori, one might anticipate that an excess of body fat would impair agility, flexibility and balance in an obese individual. Empirical data on these issues are quite limited for adults, but more readily available for children, where it has been demonstrated that obesity has a negative impact upon a variety of movement skills from an early age (Bryant et al., 2014; Okely et al., 2004).

Agility. Agility is typically measured by shuttle runs and/or sprint tests that involve rapid changes of direction, and the ratio of strength to body mass has been seen as the prime determinant of test scores. Adult test participants have typically been players of team sports, and for such individuals, excess fat is rarely a problem. Thus, Mohammed and Tareq (Mohammed and Tareq, 2016) found no relationship between shuttle run scores and skin-fold measures of body fat content in adolescent soccer players, none of whom were particularly fat.

However, the negative effects of a high BMI on participants in the Pakistani wrestling sport of Kabaddi were documented by Thakur (2016). Among those participating in this sport, a correlation of 0.54 was found between BMI and shuttle run test scores. Nevertheless, it must be noted that a substantial part of the high body mass in Kabaddi contestants is attributable to muscle rather than fat.

Data are limited for non-athletic adults, but Barbao-Castillo et al. (2011) noted limitations in agility and ability to perform the activities of daily living in obese seniors. Likewise, frequent studies of obese schoolchildren (Table 4) have shown reduced agility and speed of movement of obese students on traditional performance tests such as the shuttle run (much of this handicap reflecting the effects of a greater body mass upon sprint speeds) (Tokmakidis et al., 2006). Fidler et al. (2016) also commented that obese students found it difficult to participate in school sports programmes, with a resulting loss of self-esteem and a deterioration in their quality of life.

Flexibility. One might anticipate that severe obesity would present significant mechanical limitations to an individual's flexibility, One comparison of 10 obese adults with matched controls showed a decreased range of forward flexion of the trunk in those who were obese (Gilleard and Smith, 2007). Another study found men who were obese had impairments in the range of motion at 9 of 30 joints (Park et al., 2010). As a person ages, such initial impairments may be exacerbated by osteoarthritis, a further complication of excessive body weight (Capodaglio et al., 1010).

Table 4: Effects of overweight and obesity upon the agility of schoolchildren.

Authors	Sample	Test	Findings	Comment
Bovet et al. (2007)	2203 boys, 2143 girls aged 12-15 yr	Multi-stage shuttle run	Good score in 29.6% normal, 7.9% overweight, 1.2% obese	Similar findings for 40 m sprint and 10x5 m agility test
Ceshia et al. (2016)	2411 children aged 7-11 yr	Times on agility course	14.5% slower in overweight and obese children	Obesity determined from projection of adult BMI values
Deforche et al. (2003)	3214 children aged 14-18 yr	Speed shuttle run	Poorer performance than non-obese youth	Representative sample of Flemish children
Fidler et al. (2016)	130 children aged 7-14 yr	Side hops and shuttle run	Slowing of times correlated with BMI percentile	Obese experienced difficulties in sport participation
Malina et al. (1995)	6700 girls aged 7-17 yr	Shuttle run	Obesity explains 2-12% of variance in scores	Comparison of fattest 5% vs. leanest 5%, based on skin-folds
Tokmakidis et al. (2006)	709 primary school students	10x5m run, Shuttle run	Poorer scores in overweight and obese students	

However, empirical data in children show relatively little decrease of flexibility with the accumulation of body fat. Tokmadis et al. (2006) saw no significant loss of flexibility in obese Greek primary school students, as measured by the traditional sit-and-reach test. Likewise, Ceschia et al., (2016) and DeForche et al. (2003) saw poor scores for other fitness tests, but no differences in sit-and-reach sores in obese children. Nevertheless, Malina et al. (1995) found that obesity described a statistically significant 3-8% of the total variance in sit and reach scores.

Balance and the risk of falling. The maintenance of balance depends primarily on the strength of the postural muscles relative to the body mass, so that an adverse effect from obesity would be anticipated during disturbance of a person's equilibrium.

Jeon (2013) documented an adverse effect of obesity on the risk of falling in a sample of 351 elderly people. Himes and Reynolds (2012) also commented that obese individuals had an increased risk of falls in a study of 9621 adults aged > 65 years; on the other hand, they suggested that those with a BMI >40 kg/m² had a lower risk of fracture if a fall occurred (odds ratio 0.6). Handrigan et al. (2017) also reported that in 6399 Canadian men aged >65 years, obesity was a significant risk factor for falls, although in their study the risk was not increased in elderly obese women.

The increased propensity to falls also affects younger obese adults. A study of 8581 employees at the Idaho National Laboratory found that 51% of those slipping, tripping or falling were obese (Koepp et al., 2015). Kim et al. (2010) suggested that there was a U-shaped relationship between BMI and the risk of falls, with an increase of risk in both the underweight and those who were obese; although statistically significant, after adjusting for covariates, the obese showed only about a 4% increase in risk of falling 2 or more times per year.

Obese children also show poorer balance on physical performance tests. Ceschia et al. (2016) noted that overweight and obese children had 165% more falls than their normal weight peers while they were performing Stork stand tests. Deforche et al. (2003) also reported significantly poorer Stork stand scores in obese youth, and Malina et al. (1995) estimated that obesity contributed 0-5% to the variance in Stork stand scores.

Sleep apnoea and breathing problems

Sleep apnoea is a troublesome condition where a person's breathing is interrupted for 10 seconds or longer five or more times per hour during sleep (Bresnitz et al., 1994). The cessation of breathing causes the arterial oxygen saturation to fall, and the affected individual is awakened repeatedly throughout the night. The affected individuals are often, but not always, obese (Partinen and Telakivi, 1992).

Immediate pathology. In badly affected individuals, apnoea may occur 50 or more times per hour during sleep, with individual episodes lasting up to 30 seconds. The oximeters that are usually used in detect the impact of the apnoea upon arterial oxygen saturation lack accuracy when a marked desaturation of the blood develops. Block et al. (1979) reported saturations as low as 68-72% (more commonly in men than in women) and Sanders (1984) suggested that arterial oxygen saturations could sometimes drop below 50% during periods of breath-holding.

A thickening of the soft tissues of the mouth and throat is seen in this condition, and when the throat muscles relax during sleep, the airway becomes obstructed, wakening the patient; one important factor contributing to obstruction of the airway is a local deposition of fat (Punjabi, 2008). Neck circumference is thus one of the strongest predictors of the severity of disturbed nocturnal breathing (Resta et al., 2001).

Relationship to obesity. The association between sleep apnoea and obesity, particularly central obesity, has long been recognized (Li et al., 2010; Young, et al., 2005), and indeed substantial weight gain is the most common of a multiplicity of reasons for the development of sleep apnoea and associated hypertension (Table 5).

The prevalence of sleep apnoea in the general adult population is around 25%, but sleep apnoea is seen at least twice as frequently among those who are obese, whether the extent of fat accumulation has been assessed by BMI or by waist/hip ratio (Sharma et al., 2006). One multivariate analysis found that the odds ratio for a diagnosis of sleep apnoea was as high as 4.1 in those with a BMI >25 kg/m^2 (Reddy et al., 2009). Other reports, based on middle-aged adults, have found a prevalence of obstructive sleep apnoea of 2% in women and 4-5% in men of normal weight, but the prevalence rose to 30% in people who were classed as obese, and to

Table 5: Relationship between the frequency of sleep apnoea events, a person's initial BMI and the risk of developing hypertension (a blood pressure > 140/90 mm Hg and/or the prescription of anti-hypertensive medications) over a 4-year follow-up (based on the data of Peppard et al., 2000).

Variable	Frequency of apnoea events per hour of sleep			
	0	0-4.9	5.0-14.9	>15
BMI (kg/m^2)	27	29	32	35
Odds ratio for developing hypertension*	1.00	1.42	2.03	2.89
*Sample studied for 4 years, odds ratio adjusted for multiple variables, including BMI, waist and neck circumferences.				

50-98% in those who were morbidly obese (Newman et al., 2005; Resta et al., 2001). The Wisconsin Sleep Cohort study suggested that a 1 SD increase of BMI was associated with a 4-fold increase in the risk of sleep apnoea (Young et al., 1993).

Increased risk of daytime sleepiness. Frequent awakening and the resulting loss of night-time sleep usually results in daytime sleepiness for the affected individual, this symptom being reported at least 10 more frequently by those with severe obesity than by those with a normal body mass (Resta et al., 2001). The problem is well exemplified by Joe, the fat boy in Dicken's "Pickwick Papers," an unfortunate youth

Figure.1. Joe, the fat boy who kept falling asleep at inopportune moments in the "*Pickwick Papers*," probably suffered from sleep apnoea. Source: https://www.google.ca/search?q=Fat+boy+in+Pickwick+papers++picture

who kept falling asleep at inopportune moments (Figure 1).

Association with hypertension and other pathologies. Sleep apnoea appears to predispose to hypertension, independently of the direct impact of fat accumulation on this pathology (Peppard et al., 2000). Possibly, the obstruction of breathing has a direct immediate effect on blood pressure, and this then persists into the waking hours.

Other potential complications of sleep apnoea are pulmonary hypertension, right-sided heart failure, stroke and arrhythmias. Often, arrhythmias are provoked by the arterial desaturation (Guillenimaut et al., 1983), but associated chronic chest disease may also responsible for some of these problems (Fletcher et al., 1987).

Severity of problem and quality of life. The severity of sleep apnoea is often rated using the "respiratory disturbance index." This looks at the number of nocturnal arousals, whether caused by frank apnoea or simply by a need to increase

respiratory effort. Scores on this index increased from an average value of ~3 for those of normal weight to ~8 in women, and >10 in men who had allowed their body mass to increase by 15 kg (Newman et al., 2005).

The impact of sleep apnoea upon the quality of life in the affected individual varies, depending on the severity of the disorder. A comparison of 60 affected adults with 34 controls found impaired average scores on 6 of the 8 36-SF Health Survey scales, and these abnormalities were correlated with depression as measured on the Zung self-rating scale (Tsuneto et al., 2002). Further, a survey of 61 children aged 6 months to 12 years found that while the impact was small in 20, it was rated as moderate in 19 and severe in 22 children (Franco et al., 2000).

Relationships between body fat content and the risk of physical injury

Obesity has both negative and positive effects on the risk of physical injury during falls. There are adverse effects upon bone health, and an increased frequency of falls (as discussed above), but at the same time obesity may mitigate the risk of injury to some parts of the body if a fall occurs.

Adverse influences. Some of the adverse consequences of obesity are summarized in Table 6.

Aberrant biomechanics. Aberrant biomechanics (as discussed above) and a decrease of relative muscular strength increase the risk of falls and injuries when an obese individual engages in walking and other forms of weight-bearing exercise (Wearing et al., 2006). Weaker relative muscle strength also decreases shock attenuation at weight-bearing joints, predisposing to damage to cartilage and soft tissues (Syed and Davis, 2000). In a long-term perspective, such issues are exacerbated by an increased risk of osteoarthritis (Astephen and Deluzio, 2005; Felson et al., 1993).

Impaired balance and greater clumsiness. Poor balance and a tendency to walk more clumsily than thinner people leads to a correspondingly greater risk of falls (particularly in a sideways or backwards direction). In a study of 100 adults, Kejonen et al. (2003) demonstrated a correlation between BMI and the extent of antero-posterior movement during a bipedal stance. In a study of military recruits, Fregly et al.

Table 6: Adverse consequences of obesity in terms of fracture risk.

- Aberrant biomechanics
- Impaired balance
- Greater clumsiness and thus increased risk of falls in obese individuals
- Greater mechanical impact of a heavy person on falling
- Reduction of habitual physical activity
- Muscular weakness and Impaired protective reflexes because of reduced physically activity
- Greater mechanical impact if a fall occurs
- Reduced exposure to sunshine, and thus Vitamin D insufficiency
- Low testosterone levels in obese men
- Adverse effects of adipokines and cytokines

(1988) also identified endomorphy, abdominal circumference and total body mass as factors that negatively influenced their performance of postural tests. Corbell et al. (2001) suggested that antero-posterior stability was compromised in the obese not only by the greater body mass, but also by a forward displacement of the centre of mass, although a force platform study by Gravante et al. (2003) did not detect any forward displacement of the centre of mass during a quiet stance. Sartorio et al. (2001) further noted that there was an improvement of one-leg stance scores in a group of 230 obese individuals following their completion of a successful weight-loss programme.

Reduced habitual physical activity, poorer fitness and impaired reflexes. Despite a potential muscle training effect from the greater body mass, the overall low level of habitual physical activity (above) generally leads to weaker muscles in the obese than in those who are of normal build (Hulens et al., 2001a, b).The relative deficit of muscle strength is enhanced by the individual's greater body mass. Moreover (and probably because of limited habitual physical activity), the muscles of an obese person show a reduced capillary blood supply (Mandroukas et al., 1984) and a reduced oxidative capacity (Kirkwood et al., 1991; Newcomer et al., 2001), compromising their ability to engage in sustained activity. Early suggestions that corrective reflexes are slower in an obese than in a thinner person have not been confirmed (Burt and Stunkard, 1964) except when there are associated neuropathies or thyroid disorders, and indeed (perhaps because of the greater inertia of a heavy body) Miller et al. (2011) found no differences in recovery from small

perturbations of balance in those who were obese.

Early fatigue of weaker muscles further predisposes an obese person to physical injury (Karvonen et al., 1980).

Greater mechanical impact. The greater body weight of an obese individual necessarily results in a greater mechanical impact if a fall happens, although this may be offset by the ability of superficial fat to cushion falls (below).

Lack of exposure to sunlight. During the 1930s, it was noted with surprise that rickets occurred not only in impoverished British children with limited access to butter and milk, but also in wealthier offspring who were living in smog-laden cities. The problem was finally traced to the blocking of solar radiation by airborne particulate matter, and thus a reduction in the cutaneous synthesis of Vitamin D, a key element in maintaining bone health (Rajakumar et al., 2007).

Inadequate exposure to sunlight can also occur in very cold climates, where people rarely venture outside, and the skin is covered by heavy clothing for much of the year (Rajakumar et al., 2007). Because of self-consciousness about an ungainly figure, obese people are also hesitant to expose much of their skin to sunlight, with a corresponding reduction in the cutaneous synthesis of Vitamin D (Pereiera-Santos et al., 2015; Vanlint, 2013). However, a review of behaviour among participants in the Framingham study suggests that factors other than covering of the skin probably explain much of the Vitamin D deficiency that is commonly observed in obese individuals (Cheng et al., 2010).

Adverse hormonal changes. Testosterone is important to bone health

(Mohamad et al., 2016). Blood levels of this hormone tend to be low in men who are obese. The problem has been traced to insulin-resistance associated reductions in levels of sex-hormone binding globulin, and in more severe cases of obesity there may also be effects from a suppression of the hypothalamic pituitary axis (Fui et al., 2014).

Other hormones that are synthesized in fat (adipokines and cytokines) can have an adverse effect on bone health. Adipokines (particularly adiponectin) are negatively associated with bone density, as shown in a meta-analysis of 59 studies, and high levels of adiponectin offer a significant prediction of the risk of vertebral fractures in older men (Biver et al., 2011). Adipocytes also secrete a variety of cytokines, including resistin, TNF-α, IL-1 and IL-6; these tend to uncouple bone remodeling by enhancing bone resorption and/or suppressing bone formation (Kawai, et al., 2012).

Positive factors. Against these significant influences increasing the risk of injury, we may set several positive consequences of obesity (Table 7).

Greater mechanical loading of bones. The greater mechanical loading of the bones associated with obesity has traditionally been considered as enhancing bone health (Cao, 2011).

However, in practice this potential advantage is heavily offset by negative hormonal influences (above) and the limited volume of weight-bearing physical activity undertaken by most people who are who are obese.

Padding effects. The padding effect from subcutaneous fat provides some protection to bony structures if a fall is incurred (Bouxsein et al., 2007); indeed, some have argued from studies where weights were dropped onto various thicknesses of pork fat that the thickness of fat around the hips has a greater influence on the likelihood of fractures than does bone density. However, most authors have concluded that other adverse factors, including the greater risk of falls, more than offset any padding effects from subcutaneous fat.

Favourable hormonal changes. The main positive hormonal change associated with obesity is an increased synthesis of oestradiol from adrenal steroids in post-menopausal women. A study of 588 women aged 41-60 years found a positive influence of obesity on bone density in the spine, femoral neck, and trochanter (da Silva et al., 2007). Oestrone and oestradiol levels also bear a positive relationship to bone mineral density in elderly men (Van den Beld et al., 2000).

Table 7: Factors reducing risk of mechanical injury in an obese person.

- If an obese person is physically active, the large body mass increases mechanical loading during movement, thus tending to strengthen the bones.
- Fat provides padding on regions such as the hips, helping to create a favourable ratio between impact forces and bone strength (Bouxsein et al., 2007)
- Bone density may be increased in obese individuals, because endogenous adrenal steroids are converted to oestradiol in adipose tissue
- Lower levels of sex hormone-binding globulin in the obese may increase free levels of sex steroids, favouring an increase of bone density (Lauritzen, 1996).

Table 8: Body mass, body fat and bone mineral density.

Author	Sample	Methodology	Findings	Comments
Albala, Yanez, and Devoto, (1996)	113 obese, 51 non-obese post-menopausal women	Dual photon x-ray absorptiometry	Femoral neck +11.3%, Ward's triangle + 11.5%. lumbar spine +7.0%	Decreased sex-hormone binding globulin in obese
Casale et al. (2016)	83 Pacific Island women aged 16-45 yr	DXA	Lean mass increases bone density, fat mass decreases it	Whole body plethysmography for body composition
Castro et al. (2005)	3206 post-menopausal U.S. women	DXA	In white women only, decreased odds of low bone density, 0.9 for 1 unit BMI	Bone mineral density lower in Afro-American women
Compston et al. (1992)	97 post-menopausal British women	DXA	Correlation of 0.48 between bone density and fat mass	
Edelstein and Barrett-Connor (1993)	1492 "white" adults aged 55-84 yr	DEXA, single photon absorptiometry	Density of lumbar spine, hip, radius most closely related to body mass	Bio-impedance measure of body fat had small positive effect, esp. in F subjects
Felson et al. (1993)	693 women, 439 men, age 76 yr	Dual photon absorptiometry	Obese women- greater density at all sites, men greater density at weight-bearing sites	Obesity explained 8.9-19.8% variance in men, 2.8-6.9% in women,
Ho-Pham et al. (2010)	210 post-menopausal women aged 50-85 yr	DEXA	Lean mass and fat mass independent predictors of bone density; first more important	Lean mass and fat mass from whole body scans
Kang et al. (2015)	502 men aged 20-89 yr	DEXA	Percent body fat negatively related to bone density	
Kim et al. (2010)	907 women aged 60-79 yr	DEXA, osteocalcin data	Bone density positively related to BMI, but negatively related to % body fat and waist circumference	
Klein et al. (1998)	18 obese, 30 non-obese pre-pubertal or early pubertal children	DEXA	Bone density similar in obese and controls	Fat did not seem to influence oestradiol levels in this sample
Liu et al. (2014)	471 women aged 18-67 yr	DEXA	Fat had negative effect on bone density if >33% fat	Body composition from DEXA
Ribot et al. (1987)	176 women aged 45-71 yr	Dual photon absorptiometry. calceotonin data	Both pre- and post-menopausal obesity had favourable effect on bone density and bone turnover	Even moderate obesity protected against post-menopausal bone loss
Rocher et al. (2008)	20 pre-pubertal obese children and 23 controls	DXA	Crude values show greater bone density in obese, but lower than controls after adjusting for body mass and lean mass	
Silva et al. (2007)	588 post-menopausal women aged 41-60 yr	DXA	Obese individuals had lower risk of osteoporosis in lumbar spine and femoral neck	BMI also strong predictor of trochanter density

Empirical data on body mass, body fat and bone density

Data on bone density provide one empirical method of weighing the overall negative and positive consequences of obesity for bone health. Early empirical research (Table 8) suggested that those who were obese had a higher bone mineral density. Some subsequent research has continued to support this conclusion, although in such studies obesity has usually been diagnosed on the basis of BMI rather than on measures of body fat content. Where both lean tissue mass and fat mass have both been determined, they generally have had opposite effects on bone density.

Thus, a study of 113 obese and 50 non-obese post-menopausal women showed that obese individuals had a greater bone mineral density than their non-obese peers (Albala et al., 1996). Likewise, data from the Framingham study showed a positive association between BMI and bone mineral density at all of the sites examined in 693 women, and at the weight-bearing bones in 439 men (Felson et al., 1993), with dual photon measurements explaining a substantial fraction of the total variance in bone density.

However, an important paper by Kim et al. (2010) showed that although bone density was positively correlated with body mass index, it showed *negative* relationships to more direct measures of body fat content such as waist circumference in the same subjects. Edelstein and Barrett-Connor (1993) also included bio-impedance measures of body fat content in their study, and in a multivariate analysis they showed a substantial positive effect from body mass, with a weak positive influence also from fat mass (especially in women).

Likewise, Casale et al. (2016) found opposing relationships of bone mineral density to lean tissue mass and fat mass in pre-menopausal Pacific Island women. Further, a small study of pre-pubertal children (Rocher et al., 2008) found higher bone densities in those who were obese, but after adjusting data for total body mass and lean tissue mass, densities were lower than in controls.

Liu et al. (2014) suggested that although the added mass from moderate obesity might in itself tend to enhance bone mineral density, there was a threshold (which they set at 33% body fat) above which other consequences of fat accumulation had a negative effect.

Empirical data on relationships between the incidence of fractures and obesity. The relationship between the incidence of fractures and body fat content provides a second overall evaluation of the advantages and disadvantages of an above-average body fat content, and has considerable practical clinical significance. Recent research has emphasized that despite the potentially positive effects of moderate obesity upon bone density, particularly in post-menopausal women, the overall incidence of fractures is not any lower in those who are obese than in those of normal body weight, and particularly in the lower extremities the risk is actually above average in obese individuals. Thus, a 2-year prospective study of 44,534 post-menopausal women across 10 countries found that although there was an increase in the risk of fractures amongst those who were underweight (Table 9), in those with a BMI > 30 kg/m² , the overall incidence of fractures, at 6.4%, was closely similar to that for those who were of normal weight or overweight (6.8%). Moreover, Compston (2011) noted that if fractures

Table 9: The incidence of fractures seen over a 2-year follow-up in 44,534 post-menopausal women, classified by their initial BMI status. Based on the data of Compston et al., 2011.

Initial BMI (kg/m^2)	2-year fracture incidence (%)
Underweight (< 18.5)	7.3
Normal weight or overweight (18.5-30)	6.8
Obese (>30)	6.4

were classified by body site, several reports showed that the risk of fractures of the ankle and leg was actually greater in the obese than in those of normal weight (Compston et al., 2011), probably because the obese were falling more heavily.

Thus, Berkvist et al. (2009) found that in a sample of adults ranging in age from 20-84 years who reported to the local hospital emergency room with ankle fractures, the BMI was on average 1.9 units greater than in a control group of men and women . Again, Nielson et al. (2010) noted that in a sample of 5995 men aged > 65 years with non-spinal fractures, the incidence of was greater than average among obese individuals. In models adjusted for age, race and bone mineral density, the hazard ratios for fractures were 1.29 for those with grade I obesity, and 1.94 for those with grade II obesity. Nevertheless, a part of this association reflected the poor physical function of those who were obese; hazard ratios for those with grade I and grade II obesity dropped to 1.12 and 1.44 when data were adjusted for differences in walking pace and limitations of mobility.

Several other reports (primarily on post-menopausal women) have underlined that obesity increases the risk of fractures in some parts of the body, particularly the extremities, but analyses often show also a reduction in the incidence of hip fractures (Table 10). Some reports have complicated the interpretation of their data by adjusting the risk of fractures for the individual's bone density (for example, Ishii et al., 2014). Unfortunately, many studies have also based fat assessments on BMI rather than direct measurements of body fat content. Where both BMI and body fat have been determined (for example, Laslett et al. (2012)), any association with the risk of vertebral fractures has appeared greater for BMI than for percentage body fat.

Problems of thermoregulation in the obese. Fat in itself has a low thermal conductivity, and it also has only a limited vascularity. Thus the thermal insulation of the body increases in proportion to the thickness of the subcutaneous fat layer. As discussed below, this may confer some advantage when undertaking ultra-long distance swimming in frigid waters, but in most circumstances the necessary protection against cold is better achieved by layers of clothing that can be removed when the rate of body heat production is increased sufficiently by exercise.

Sub-cutaneous fat is a particular handicap when a person is attempting to exercise at high environmental temperatures, as in such individuals a

Table 10. Impact of obesity upon the risk of fractures.

Author	Sample	Fat assessment method	Findings
Beck et al. (2009)	78,013 women aged 50-79 yr	BMI	Longitudinal study. Increased fractures of lower extremity, reduced fractures of hips and vertebrae
Bergkvist et al. (2009)	79 women, aged 29-47 yr	BMI	Cross-sectional study. Increased ankle fractures in obese
Compston et al. (2011)	60,393 women > 55 yr	BMI	Longitudinal study. Fracture incidence of obese increased in leg and ankle, but reduced at wrist
Gnudi et al. (2009)	2235 post-menopausal women	BMI	Cross-sectional study. Increased fractures of humerus, reduced fractures of hip in obese
Hsu et al. (2006)	2248 women, 7137 men aged 25-64 yr.	% body fat	Cross-sectional study. Increased non-spinal fractures in obese (men only)
Johansson et al. (2014)	389,610 women aged 20-105 yr	BMI	Meta-analysis. Hugh BMI a risk factor for arm fractures
Kim et al. (2009)	907 women aged 50-80 yr	%body fat, waist circumference	Cross-sectional study. Increased fractures of vertebrae in obese
Laslett et al. (2012)	522 women, 489 men aged 50-80 yr	BMI, total fat, waist circumference	Cross-sectional study. Increased fractures of vertebrae in obese women, but decreased in obese men.
Moayyeri et al. (2012)	8319 women, 6470 men aged 42-82 yr	% body fat	Longitudinal study. Decreased hip fractures in obese women, no effect of obesity in men
Nielson et al. (2010)	5995 men aged >65 yr	BMI	Longitudinal study. Increased of hip and non-spinal injuries in obese
Pirro et al. (2010)	362 women	BMI	Cross-sectional study. Increased fractures of vertebrae in obese
Prieto-Alhambra et al. (2012)	832,755 women aged > 50 yr	BMI	Cross-sectional study. Increased fractures of humerus, reduced fractures hips and pelvis in obese.

greater proportion of the total blood flow must be diverted from the muscles to the skin, in an attempt to dissipate metabolic heat (Haymes et al., 1974; Vroman et al., 1983). Fat people face the double challenge of the need to perform more physical work than a slimmer person during any form of body movement, and also greater difficulty in heat elimination (Tables 11 and 12).

Table 11: Reports that obesity is a factor predisposing to heat stress.

- Rigorous military training (Chung and Pin, 1996, Gardner et al., 1996; Wallace et al., 2006)
- Hot industrial environments (Donoghue and Bates, 2000; Leithead and Lind, 1964).
- Environmental chamber tests (Bar-Or et al.,1969; Dehghan et al., 2013; Epstein et al.,1983; Havenith et al., 1998; Havenith and Van Middendorp, 1990; Haymes et al., 1975; Hayward et al.,1986)
- Elderly people during heat waves (Green et al., 2001; Vandentoreen et al., 2006)

Fat people are thus particularly liable to collapse when they exercise in very hot environments (Adams, 1992). This has been most evident during arduous military training programmes. One study of U.S. soldiers found that the odds ratio of developing a heat-related disorder was 3.53 in personnel with a BMI >27 kg/m^2 (Chung and Pin, 1996). Gardner et al. (1996) and Wallace et al. (2006) also noted that in Marine recruits, the incidence of heat illness during rigorous training exercises increased in direct proportion to the individual's BMI at recruitment. Nevertheless, the interpretation of these findings is somewhat complicated by the fact that obese recruits also tended to be substantially less physically fit than their peers.

Likewise, obese workers are likely to face problems of heat stress in industries where heavy physical work is combined with exposure to a hot environment. For instance, in Australian deep mining operations, the odds ratio of developing an acute heat disorder was 3.63 in employees with a BMI of 32-37 kg/m^2, relative to individuals with a BMI <27 kg/m^2 (Donoghue and Bates, 2000). Leithead and Lind (1964) considered the handicap of obesity to be such that wherever possible obese individuals should not be employed in hot environments.

Experimental studies of individuals working in environmental chambers have also shown consistently that environmental stress occurs earlier in obese individuals than in their leaner peers who are working at the same intensity and in the same climate (Bar-Or et al., 1969; Epstein et al., 1983; Havenith and Van Middendorp, 1990; Hayward et al., 1986). One report from Iran noted a working heart rate of 127 beats/min in those with a BMI > 25 kg/m^2, as compared with only 108 beats/min in individuals who were performing the same task but had a BMI<25 kg/m^2 (Dehghan et al., 2013).

During summer heat waves, many elderly individuals face heat stress and even death when living quietly in homes that lack air conditioning. One would anticipate again that the problem would be exacerbated for those who are obese, but because the number of deaths in any one episode is relatively small, information on such a linkage is limited. During the French heat wave of 2003, the odds ratio of death was doubled in those who were obese (Vandentoreen et al., 2006). Obesity was also one of the contributory factors in an analysis of heat-related deaths in Australia over an 8-year period (Green et al., 2001).

Table 12: Changes in the summed thickness of 3 skin-folds (mm) as seen in a crossover trial of fat loss. Young men undertook one week of walking 2.5 hours per day at -40°C (COLD) versus a similar volume of exercise performed for one week at normal ambient temperatures (CONTROL). Based on the data of O'Hara et al. (1977).

Group & environmental sequence	Skin-fold change (mm) Week 1	Significance of change	Skin-fold change (mm) Week 2	Significance of change
A (control/cold)	-2.0	ns	-9.3	$p < 0.001$
B (cold/control)	-6.9	$p < 0.001$	+3.0	$p < 0.05$

Protection against excessive heat loss in cold environments. In contrast to the problems encountered when working in a hot environment, those who are obese might be thought to have some advantage when exposed to very cold conditions. For any given thermal gradient, a large body mass (whether fat or muscle) takes longer to cool than a smaller one, and superficial fat also increases the individual's insulation because fat has a poor thermal conductivity. Further, there is greater possibility of generating heat in brown fat, although the relative amount of brown fat is not necessarily greater in someone who is obese.

Effect of body mass upon the rate of heat loss from the body. Heat loss from the human body is complicated by considerations of body shape, surface area and a film of still air or water that, under resting conditions, remains in immediate contact with the skin surface. If the body as a whole were to keep strictly to Newton's law of cooling for a spherical object, then the rate of heat loss would be proportional to the temperature gradient between the body surface (T_b) and the ambient air or water (T_a), multiplied by the exposed surface area (A) and divided by the product of body mass (m) and the average specific heat capacity of the body tissues (s_h):

$$\text{Heat loss} = kA(T_b - T_a)/ms_h$$

Sloan and Keatinge (1973) noted that there are substantial inter-individual differences in the ratio of surface area to body mass, and that these differences influence a person's tolerance of a cold environment. However, the body area is roughly proportional to the 2/3rd power of body mass:

$$A = m^{2/3}$$

Thus, if the body mass were to increase from m_1 to m_2 (for example, with an accumulation of muscle or body fat), then the surface area would change from A_1 to A_2 and:

Delta heat loss (%) = $[(A_2/m_2 - A_1/m_1)/(A_1/m_1)]$

or $[(A_2/m_2)/(A_1/m_1)] - 1$

Thus, if $m_2 = 1.1\ m_1$ (a 10% increase of body mass), then the change in rate of heat loss should become:

$$[(1.1^{2/3}A_1/1.1m_1)/(A_1/m_1)] - 1$$

or

$$[(1/1.1^{1/3}) - 1] \times 100 = -3.1\%\ \text{change}$$

In practical terms, a person who previously could swim in cold water for an hour before body temperature fell to a dangerously low level should (other factors being equal) be able to continue swimming for a further 3.1% of 60 minutes (1 min 52 s) with a 10% increase of body mass, whether the weight gain was due to an accumulation of fat or lean tissue.

Heat loss by conduction. Heat loss from the skin surface occurs through a combination of convection, conduction, radiation and evaporation. Under resting conditions (and in the absence of wind or wave motion), then the surrounding film of air or water in immediate contact with the skin is motionless, and it provides a substantial convective barrier to heat loss. However, for the active individual who is running in cold air or swimming in cold water, the still film is largely dispersed. Conduction through the underlying tissues to air or water at the skin surface imposes an important limitation on the rate of heat loss from the body, and here the modulating influence of subcutaneous fat is important (Buskirk et al., 1963; Quarade, 1963; Wyndham et al., 1968) (Figure 2). Quarade (1963) noted that during cold exposure, the metabolism of a normal weight person was increased by 33%, but- presumably because of greater insulation at the skin surface- the increase in those who were obese was only 11%. Quarade (1963) reported corresponding inter-group differences in skin and deep tissue temperatures. The lesser heat loss and the smaller requirement to increase heat production could be one factor helping to perpetuate obesity (Jequier et al., 1974).

Depending in part on skin-fold thickness, Smith and Hanna (1975) found that the heat loss of stationary subjects was on average 3.3 times greater in water than in air. Such differences are probably even greater for active individuals, since they disturb the film of water or air in immediate contact with the skin surface. Subcutaneous fat has indeed been

Figure 2: Body fat offers buoyancy and thermal insulation, important assets for long-distance swimmers such as Marilyn Bell, who crossed both Lake Ontario and the English Channel. Source: http://lostswimming.com/2014/02/03/swimmer-marilyn-bell-recalls-

considered an important source of insulation for athletes who are attempting to swim long distances in open water. In fact, some cross-channel swimmers have sought to increase their thermal protection by covering their bodies with a layer of up to 10 mm of grease.

However, there are only limited data on the relative thermal conductivities of body fat and muscle. Early analyses, based on excised tissue samples, suggested that muscle conducted heat 1.8 times as well as fat (Hatfield and Pugh, 1951). Mitchell et al. (1970) set the thermal conductivity of fat at 0.21 W/[m.°C], as opposed to 0.50 W/[m.°C] for muscle. A ratio of this order has been adopted in the more recent analyses of Zhang et al. (2001). Nevertheless, the thermal conductivity of fat and muscle both depend greatly on the rate of tissue perfusion; in circumstances where muscle

is poorly perfused, it can contribute a fair amount to the overall insulation of the human body (Cohen, 1977).

In vivo measurements suggest that the differential conductivity of fat and muscle persist when these tissues have their normal blood supply (Cohen, 1977). But ignoring issues of perfusion, what is the effect of an increase in the percentage of body fat on the thickness of the body's layer of superficial fat? If fat has a density of D, then the total volume of body fat V is given by the product of body mass m and the fraction (f) of this mass that attributable to fat:

$$V = mf/D$$

Ignoring internal deposits of fat, the average thickness of subcutaneous fat can then be approximated as:

$$t = mf/DA$$

As in earlier calculations (above), If fat mass is increased by 10%, $m_2 = 1.1\ m_1$, and $A_2 = 1.1^{2/3}\ (A_1)$, so that the resulting percentage change in fat thickness is given by:

Delta thickness (%) = $[(1/1.1^{1/3}) - 1] \times 100 = 3.1\%$ change

Empirical data on heat loss and body build. Early empirical data were collected on subjects with substantial differences of body build. Cannon and Keating (1960) found that the metabolic rate of fat men rose less than that of their thinner colleagues when they were immersed in water at a temperature of 33° C or lower. Although there may also have been inter-group differences in skin reflexes, they attributed most of the difference in metabolic stimulation to the greater insulation of the obese men. The obese group were thus able to stabilize body temperatures to water temperatures as low as 10-12° C.

When swimming in cold water, rates of cooling were also found to be substantially slower in fatter subjects. Thus, Keatinge et al. (2001) compared the tolerance of cold water (9-11° C) among a group of experienced swimmers. Although the final rectal temperatures did no differ between groups, those with less subcutaneous fat were able to spend much less time in the water than their fatter peers. Tolerance times ranged from 23 minutes (in a person with an average skin-fold reading of 8.6 mm) to 65 minutes (for a person with an average skin-fold of 21.5 mm). Knechtle et al. (2009) also found that the tolerance of swimming in water at 4° C was almost doubled by even a small advantage of skin-fold thickness (average of 8 folds, 17.4 vs. 15.6 mm).

Branningan et al. (2009) studied 75 swimmers who were immersed in water at 19-22 ° C for 5-12 hr. They concluded that for each additional unit of BMI there was a 43% decrease in the rate of developing hypothermia. However, their study has been criticized on technical grounds; the use of BMI as an index failed to distinguish between muscle and fat, and most of the body temperatures were measured with oral rather than rectal thermometers.

Pugh and Edholm (1955) found that the typical skin-fold thickness (the average of 6 folds) was 13.2 mm in male cross-channel swimmers and 15.0 mm in female swimmers, much higher than in many other categories of athlete, and also relative to values of 7.8 and 9.0 mm seen in their control groups.

Fat as a source of metabolic heat and a component of cold acclimation. White adipose tissue has quite a low metabolic rate compared with most body tissues,

only around 17 kJ/kg/day, although this value becomes larger if it is expressed relative to the mass of the thin film of protein surrounding the adipocyte's fat globule.

Brown adipose tissue has a specific function of increasing body heat production when an animal or person is challenged by a cold environment (Cannon and Nedergaard, 2004; Shephard, 1985). At least in small mammals, it is an important site of non-shivering thermogenesis during cold exposure. It often accounts for no more than 2% of an animal's body mass, but when stimulated by noradrenaline secretion, it can double the basal metabolic rate (Foster and Frydman, 1979).

Extent of brown fat deposits in humans. The presence of brown fat in young children is well documented, but extent of its role in the adaptation of adult humans to cold environments and any modification of this role by obesity are more controversial issues. Brown fat is certainly important in maintaining the thermal balance of young children, before they become physically very active. Some investigators have suggested that brown fat has little importance in mature individuals, since when they are exposed to severe cold they can increase the amount of clothing that is worn, the surface area/body mass ratio is more favourable to thermal balance than in a youngster, and they can augment their heat production by engaging in vigorous physical exercise (Cunningham et al., 1985; Jung et al., 1988). Nevertheless, other mammals also engage in physical activity, and yet they maintain brown fat as adults, and indeed seem able to augment this tissue during cold acclimation (Chaffee and Allen, 1973).

Cunningham et al. (1985) identified small amounts of the chemical marker of brown adipocytes, uncoupling protein, in the perinephric fat of adults; however, they estimated that the total amount of brown adipose tissue in an adult was so small that it could account for only 0.2% of the thermal response to noradrenaline. Nevertheless, some more recent evidence suggests that adults retain sufficient brown fat to make a significant contribution to their cold tolerance (Cypess et al., 2009; Nedergaard and Cannon, 2010). Further, the quantity of brown fat in the body may increase during cold acclimation.

Unfortunately for those who are obese, it seems that they have less brown fat than those who are leaner (Cypess et al., 2009; Himms-Hagen, 1979; Nedergaard and Cannon, 2010), and indeed the resulting difference in metabolic rate may have been one factor contributing to their development of obesity (Himms-Hagen, 1979; Nedergaard and Cannon, 2010).

Heat generation in brown fat. Large amounts of heat are generated in brown fat in essence by the operation of an ineffective sodium pump at the cell membranes of the adipocytes. Normally, metabolic processes build up a proton gradient at cell membranes. However, during a period of cold exposure, the secretion of adrenaline stimulates brown fat cells to produce an uncoupling protein 1 (UCP1), and this dissipates the gradient that otherwise would have been generated by the NADH-powered pumping of electrons across mitochondrial membrane (Figure 3).

The process, also known as "futile cycling," is well-developed in small mammals, where the extra heat that is produced and the resulting decrease in overall metabolic efficiency have
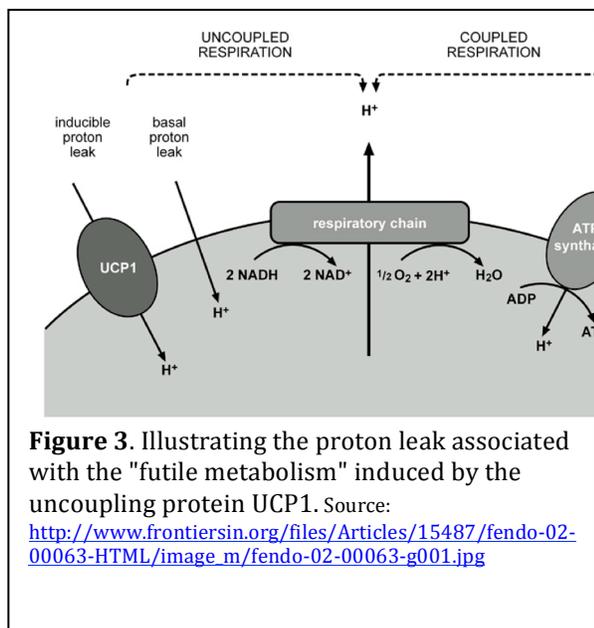
Figure 3. Illustrating the proton leak associated with the "futile metabolism" induced by the uncoupling protein UCP1. Source: http://www.frontiersin.org/files/Articles/15487/fendo-02-00063-HTML/image_m/fendo-02-00063-g001.jpg

considerable significance in terms of non-shivering thermogenesis, acclimation to cold conditions, and a facilitation of fat loss during periods of overfeeding (Girardier and Stock, 1983).

Nevertheless, there is evidence that the FTO gene (Srivastnava et al., 2016), which has a strong association with obesity, can mediate a browning of white fat in human adults (Claussnitzer et al., 2015; Pigeyre et al., 2016), and the mRNA of the UCP1 responsible for futile cycling has been detected in adult white adipose tissue (Cinti, 1999). Furthermore, the injection of noradrenaline induces substantial non-shivering thermogenesis in adults (Kurpad et al., 1994).

Not all of the added heat production of non-shivering thermogenesisis is necessarily generated by brown fat, but radio-isotope studies have shown that with controlled cold exposure much of the metabolism of radio-actively tagged fatty acids occurs in those regions of the body where brown adipose tissue is located. One report indicated that during acute exposure to a cold environment there was a 17% increase in the radio-density of brown fat, but only a 3% change in the radio-density of white fat (Ouellet et al., 2012).

Reactions of body fat to cold exposure. In a cross-over trial with 2 small groups of young men, O'Hara et al. (1977) made the intriguing discovery that the rate of fat loss during a programme of vigorous exercise was increased if the physical activity was performed in a very cold environment (Table 12). Their subjects exercised for one week, 2.5 hours per day, at a temperature of -40°C (in still air, and when wearing standard military arctic clothing). In the second leg of the trial, they performed the same exercise for 2.5 hours/day at normal ambient temperatures.

In support of the data of O'Hara et al. (1977), van der Lans et al. (2013) exposed a small group of adults to a temperature of 15-16° C for 6 hours/day. This challenge significantly increased both their total energy expenditures over a 10-day period (in women, from 6.2 to 6.9 MJ/day, and in men from 7.6 to 8.5 MJ/day) and it also increased their non-shivering thermogenesis, although no "browning" of white adipocytes was detected. On the other hand, a small trial where obese subjects performed 40 sessions of quite light exercise (insufficient to augment their maximal oxygen intake) while immersed in cool water (17-22° C) did not induce any significant loss of body fat from either white or brown adipocytes (Sheldahl et al., 1982).

The role of beige cells. There is growing evidence that the capacity to synthesize uncoupling protein can be induced in a sub-population of white adipose tissue termed "beige" or "brite" adipocytes, if they are exposed to either challenging cold conditions (Frontini and Cinti, 2010)

or the prolonged administration of adrenergic compounds (Fisher et al., 2012). Indeed, these beige cells may account for much of the supposed brown fat that has been described in adult humans.

White adipocytes can be transformed into beige cells over as little as 24 hours, and reversion to white adipocytes can occur over a few weeks of exposure to a more temperate climate (Gospodarska et al., 2015). However, there is as yet little evidence on the relative importance of beige adipocytes in obese people versus those of normal body mass.

Adipose tissue as a source of buoyancy
Fat has a density of around 0.90 g/mL, so that a person with a large body fat content has a much greater buoyancy than a lean and muscular athlete. I remember that in our Kinesiology programme at the University of Toronto a number of well-muscled students had great difficulty in meeting a mandatory first-year swimming requirement for their Red Cross certification. On entering the pool, the muscular but lean students quickly found themselves sinking underwater, because their overall specific gravity was high relative to that of their more obese classmates. Forensic studies have thus shown that accidental drowning is a more common occurrence for thin than for obese individuals (Roger, 2017).

Because buoyancy reduces the load on aging joints, pool exercises are an effective and well-accepted form of physical activity for those who are obese, particularly if they suffer from osteo-arthritis of the knee or hip joints (Avelar et al., 2018; Brandt, 1998; Gill et al., 2009). However, a study of adolescent swimmers found no significant correlations between body fat content

and 100 m swim performance (Geladas et al., 2005).

Moderate obesity could in theory be an asset to the long distance swimmer, not only because of greater buoyancy, but also because of increased protection against cold; as noted above, the latter factor seems important in ultra-long events. Whereas most female athletes have a body fat content of 5-15%, that of female long-distance swimmers tends to fall in the range 14-24% (Shephard et al., 1973). However, empirical data show little influence of body fat content on swimming performance over most competitive distances. No correlation was seen between body fat content and the distance covered during a 12-hour swim (Knechtle et al., 2008), and a study of 2012 Olympic contestants found little correlation between BMI and performance over distances from 50 m to 10,000 m (Gagnon et al., 2018).

Although an increased body fat content lifts a swimmer higher out of the water, tending to reduce drag, this effect seems to be more than offset by an increased body cross-section to forward movement. In the one available experimental study, the buoyancy of 10 university athletes was increased by fitting latex/air balloon pads beneath their swimsuits. The net effect was to increase times for a 50-yard swim by 3.1% (Lowensteyn et al., 1994). Likewise, Siders and colleagues (1993) found a positive correlation of 0.35 between body fat content and 100 m swim times in female (but not male) collegiate level swimmers.

It remains to be established whether an increase of normal subcutaneous body fat would have a similar negative effect to externally applied buoyancy pads, but in any event a swimmer can increase buoyancy without accumulating fat if he or she develops a large vital capacity, and

maintains a large mean volume of air in the lungs while swimming.

Adipose tissue as an energy reserve during prolonged exercise Although athletes seek to develop carbohydrate stores as a source of energy for brief bouts of heavy physical activity, even optimized body reserves of carbohydrate are quite limited. Typically, some 400 g of glycogen is found in the skeletal muscles, and a further 100 g in the liver (Shephard, 1982). Each gram of glycogen provides about 16.5 kJ of energy, so that even if the entire 500 g reserve were to be depleted, it would only provide some 8 MJ of energy. In practice, the sedentary person rarely depletes this reserve, but the endurance athlete can use up most of the stored carbohydrate by undertaking around 90 min of sustained exercise at an intensity demanding of 75% of his or her maximal aerobic power.

Once carbohydrate stores have been depleted, the aerobic contraction of muscle is sustained largely by the metabolism of fat, including a breakdown of both the small amounts of fat contained within resting muscle fibres, and a lipolysis of the triglycerides found in adipose tissue. Over an event such as a marathon or ultra-marathon run, a substantial part of the energy required to sustain such activity depends on the relatively slow process of lipolysis. During sustained and vigorous physical activity, increased quantities of catecholamines are secreted, and their beta-adrenergic stimulatory action (Arner et al., 1990; Horowitz and Klein, 2000; Shephard and Johnson, 2015) up-regulates the hormone sensitive lipase, releasing free fatty acids into the blood stream. The fatty acids are initially largely (99%) bound to albumin, before being conveyed to liver and muscle. Subsequently, they cross the cell wall (which also contains fatty acid binding proteins), and then enter the Krebs metabolic cycle, providing the energy needed by the liver and working muscles. Perhaps because of the needs of lactation, fat mobilization (as monitored by accumulation of glycerol in the plasma) occurs more readily in women than in men, and in women the process is also more marked in the abdominal fat (Blaak, 2001; White and Tchoukalova, 2014).

The neutral fat content of adipose tissue provides a total reserve of at least 500 MJ of energy, even in a relatively thin person (20% fat, 15 kg of fat). In this regard, fat stores have one important advantage over carbohydrate: a high energy density (Joules per g). Fat thus provides a much less heavy means of storing energy than if an equivalent metabolic reserve were to be carried in the form of glycogen and its associated water molecules.

Unfortunately, the human brain cannot metabolize triglycerides, nor can muscle if its oxygen supply is compromised. Thus, there is a continuing need for glucose to sustain both cerebral function and anaerobic muscular activity. Glucose must be synthesized in the liver by the process of gluconeogenesis, drawing upon circulating triglycerides, glycerol and amino acids derived from a breakdown of skeletal muscle (Shephard and Johnson, 2015). Mobilization of the stored neutral fat is mediated by a variety of hormones (glucagon, adrenaline, noradrenaline, growth hormone, and cortisol), acting on two main enzyme systems (hormone-sensitive lipase and lipoprotein lipase).

Despite the potential of body fat as a long-term energy reserve, its practical value is limited. Few people are likely to face the need of surviving for several weeks without eating.

Discussion and Conclusions

Although popular opinion sometimes regards a moderate level of obesity with indulgence, comparisons between lean and obese individuals show many that there are important disadvantages to allowing an excess of body fat to accumulate in the body. Moreover, these disadvantages include not only negative psychosocial influences and an increased risk of chronic ill health, but also problems in many domains of biomechanical and physiological function. A few positive physiological consequences of obesity have also been suggested, but many of these supposed benefits do not stand up to close examination.

The mechanical efficiency of movement is impaired by obesity, with an increased energy cost of body displacement, and a consequent reluctance to engage in that minimum volume of daily physical activity that is important to health maintenance. Agility, flexibility and balance are impaired, with a reduction of functional capacity in terms of physically demanding employment, and a diminished ability to engage in enjoyable physically active leisure activities. Deposition of fat around the airways leads to nighttime bouts of apnoea, and a resulting reduction of daytime mental alertness. Finally, the insulating properties of sub-cutaneous fat lead to thermal overloading in a hot environment, an important consideration in times of rising global temperatures.

Among possible advantages, there have been suggestions that body fat can protect the hip bones in the event of a fall, but against this must be set the poorer agility and balance of an obese person, and an increased tendency to falls. Subcutaneous fat may increase thermal protection during exposure to severe cold, but since the amount of insulation required depends greatly upon the amount of physical activity that is being performed, it is preferable to obtain the necessary thermal protection from readily adjustable layers of clothing, rather than rely upon a thick and often excessive layer of fat. Brown fat plays some role in cold acclimation, but again the fat person seems at a disadvantage, since the brown fat response appears to be attenuated in those who are obese. Subcutaneous fat provides a thermal advantage to the very rare person who attempts an ultra-endurance swimming event such as a Cross-Channel swim, but again it seems possible that equal benefit could have been obtained by the short-term external application of an equivalent layer of grease. Further, the greater buoyancy of an obese person may increase flotation, but this benefit seems offset by a greater drag as the body rises out of the water. Even the energy reserves offered by an excess of body fat have little practical value unless a period of prolonged starvation is anticipated.

Given the extensive range of chronic illnesses provoked by fat accumulation, these seem no significant physiological arguments to warrant ignoring the need for measures to maintain an optimal body mass.

Acknowledgments

Author's qualifications

The author's qualifications are as follows: Roy J. Shephard, C.M., Ph.D., M.B.B.S., M.D. [Lond.], D.P.E., LL.D., D.Sc., FACSM, FFIMS.

References

Adams, T. (1992). *Guidelines for surviving heat and cold*. Champaign, IL: Stipes.

Albala, C., Yanez, M., Devoto, E., Sostin, C., Zeballos, L., and Santos, J.L. (1996). Obesity as a protective factor for post- menopausal osteoporosis *Int J Obesity Relat Metab Disord, 20,* 1027-1032. PMid:8923160

Arner, P., Kriegholm, E., Engfeldt, P., and Bolinder, J. (1990). Adrenergic regulation of lipolysis in situ at rest and during exercise. *J Clin Invest, 85,* 893-898. https://doi.org/10.1172/JCI114516

Astephen, J.L., and Deluzio, K.J. (2005). Changes in frontal plane dynamics and the loading response phase of the gait cycle are characteristic of severe knee osteoarthritis application of a multidimensional analysis technique. *Clin Biomech, 20,* 209–217. https://doi.org/10.1016/j.clinbiomech.2004 .09.007

Avelar, I.S., Soares, V. Barbosa, R.C., Andrade, S.R., Silva, M.S. (2018). The influence of a protocol of aquatic exercises in postural control of obese elderly. *Rev Adalusia Med Deporte, 11(2),* 69-74. https://doi.org/10.1016/j.ramd.2016.01.00 3

Ayub, B. and Bar-Or, O. (2003). Energy cost of walking in boys who differ in adiposity but are matched for body mass. *Med Sci Sports Exerc, 35(4,)* 669-674.

Bar-Or, O., Lundegren, H.M., and Buskirk, E.R. (1969). Heat tolerance of exercising obese and lean women. *J Appl Physiol, 54,* 403-409. https://doi.org/10.1152/jappl.1969.26.4. 403

Beck, T.J., Petit, M.A., Wu, G., LeBoff, M.S., Caulkey, J.A., and Chen, Z. (2009). Does obesity really make the femur stronger? BMD, geometry, and fracture incidence in the women's health initiative-observational study. *J Bone Miner Res, 24(8)*, 1369–1379.https://doi.org/10.1359/jbmr.09037

Bergkvist, D., Hekmat , K., Svensson, T., and Dahlberg, L. (2009). Obesity in orthopedic patients. *Surg Obes Relat Dis, 5(6),* 670-672. https://doi.org/10.1016/j.soard.2009.05.0 14

Biver, E., Salliot, C., Combescure, C., Gossec, L., Hardouin, P., Legroux,-Gerot, I., and Cortet, B. (2011). Influence of adipokines and ghrelin on bone mineral density and fracture risk: a systematic review and meta-analysis. *J Clin Endocrinol Metab, 96(9)*, 2703-2713. https://doi.org/10.1210/jc.2011-0047

Blaak, E. (2001). Gender differences in fat metabolism, *Curr Opin Clin Nutr Metab Care, 4(6),* 499-502. https://doi.org/10.1097/00075197-200111000-00006

Block, A.J., Boysen, P.G., Wynne, J.W. , and Hunt, L.A. (1979). Sleep apnea and oxygen desaturation in normal subjects. A strong male predominance. *N Engl J Med, 300,* 513-517. https://doi.org/10.1056/NEJM197903083 001001

Bonomi, A.G., Soenen, S., Goris, A.H.C, and Westerterp, K.R. (2013). Weight loss induced changes in physical activity and active energy expenditure in overweight and obese subjects before and after energy restriction. *PLoS ONE, 8(3), e59641*. https://doi.org/10.1371/journal.pone.005 9641

Bouxsein, M.L., Szulc, P., Munoz, F., Thrall, E., Sornay-Rendu, E., and Delmas, P.D. (2007). Contribution of trochanteric soft tissues to fall force estimates, the factor of risk, and prediction of hip fracture risk. *J Bone Miner Res, 22(6),* 825-831. https://doi.org/10.1359/jbmr.070309

Bovet, P., Auguste, R., and Burdette, H. (2007). Strong inverse association between physical fitness and overweight in adolescents. *Int J Behav Nutr Phys Activ, 4*: 24. https://doi.org/10.1186/1479-5868-4-24

Brandt, K.D. (1998). The importance of nonpharmacologic approaches in management of osteoarthritis 1. *Am J Med, 105(1,)* 39S-44S. PMID 9715833

Brannigan, D., Rogers, I.R., Jacob, I., Montgomery, A., Williams, A., and Khngure, N. (2009). Hypothermia is a significant medical risk of mass participation long distance open water swimming. *Wilderness Environ Med, 20(1),* 14-18. https://doi.org/10.1580/08-WEME-OR-214.1

Bresnitz, E.A., Goldberg, R., and Kosinski, R.M. (1994). Epidemiology of obstructive sleep apnea. *Epidemiol Rev, 16(2),* 210-227. https://doi.org/10.1093/oxfordjournals.ep irev.a036151

Brown, J.R. (1966). The metabolic cost of industrial activity in relation to weight. *Med Serv J Canada, 22*, 262-272. PMid:5943242

Browning, R.C., Baker, E.A, Herron, J.A., and Kram, R. (1985). Effects of obesity and sex on the energetic cost and preferred speed of walking. *J Appl Physiol, 100(2)*, 390-398. https://doi.org/10.1152/japplphysiol.00767.2005

Bryant, E.S., Duncan, M.J., and Birch, S.L. (2014). Fundamental movement skills and weight status in British primary school children. *Eur J Sport Sci, 14(7),* 730-736. https://doi.org/10.1080/17461391.2013.87 0232

Burt, V., and Stunkard, A. (1964). Body weight and Achilles reflex time. *Ann Intern Med, 60(5),* 900-902. https://doi.org/10.7326/0003-4819-60-5-900

Buskirk, E.R., Thompson, R.H., and Whedon, G.D. (1963). Metabolic response to cold air in men and women in relation to body fat content. *J Appl Physiol, 18,* 603-612. https://doi.org/10.1152/jappl.1963.18.3.603

Cannon, B., and Nedergaard, J. (2004). Brown adipose tissue: Function and physiological significance. *Physiol Rev, 84,* 277-359. https://doi.org/10.1152/physrev.00015.2003

Cannon, P., and Keatinge, W.R. (1960). The metabolic rate and heat loss of fat and thin men in heat balance in cold and warm water. *J Physiol, 154*, 329-344. https://doi.org/10.1113/jphysiol.1960.sp006582

Cao, J.J. (2011). Effects of obesity on bone metabolism. *J Orthop Surg, 6,* 30 https://doi.org/10.1186/1749-799X-6-30.

Capodaglio, P., Castelnuovo, G., Brunani, A., Vismara, L., Villa, V., and Capodaglio, E.M. (2010). Functional limitations and occupational issues in obesity. *Int J Occup Safety Ergon, 16(4),* 507-523. https://doi.org/10.1080/10803548.2010.11076863

Casale, M., von Hurst, P.R., Beck, K.L., Shultz, S., Kruger, M.C., O'Brien, M., Conlon, C.A., and Kruger, R. (2016). Lean body mass and body fat percentage are contradictory predictors of bone mineral density in premenopausal Pacific Island women. *Nurients, 8(8),* 470. https://doi.org/10.3390/nu8080470

Castro, J.P., Joseph, L.A., Shin, J.J., Arora, S.K., Nicasio, J., Shtazkes, J., et al. (2005). Differential effect of obesity on bone mineral density in White, Hispanic and African American women: a cross sectional study. *Nutr Metab, 2,* 9. https://doi.org/10.1186/1743-7075-2-9

Ceschia, A., Giaconmini, S., Santarossa, S., Rugo, M., Salvadego, D., and Da Ponte, A. (2016). Deleterious effects of obesity on physical fitness in pre-pubertal children. *Eur J Sport Sci, 16(2),* 271-278. https://doi.org/10.1080/17461391.2015.1030454

Chaffee, R.R., and Allen, J.R. (1973). Effects of ambient temperature on the resting metabolic rate of cold and heat acclimated Macaca mukatta. *Comp Biochem Physiol A (Physiol), 50,* 303-306. https://doi.org/10.1016/0300-9629(75)90017-1

Chen, K.Y., Acra, S.A., Donahue, C.A., Sun, M., and Buchowski, S.M. (2004). Efficiency of walking and stepping: relationship to body fatness. *Obes Res, 12(6)*, 982-989. https://doi.org/10.1038/oby.2004.120

Cheng, S., Massaro, J.M., Fox, C.S., Larson, M.G., Keyes, M.J„ McCabe, E.L. et al. (2010). Adiposity, cardiometabolic risk, and vitamin D status: the Framingham Heart Study. *Diabetes Care, 59,* 242-248. https://doi.org/10.2337/db09-1011

Chung , N.K., and Pin, C.H. (1996). Obesity and the occurrence of heat disorders. *Mil Med, 161(12)*, 739-742. https://doi.org/10.1093/milmed/161.12.739

Cinti, S. (1999). *The adipose organ.* Milan, Italy, Kurtis SRL, pp. 1-94.

Claussnitzer, M., Dankel, S.N., Kim, K.H., Quon, G., Meuleman, W., Haugen, C. et al. (2015). FTO obesity variant circuitry and adipocyte browning in humans. *N Engl J Med, 373*, 895-907. https://doi.org/10.1056/NEJMoa1502214

Cohen, M.L. (1977). Measurement of the thermal properties of the skin: A review. *J Invest Derm, 69,* 333-338. https://doi.org/10.1111/1523-1747.ep12507965

Compston, J.E., Bhambhani, M., Laskey, M.A. , Murphy, S., and Khaw, K.T. (1992). Body composition and bone mass in post-menopausal women. *Clin Endocrinol (Oxford), 37(5),* 426-431.

https://doi.org/10.1111/j.1365-2265.1992.tb02354.x

Compston, J.E., Watts, N.B., Chapurlat, R., Cooper, C., Boonen, S., Greenspand, S., et al. (2011). Obesity is not protective against fracture in post-menopausal women. *Am J Med, 124(11)*,1043-1050. https://doi.org/10.1016/j.amjmed.2011.06.013

Corbeil, P., Simoneau, M., Rancourt , D., Tremblay, A., and Teasdale, N. (2001). Increased risk for falling associated with obesity: mathematical modelling of postural control. *IEEE Trans Neural Syst Rehabil Eng, 9,* 126-136. https://doi.org/10.1109/7333.928572

Cunningham, S.A., Leslie, P., Hopwood, D., Illingworth, P., Jung, R.T., Nichols, D.G., et al. (1985). The characterization and energetic potential of brown adipose tissue in man. *Clin Sci, 69*, 343-348. https://doi.org/10.1042/cs0690343

Cypess, A.M., Lehman, S., Williams, G., Rodman, D., Goldfine, A.B., Kuo, F.C., et al. (2009). Identification and importance of brown adipose tissue in adult humans. *N Engl J Med, 360(15),* 1509-1517. https://doi.org/10.1056/NEJMoa081070

da Silva, H.G.V., Mendonça, L.M.C., Conceição, F.L. Zahar, S.E., and Farias, M.L. (2007). Influence of obesity on bone density in postmenopausal women. *Arq Bras Endocrinol Metab, 51(6),* 943-949. https://doi.org/10.1590/S0004-27302007000600008

Davies, C.T.M., Godfrey, S., Light, M., Sargeant, A.J., and Zeidifard, A. (1975). Cardiorespiratory responses to exercise in obese girls and young women. *J Appl Physiol, 38*, 373-376. https://doi.org/10.1152/jappl.1975.38.3.373

Deforche, B., Lefevre, J., and De Bourdeaudhuij, I. (2003). Physical fitness and physical activity in obese and non-obese Flemish youth. *Obes Res, 11(3),* 434-441. https://doi.org/10.1038/oby.2003.59

Dehghan, H., Mortazavi, S.B., Jafati, M.J., and Maracy, M.R. (2013). Cardiac strain between normal weight and overweight workers in hot/humid weather in the Persian Gulf. *Int J Prev Med, 4(10),* 1147-1153. PMid:24319554

Delextrat, A., Matthew, D., Cohen, D.D., and Brisswalter, J. (2011). Effect of stride frequency on the energy cost of walking in obese teenagers. *Human Movement Sci 30(1)*, 115-124. https://doi.org/10.1016/j.humov.2010.10.004

Dempsey, J.A., Reddan, W., Blake, B., and Rankin, J. (1966). Work capacity determinants and physiologic cost of weight-support work in obesity. *J Appl Physiol, 21,* 1815-1820. https://doi.org/10.1152/jappl.1966.21.6.18 15

Donoghue, A.M., and Bates, G.P. (2000). The risk of heat exhaustion at a deep underground metalliferous mine in relation to body-mass index and predicted VO2max. *Occup Med (Lond), 50(4),* 259-263. https://doi.org/10.1093/occmed/50.4.259

Edelstein, S.L., and Barrett-Connor, E. (1993). Relationship between body size and bone mineral density in elderly men and women. *Am J Epidemiol, 138(3)*, 160-169. https://doi.org/10.1093/oxfordjournals.aj.a116842

Epstein, S., Shapiro, Y., and Brill, S. (1983). Role of surface area to mass ratio and work efficiency in heat intolerance. *J Appl Physiol, 54,* 831-836. https://doi.org/10.1152/jappl.1983.54.3.831

Felson, D.T., Zhang, Y., Hannan, M.T. and Anderson, J,J. (1993). Effects of weight and body mass index on bone mineral density in men and women: the Framingham study. *J Bone Miner Res, 8(5),* 567-573. https://doi.org/10.1002/jbmr.5650080507

Fidler, J., McLaughlin, P., Bubela, D., Scarneo, S.E., McGrry, J.., Evanovich, J., and diStefano, L. (2016). An exploration of the relationship of body mass index with motor performance measures and quality of life in children in an urban setting. *J Childh Obes, 1(4),* 20. https://doi.org/10.21767/2572-5394.100019

Fisher, H.M., Kleinwer, S., Douris, N., Fox, E.C., Mepani, R,J., Verdeguer, F., et al. (2012). FGF21 regulates PGC-1α and browning of white adipose tissues in adaptive thermogenesis. *Genes Dev, 26(3),* 2871-2281. https://doi.org/10.1101/gad.177857.111

Fletcher, E.C., Schaaf, J.W., Miller, J. , and Fletcher, J.G. (1987). Long-term cardiopulmonary sequelae in patients with sleep apnea and chronic lung disease. *Am Rev Resp Dis, 135(3),* 525-533. PMid:3826878

Foster, D.O., and Frydman, M.L. (1979). Tissue distribution of cold-induced thermogenesis in conscious warm- or cold-acclimated rats reevaluated from changes in tissue blood flow: the dominant role of brown adipose tissue in the replacement of shivering by nonshivering thermogenesis. *Can J Physiol Pharmacol, 57,* 257-270. https://doi.org/10.1139/y79-039

Franco, R.A., Rosenfeld, R.M., and Rao, M. (2000). Quality of life for children with obstructive sleep apnea. *Otoloaryngology- Head Neck Surg, 123(1),* 9-16. https://doi.org/10.1067/mhn.2000.105254

Fregly, A.R., Oberman, A., Graybiel, A., and Mitchell, R.E. (1988). Thousand aviator study: nonvestibular contributions to postural equilibrium functions. *Aero Med, 39,* 33-37.

Frontini, A., and Cinti, S. (2010). Distribution and development of brown adipocytes in the murine and human adipose organ. *Cell Metab, 11(4,)* 253-256. https://doi.org/10.1016/j.cmet.2010.03.004

Fui, M.N.T., Dupuis, P., and Grossmann, M. (2014). Lowered testosterone in male obesity: mechanisms, morbidity and management. *Asian J Andro,l 16(2),* 223-231. https://doi.org/10.4103/1008-682X.122365

Gagnon, C.M., Steiper, M.E., and Pontzer, H. (2018). Elite swimmers do not exhibit a body mass index trade-off across a wide range of event distances. *Proc Roy Soc B, 285(1882), pii:20180684.*

Garby, L. (1982). Relationship between energy expenditure, expressed as the physical activity index, and body weight at low physical activity programmes. *Hum Nutr Clin Nutr, 39(6)*, 455-458.

Gardner, K.W., Kark, J.A., Kamei, K., Sanborn, J.S., Gastaldo, E., Burr, P.. and Wenger, C.E. (1996). Risk factors predicting exertional heat illness in male Marine Corps recruits. *Med Sci Sports Exerc 28(8)*, 939-944. https://doi.org/10.1097/00005768-199608000-00001

Geladas, N.D., Nassis, G.P., and Pavlicevic, S. (2005). Somatic and physical traits affecting sprint swimming performance in young swimmers. *Int J Sports Med, 26(2)*,139-144. https://doi.org/10.1055/s-2004-817862

Gill, S.D., McBurney, H., and Schulz, D.L. (2009). Land-based versus pool-based exercises for people awaiting joint replacement surgery of the hip or knee: Results of a random trial. *Arch Phys Med Rehab, 90,* 388-394. https://doi.org/10.1016/j.apmr.2008.09.561

Gilleard, W., and Smith, T. (2007). Effect of obesity on posture and hip joint movements during a standing task, and trunk forward flexion motion. *Int J Obes, 31*, 267-271. https://doi.org/10.1038/sj.ijo.0803430

Girardier, L., and Stock, M.J. (1983). *Mammalian thermogenesis.* London, U.K., Chapman & Hall, pp. 1-360. https://doi.org/10.1007/978-94-011-6032-2

Gnudi, S., Sitta, E., and Lisi, L. (2009). Relationship of body mass index with main limb fragility fractures in postmenopausal women *J Bone Min Metab, 27,* 479-484.

Godin, G., and Shephard, R.J. (1973). Body weight and the energy cost of activity. *Arch Env Health, 27,* 289-293. https://doi.org/10.1080/00039896.1973.10 666383

Gospodarska, E., Nowialis, P., and Kozak, L.P. (2015). Mitochondrial turnover: a phenotype distinguishing brown adipocytes from interscapular brown adipose tissue and white adipose tissue. *J Biol Chem, 290(13),*8243–8255. https://doi.org/10.1074/jbc.M115.637785

Gravante, G., Russo, G., Pomara, F., and Ridola, C. (2003). Comparison of ground reaction forces between obese and control young adults during quiet standing on a baropodometric platform. *Clin Biomech, 18,* 780-782. https://doi.org/10.1016/S0268-0033(03)00123-2

Green, H., Gilbert, J., James, R., and Byard, R. (2001). An analysis of factors contributing to a series of deaths caused by exposure to high environmental temperatures. *Am J Forens Med Pathol, 22(2),* 196-199. https://doi.org/10.1097/00000433-200106000-00018

Guilleminaut, C., Connolly, S.J., and Winkle, R.A. (1983). Cardiac arrhythmia and conduction disturbances during sleep in 400 patients with sleep apnea syndrome. *Am J Cardiol, 52(5,)* 490-494. https://doi.org/10.1016/0002-9149(83)90013-9

Gutiérrez-Fisac, J.L., Guallar-Castillón, P., Diez-Gañán, L., Garcia, E.L., Banegas-Banegas, J.R., and Artalego, F.R. (2002). Work-related physical activity is not associated with body mass index and obesity. *Obes Res, 10(4),* 270-276. https://doi.org/10.1038/oby.2002.37

Hanson, J.S. (1973). Exercise responses after experimental production of obesity. *J Appl Physiol, 35*, 587-591. https://doi.org/10.1152/jappl.1973.35.5.587

Hatfield, H.S., and Pugh, L.G.C.E. (1951). Thermal conductivity of human fat and muscle. *Nature, 168*, 918-919. https://doi.org/10.1038/168918a0

Havenith, G., Coenen, J.M., Kistemaker, L., and Keenny, W.L. et al. (1998). Relevance of individual characteristics for human heat stress response is dependent on exercise intensity and climate type. *Eur J Appl Physiol, 77,* 231-241. https://doi.org/10.1007/s004210050327

Havenith, J., and Van Middendorp, H. (1990). The relative influence of physical fitness, acclimatization state, anthropometric measures and gender on individual reactions to heat stress. *Eur J Appl Physiol, 61*, 419-427. https://doi.org/10.1007/BF00236062

Haymes, E.M., Buskirk, E.R., Hodgson, J.L., Lundegren, H.M., and Nicholas, W.C.(1974). Heat tolerance of exercising lean and heavy prepubertal girls. *J Appl Physiol, 36* 566-571. https://doi.org/10.1152/jappl.1974.36.5.566

Haymes, E.M., McCormick, R.J., and Buskirk, E.R. (1975). Heat tolerance of exercising lean and obese prepubertal boys. *J Appl Physiol, 39(3),* 457-461. https://doi.org/10.1152/jappl.1975.39.3.457

Hayward, J.S., Eckerson, J.D., and Dawson, B.T. (1986). Effect of mesomorphy on hyperthermia during exercise in a warm and humid environment. *Am J Phys Anthropol, 70,* 11-17. https://doi.org/10.1002/ajpa.1330700104

Himms-Hagen, J.. (1979). Obesity may be due to a malfunctioning of brown adipose tissue. *Can Med Assoc J, 121*, 1361-1364. PMid:391377

Ho-Pham, L.T., Nguyen, N.G., Lai, T.Q., and Nguyen, T.V. (2010). Contributions of lean mass and fat mass to bone mineral density: a study in postmenopausal women. *BMC Musculoskeletal Disord, 11*, *59.* https://doi.org/10.1186/1471-2474-11-59

Horowitz, J.F., and Klein, S. (2000). Lipid metabolism during endurance exercise. *Am J Clin Nutr,72 (Suppl. 2),* 558S-563S. https://doi.org/10.1093/ajcn/72.2.558S

Hsu, Y.H., Venners, S.A., and Terwedow, H.A. (2006). Relation of body composition, fat mass, and serum lipids to osteoporotic fractures and bone mineral density in Chinese men and women. *Am J Clin Nutr, 83,* 146–154. https://doi.org/10.1093/ajcn/83.1.146

Huang, L., Chen, P., Zhuang, J., and Walt, S. (2013). Metabolic cost, mechanical work, and efficiency during normal walking in obese and normal-weight children. *Res Quart Exerc Sport, 84(Suppl. 2),* S72-S79. https://doi.org/10.1080/02701367.2013.849159

Hulens, M.J, Vansant, G. , Lysens. R., Claessens, A.L., Muls, E., and Brumagne, ,S (2001a). Study of differences in peripheral muscle strength of lean versus obese women: an allometric approach. *Int J Obes Relat Metab Disord, 25,* 676-681. https://doi.org/10.1038/sj.ijo.0801560

Hulens, M.J., Vansant, G., Lysens, R., Claessens, A.L.., and Muls, E. (2001b). Exercise capacity in lean versus obese women. *Scand J Med Sci Sports, 11(5),* 305-309. https://doi.org/10.1034/j.1600-0838.2001.110509.x

Ishii, S., Cauley, J.A., Greendale, G.A., Nielsen, C., Karvonen-Gutierez, C., Ruppert, K., and Karlamangla, A.S. (2014). Pleiotropic effects of obesity on fracture risk. The study of women's health across the nation. *J Bone Min Res, 29(12)*, 2561-2570. https://doi.org/10.1002/jbmr.2303

Jabbour, G., Lambert, M., O'Loughlin, J., Tremblay, A., and Mathieu, M.E. (2013). Mechanical efficiency during a cycling test is not lower in children with excess body weight and low aerobic fitness. *Obesity (Silver Spring), 21(1)*, 107-114. https://doi.org/10.1002/oby.20241

Jeon, B-J. (2013). The effects of obesity on fall efficacy in elderly people. *J Phys Ther Sci, 25(11)*, 1485-1489. https://doi.org/10.1589/jpts.25.1485

Jequier, E., Gygax, P-H., Pittet, P., and Vannotti, A. (1974). Increased body insulation:

relationship to the development of obesity. *J Appl Physiol, 36(6),* 674-678. https://doi.org/10.1152/jappl.1974.36.6.674

Jimenez-Pavon, D., Kelly, J., and Reilly, J.J. (2010). Associations between objectively measured habitual physical activity and adiposity in children and adolescents: Systematic review. *Int J Pediatr Obes, 5(1),* 3-18. https://doi.org/10.3109/17477160903067601

Johansson, H., Kanis, J.A., Oden, A., McKloskey, E., Chapuriat, R.D., Christ9iansen, C. et al. (2014). A meta-analysis of the association of fracture risk and body mass index in women. *J Bone Min Res, 29(1)*, 223-233. https://doi.org/10.1002/jbmr.2017

Jung, R.T., Leslie, P., Nicholls, D.G., Cunningham, S., and Isles, T.E. (1988). Energy expenditure in normal and diabetic man: the role of brown adipose tissue. *Health Bull, 46,* 55-62.

Kang, D.H., Guo, L.F., Guo, T. , Wang, Y., Liu, T., Feng, X.Y., and Che, X.Q. et al. (2015). Association of body composition with bone mineral density in northern Chinese men by different criteria for obesity. *J Endocrinol Invest, 38(3),* 323-331.https://doi.org/10.1007/s40618-014-0167-5

Karvonen, M.J., Viitasalo, J.T., Komi, P.V., Nummi, J., and Järvinen, T. (1980). Back and leg complaints in relation to muscle strength in young men. *Scand J Rehabil Med, 12,* 53-59. PMid:6451926

Kawai, M., DePaula, F.J.A., and Rosen, C.J. (2012). New insights into osteoporosis: The bone-fat connection. *J Intern Med, 272(4),* 317-329. https://doi.org/10.1111/j.1365-2796.2012.02564.x

Keatinge, W.R., Khartchenko, M., Lando, N., and Lioutov, V. (2001). Hypothermia during sports swimming in water below 11° C. *Br J Sports Med, 35,* 352-353. https://doi.org/10.1136/bjsm.35.5.352

Kejonen, P., Kauranen, K., and Vanharanta, H. (2003). The relationship between anthropometric factors and body-balancing movements in postural balance. A*rch Phys Med Rehabil, 84,* 17-22. https://doi.org/10.1053/apmr.2003.50058

Kim, C.J., Oh, K.W., Rhee, E.J. , Kim. K.H., Jo, S.K., Jung, C.H. et al. (2009). Relationship between body composition and bone mineral density (BMD) in peri-menopausal Korean women. *Clin Endocrinol (Oxf), 71,* 18-26. https://doi.org/10.1111/j.1365-2265.2008.03452.x

Kim, K-C., Shin, D-H., Lee, S-Y., Im, J.A., and Lee, D.C. (2010). Relationship between obesity and bone mineral density and vertebral fractures in Korean postmenopausal women. *Yonsei Med J, 51(6,)* 857-863. https://doi.org/10.3349/ymj.2010.51.6.857

Kirkwood, S.P., Zurlo, F., Larson, K. , and Ravussin, E. (1991). Muscle mitochondrial morphology, body composition, and energy expenditure in sedentary individuals. *Am J Physiol, 260,* E89-E94. https://doi.org/10.1152/ajpendo.1991.26.1.E89

Klein, K.O., Larmore, K.A., de Lancey, E., Brown, J.M., Cinsidine, R.V., and Hassink, S.G. (1998). Effect of obesity on estradiol level, and its relationship to leptin, bone maturation, and bone mineral density in children. *J Clin Endocrinol Metab, 83(10)*, 3469-3475. https://doi.org/10.1210/jcem.83.10.5204

Knechtle, B., Christinger, N., Kohler, G., Knechtle, P., and Rosemann, T. (2009). Swimming in ice cold water. *Irish J Med Sci, 178,* 507-511. https://doi.org/10.1007/s11845-009-0427-0

Knechtle, B. , Knechtle, P., and Kohler, G. (2008). No correlation of anthropometry and race performance in ultra-endurance swimmers at a 12-hours-swim. *Anthropol Anz, 66(1),* 73-79. PMid:18435207

Ko, S-U., Stenhom, S., and Ferrucci, L. (2010). Characteristic gait patterns in older adults with obesity- results from the Baltimore longitudinal study. *J Biomech, 43(6),* 1104-1110. https://doi.org/10.1016/j.jbiomech.2009.12.004

Koepp, G.A., Snedden, B.J., and Levine, J.A. (2015). Workplace slip, trip and fall injuries and obesity. *Ergonomics, 58(5),* 674-679. https://doi.org/10.1080/00140139.2014.985260

Kurpad, A.V., Khan, K., Calder, A.G., and Elia, M. (1994). Muscle and whole body metabolism after norepinephrine. *Am J Physiol, 266*, E877-E884. https://doi.org/10.1152/ajpendo.1994.266.6.E877

LaFortuna, C., Proletti, M., Agosti, F., and Sartorio, A. (2006).The energy cost of cycling in

obese young women. *Eur J Appl Physiol, 97(1)*, 18- 25. https://doi.org/10.1007/s00421-006-0137-5

LaFortuna, C., Agosti, F., Busti, C., Galli, R., and Sartotio, A. (2009). The energy cost of cycling and aerobic performance of obese adolescent girls. *J Endocrinol Invest, 32*, 647-652. https://doi.org/10.1007/BF03345735

LaRoche, D.P., Marques, N.R., Shumila, H.N., Logan, C.R., St. Laurent, R., and Gonçalves, M. (2015). Excess body weight and gait influence eergy cost of walking in older adults. *Med Sci Sports Exerc, 47(5)*, 1017-1025. https://doi.org/10.1249/MSS.0000000000000501

LaRoche, D.P., Kralian, R.J., and Millett, E.D. (2011). Fat mass limits lower-extremity relative strength and maximal walking performance in older women. *J Electromyogr Kinesiol, 21(5,)* 754-761. https://doi.org/10.1016/j.jelekin.2011.07.006

Laslett, L.L., Just Nee Foley, S.J., Quinn, S.J., Winzenberg, T.M., and Jones, G. (2012). Excess body fat is associated with higher risk of vertebral deformities in older women but not in men: a cross-sectional study. *Osteoporos Int, 23(1,)* 67-74. https://doi.org/10.1007/s00198-011-1741-8

Lauritzen, J.B. (1996). Hip fractures: incidence, risk factors, energy absorption, and prevention. *Bone, 18(1)*, S65-S75. https://doi.org/10.1016/8756-3282(95)00382-7

Leithead, C.S., and Lind, A.R. (1964). *Heat stress and heat disorders.* Philadelphia, PA, F.A. Davis, pp. 1-303.

Li, C., Ford, E.S., Zhao, G. , Croft, J.B., Balluz, L.S., and Mokdad, A.H. 2010). Prevalence of self-reported clinically diagnosed sleep apnea according to obesity status in men and women: National Health and Nutrition Examination Survey, 2005-2006. *Prev Med, 51*, 18-23. https://doi.org/10.1016/j.ypmed.2010.03.0 16

Liu, P.Y., Ilich, J.Z., Brummel-Smith, K.. and Ghosh, S. (2014). New insight into fat, muscle and bone relationship in women: determining the threshold at which body fat assumes negative relationship with bone mineral density. *Int J Prev Med, 5(11),* 1452-1463. PMid:25538842

Lowensteyn, I., Signorile, J., and Giltz, K. (1994). The effect of varying body composition on swimming performance. *J Strength Cond Res, 8(3),* 149-154.

Maciejczyk, M., Wiecek, M., Szymura, J., and Szygula, Z. (2016). Effect of body composition on walking economy. *Hum Movement, 17(4),* 222-228. https://doi.org/10.1515/humo-2016-0034

Malhotra, M.S., Ramaswamy, S.S., and Ray, S.N. (1962). Influence of body weight on energy expenditure. *J Appl Physiol, 17*, 355-364. https://doi.org/10.1152/jappl.1962.17.3.433

Malina, R.M., Neunen, G., Claessens, A.L., Lefevre, J., Vanden Eyende, B.V., Renson, R., Vanreusel, B., and Simons, J. (1995). Fatness and physical fitness of girls 7 to 17 years. *Obes Res, 3(3),* 221-231. https://doi.org/10.1002/j.1550-8528.1995.tb00142.x

Mandroukas, K., Krotkiewski, M., Hedberg, M. , Wroblewski, Z., Björntorp, P., and Grimby, G. et al. (1984). Physical training in obese women: effects of muscle morphology, biochemistry and function. *Eur J Appl Physiol, 52,* 355-361. https://doi.org/10.1007/BF00943363

McKee, W.P., and Bollinger, R.E. (1960). Caloric expenditure of normal and obese subjects during a standard work test. *J Appl Physiol, 15*, 197-200. https://doi.org/10.1152/jappl.1960.15.2.197

Miller, E.M., Matrangola, S. L., and Madigan, M.L. (2011). Effects of obesity on balance recovery from small postural perturbations. *Eronomics, 54(6),* 547-554. https://doi.org/10.1080/00140139.2011.582959

Mitchell, J.W., Galvez, T.L., Hengje, J., Myers, G.E., and Siebecker, K.L. et al. (1970). Thermal response of human legs during cooling. J Appl Physiol, 29(6), 859-865. https://doi.org/10.1152/jappl.1970.29.6.859

Moayyeri, A., Luben, R.N., Wareham, N.J., and KHaw, K.T. (2012). Body fat mass is a predictor of risk of osteoporotic fractures in women but not in men: a prospective population study. *J Intern Med, 271(5),* 472-

480. https://doi.org/10.1111/j.1365-2796.2011.02443.x

Mohamad, N.V., Soelaiman, I.N., and Chin, K.Y. (2016). A concise review of testosterone and bone health. *Clin Interv Aging, 11*, 1317-1324.
https://doi.org/10.2147/CIA.S115472

Mohammed, A. , and Tareq, A. (2016). The relationship between body fat percentage with speed, agility and reaction time of male football players of Bangladesh. *Int J Sci Culture Sport, 4(4)*, 453-460.
https://doi.org/10.14486/IntJSCS601

Morris, J.N., Heady, J.A., and Raffle, P.A. (1956). Physique of London busmen: The epidemiology of uniforms. *Lancet, 271 (6942),* 569-570.
https://doi.org/10.1016/S0140-6736(56)92049-9

Myers, M.J., and Steudel, K. (2005). Effect of limb mass and its distribution on the energetic cost of running. *J Exp Biol, 116(1),* 363-373.

Nedergaard, J., and Cannon, B. (2010). The changed metabolic world with human brown adipose tissue: Therapeutic visions. *Cell Metab, 11(4),* 268-272.
https://doi.org/10.1016/j.cmet.2010.03.007

Newcomer, B.R., Larson-Meyer, D.E., Hunter, G.R., and Weinsier, R.L. (2001). Skeletal muscle metabolism in overweight and post-overweight women: an isometric exercise study using ^{31}P magnetic resonance spectroscopy. *Int J Obes Relat Metab Disord, 25,* 1309-1315.
https://doi.org/10.1038/sj.ijo.0801673

Newman, A.B., Foster, G., Givelber, R., Nieto, F.J., Redline, S., and Young, T.. (2005). Progression and regression of sleep disordered breathing with changes in weight: the Sleep Heart Health Study. *Arch Int Med, 165(20),* 2408-2413.
https://doi.org/10.1001/archinte.165.20.2408

Nielson, C.M., Marshall, L.M., Adams, A.L. , LeBlanc, E.S., Cawthon, P.M., Ensrud, K., et al. (2010). BMI and fracture risk in older men: the osteoporotic fractures in men (MrOS) study. *J Bone Miner Res, 26(3),* 496-502.
https://doi.org/10.1002/jbmr.235

O'Hara, W.J., Allen, C., and Shephard, R.J. (1977). Loss of body weight and fat during exercise in a cold chamber. *J Appl Physiol, 37*, 205-218. https://doi.org/10.1007/BF00421776

Okely, A.D., Booth, M.L., and Chy, T. (2004). Relationships between body composition and fundamental movement skills among children and adolescents. *Res Quart Exerc Sport, 73(3)*, 238-244.
https://doi.org/10.1080/02701367.2004.10609157

Ouellet, V., Abbé, S.M., Blondin, D.P., Phoenix, S., Guérin, B., Haman, F., et al. (2012). Brown adipose tissue oxidative metabolism contributes to energy expenditure during acute cold exposure in humans. *J Clin Invest, 122(2),* 545-552.
https://doi.org/10.1172/JCI60433

Park, W., Ramachandran, J., Weisman, P. and Jung, E.S. (2010). Obesity effect on male active joint range of motion. *Ergonomics, 53(1),* 102-108.
https://doi.org/10.1080/00140130903311617

Partinen, M., and Telakivi, T. (1992). Epidemiology of obstructive sleep apnea syndrome. *Sleep, 16 (Suppl. 6)*, S1-S4.
https://doi.org/10.1093/sleep/15.suppl_6.S1

Passmore, R., and Durnin, J.V.G.A. (1955). Human energy expenditure. *Physiol Rev, 35*, 801-840.
https://doi.org/10.1152/physrev.1955.35.4.801

Pataky, A., Armand, S., Müller-Pinget, S., Golay, A., and Allet, L. (2014). Effects of obesity on functional capacity. *Obesity, 22*, 56-62.
https://doi.org/10.1002/oby.20514

Paterson, D.H., and Stathokostas, L. (2001). Physical activity, fitness, and gender in relation to morbidity, survival, quality of life and independence in old age. In: R.J. Shephard (Ed.), *Gender, physical fitness and aging*. Boca Raton, FL, CRC Press, pp. 99-120. https://doi.org/10.1201/b14223-6

Peppard, P.E., Young, T., Palta, M., and Skatrud, J. (2000). Prospective study of the association between sleep-disordered breathing and hypertension. *N Engl J Med, 342(19)*, 1378-1384.
https://doi.org/10.1056/NEJM200005113421901

Pereiera-Santos, M., Costa, P.R.F., Assis, A.M.O., Santos, C.A., and Santos, D.B. (2015). Obesity and vitamin D deficiency: a systematic review and meta-analysis. *Obes Rev, 16(4)*, 341-349.
https://doi.org/10.1111/obr.12239

Peyrot, N., Thivel, D., Isacco, L., Morin, J.B., Duche, P., and Belli, A. (1985). Do mechanical gait parameters explain the higher metabolic cost of walking in obese adolescents? *J Appl Physiol, 106(6)*, 1763-1770. https://doi.org/10.1152/japplphysiol.91240.2008

Pigeyre, M., Yazdi, F.T., Kaur, Y., and Meyre, D. (2016). Recent progress in genetics, epigenetics and metagenetics in the pathophysiology of human obesity. *Clin Sci, 130,* 943-986. https://doi.org/10.1042/CS20160136

Pirro, M., Fabbriciani, G., Leli, C., Callarelli, L., Manfredelli, M.R., Fioroni, C., et al. (2010). High weight or body mass index increase the risk of vertebral fractures in postmenopausal osteoporotic women. *J Bone Miner Metab, 28(1),* 88-93. https://doi.org/10.1007/s00774-009-0108-0

Prentice, A.M., Black, A.E., Coward, J.A., and Cole, T.J. (1996). Energy expenditures in overweight and obese adults in affluent societies: ananalysis of 319 doubly-labelled water measurements. *Eur J Clin Nutr, 50*, 93-97. PMid:8641251

Prieto-Alhambra, D., Premaor, M.O., Fina Avilés, F., Hermosilla, E., Martinez-Laguna, D., Carbonella-Abella, C., et al. (2012). The association between fracture and obesity is site-dependent: a population-based study in postmenopausal women. *J Bone Miner Res, 27(2),* 284-300. https://doi.org/10.1002/jbmr.1466

Pugh, L.G.C.E., and Edholm, O.G. (1955). The physiology of channel swimmers. *Lancet, 269 (6893)*, 761-768. https://doi.org/10.1016/S0140-6736(55)92454-5

Punjabi, N.M. (2008). The epidemiology of adult obstructive sleep apnea. *Proc Am Thor Soc, 5(2),* 136-143. https://doi.org/10.1513/pats.200709-155MG

Quarade, F. (1963). Insulation in leanness and obesity. *Lancet, 2(7305)*, 429-432. https://doi.org/10.1016/S0140-6736(63)92172-X

Rajakumar, K., Greenspan, S.L., Thomas, S.B., and Holick, M.F. (2007). SOLAR ultraviolet radiation and vitamin D: a historical perspective. *Am J Publ Health, 97(10),* 1746-1754.

https://doi.org/10.2105/AJPH.2006.091736

Reddy, E.V., Kadhiravan, T., Mishra, H.K., Sreenivas, V., Handa, K.K., Sinha, S., and Sharma, S.K. (2009). Prevalence and risk factors of obstructive sleep apnea among middle-aged urban Indians: a community-based study. *Sleep Med, 10(8),* 913-918. https://doi.org/10.1016/j.sleep.2008.08.01

Resta, O., Foschino-Barbaro, M.P., Legari, G., Talamo, S., Bonfitto, P., Palumbo, A. et al. (2001). Sleep-related breathing disorders: loud snoring and excessive daytime sleepiness in obese subjects. *Int J Obes Rel Metab Disord, 25(5)*, 669-675.

Ribot, C., Tremollieres, F., Pouilles, J.M., Bonneu, M., Germain, F., and Louvet, J.P. (1987). Obesity and postmenopausal bone loss: the influence of obesity on vertebral density and bone turnover in postmenopausal women. *Bone, 8(6),* 327-331. https://doi.org/10.1016/8756-3282(87)90062-7

Rocher, E., Chappard, C., Jaffre, C., Benhamou, C.L., an Courteix, D. (2008). Bone mineral density in prepubertal obese and control children: relation to body weight, lean mass, and fat mass. *J Bone Min Metab, 26(1)*, 73-78. https://doi.org/10.1007/s00774-007-0786-4

Roger, B.. (2017). Body mass index and saltwater drowning. *Am J Forens Med Pathol, 38(4),* 304-305.

Salvadori, A., Fanari, P., Fontana, M., Buontempi, L., Saezza, A., Baudo9, S., Miserocchi, G., and Longhini, E. (1999). Oxygen uptake and cardiac performance in obese and normal subjects during exercise. *Respiration, 66(1)*, 25-33. https://doi.org/10.1159/000029333

Sanders, M.K. (1984). Nasal CPAP effects on patterns of sleep apnea. *Chest, 88(6)*, 839-844.
https://doi.org/10.1378/chest.86.6.839

Sartorio, A., Lafortuna, C.L., Conte, G., Faglia, G., and Narici, M.V. (2001). Changes in motor control and muscle performance after a short-term body mass reduction program in obese subjects. *J Endocrinol Invest, 24,* 393-398. https://doi.org/10.1007/BF03351039

Sharma, S.K., Kumpawat, S., Banga, A., and Goel, A. (2006). Prevalence and risk factors for obstructive sleep apnea syndrome in a population of Delhi, India.*Chest, 130(1)*,

149-156. https://doi.org/10.1378/chest.130.1.149

Sheldahl, L.M., Buskirk, E.R., Loomis, J.L., Hodgson, J.L., and Mendez, J. (1982). Effects of exercise in cool water on body weight loss. *Int J Obes, 6(1),* 29-42. PMid:7068314

Shephard, R.J. (1982). *Physiology and Biochemistry of Exercise*. New York, NY, Praeger, pp. 1--672.

Shephard, R.J. (1985). Adaptation to exercise in the cold. *Sports Med, 2(1)*, 59-71. https://doi.org/10.2165/00007256-198502010-00006

Shephard, R.J. (1997). *Aging, physical activity and health*. Champaign, IL, Human Kinetics, pp. 1-487.

Shephard, R.J., Godin, G., and Campbell, R. (1973). Characteristics of sprint, medium and middle distance swimmers. *Int Z Angew Physiol, 32*, 1-19.

Shephard, R.J. , and Johnson, N. (2015). Effects of physical activity upon the liver. *Eur J Appl Physiol, 115(1)*, 1-46. https://doi.org/10.1007/s00421-014-3031-6

Siders, W.A., Lukaski, H.C., and Bolonchuk, W.W. (1993). Relationships among swimming performance, body composition and somatotype in competitive collegiate swimmers. *J Sports Med. Phys Fitness, 33(2)*, 166-171. PMid:8412052

Silva, K.S., Mendonça, L.M., Conceição, F., Zahar, S.E., and Farias, M.L. (2007). Influence of obesity on bone density in postmenopausal women. *Arq Bras EndocrinolMetabol, 51(6)*, 943-949. https://doi.org/10.1590/S0004-27302007000600008

Sloan, R.E.G., and Keatinge, W.R. (1973). Cooling rates of young people swimming in cold water. *J Appl Physiol, 35,* 371-375. https://doi.org/10.1152/jappl.1973.35.3.371

Smith, R.M., and Hanna, J.M. (1975). Skinfolds and resting heat loss in cold air and water: temperature equivalence. *J Appl Physiol, 39(1),* 93-102. https://doi.org/10.1152/jappl.1975.39.1.93

Srivastnava, A., Srivastava, N, and Mittal, B. (2016). Genetics of obesity. *Int J Clin Biochem, 31(4),* 361-371. https://doi.org/10.1007/s12291-015-0541-x

Syed, I.Y., and Davis, B.L. (2000). Obesity and osteoarthritis of the knee: hypotheses concerning the relationship between ground reaction forces and quadriceps fatigue in long-duration walking. *Med Hypotheses, 84,* 182–185. https://doi.org/10.1054/mehy.1999.0013

Thakur, J.S. (2016). Association of obesity with agility and speed of university level Kabaddi players. *Int J Phys Ed Sports Health, 3(2),* 254-256.

Tokmakidis, S.P., Kasambalis, A., and Christodoulos, A.D. (2006). Fitness levels of Greek primary schoolchildren in relationship to overweight and obesity. *Eur J Pediatr, 165*, 867-874. https://doi.org/10.1007/s00431-006-0176-2

Tsuneto, A., Seiji, K., Toshiki, A., Omori, C., Saito, O., Majima, T., and Horie, T. (2002). Relationship between quality of life and mood or depression in patients with severe obstructive sleep apnea syndrome. *Chest, 122(3)*, 861-865. https://doi.org/10.1378/chest.122.3.861

Turell, D.J., Austin, R.C., and Alexander, J.K. (1964). Cardio-respiratory response of very obese subjects to treadmill exercise. *J Lab Clin Med, 64*, 107-116. PMid:14192555

Van den Beld, A.W., de Jong, F.H., Grobbee, D.E., Pols, H.A., and Lamberts, S.W. (2000). Measures of bioavailable serum testosterone and estradiol and their relationships with muscle strength, bone density, and body composition in elderly men. *J Clin Endocrinol Metab, 85(9),* 3276-3282. PMid:10999822

Van der Lans, A.A.J.J., Hoeks, J., Brans, B., Vijgen, B.B., Visser, M.G.W., Vosselman, M.J. et al. (2013). Cold acclimation recruits human brown fat and increases non-shivering thermogenesis. *J Clin Invest, 123(8)*, 3395-3403. https://doi.org/10.1172/JCI68993

Vandentoreen, S., Bretin, P., Zeghnoun, A., Mandereau-Bruno, L., Croisier, A., Cochet, C., et al. (2006). August 2003 heat wave in France: Risk factors for death of elderly people living at home. *Eur J Publ Health, 16(6),* 583-591. https://doi.org/10.1093/eurpub/ckl063

Vanlint, S.. (2013). Vitamin D and obesity. *Nutrients, 5(3),* 949-956. https://doi.org/10.3390/nu5030949

Vroman, N.B., Buskirk, E.R., and Hodgson, J.L. (1983). Cardiac output and skin blood flow

in lean and obese individuals during exercise in the heat. *J Appl Physiol, 55 (1 Pt. 1),* 69-74. https://doi.org/10.1152/jappl.1983.55.1.69

Wallace, R.F., Knebel, D., Punnett, L., Wegman, D.H., Wenger, C.B., Gardner, J.W., and Kark, J.A. (2006). Risk factors for recruit exertional heat illness by gender and training period. *Aviat Space Environ Med, 77(4),* 415-421. PMid:16676653

Wearing, S.C., Hennig, E.M., Byrne, N.M. , Steele, J.R., and Hills, A.P. (2006). The biomechanics of restricted movement in adult obesity. *Obes Rev, 7(1)*, 13-24. https://doi.org/10.1111/j.1467-789X.2006.00215.x

Westerterp, K.R., and Speakman, J.R. (2008). Physical activity energy expenditure has not ceclined since the 1980s, and matches energy expenditures of wild mammals. *Int J Obes, 32*, 1256-1273. https://doi.org/10.1038/ijo.2008.74

Weyand, P.G., Smith, B.R., Puyau, M.R. , and Butte, N.F. (2010). The mass specific energy cost of human walking is set by stature. *J Exp Biol, 213*, 3972-3979. https://doi.org/10.1242/jeb.048199

White, U.A., and Tchoukalova, Y.D. (2014). Sex dimorphism and depot differences in adipose tissue function. *Biochim Biophys Acta, 1842(3)*, 377-392. https://doi.org/10.1016/j.bbadis.2013.05.006

Wyndham, C.H., Williams, C.G., and Loots, H. (1968). Reactions to cold. *J Appl Physiol, 24,* 282-287. https://doi.org/10.1152/jappl.1968.24.3.282

Young, L.R., Plata, M., Dempsey, J., Skatrud, J., Weber, S., and Badr, S. (1993). The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med, 328*, 1230–1235. https://doi.org/10.1056/NEJM199304293281704

Young, T., Peppard, P.E., and Taheri, S. (2005). Excess weight and sleep disordered breathing. *J Appl Physiol, 99,* 1592-1599. https://doi.org/10.1152/japplphysiol.00587.2005

Zhang, H., Huizenga, C., Arens, E., and Yu, T. (2001). Considering individual physiological differences in a human thermal model. *J Thermal Biol, 26*, 401-

448. https://doi.org/10.1016/S0306-4565(01)00051-1