1-1-2011

A Test of an Evolutionary Theory of Adiposity Gain Induced by Long Sleep in Descendants of European Hunter-Gatherers

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2013

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Dissertation Submitted in Partial Fulfillment

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Doctor of Philosophy

Psychology

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Abstract

Researchers have identified inadequate sleep duration as one of the factors contributing to global obesity. The purpose of this study was to test a hypothesis deduced from a new sleep-duration-based evolutionary theory claiming that sleep extension in response to lengthening night duration in early fall evolved into a behavioral marker of an approaching winter; this adaptive trait was theorized to produce adiposity gain in White men in response to sleep extension. The hypothesis was that White Americans would show a greater increase in the age-adjusted fat mass index per unit of sleep duration compared to that of Black Americans. Data were part of the National Health and Nutrition Examination Survey (NHANES) study between 2005 and 2010. The multiple regression analysis did not support the study hypothesis. The results indicated that habitual sleep duration had no effect on the annual rate of adiposity gain in White men, while in Black men, longer sleep was associated with significantly higher annual rates of adiposity gain. Implications for social change include the case for population-specific antiobesity interventions in Black men, including closer monitoring of sleep duration in order to prevent adverse habitual sleep extension and to improve time budgeting for physical exercise.

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Dedication

I dedicate this work to the memory of my mother who would love to have seen it completed.

Acknowledgments

I would like to thank my dissertation chair Dr. Gary J. Burkholder for his eagerness to comprehend the meaning of my half-formed ideas and his vision and encouragement in bringing them forward, towards a glimmer of clarity. I would like to thank my dissertation committee members, Dr. Rachel Piferi and Dr. Marites Pinon, and all faculty and staff at Walden University for being such amazing partners in this journey.

Always, my gratitude goes to my wife and dearest friend Irina Chadyuk for expecting only the very best from me and for her incredible drive to turn our dreams into reality. I am grateful to my children Nikita and Dasha who admired this work when it was merely a verb, to my father who set a standard of dedication to knowledge in our family, and to my sisters who never fail to aspire to it.

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Chapter 1: Introduction to the Study

The recent surge in prevalence of obesity transcends national, cultural, and socioeconomic boundaries (Swinburn et al., 2011). Despite growing health risks (Reilly & Kelly, 2011) and mounting economic costs associated with obesity (Y. C. Wang, McPherson, Marsh, Gortmaker, & Brown, 2011), no reliable method of obesity treatment and prevention has so far been found (Wells, 2013). With a possible exception of bariatric surgery, most interventions proposed to date do not produce lasting effects (Wells & Siervo, 2011). Although researchers agree on the overall principles of obesity (Hall et al., 2012), there is little theoretical consensus on obesity's etiology (Knutson, 2012). In this study, I aspired to contribute to theoretical understanding of the causal mechanisms in the development of obesity.

This chapter will position the study against the background of the current scientific knowledge about obesity. The purpose of the study, which is the formulation and testing of a theory of sleep-induced obesity, will be discussed; the research question and hypothesis will be posed. A more detailed description of the proposed theory will be followed by the review of the study's methodology, as well as the operational definitions used in this work. A discussion of the assumptions and the scope of the study will be concluded by an explanation of the study's limitations and scientific significance.

Background

Despite being a subject of active research, obesity remains poorly understood and thus far impervious to treatment and prevention (Marcus & Wildes, 2012; Wells & Siervo, 2011). Paradoxically, the macro level physics of obesity, captured by the

dominant energy-balance theory, is fairly straightforward (Wells, 2013). However, the mechanics of obesity-promoting energy imbalance remains ambiguous (Knutson, 2012).

A human organism constantly expends energy to fuel its movement, thermoregulation, and various internal physiological processes (Hall et al., 2012). Energy intake, on the other hand, happens only at certain intervals (Dunbar, Korstjens, & Lehmann, 2009). In the view of this mismatch, the task of the adipose tissue, generally known as fat, is to buffer the body's constant energy demands against the vagaries of irregular supply (Wells, 2009a).

The total usable energy in the adipose tissue of a lean person is around 130,000 kcal; in contrast, an extremely obese adult carries almost 1 million kcal of energy reserves (Hall et al., 2012). According to the law of conservation of energy, such reserves can be accumulated only if the organism's energy intake chronically exceeds expenditure; this, in a nutshell, is the logic of the energy-balance theory of obesity (Siervo, Wells, & Cizza, 2009).

A slight imbalance between energy acquisition and consumption results in a long-term accumulation of the unused surplus. According to Ulijaszek and Lofink (2006), the current rates of adult obesity in the industrialized nations may be caused by an energy imbalance of less than 1%. Indeed, a positive caloric balance of +0.3% produces a weight gain of 9 kg over 30 years (Flier & Maratos-Flier, 2008), so a lean person's stable weight must be, in its own right, an incredible feat of energy balancing (Taubes, 2007). Given that a human organism is able to manage its energy supply and demand in such a narrow equilibrium, the main task of obesity research is to understand the factors that

upset the fine balance of the body's energy homeostasis and cause a runaway epidemic of obesity in modern humans (Hall et al., 2012).

A possible approach to this task was proposed by Neel (1962), who theorized that humans have evolved a simple adaptive strategy to deal with the challenge of balancing almost constant energy expenditure with occasional and often uncertain energy intake. Neel (1962) postulated that this strategy, embodied in a so-called *thrifty gene*, is designed to stimulate accumulation of energy by the organism when a future nutritional deficit is anticipated (Bouchard, 2007). According to Neel (1962), this innate bias for energy acquisition was counterbalanced by natural food scarcity rather than genuine moderation in food consumption. Thus, the people who carry the thrifty genotype are naturally prone to obesity, and the factor disrupting their energy equilibrium is the "progress" itself (Neel, 1962, p. 353), which has made a widespread famine all but nonexistent.

Such evolutionary articulation of the problem suggests that the propensity to become obese may be genetic. Indeed, a fair amount of evidence suggests that up to 70% of variation in body weight can be attributed to heredity (Lavebratt, Almgren, & Ekström, 2012). Yet, in defiance of this observation, the genes, which have so far been implicated in obesity, account only for 5% of the estimated heritability (O'Malley & Stotz, 2011). It has been conjectured that this "missing heritability" (Manolio et al., 2009, p. 747) is observed because the elusive thrifty genes are expressed only under certain environmental conditions. For example, it has been speculated that seasonal environments where food availability was governed by a circannual cycle of lean and

plenty must have resulted in a cyclical pattern of expression of obesity-promoting genes (Gangwisch, 2013).

In parallel to these theoretical developments, many empirically-oriented researchers have looked for specific environmental factors disrupting energy homeostasis in modern humans. Research studies have probed in a multitude of directions, ranging from the most obvious targets like diet and physical activity, to the most audacious ones, like a globally proliferating viral infection (Gabbert, Donohue, Arnold, & Schwimmer, 2010). Among these empirically-derived factors, inadequate sleep has been identified as one of several behavioral contributors to onset and progression of obesity (Golley, Maher, Matricciani, & Olds, 2013). However, the mechanisms of the apparent relationship between sleep and obesity are still not fully understood (St-Onge, 2013).

Problem Statement

Many current studies associating sleep and obesity have focused on obesity-promoting effects of voluntary sleep restriction (Chaput, Després, Bouchard, & Tremblay, 2012). In addition, some researchers found long sleep (generally, 9 hours or longer; Magee, Kritharides, Attia, McElduff, & Banks, 2012) to be likewise implicated in obesity progression (Yiengprugsawan, Banwell, Seubsman, & Sleigh, 2012). However, research attempting to uncover this apparent relationship between long sleep and obesity has produced mixed results. Although some researchers have found long-sleep-related obesity both cross-sectionally (Buxton & Marcelli, 2010) and longitudinally (Hairston et al., 2010), other researchers found no relationship (Anic, Titus-Ernstoff, Newcomb, Trentham-Dietz, & Egan, 2010). Consequently, very few theories have been proposed to

predict whether and why long sleep influences the development of obesity (Knutson, 2012). As a result, existing behavioral antiobesity interventions generally disregard possible negative effects of sleep extension (Cizza et al., 2010).

Purpose of the Study

The purpose of this quantitative study was to propose and test a new theory claiming that, in certain populations, obesity is promoted by long sleep. This theory postulated that in the populations descending from European Upper-Paleolithic hunter-gatherers, accumulation of fat in response to behavioral extension of sleep evolved as an adaptation to cold winter. As a further qualification of this conjecture, this study assumed that the human multiyear reproductive cycle resulted in a suppression of seasonal adiposity fluctuation in women, thus restricting the proposed theory's applicability to men.

An empirical goal of the study was to test the proposed theory by probing a directional hypothesis comparing two modern US populations: the one in which the theorized adaptation is most likely to be found (White male Americans) and the one in which it is least likely to be found (Black male Americans). The hypothesized difference in sleep-related adiposity was estimated from anthropometric measurements and participant-reported sleep duration, age, sex, and race obtained as part of the National Health and Nutrition Examination Survey (NHANES) conducted by Centers for Disease Control and Prevention (CDC, National Center for Health Statistics, 2013b) in the United States.

Research Question and Research Hypothesis

The research question addressed by this dissertation was as follows: Is there a racial-group difference in the relationship between habitual sleep duration and adiposity, as would be predicted by the proposed theory of long-sleep-induced adiposity gain?

Research hypothesis. In Whites, compared to Blacks, there is a stronger increase in the age-adjusted fat mass index (FMI) per unit of sleep duration as measured in male participants of NHANES (CDC, National Center for Health Statistics, 2013b) aged 16 years and older.

Null hypothesis. There is no significant difference between Black and White male participants of NHANES (CDC, National Center for Health Statistics, 2013b) aged 16 years and older, in the relationship between self-reported sleep duration and age- and race-adjusted FMI assessed from anthropometry.

Theoretical Framework

According to the energy-balance theory, obesity results from accumulation of large amounts of excess energy, which is a consequence of a chronic imbalance between the energy deposited in the adipose tissue and the energy taken out of it (Hall et al., 2012). The theory of the thrifty genotype predicts that, as a result of surviving regular periods of food scarcity, humans evolved a set of adaptations that tilt the internal energy homeostasis and result in weight gain (Bouchard, 2007). It has recently been theorized that a seasonal nature of these adaptations might provide clues to environmental and behavioral factors that promote and sustain an imbalance of energy equilibrium in humans (Gangwisch, 2013). A theory proposed here extends the idea of adaptive

seasonal adiposity in order to highlight the pathway that associates fat accumulation and sleep.

In adaptation to warm/cold seasonality that was necessary for survival of human hunter-gatherers in Europe in the Upper-Paleolithic period (Higham et al., 2011), the optimum period for adiposity gain was late summer and early fall when nutrition from both animal and plant sources was relatively plentiful (Gangwisch, 2013). Another apparent factor of European seasonality is a rapid contraction of the day length that occurs during the same period in late summer and early fall. At the latitude of Lyon, France, where humans most likely remained throughout the last glacial maximum (J. R. Stewart & Stringer, 2012), the period of complete darkness lengthens from just over 7 hours in June to almost 10 hours in September (Broomberg, 2008; Morrissey, 2012).

The key element of the theory advanced here is the observation that humans tend to extend habitual sleep duration when exposed to prolonged periods of darkness (Peixoto, da Silva, Carskadon, & Louzada, 2009). It is possible that for early humans living in Europe, late summer and early fall was not only the most advantageous period for adaptive adiposity gain, but was also associated with the extension of sleep duration (Cizza, Requena, Galli, & de Jonge, 2011).

The theory proposed here postulates that such sleep extension acted as a behavioral cue for the expression of the seasonal thrifty gene, which promoted adaptive adiposity gain. If this mechanism still operates in the present-day descendants of European hunter-gatherers, the theory predicts that habitually long sleep in these populations will be associated with chronic expression of the theorized thrifty gene, and

thus, obesity. Thus, to summarize, the theory proposed here extends the energy balance theory—and the theory of a thrifty genotype derived from it—to postulate that in the seasonal European environment, Upper-Paleolithic humans evolved a trait of adaptive adiposity gain activated by a behavioral cue of sleep extension.

Nature of the Study

This study involved the test of a research hypothesis predicting an interaction between race and sleep duration in the model for adiposity in adult male participants of NHANES (CDC, National Center for Health Statistics, 2013b). The archival dataset containing the questionnaire and anthropometric data was downloaded from the CDC website (CDC, National Center for Health Statistics, 2013b). As part of the NHANES study, age, race, sex, and habitual sleep duration were obtained from participants via interview (CDC, National Center for Health Statistics, 2012b). Fat-mass index (FMI), the operationalization of the degree of adiposity, was calculated according to Davidson et al. (2011) from the NHANES anthropometric data (skinfolds and body height and weight; CDC, National Center for Health Statistics, 2007). Given that age has been demonstrated to have a systematic influence on a person's degree of adiposity and sleep habits (Heo, Faith, Pietrobelli, & Heymsfield, 2012), age was controlled in the analyses (Berry, 1993). The analysis was restricted to men because the evolution of a multi-year female reproductive cycle in humans was assumed to have resulted in a suppression of seasonal variation in female adiposity (Jasienska, 2011). Thus, the theorized trait of sleep-induced adiposity gain was not expected to be found in women.

Philosophical Justification of the Proposed Design

The present study follows the tradition in scientific discovery initially proposed by Popper (1959). This tradition stipulates that a new theory is first proposed within the generally accepted paradigm describing the phenomenon (Kuhn, 1970). In order to corroborate the theory empirically, it is subjected to a series of attempts to falsify it (Popper, 1959). Such falsification attempts involve a number of independent research hypotheses logically derived from the theory in question. If these hypotheses withstand multiple empirical tests with a high degree of success, the theory underlying them is viewed as tentatively corroborated (Popper, 1959).

Generally speaking, it is in the interests of the science that the odds of scientific acceptance are stacked against a newly proposed theory. From the point of view of scientific progress, the risks of widely adopting a wrong theory are much higher than those of mistakenly rejecting a genuine one. This logic demands that the empirical hypotheses derived from theory must make falsification of the theory relatively easy and avoidance of falsification relatively difficult (Meehl, 1997). In contrast, proposing a research hypothesis that is likely to be confirmed, in epistemological terms, amounts to a straw man fallacy: substituting a real opposition to the proposed theory with an easily defeatable one (Copi, Cohen, & McMahon, 2011).

This logic leads to a conclusion that proposing an obvious research hypothesis, for example, that White Americans sleeping longer hours would have higher adiposity than those getting a modal amount of sleep, would have been inadequate for the purposes of rigorous testing of the proposed theory. Prior studies have shown that it is quite often

the case that longer sleep is associated with higher adiposity (e.g., Chaput, Després, Bouchard, & Tremblay, 2008), so proposing this as a research hypothesis would indeed result in a straw man fallacy. A more rigorous test of the proposed theory had to probe a hypothesis that had had a low a-priori likelihood of being supported.

The research hypothesis advanced here has not been widely tested before, so there was little a-priori reason to believe that an interaction between race and sleep duration in predicting sex- and age-adjusted fat-mass index (FMI) would be discovered in the US population. Few researchers have attempted to test similar hypotheses and only one of these researcher teams (Meyer, Wall, Larson, Laska, & Neumark-Sztainer, 2012) found corroborating evidence (albeit for body-mass index rather than FMI). Therefore, the probability that the proposed null-hypothesis would be rejected was fairly small, which is argued to be a methodological strength of this study's design.

Operational Definitions

Degree of adiposity. Adiposity is defined as the total amount of adipose tissue in the body. Within the framework of the energy-balance theory, adipose tissue is viewed as the storage of body's energy reserves (Hall et al., 2012; although small short-term stores of energy also appear in the form of glycogen in muscle and liver; Wells, 2009a). Strictly speaking, stored energy is better represented by total body lipid or fat mass, because living adipose tissue also includes water and small blood vessels (Wells, 2009a). In the present study, however, this difference between total fat mass and total adipose tissue was ignored.

Measurement of adiposity poses a practical problem, because the only direct method involves full cadaver dissection (Wells, 2009a). Indirect measurement methods rely on assessment of the fat mass from molecular body composition, body density (air displacement plethysmography or hydrodensitometry), x-ray absorption (dual-energy x-ray absorptiometry), imaging (computed tomography or magnetic resonance imaging), body conductivity (bioelectrical impedance analysis), as well as anthropometric measurements, including skinfolds and circumferences (Ackland et al., 2012; Heymsfield et al., 2007; Rutherford & Diemer, 2011). In this study, the participants' fat mass was estimated from skinfold measurements, the most widely used epidemiological method of fat mass assessment (Ackland et al., 2012).

When comparing two individuals, the degree of adiposity is a more valid basis for comparison than the absolute fat mass; therefore an adjustment of fat mass for the size of the individual's body was made (Deurenberg & Deurenberg-Yap, 2007). In this study, fat mass index (FMI) was used as the operationalization of the degree of adiposity. FMI is calculated as the total fat mass in kilograms divided by squared body height in meters. FMI has been shown to provide a more accurate measure of the degree of adiposity compared to body mass index (BMI) and body fat percentage (BF%; Peltz, Aguirre, Sanderson, & Fadden, 2010; Rao et al., 2012).

Sleep duration. Habitual sleep duration was defined here as the self-reported number of hours of sleep during weeknights (CDC, National Center for Health Statistics, 2008). Although some normative data on sleep duration have been published (particularly, for children and adolescents; Iglowstein, Jenni, Molinari, & Largo, 2003;

Olds, Maher, Blunden, & Matricciani, 2010) most researchers in the studies reviewed here, selected the cutoffs for their definitions of short and long sleep arbitrarily. In adults, long sleep has most frequently been defined as 9 hours or more (Magee et al., 2012), short as 6 hours or less (Chaput, Després, Bouchard, & Tremblay, 2011a).

Assumptions

The proposed theory of sleep-induced adiposity gain postulated that human survival in the seasonal European environment resulted in the evolution of a trait of adaptive fat accumulation primed by extension of sleep duration. The theory claimed that sleep extension provided a behavioral cue to the organism about an approaching winter and, consequently, about an imminent change in food availability. Such sleep extension was assumed to have occurred naturally as a result of contraction of light day in late summer and early fall (Morrissey, 2012; Peixoto et al., 2009). A number of assumptions were made in order to support the logic of the proposed theory. These assumptions are discussed in this section.

European winters are associated with a sharp reduction in ambient temperatures, which must have produced an unprecedented set of survival challenges for anatomically modern humans inhabiting European continent from about 50,000 years ago (Lukic & Hey, 2012; J. R. Stewart & Stringer, 2012). Subsistence of these humans is believed to have been obtained through hunting and gathering (Dunbar et al., 2009). Agriculture was unknown at the time: its proliferation is believed to have started much later, about 10,000 years ago (Gignoux, Henn, & Mountain, 2011). Therefore, one can thus assume that Upper-Paleolithic humans could not rely on long-term stores of grain to offset any

irregularities in food provisioning and so the adiposity buffer was the main protection for early humans against the vagaries of energy supply (Wells, 2009a).

This line of reasoning led to the assumption that, in order to deal with the challenge of cold seasonality in their new European habitat, Upper-Paleolithic humans developed certain physiological mechanisms that helped them to predict approaching winter and to initiate gradual accumulation of adiposity early enough, so that they could support themselves through the period of temporary food scarcity. In the present work it was assumed that the annual change in the duration of night had been a reliable source of environmental information with good predictive value (Gangwisch, 2013). It was further assumed that a coordinated change in habitual sleep duration had been one of the methods that the evolving human organism developed to capture this season-predicting information and to feed it to the relevant physiological processes in the human body. The precise detail of this mechanism was outside of the scope of this work, but the main logic of its operation was as follows: (1) sleep is extended in response to lengthening nights; (2) sleep extension emerges as a behavioral predictor of an approaching winter; (3) sleep extension is used to induce adaptive seasonal adiposity gain needed to survive the imminent energy deficit. To conduct an empirical test of this theory, another set of assumptions was made.

Anatomically modern humans colonized Europe approximately 50,000 years ago in what is believed to be a gradual process of migration from their original habitat in equatorial Africa (Lukic & Hey, 2012). European climate is characterized by both warm/cold seasonality (J. R. Stewart & Stringer, 2012) and a substantial annual variation

in night duration (Morrissey, 2012). In contrast, equatorial Africa has barely any variation of night duration throughout the year. Therefore, the population that continued to evolve in its original habitat in Africa could not have acquired the theorized trait, whereas the population surviving in Europe should have done so.

It is generally assumed that the modern-day White Americans are most likely to be descendants of the European Upper-Paleolithic hunter-gatherers—whereas Black Americans, those of the equatorial-African ones (McEvoy, Powell, Goddard, & Visscher, 2011; problems with this assumption are discussed in more detail in the *Limitations* section in this chapter.) This assumption allowed to build an empirical test of the proposed theory based on the comparison between Blacks and Whites, because, if the theory were true, long habitual sleep would lead to higher adiposity in Whites compared to Blacks.

Inevitably, one has to assume that 50,000 years of geographical separation between the two populations was long enough for any noticeable difference in sleep-induced adiposity to appear and stabilize in the population genotype. However, as noted by Futuyma (2010), the main factor in genetic evolution was not the time period, but the strength of natural selection, in other words, the difference in the likelihood of survival with and without the focal trait. The logic of the empirical test proposed here relied on the assumption that the theorized trait of sleep-induced adiposity gain provided a survival advantage strong enough for this trait to result in a distinguishable difference between the present-day Blacks and Whites.

Yet, it would be wrong to assume that the theorized trait could never evolve in Blacks and its existence in Whites would become immediately apparent through statistical comparison. A more accurate way of looking at the problem was to assume that the theorized trait appeared in both Whites and Blacks with an equal likelihood, but that all Whites who *did not* possess this trait have been culled out by natural selection (Futuyma, 2010). Empirical evidence has shown that the genotypes of populations who survived in cold climates are indeed a fairly narrow subset of the genetic variation seen in less environmentally-stressed populations (Nishimura et al., 2012). Therefore, the challenge of this work was not to find a specific difference between White and Black genotypes as they are expressed in today's population phenotypes, but to distinguish the narrow genetic cluster expressed by Whites, from a more uniformly distributed genetic palette expressed by Blacks.

From the point of view of the modern populations that were compared, it had to be further assumed that the theorized trait of seasonal sleep-induced adiposity gain expressed in modern environment resulted in a gradual progression of obesity, rather than a seasonal pattern of weight gain and loss. Today, the ubiquitous use of electric lighting suppresses the seasonal influence of environmental light factors (Peixoto et al., 2009). As a consequence, seasonal variation in sleep duration becomes fairly insignificant (O'Connell, Griffiths, & Clemes, 2012). In this situation, habitually long sleep must result in constant activation of the hypothesized mechanism of adiposity gain. Such activation must lead to chronic obesity. The described assumption provided a logical link from the theory of seasonal fat accumulation primed by sleep extension in hunter-

gatherers—to a hypothesis of chronic obesity caused by habitually long sleep, in their modern descendants. The issues associated with this assumption are also discussed in the *Limitations* section.

Finally, this work's underlying assumption of ceteris paribus (everything else being equal) pre-supposed that, apart from the postulated trait of sleep-induced obesity, the metabolism of Blacks and Whites works exactly the same. This generalization was perhaps unwarranted (Itan, Jones, Ingram, Swallow, & Thomas, 2010), especially since the present work did in fact postulate at least one difference in sleep-associated metabolism in these two populations.

In summary, the assumptions of the present study included (a) out-of-Africa migration of early humans resulting in long-term geographic separation between hunter-gatherer populations in Continental Europe and Sub-Saharan Africa leading to (b) genetic differences between these two populations resulting from differences in seasonality-related natural selection. These genetic differences were further assumed to produce (c) cross-sectional phenotypic differences in sleep-related energy homeostasis in descendants of these two populations found among the present-day residents of the United States. Finally, it was assumed that (d) the phenotypic differences between Blacks and Whites, if they had been discovered, could be attributed to the theorized genetic differences, rather than an unrelated metabolic difference.

Scope and Delimitations

Race

This study's analysis was restricted to comparisons between Black and White Americans. It is generally assumed that other human races have experienced a far more complex paths of migration and interbreeding (McEvoy et al., 2011). These paths do not allow for an equally serendipitous north-south separation between races, as afforded by Sahara Desert and the Mediterranean Sea, in the case of African and European hunter-gatherers. Therefore, other races and ethnicities were excluded from the analysis.

Sex

If early humans maintained stable average weight throughout adulthood, as virtually all vertebrates do (Pond, 1998, 2007; Wells, 2009a), the theorized seasonal weight gain must have been balanced by a counter-seasonal weight loss. It is possible that the survival advantage afforded by circannual fluctuation of adiposity might not be the same for males and females, given the intricate relationship between adiposity, reproductive function, and sexual selection (Wells, 2006).

For human males living in Europe and engaged in hunting and gathering (Dunbar et al., 2009; Hockett & Haws, 2009), an improved muscle-to-fat ratio was clearly advantageous in late spring and early summer when ambient air was warmer but individuals still had to rely on animal food sources (Allué, Ibáñez, Saladié, & Vaquero, 2010; Gangwisch, 2013). In this case, carrying large adiposity stores did not provide additional survival advantage – or may have even been a disadvantage, if it impaired the ability to migrate, chase prey, or escape predators (Heyer, Chaix, Pavard, & Austerlitz,

2012; Wells, 2009a). In contrast, in late summer and fall, as more plant fruit became available, physical agility required to chase prey became less critical for survival. At the same time, the ability to accumulate energy in the form of adiposity stores in preparation for the coming winter became advantageous (Wells, 2009a). Thus, for human males, the strategy of seasonal variation in adiposity would make good evolutionary sense.

Such adiposity strategy would be equally well-aligned with seasonal reproductive strategy. From the reproductive point of view, this strategy involves reducing adiposity stores in late spring and investing energy obtained from food into reproductive effort (Ellison, 2011); in late summer and fall, reproductive activity is suppressed (Eriksson, Fellman, Jorde, & Pitkänen, 2008; Turnbull, Nguyen, Jamieson, & Palerme, 2010), and surplus energy is invested in growing adiposity stores (Bribiescas, 2011). Thus, for human males, seasonal variation in adiposity seems to have been advantageous.

For women, however, seasonal energy balancing must be quite different (Davis et al., 2010). While accumulating additional adiposity was beneficial in preparation for approaching winter, losing it in spring did not provide women with any survival advantage (Jasienska, 2011). If for men increased reproductive activity implied migrating in search of new mates (Heyer et al., 2012), courting them, fending off competing males, and producing large amounts of energy-costly sperm—all of which was discordant with long-term fat accumulation (Bribiescas, 2011)—for women, investment in reproduction was directly associated with maintenance of a constantly high level of adiposity (Jasienska, 2011; Pike, 2011). Given the duration of the female reproductive cycle, which demands high energy expenditure throughout in-utero gestation and further

2–3 years of breastfeeding (Sellen, 2001; Valeggia & Ellison, 2011), a seasonal variation in adiposity would imply a potentially risky energy instability, which seems to confer no additional survival advantage (Valeggia & Ellison, 2011). Thus, assuming that stable weight was maintained by humans throughout a healthy adult life, no seasonal variation in adiposity was expected to be found in women.

Thus, a trait of seasonal variation in adiposity must have been advantageous for male hunter-gatherers, because it reflected a circannual cycle of reproduction and energy conservation. In women, on the other hand, the presence of the menstrual, rather than circannual, reproductive cycle suggested that there was no major circannual variation in energy-conservation strategies in females during Upper Paleolithic period and beyond. It was thus possible to conclude that the female reproductive strategy presupposed constant maintenance of a relatively high level of adiposity (Jasienska, 2011). Thus, the present study included only men, because the expression of the theorized trait of sleep-induced adiposity gain was assumed to be suppressed in women.

Other Sleep-Related Factors

Other sleep-related factors have been associated with onset and progression of obesity. These include chronotype (also called eveningness / morningness, which categorizes the shift in the median of the sleep period towards either earlier or later in the morning; Chung, Kan, & Yeung, 2013), variability of sleep duration from day to day (Kobayashi, Takahashi, Shimbo, et al., 2012), sleep debt and sleep compensation during weekends and holidays (Spruyt, Molfese, & Gozal, 2011), shift work and the consequent substitution of sleep and waking activities within the day cycle (Macagnan et al., 2012),

duration of different stages of sleep (particularly, rapid eye movement and slow-wave sleep; Wojnar et al., 2010), sleep quality (Wilsmore et al., 2012), perceived adequate quantity of sleep (Wheaton et al., 2011), excessive daytime sleepiness (Vgontzas, Bixler, Chrousos, & Pejovic, 2008), and napping during the day (Picarsic et al., 2008). Because voluntary variation in sleep duration was the main interest of the present study, the total nocturnal sleep duration was the only sleep variable analyzed.

Limitations

Americans vs Europeans and Africans

It was assumed that modern-day Black and White Americans would provide an adequate representation of adaptive traits that evolved in African and European Upper-Paleolithic hunter-gatherers. The problem with this assumption was that ancestral genotypes of Black and White Americans have inevitably been confounded by many generations of genetic intermixing between the two races living in the same country (McEvoy et al., 2011). Arguably, a much better design would be afforded by a comparison of actual European and African populations, rather than White and Black Americans. However, conducting multi-country epidemiological data collection for this study was not feasible, and comparable sets of prior data—with adequately large sample sizes and equivalent data collection methodologies—were not available.

Cross-Sectional Nature of the Design

Another assumption was that the existence of the theorized causal relationship between sleep extension and fat accumulation could be inferred from a cross-sectional relationship between the habitual sleep duration and the fat-mass index. This is technically not so; however, cross-sectional data could provide insight into potential causal relationships. Higher validity of inference would have been achieved from a longitudinal experimental study of the relationship between sleep extension and adiposity gain. However, such study might be impossible to conduct due to ethical considerations (Nielsen, Danielsen, & Sørensen, 2011).

Wells and Siervo (2011) theorized that the relationship between sleep duration and adiposity could be spurious, with diet high in refined carbohydrates leading to both reduction in habitual sleep and to obesity. Alternatively, Siervo et al. (2009) proposed that frequent exposure to psychological stress in modern societies may result in both sleep loss and weight gain (Elder et al., 2012). This again implies that short sleep duration and obesity are correlated without causing each other (Siervo et al., 2009). It has to be noted that the energy-balance theory can predict an equally plausible account of the reverse relationship, in other words, obesity leading to short sleep. It is possible to argue that obese people have enough energy reserves to allow them to stay more active during the day—and, therefore, sleep less (Wells et al., 2008).

Despite logical plausibility of this theory, there has been little interest in pursuing it in the present scientific environment. First, it does not solve the problem of obesity (and, hence, that of extramural funding). Second, it has dubious practical significance:

does it make sense to reduce global obesity simply to extend the average sleep duration? The present work has been equally affected by this scientific-interest bias, because it likewise predicted that extended sleep duration led to obesity, rather than vice versa. As a consequence, the present study tested the hypothesis that Whites—more than Blacks—would be prone to develop obesity in response to long sleep. A reverse interpretation of the same finding would have been that Blacks—compared to Whites with the same degree of obesity—would have a stronger tendency to extend their sleep. Note that such interpretation was equally plausible due to a cross-sectional design of this study.

Culture

Culture has been shown to have an impact on habitual sleep duration as well as on attitudes towards obesity (Trigwell, Watson, Murphy, Stratton, & Cable, 2013). Earlier studies found systematic differences between American Blacks and Whites in the population distributions of both obesity and self-reported sleep duration (Heo et al., 2012; Krueger & Friedman, 2009). There are consistent differences between American Blacks and American Whites in obesity-related body-image satisfaction, culturally specific perceptions of healthiness and attractiveness of high levels of adiposity (Ulijaszek & Lofink, 2006)—as well as in normative sleep practices (Nielsen et al., 2011). It is possible that cultural differences between Black and White Americans could result in a racial difference in the sleep–adiposity relationship. From this point of view, culturally prescribed attitudes and behaviors would act as confounding factors that an epidemiological study like this would be unable to control. Indeed, race and culture are

so inextricably linked that separating one from the other would be methodologically impossible.

Participants of NHANES Study

The present study was based on the analysis of archival data from NHANES survey (CDC, National Center for Health Statistics, 2013b). Therefore, this study's scope was inevitably be restricted by the scope of the NHANES. One of such restrictions resulted from the fact that sleep duration data were collected only from participants aged 16 years or older.

Only Weeknight Sleep Reported in NHANES

Another restriction of NHANES survey design was that the sleep questionnaire asked participants only about their habitual sleep during weeknights (CDC, National Center for Health Statistics, 2008). Wing, Li, Li, Zhang, and Kong (2009) showed that weekend and holiday sleep had a moderating effect on the relationship between weeknight sleep and obesity risk in children and adolescents. The definition of habitual sleep used by NHANES improved precision and allowed to avoid construct confounding, which could result from participants' reporting sleep duration averaged over the entire week. However, additional information about weekend sleep, which would provide a potentially important control variable for the prediction of sleep-related adiposity, was not collected by NHANES.

Clinical Underweight

Additional limitation of this study was the lack of control for clinical conditions in participants that could lead to both underweight and long sleep. Analyzing a survey

similar to NHANES, Krueger and Friedman (2009) found a significant prevalence of clinical underweight in participants reporting long sleep. The presence of morbidities associated with under-nutrition may have depressed estimates of mean adiposity in participants reporting longer sleep.

Significance

Obesity is associated with a number of negative health outcomes, increased mortality, and impaired psycho-social well-being (Arterburn et al., 2012; Berrington de Gonzalez et al., 2010). Although inadequate sleep has been identified as a behavioral factor contributing to onset and progression of obesity, the mechanism of this relationship is still not known (St-Onge, 2013). The present study, which included a test of a hypothesis based on evolutionary theory, represents an attempt to fill this explanatory gap by postulating a trait of sleep-induced adiposity gain that evolved in early humans living in Europe. If this genetic mechanism was discovered in modern descendants of the European Upper-Paleolithic hunter-gatherers, the proposed theory could provide much stronger support for the development of population-specific recommendations for the optimum duration of sleep (Johnsen, Wynn, & Bratlid, 2013).

Although this study did not deal with genetic causes of obesity as such, it might have pinpointed one specific behavioral factor leading to the expression of the hypothesized thrifty genes (Gangwisch, 2013). In this case, such behavioral factor was long habitual sleep. In this context, a better understanding of the dynamics of thrifty genes could help fill the “missing heritability” gap (Manolio et al., 2009, p. 747).

Finally, the proposed theory would inform more effective sleep interventions aimed at treatment and prevention of obesity (Cizza et al., 2010), that may have eventually led to the reduction of the global burden of obesity. Although anti-obesity treatments have traditionally been race-agnostic (Göhner et al., 2012; McConnon et al., 2012), there is an emerging trend for cultural adaptation of psychological interventions (Corsino et al., 2012). Race differences have been observed in attitudes towards obesity (Trigwell et al., 2013), body-image perceptions (Ulijaszek & Lofink, 2006), as well as self-monitoring of energy intake during anti-obesity interventions (A. Kong et al., 2012), all of which may have important implications for the effectiveness of anti-obesity programs. Since sleep-duration monitoring is becoming an important element of anti-obesity hygiene (Cizza et al., 2010), the findings of this study may have informed the development of race-specific sleep interventions. Specifically, if longer sleep duration was producing a higher weight gain in Whites, as was hypothesized in this work, a closer control of sleep duration during holidays and weekends should have been given priority in behavioral interventions designed for treatment and prevention of obesity in White men.

Summary

Obesity has reached the proportions of a global epidemic, but a safe and reliable method for its treatment and prevention is yet to be found. Although the general physics of obesity is fairly straightforward, scientists are still working out specific theories that would explain the mechanisms of its onset and progression. This work postulated an evolutionary theory of adiposity gain induced by long sleep in the present-day

descendants of European hunter-gatherers. The proposed theory, if it had been corroborated, would explain one of the pathways that sleep behavior may influence the progression of obesity in one specific population domain: White males. A successful corroboration of this theory would inform the development of efficient domain-specific treatment and prevention protocols.

In order to subject this theory to a rigorous empirical test, this study advanced a low-likelihood research hypothesis. This hypothesis postulated the existence of an interaction between sleep duration and race (American Black and American White) in prediction of age-adjusted adiposity, operationalized via skinfold-derived fat-mass index. The low likelihood of the proposed hypothesis implied that an empirical support of this hypothesis would constitute a strong corroboration of the proposed theory. In this study, the analysis was restricted to men, because the postulated trait of sleep-induced adiposity was assumed to be attenuated in women. Archival data, collected as part of National Health and Nutrition Survey (CDC, National Center for Health Statistics, 2013b), was used for the analyses.

The following chapter will provide a review of scientific literature relevant for the ideas advanced above. The association between sleep and obesity is a subject of active research by many teams worldwide. A large number of conflicting theories proposed to explain this association, may be viewed as a sign of an imminent breakthrough in scientific understanding of this phenomenon.

Chapter 2: Literature Review

A number of researchers have identified a relationship between sleep duration and various markers of adiposity (Wilsmore et al., 2012). Most researchers have associated the progression of obesity with short sleep; the results for long sleep have been less consistent (Magee et al., 2012). As a consequence, very few explanatory theories about the influence of long sleep on obesity development have been advanced (Knutson, 2012).

To fill this gap, the present work proposed a theory about the relationship between sleep duration and obesity. According to this theory, long sleep provided a behavioral cue for seasonal fat accumulation in European Upper-Paleolithic hunter-gatherers. This mechanism was adaptive for early humans living in the seasonal European environment (J. R. Stewart & Stringer, 2012), but is theorized to have become deleterious for their present-day descendants. This is because such mechanism induces obesity in response to habitually long sleep. The purpose of this chapter is to review the prior theoretical and empirical work that has to be considered in evaluating the merits of the proposed theory and the research hypothesis derived on its basis.

Organization of the Chapter

After an outline of the literature search strategy, this review includes a discussion of the research problem in the context of global proliferation of obesity and its growing financial and personal costs resulting from obesity-related morbidity and mortality. Following this is a summary of the energy-balance theory of obesity, including its major tenets, physical foundations, and empirical evidence, as well as treatments and interventions informed by it. Following this is a description of the limitations of the

energy-balance theory and the way these limitations have been addressed in modern research, including the present study. Research relating to long sleep is presented. Research findings on the association of obesity to long and short sleep duration are integrated with the existing theoretical explanations of obesity, and the main tenets of the proposed evolutionary theory of sleep-induced adiposity gain. Empirical studies are then examined related to the race–sleep interaction hypothesized in this work. In conclusion, this review will provide a description of a theory of lactose persistence that is cited as an evolutionary analogy to the trait theorized here.

Literature Search Strategy

The literature search was conducted using EBSCO and Embase search engines for peer reviewed articles from January 2008 to September 2013 in the following databases: Academic Search Complete, CINAHL Plus, Embase, MEDLINE, PsycARTICLES, and PsycINFO. For the review of literature related to sleep and obesity the term *sleep* was used in combination with *obesity*, *overweight*, *obese*, *weight*, *BMI*, *body*, *mass*, *adiposity*, and *fat*. Because of the focus on adaptive mechanisms of sleep-induced adiposity gain that evolved in humans during the Upper Paleolithic period (50–10 thousand years ago; Pinhasi, Thomas, Hofreiter, Currat, & Burger, 2012), animal studies were excluded. Further, because the hypothesized mechanism was assumed to be present in general population and not related to particular disorders, the review did not include studies of obstructive sleep apnea, sleep-disordered breathing, obesity hypoventilation syndrome, and sleep disorders.

For the review of health issues related to long sleep, the term *long sleep duration* was searched. For the review of adiposity measurement, the search term *fat* was used in combination with *mass* and *BMI*, as well as *body composition* and *assessment*. For the review of obesity-related health issues and interventions, the search terms *obesity*, *obese*, and *overweight* were used in combination with *health*, *morbidity*, *diabetes*, *heart failure*, *stroke*, *fatty liver*, *pancreatitis*, *renal*, *cancer*, *gallbladder*, *mortality*, *cognitive*, *dementia*, *Alzheimer*, *well being*, *body image*, *mood*, *depression*, *mania*, *anxiety*, *panic*, *phobia*, *suicide*, *policy*, *cost*, *intervention*, *satiety*, *gastric bypass*, *sleeve gastrectomy*, *gastric banding*, *biliopancreatic diversion*, *duodenal switch*, *theory*, *seasonality*, and *gene*. The reference lists of selected articles were analyzed, and notable studies published prior to 2008 were also included in this review.

Obesity and its Implications

This review begins with a discussion of the phenomenon of obesity in the context of its global prevalence, associated health risks, and personal and societal costs.

Global Obesity Epidemic

Obesity has emerged as a major health hazard for both developed and developing nations (Bamidele, Olarinmoye, Olajide, & Abodunrin, 2011; Finucane et al., 2011). At the current rate of obesity's progression, all American adults (Y. Wang, Beydoun, Liang, Caballero, & Kumanyika, 2008) and 90% of British adults (McPherson, Marsh, & Brown, 2007) will be overweight or obese by 2050. Y. C. Wang et al. (2011) estimated that by 2030, obesity related diseases would result in additional healthcare costs of $48–66 billion a year in the United States alone. Although recent evidence showed signs of

stabilization or even reduction in the rates of obesity in the developed world (Harbaugh et al., 2011; Nichols et al., 2011), it has been noted that earlier stabilizations have often been followed by increases in prevalence (Rokholm, Baker, & Sørensen, 2010).

A special concern has been the prevalence of obesity in childhood and adolescence—because it usually persists into adulthood (M. H. Park, Falconer, Viner, & Kinra, 2012). Rates of childhood obesity continue to grow globally (Armstrong, Lambert, & Lambert, 2011; Midha, Nath, Kumari, Rao, & Pandey, 2012), although some populations (for example, Australian preschool children; Nichols et al., 2011) have also shown stabilization or decrease in prevalence (see Rokholm et al., 2010, for a detailed review).

Obesity and Negative Health Outcomes

Often associated with general morbidity and disability (Arterburn et al., 2012), obesity has been found a predisposing factor for a large number of physiological and psychological disorders. Although not a disease proper (Moffat, 2010), obesity has so often been associated with hypertension (Hwang, Bai, Sun, & Chen, 2012), dyslipidemia (Nahar, Dubey, Joshi, Phadnis, & Sharma, 2012), and insulin resistance (Ghandehari, Le, Kamal-Bahl, Bassin, & Wong, 2009) that these four have been used as the diagnostic axes of so called *metabolic syndrome* (Gupta, Prieto-Merino, Dahlöf, Sever, & Poulter, 2011). Metabolic syndrome is a combination of factors associated with a high risk of developing Type 2 diabetes mellitus (Langenberg et al., 2012) and cardiovascular disease (de Groot, Dekkers, Romijn, Dieben, & Helmerhorst, 2011). These factors can lead to

venous thrombosis (Allman-Farinelli, 2011), heart failure (Loehr et al., 2010), and stroke (Towfighi, Zheng, & Ovbiagele, 2010).

Maternal obesity has been associated with an increased risk of gestational hypertension (Kazemian, Sotoudeh, Dorosti-Motlagh, Eshraghian, & Bagheri, 2012) and fetal anomalies (Stothard, Tennant, Bell, & Rankin, 2012). Childhood obesity has also been linked with increased risk of Type 1 diabetes (Verbeeten, Elks, Daneman, & Ong, 2011) and double diabetes, i.e. both Type 1 and 2 present concurrently (Pozzilli, Guglielmi, Caprio, & Buzzetti, 2011).

Inflammatory processes in the adipose tissue make obesity, in essence, equivalent to low-grade systemic inflammation (Roth, Kratz, Ralston, & Reinehr, 2011), which is seen as a key causal link between insulin resistance, Type 2 diabetes, and cardiovascular disease (Winer & Winer, 2012), non-alcoholic fatty liver disease (Kelishadi & Poursafa, 2011), renal disease (Gilardini et al., 2010), pancreatitis (Shin et al., 2011), gallbladder disease (Di Ciaula, Wang, & Portincasa, 2012), respiratory disease (including asthma and obstructive sleep apnea; Yeh et al., 2011), musculoskeletal problems, and gout (Maynard et al., 2012). Inflammation also contributed to perioperative and postoperative complications associated with obesity, such as substantial blood loss (Williams et al., 2009), higher operation complexity (Jayachandran et al., 2008) and duration (M. G. Kim et al., 2011), longer hospital stay (Kalanithi, Arrigo, & Boakye, 2012), higher rates of readmission (Giugale, Di Santo, Smolkin, Havrilesky, & Modesitt, 2012) and reoperation (Hirose, Shore, Wick, Weiner, & Makary, 2011), wound infections (Leth, Uldbjerg, Nørgaard, Møller, & Thomsen, 2011), thromboembolism (Davenport et al., 2009),

periprosthetic joint infections (Jämsen et al., 2012), and lower rates of transplant survival (Cacciola et al., 2008).

Obesity and obesity-induced chronic inflammation have been associated with higher incidence of certain types of cancer (Hemminki, Li, Sundquist, & Sundquist, 2011), including liver (Schlesinger et al., 2013), breast (Amir et al., 2012), kidney (Attner, Landin-Olsson, Lithman, Noreen, & Olsson, 2012), thyroid (Kitahara et al., 2011), ovarian (Olsen et al., 2007), prostate (Hoda, Theil, Mohammed, Fischer, & Fornara, 2012), colorectal (Ho et al., 2012), pancreatic (D. Wang et al., 2012), and endometrial (Reeves et al., 2011) cancers. Connections also have been established between obesity and leukemia (Castillo et al., 2012) and adenocarcinoma of esophagus (Doyle et al., 2012). In fact, almost a half of all cases of post-menopausal endometrial cancer and adenocarcinoma of esophagus in women have been attributed to overweight and obesity (Meredith, Sarfati, Ikeda, Atkinson, & Blakely, 2012; D. Wang et al., 2012).

Mortality

Obesity is associated with an increased risk of all-cause mortality (Berrington de Gonzalez et al., 2010), as well as specific-cause mortality due to diabetes (Abdullah et al., 2011), cardiovascular disease (Schneider et al., 2012), respiratory disease (Jordan & Mann, 2010), liver disease (Batty et al., 2008), kidney disease (Flegal, Graubard, Williamson, & Gail, 2007), and obesity-related cancers (Castillo et al., 2012). The speculated causal link between obesity and mortality is supported by the apparently protective effects of bariatric surgery (Plecka Östlund, Marsk, Rasmussen, Lagergren, & Näslund, 2011).

Psychological Effects of Obesity

Childhood. Childhood obesity and metabolic syndrome have been associated with deficiencies in cognitive development, including impaired attention and executive function (Verdejo-García et al., 2010), possibly mediated by sleep disordered breathing (Beebe, Ris, Kramer, Long, & Amin, 2010). Obesity in childhood and adolescence has also been linked with emotional and behavioral problems (Griffiths, Dezateux, & Hill, 2011), anxiety (Pitrou, Shojaei, Wazana, Gilbert, & Kovess-Masféty, 2010), depression (Anderson et al., 2010), peer censure and social isolation (Brixval, Rayce, Rasmussen, Holstein, & Due, 2012), body-image dissatisfaction, low self-esteem, and poor subjective well-being (Mériaux, Berg, & Hellström, 2010; Mond, van den Berg, Boutelle, Hannan, & Neumark-Sztainer, 2011). The perception of being overweight has been found to correlate with lower academic performance (Florin, Shults, & Stettler, 2011). Overweight in adolescents have been implicated in the increased risk of suicide attempts (Swahn et al., 2009).

Adulthood. In adulthood, obesity has been linked to weaker cognitive performance (Sabia, Kivimaki, Shipley, Marmot, & Singh-Manoux, 2009), including impaired executive functioning (Cserjési, Luminet, Poncelet, & Lénárd, 2009), attention (Siervo et al., 2011), and memory (Nilsson & Nilsson, 2009). Obesity has also been related to the erosion of subjective well-being (Shebini, Kazem, Moaty, & El-Arabi, 2011), low self-esteem, and deepening body image dissatisfaction (Brytek-Matera, 2011; Gavin, Simon, & Ludman, 2010). Obesity has also been associated with anxiety disorders (Brunault et al., 2012), social phobia (Mather, Cox, Enns, & Sareen, 2009), and

stress (Elder et al., 2012). Women with overweight and obesity were found to be more susceptible to mood disorders (Pickering et al., 2011) and depression (Hamer, Batty, & Kivimaki, 2012). In addition, obesity has been one of the prime suspects in suicide ideation and suicide attempts, especially in women (Klinitzke, Steinig, Blüher, Kersting, & Wagner, 2013).

Late adulthood. With advancing age, obesity has been associated with deteriorating cognitive performance (Hassing, Dahl, Pedersen, & Johansson, 2010), depression (Almeida, Calver, Jamrozik, Hankey, & Flicker, 2009), higher incidence of dementia and Alzheimer disease (Luchsinger, Cheng, Tang, Schupf, & Mayeux, 2012; W. L. Xu et al., 2011), impaired memory, language, executive function (Gunstad et al., 2007; Gunstad, Lhotsky, Wendell, Ferrucci, & Zonderman, 2010), and psychomotor speed (E. Kim et al., 2008). Obesity also has been linked with higher rates of completed suicide among the elderly (A. Shah, 2010).

Given the growing evidence of correlation between obesity and mental disorders, some scholars raised the question whether obesity is itself a mental disorder (Marcus & Wildes, 2012). However, the recent analysis of this issue by the Eating Disorders Work Group of the *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition (*DSM-5*) Task Force resulted in a decision not to classify obesity as a mental disorder at present (Marcus & Wildes, 2012).

Costs of Obesity

Costs of obesity are classified as direct, indirect, and personal (Caterson, Franklin, & Colditz, 2007).

Direct costs of obesity. Direct costs are those attributed to obesity and its associated conditions (Au, 2012). In the developed economies, direct healthcare costs of a person with obesity were found to be on average 30-60% higher than those of a person with BMI in the normal range (Withrow & Alter, 2011). Finkelstein et al. (2008) estimated lifetime medical costs for a 20 year old attributable to obesity I (BMI from 30 to 35 kg/m^2) between $5,340 and $21,550, and obesity II and III (BMI over 35 kg/m^2), between $14,580 and $29,460 in 2007 dollars. Y. C. Wang et al. (2011) estimated additional healthcare costs directly attributable to obesity in the United States at $22–28 billion a year in 2020 and $48–66 billion a year in 2030, or between 0.8 and 2.6% of total healthcare spending (see also Au, 2012; Withrow & Alter, 2011). In comparison, McPherson et al. (2007) projected that in 2025 British total healthcare costs attributable to obesity will reach £37.2 billion a year, or 11.9% of the total healthcare bill.

Indirect costs of obesity. Indirect costs of obesity are defined as "the reduction in the level of economic activity due to the illness and premature death attributable to obesity" (Caterson et al., 2007, p. 150). The indirect costs of obesity (Knai, Suhrcke, & Lobstein, 2007) are further classified into absenteeism (Arterburn et al., 2012) and presenteeism (i.e. malingering while at workplace; Finkelstein, DiBonaventura, Burgess, & Hale, 2010). The annual cost of excess absenteeism attributable to obesity in the United States has been estimated between $3.4 billion (Trogdon, Finkelstein, Hylands, Dellea, & Kamal-Bahl, 2008) and $12.8 billion (Finkelstein, DiBonaventura, et al., 2010); that of presenteeism from $9.1 billion (Trogdon et al., 2008) to $30bn (Finkelstein, DiBonaventura, et al., 2010). Y. C. Wang et al. (2011) estimated current

indirect costs attributable to obesity in the United States at 1.7–3.0 million productive person years, equivalent to a $390–580 billion cost to the US economy between 2010 and 2030. Lightwood et al. (2009) estimated annual indirect costs of productivity lost specifically due to adolescent obesity in the range from $942 million in 2020 to $36 billion in 2050.

Personal costs of obesity. Personal or intangible costs of obesity are assessed via estimating obesity-related reduction in subjective quality of life (Grammer et al., 2010). More objective estimates include *years lost to disability* (YLD), *disability-adjusted life years* (DALY), and a hybrid measure of *quality-adjusted life years* (QALY; S. T. Stewart, Cutler, & Rosen, 2009). Y. C. Wang et al. (2011) estimated that, at the current rate of progression, obesity will cost the United States population between 24.5 and 48.2 million quality-adjusted life years from 2010 to 2030. Further, Lightwood et al. (2009) estimated that 1.48 million life years will be lost due to obesity-related mortality between 2020 and 2050. According to another estimate, if the obesity prevalence remains unchanged, the 2008 US population will lose almost 95 million years of its lifetime due to obesity II and III (Finkelstein, Brown, Wrage, Allaire, & Hoerger, 2010; see also Jia & Lubetkin, 2009).

Government Policy Interventions

In the view of mounting costs of obesity, some countries have urged government intervention (Dean & Elliott, 2012; Gortmaker et al., 2011; Loureiro & Freudenberg, 2012). Various policies have been proposed and implemented, including taxes on caloric sweetened beverages (Jou & Techakehakij, 2012); ice cream, chocolate, and sweets

(Wilkins, 2010); as well as junk foods (Sacks, Veerman, Moodie, & Swinburn, 2011). Some governments offered subsidies to stimulate consumption of fruit and vegetables (Faulkner et al., 2011), introduced additional requirements for food labeling (MacKay, 2011), restricted TV advertising targeting children (Magnus, Haby, Carter, & Swinburn, 2009), and prescribed lists of foods that can be sold in school canteens (Sanchez-Vaznaugh, Sánchez, Baek, & Crawford, 2010).

Government efforts have also been directed at raising public awareness of risks of obesity (Salopuro et al., 2011), funding school-based sports (Moodie, Carter, Swinburn, & Haby, 2010), educational (P. Shah et al., 2010), and health-screening programs (Zhang et al., 2010). Some authors even tried to justify court interventions (Mitgang, 2011; Pomeranz & Brownell, 2011).

To summarize, the prevalence of obesity continues to grow around the world. Although not a disease as such, obesity is associated with significant morbidity, increased mortality, impaired psychosocial functioning, and growing personal and financial costs.

Theoretical Foundation: The Energy-Balance Theory of Obesity

Having put the global obesity epidemic in the context of its impact on health, quality of life, and economic productivity, the present discussion will move to an overview of the energy-balance theory. This theory forms the foundation of the mainstream scientific framework used to explain the development of obesity and to inform treatments and interventions (Hall et al., 2012). This section includes the key elements of this theory, supporting empirical evidence, and theory's limitations.

Foundations of the Energy-Balance Theory

Obesity is defined as abnormal accumulation of excess adipose tissue (Flier & Maratos-Flier, 2008). Since the main functional purpose of adipose tissue is storage of triglycerides (Lafontan & Langin, 2009)—and triglycerides form the primary vehicle for long-term energy accumulation in vertebrates (Pond, 1998; Wells, 2009a)—excess adiposity is thus construed as excess energy accumulated in the body (Hall et al., 2012). The modern conceptualization of obesity as a metabolic disorder resulting in positive energy balance was formulated in the 1930s (Campbell, 1936; Lambie, 1935), and it is now widely accepted that the progression of obesity is caused by a disruption of an otherwise stable energy equilibrium: this disruption prompts the organism to accumulate more energy than it spends (Hall et al., 2012).

The need for energy balancing follows from the law of conservation of energy (Hess, 1839), which postulates that energy equilibrium is maintained only when energy expenditure is compensated by energy intake. Mere maintenance of basic physiological processes, which in terms of body energetics appears as resting energy expenditure, takes up around two thirds of the total energy budget of the body (de Jonge et al., 2012). On the other hand, energy intake in the form of nutrition happens only at certain intervals, because feeding is only one of several competing functional tasks performed by an organism (Dunbar et al., 2009). Because an individual often cannot predict the next opportunity for energy intake, maintaining a constant positive energy balance results in improved survival (Wells, 2009b).

This evolutionary logic was used by Neel (1962) to postulate the existence of a thrifty genotype. Those with this genotype are particularly good at utilizing available food sources and converting them into adiposity depots. As an unintended consequence, these people are more prone to develop obesity. According to Neel, famines during the human evolutionary history provided the selection pressure that supported the proliferation of thrifty genes. Bouchard (2007) proposed that several thrifty genotypes—not necessarily mutually exclusive—could have evolved. They could use different approaches to supporting positive energy balance, including a propensity for (a) having low base metabolic rate, (b) overfeeding, (c) being physically inactive, (d) maintaining low lipid oxidation rate, and (e) developing a high lipid storage capacity (Bouchard, 2007).

Neel's (1962) theory of evolving gene for energy thriftiness prompted a search for signs of genetic predisposition to obesity. This search, however, produced mixed results (Speakman, 2006). Heritability of obesity estimated from twin studies was found to be around 70% (O'Malley & Stotz, 2011), which seems to support the idea of a thrifty genotype. Nearly 60 genetic loci have been associated with obesity (Choquet & Meyre, 2011). The problem is that the identified obesity-related genes account for about 5% of heritability predicted from twin studies, resulting in so-called "missing heritability" (Manolio et al., 2009 p. 747).

To deal with this issue, more recent obesity-related genetic research concentrated on gene-environment interactions (Bouchard, 2008). In this context, Gangwisch (2013) hypothesized that seasonal variation in nutrition, temperature, sleep duration, and

physical activity may have circannually varying effects on the expression of the thrifty genotype in humans. This line of reasoning is further extended in the present work.

The major theoretical propositions of the energy-balance theory of obesity—widely accepted for almost a century—can be summarized in the following manner (Kennedy, 1966; Lafontan & Langin, 2009; Lambie, 1935). First, any organism maintains a close control of the balance sheet of its energy acquisition and expenditure. Second, obesity results from chronic positive energy balance. Third, the amount of energy assimilated from ingested food is only weakly related to the food's total caloric content, just like energy expended by the organism is weakly related to the person's physical exertion. This is because an organism's energy balancing is moderated by complex hormonal processes orchestrated by various brain structures (the fourth point). While the first two propositions follow from the second law of thermodynamics and the law of conservation of energy, the latter two have been established empirically (Kondoh, Mallick, & Torii, 2009; Siervo et al., 2009). The next section contains a review of the empirical evidence for the relationship of energy intake and expenditure to obesity as predicted by the energy-balance theory; the following sections will cover the evidence-based interventions, as well as the limitations to the theory's explanatory power.

Empirical Evidence Supporting Energy-Balance Theory

Energy-balance theory of obesity predicts that reduction in energy intake and increase in energy expenditure will lead to energy deficit and the consequent reduction of adiposity; likewise, balancing energy intake with expenditure leads to energy equilibrium and, consequently, stable levels of adiposity (Hall et al., 2011; Hall et al., 2012).

Energy intake. Although the correlation between food intake and energy absorption follows logically from the law of conservation of energy (Swinburn, Sacks, & Ravussin, 2009), a human organism exerts complex control over the energy intake. This control is implemented through the psychological mechanisms of appetite and satiety (Gilbert et al., 2011), as well as physiological mechanisms adjusting the degree of energy absorption from the food that has been ingested (Schwartz, 2011). Given a real or perceived energy deficit, a human body is equally capable of transforming dietary fat (Ambrosini et al., 2012), protein (Eloranta et al., 2012), and carbohydrates (F. M. Silva et al., 2011) into adiposity stores, albeit with differing degrees of efficiency (Fabricatore et al., 2011). Caloric energy content of food is one of well-established common factors in the relationship between macronutrient intake and adiposity growth (Ambrosini et al., 2012).

Energy expenditure. A higher level of physical activity leads to energy deficit, which—if not compensated by additional calorie intake—results in reduced adiposity (John et al., 2011; Kriemler et al., 2010). Conversely, longer time spent in sedentary activities has been associated with progression of obesity (Fulton et al., 2009; Thorp et al., 2010). These empirical observations have provided additional support for the energy-balance theory of obesity. It should be noted, however, that even vigorous physical activity is only a modest contributor to the overall energy expenditure budget. Resting energy expenditure involves about 2/3 of the total expenditure (with two other notable energy consumers being thermoregulation and food digestion; de Jonge et al., 2012; Hall et al., 2012). Incidentally, it has been speculated that temperature-controlled

environments reduce modern humans' thermoregulation energy expenditure, thus contributing to obesity development (McAllister et al., 2009).

Interventions Informed by the Energy-Balance Theory

The two factors on the earnings side of the energy balance sheet—food intake and nutrient absorption—have been the targets of two types of currently approved drugs: (1) sympathomimetics, which act as appetite suppressors (Aronne, Powell, & Apovian, 2011; Ioannides-Demos, Piccenna, & McNeil, 2011), and (2) pancreatic lipase inhibitors, which reduce absorption of dietary fat (Tucci, Boyland, & Halford, 2010; Unick et al., 2011). However, despite notable advances, pharmacotherapy for obesity has been associated with serious side effects (Ioannides-Demos et al., 2011).

The most remarkable support for the energy-balance conceptualization of obesity has been provided by bariatric surgery, which demonstrated dramatic—and lasting—adiposity reduction in people with obesity and diabetes (Eid et al., 2012; Spivak, Abdelmelek, Beltran, Ng, & Kitahama, 2012). Bariatric surgery reduces energy intake through alteration of the gastro-intestinal tract in order to restrict its throughput capacity, reduce its ability to absorb nutrients, or both (Dorman et al., 2012; Leonetti et al., 2012;). Surgical methods have included gastric banding (G. M. M. Silva et al., 2012), sleeve gastrectomy (Boza et al., 2012), Roux-en-Y gastric bypass (Ullrich, Ernst, Wilms, Thurnheer, & Schultes, 2012), and biliopancreatic diversion (Hedberg & Sundbom, 2012). Less invasive bariatric methods, which have been tested in animal studies, involved coating the proximal intestine with a resin (Abolmaali, Xu, Karp, & Tavakkolizadeh, 2010) or manipulating gut microbiota with antibiotic and probiotic

agents (Murphy et al., 2013); both methods attempt to influence nutrient absorption in order to reduce energy intake.

The idea of reducing energy intake has been found behind a large number of diets focusing on calorie restriction (Chaput & Tremblay, 2012) and reduced consumption of fat and refined carbohydrates, as the most calorie-dense macronutrients (Fabricatore et al., 2011). The energy-balance theory has provided a theoretical foundation for a large number of combined interventions involving diet and exercise aiming at both reduced intake and increased expenditure of energy (Helmink et al., 2012). Likewise, obesity prevention efforts have been based on the concept of energy equilibrium: they have stressed the task of balancing energy intake and expenditure (Johnson, Kremer, Swinburn, & de Silva-Sanigorski, 2012).

The impact of appetite and satiety on energy intake has informed a number of diets involving supplementation with polyunsaturated fatty acids (Mohamed, El-Swefy, Rashed, & Abd El-Latif, 2010), milk (Gilbert et al., 2011), and even tea (Vernarelli & Lambert, 2012). More radically, deep brain stimulation has been proposed as a possible treatment strategy to manipulate hunger, satiety, palatability, and metabolic signals in the brain (Halpern et al., 2008). Current diet research has focused not only on the way different diets affect the energy-intake part of the balance equation, but also on their influence on resting energy expenditure, the biggest contributor to the energy outflow (de Jonge et al., 2012). Energy homeostasis mechanisms in the human body are complex (Hall et al., 2011); they reflect multiple roles played by adipose tissue in the functioning of a human organism (Wells, 2009a).

To summarize, the energy-balance theory conceptualizes obesity as a consequence of imbalance of energy intake and expenditure. This conceptualization has been supported by the experimental evidence and by the modern theoretical understanding of physical, chemical, and physiological processes at work in the human body.

Limitations of the Energy Balance Theory

Despite a high level of plausibility of the energy-balance theory, anti-obesity interventions have rarely been effective (Bäcklund, Sundelin, & Larsson, 2011; Faria, Kelly, & Faria, 2009). Even successful ones often result in later weight regain (Odom et al., 2010)—usually with negative health outcomes (Hinton et al., 2012). It has been argued that mechanistic interventions translating the energy-balance conceptualization into prescriptions of intake reduction or expenditure boosting will not be effective in the long term (Siervo et al., 2009).

A more fundamental accusation leveled against the energy balance theory of obesity is that this theory is a truism that gives a scientifically immaculate account of how obesity develops—but cannot provide the answer why (Wells, 2013). It is fair to say that, given that obesity is defined as a consequence of energy imbalance, an attempt to *explain* obesity by energy imbalance indeed begs the question. Nevertheless, for evolutionary theories of obesity, like the one proposed here, the energy-balance conceptualization provides a logical connection between fat mass and usable energy stored in the body. This connection, in its turn, allows researchers to make meaningful assumptions about survival tradeoffs a biological organism has to make between weight

and mobility, on one hand, and energy reserves, on the other. An example of such assumption is the idea of seasonal variation in adiposity.

The resolution of the above problem of circular reasoning associated with the energy balance theory (Wells, 2013) is thus achieved by expanding the scope of the discussion to include independent predictive factors (Beck, 2004) that modulate the energy balance within the human body. Once a justifiable assumption is made on the way a given factor influences the energy homeostasis of the organism, the energy-balance framework can provide a valid prediction about the development of obesity.

Independent Predictive Factors

Many researchers examining causes of obesity have tried to find evidence for such independent factors that disrupt the energy homeostasis and result in the long-term adiposity gain (Flier & Maratos-Flier, 2008). The factors that have been investigated included environmental influences affecting metabolism and energy homeostasis throughout the entire human lifespan. These included gestational environment (Schnitzler & Fisch, 2012), smoking during pregnancy (Hawkins, Cole, & Law, 2009), maternal diabetes (Mendelson et al., 2011), maternal age at birth (McAllister et al., 2009), duration of breastfeeding (Feig, Lipscombe, Tomlinson, & Blumer, 2011), endocrine-disruptive chemicals (Thayer, Heindel, Bucher, & Gallo, 2012), including pharmaceutical iatrogenesis (Bond, Kauer-Sant'Anna, Lam, & Yatham, 2010), social environment (Kestilä, Rahkonen, Martelin, Lahti-Koski, & Koskinen, 2009), stress (Elder et al., 2012), macronutrient intake (Goyenechea et al., 2011), ambient light exposure (Danilenko,

Mustafina, & Pechenkina, 2013), and even viral infection (Na et al., 2010), among many others.

Sleep has long been suspected to be a behavioral factor associated with the development of obesity (Golley et al., 2013). Different sleep-related variables that have been under investigation included not only habitual sleep duration, the main focus of the present study, but also variability of sleep duration (Kobayashi, Takahashi, Shimbo, et al., 2012), sleep quality (Thomson et al., 2012), sleep architecture (Sahlin, Franklin, Stenlund, & Lindberg, 2009), perceived sleep adequacy (Wilsmore et al., 2012), excessive daytime sleepiness (Vgontzas et al., 2008), napping during the day (Picarsic et al., 2008), sleep debt (cumulative effect of sleep restriction over several days; Liou, Liou, & Chang, 2010), shift work and a consequent sleep–wake cycle substitution (Kawada & Otsuka, 2014), as well as chronotype (i.e. morningness and eveningness; Baron, Reid, Kern, & Zee, 2011; Randler, Haun, & Schaal, 2013).

In summary, the main limitation of the energy-balance theory has been found in the apparent circularity in the explanation of the causes of obesity. A widely-used approach to deal with this limitation has been the development of independent theories about the factors that alter the energy homeostasis and, thus, promote obesity. The following section will discuss theories—and the supporting empirical evidence—related to one of such factors, habitual sleep duration.

Sleep Duration and Obesity

Both short and long sleep have been found to be associated with obesity (Buxton & Marcelli, 2010). An unusual bias has been noted in the cultural norms related to sleep

duration. In Western culture, people have generally been more concerned about deleterious effects of short sleep, whereas long sleep has mostly been seen as harmless indolence. As a consequence, the Western scientific tradition has almost exclusively advocated sleep *extension* as a main priority in sleep hygiene (Cassoff, Knäuper, Michaelsen, & Gruber, 2012). A historical analysis of childhood-sleep recommendations from 1897 to 2009 showed that "no matter how much sleep children are getting [as a population average], it has always been assumed that they need more" (Matricciani, Olds, Blunden, Rigney, & Williams, 2012, p. 548). Most studies focusing on deleterious effects of short sleep, with few exceptions, were conducted in Europe and North America, whereas studies concerning morbidities associated with long sleep more often than not came from Japan.

The latter is perhaps not surprising because, in the Japanese culture, long sleep has been associated with a certain degree of social stigma (Steger, 2003). Confucian tradition stipulates that "reducing sleep by rising early and going to bed late is […] a basic requirement for proficient rulers, filial sons, and devoted wives" (Steger, 2003, p. 186). It is difficult to say to what extent such cultural bias (rather than a genuine population difference) motivated Asian researchers to look for—and find—deleterious effects of long sleep that were stronger and more prevalent in Asian populations than elsewhere in the world (Cappuccio, D'Elia, Strazzullo, & Miller, 2010). In the view of this difference, it is worthwhile to put the present discussion of the relationship between long sleep and obesity in the context of findings (both Japanese and otherwise) on morbidity and mortality associated with long sleep.

Long Sleep, Morbidity, and Mortality

It has long been accepted that habitual sleep *restriction* is associated with a large number of negative health outcomes, including diabetes, hypertension, cardiovascular disease, and well as higher injury risk and mortality (Knutson, Van Cauter, Rathouz, DeLeire, & Lauderdale, 2010). The health risks associated with short sleep have been high on the agenda of public healthcare and public media alike (Matricciani et al., 2012).

The Western public has mostly been unaware (Matricciani et al., 2012) that long sleep has been likewise associated with increased prevalence of hypertension (Fang et al., 2012; Magee et al., 2012), cardiovascular disease (Buxton & Marcelli, 2010; Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011), Type 2 diabetes (Chao et al., 2011; Kachi, Ohwaki, & Yano, 2012), metabolic syndrome (Arora et al., 2011), hyperlipidemia (Kaneita, Uchiyama, Yoshiike, & Ohida, 2008), hyperleptinemia (Charles et al., 2011), insulin resistance (Javaheri, Storfer-Isser, Rosen, & Redline, 2011), and higher levels of inflammation markers (Dowd, Goldman, & Weinstein, 2011). In adults over 40, longer sleep has also been implicated in higher odds for risk of cancer (Gangwisch et al., 2008; Tu et al., 2012), pneumonia (Patel et al., 2012), rheumatism, arthritis, arthrosis (Lima, Bergamo Francisco, & de Azevedo Barros, 2012), osteoporosis (Kobayashi, Takahashi, Deshpande, Shimbo, & Fukui, 2012), increased risk of recurrent falls (Mesas, López-García, & Rodríguez-Artalejo, 2011), and bone fracture (Tu et al., 2012). In older adults, long sleep has often been linked with physical function decline (Stenholm, Kronholm, Bandinelli, Guralnik, & Ferrucci, 2011), lower psychomotor speed (Kronholm, Sallinen, et al., 2011), poorer cognitive performance (Kronholm et al., 2009), memory impairment

(L. Xu et al., 2011), and dementia (Auyeung et al., 2013; Benito-León, Bermejo-Pareja, Vega, & Louis, 2009). Long sleep has been found associated with increased daytime sleepiness (Mesas, López-García, León-Muñoz, et al., 2011), poorer self-reported health (Shankar, Charumathi, & Kalidindi, 2011), lower subjective quality of life (Magee et al., 2011a), depression (Krueger & Friedman, 2009; Ryu, Kim, & Han, 2011), and anxiety (S. Park et al., 2010). Many researchers found a correlation between long sleep and all-cause mortality (Castro-Costa et al., 2011; Gallicchio & Kalesan, 2009; Y. Kim et al., 2013). The relationship held even when it was controlled for overall poor health, impaired cognitive function, and depression (Kakizaki et al., 2013). However, as was mentioned above, a meta-analysis conducted by Cappuccio et al. (2010) found that the effect of long sleep on mortality risk was stronger in East Asia compared to Europe and the US.

To summarize, both short and long sleep have been associated with increased morbidity and mortality, although a population bias has been noted with regards to the long sleep. The following discussion will concentrate on the relationship between sleep duration and obesity.

Problems of Comparison of Sleep–Adiposity Studies

A review of research relating sleep duration and adiposity has been complicated by the lack of consistency in the definition of *short* and *long* durations. The definition of *short* sleep has been set fairly arbitrarily, ranging from 8 hours (Tian et al., 2010) to 11 hours (Padez, Mourao, Moreira, & Rosado, 2009) in children; from 5 hours (Seicean et al., 2007) to 9 hours (Hitze et al., 2009) in adolescents; and from 5 hours (Theorell-

Haglöw, Berglund, Janson, & Lindberg, 2012) to 7 hours (Thomson et al., 2012) in adults. *Long* sleep has also been defined equally arbitrarily at 12 hours for children (Chaput, Lambert, et al., 2011), from 8 hours (Seicean et al., 2007) to 11 hours (Calamaro et al., 2010) in adolescents, and from 8 hours (Hairston et al., 2010) to 10 hours (Theorell-Haglöw et al., 2012) in adults.

A further complication has stemmed from the difference in methods of assessment of sleep duration. Many studies used self- and parent-report, as well as time diaries for subjective assessment of sleep duration (Yang, Matthews, & Chen, 2013). It has been shown that measurements obtained by these subjective methods were not equivalent to those obtained from actigraphy (objective measurement of body movement; Lauderdale, Knutson, Yan, Liu, & Rathouz, 2008) or polysomnography (measurement of electromagnetic brain activity; G. E. Silva et al., 2007).

Assessment of adiposity in the studies reviewed below likewise did not yield to straightforward comparison. Researchers operationalized adiposity as fat mass (Ackland et al., 2012), fat volume (Hairston et al., 2010), body fat percentage (Chaput, Lambert, et al., 2011), fat-mass index (FMI; Carter, Taylor, Williams, & Taylor, 2011), body-mass index (BMI; Meyer et al., 2012), body circumferences (Garaulet et al., 2011), as well as their ratios (Al-Hazzaa, Musaiger, Abahussain, Al-Sobayel, & Qahwaji, 2012). In addition, many studies replaced continuous adiposity variables with categorical states of *normal*, *overweight*, and *obesity* based on the respective clinical definitions of these states (which have not always been consistent either; Kobayashi, Takahashi, Deshpande, Shimbo, & Fukui, 2011; World Health Organization Consultation on Obesity, 2000).

These studies then used logistic regression and odds ratios, instead of simple multivariate regression, for the purposes of statistical hypothesis testing. The use of this methodology has been argued to be an additional source of statistical error (Berry, 1993).

The following reviews of short and long sleep are organized by method of sleep measurement (self-report, then actigraphy, and then polysomnography). Studies that used continuous BMI precede those that categorized participants by obesity status, followed by those that used other markers of adiposity.

Short Sleep Duration and Obesity

Self- and parent-reported short sleep. When sleep duration was assessed via parent- or self-report, shorter sleep was associated with higher age-adjusted BMI scores in cross-sectional studies of children (Börnhorst et al., 2012), adolescents (Garaulet et al., 2011) and adults (Ford et al., 2013; Magee, Caputi, & Iverson, 2011b; Theorell-Haglöw et al., 2012), although findings of other studies have been ambiguous (Guo et al., 2013; Lytle, Pasch, & Farbakhsh, 2011; Meyer et al., 2012; Storfer-Isser, Patel, Babineau, & Redline, 2012). A higher prevalence of BMI overweight and obesity was also found to correlate with shorter self-reported sleep in children (Carter et al., 2011), adolescents (Olds, Blunden, Dollman, & Maher, 2010), and adults (Buxton & Marcelli, 2010), although, again, other findings were mixed (Calamaro et al., 2010; Magee, Caputi, & Iverson, 2010; Shi et al., 2010). Cross-sectional studies found shorter self- and parent-reported sleep to be associated with higher waist circumference in children (Chaput, Lambert, et al., 2011), adolescents (Al-Hazzaa et al., 2012), and adults (Chaput, Després, Bouchard, & Tremblay, 2011b). In some studies, however, this relationship was

significant only in male participants (Garaulet et al., 2011; Ozturk et al., 2009; Yi et al., 2013).

Some researchers found a relationship between short sleep and neck (Theorell-Haglöw et al., 2012) and hip circumference (Garaulet et al., 2011), as well as waist-to-height ratio (Al-Hazzaa et al., 2012). Short sleep has also been implicated in higher body fat percentage in children (Börnhorst et al., 2012), adolescents (mostly girls; Garaulet et al., 2011), and adults (Chaput, Després, Bouchard, Astrup, & Tremblay, 2009). The highest sleep-related variation in adiposity was discovered in subcutaneous fat depots (Bayer, Rosario, Wabitsch, & von Kries, 2009; Chaput, Després, Bouchard, & Tremblay, 2007). Results from some cross-sectional studies indicated a correlation between short parent-reported sleep and higher fat mass index in children (Carter et al., 2011; Diethelm, Bolzenius, Cheng, Remer, & Buyken, 2011).

In longitudinal studies, researchers found shorter sleep to be associated with faster BMI gain in infants (Taveras, Rifas-Shiman, Oken, Gunderson, & Gillman, 2008), children (Snell, Adam, & Duncan, 2007), and adolescents (Magee & Lee, 2013; Mitchell, Rodriguez, Schmitz, & Audrain-McGovern, 2013; Storfer-Isser et al., 2012). Landhuis, Poulton, Welch, and Hancox (2008) reported that short sleep in childhood predicted higher BMI at the age of 32 years. Short-sleep-related gain in weight, waist-circumference, body-fat percentage, subcutaneous fat and visceral fat was also found in adults under 40 (Chaput, Després, et al., 2011a; Kobayashi, Takahashi, Shimbo, et al., 2012; Yiengprugsawan et al., 2012); findings in adults over 40 have been mixed (Hairston et al., 2010; Lyytikäinen, Rahkonen, Lahelma, & Lallukka, 2011; Stranges,

Cappuccio, et al., 2008). Postpartum women sleeping shorter hours were found to retain more weight and body fat after giving birth (Taveras et al., 2011). Shorter self-reported sleep has been associated with lower weight loss in various anti-obesity interventions (Clifford et al., 2012; Logue et al., 2012). Longitudinal studies have found higher odds of onset of BMI obesity in shorter-sleeping, as compared to longer-sleeping, infants (Bell & Zimmerman, 2010) and children (Magee, Caputi, & Iverson, 2013; Seegers et al., 2011)—but not adolescents (Calamaro et al., 2010) or adults (Chaput et al., 2008; Itani, Kaneita, Murata, Yokoyama, & Ohida, 2011), although Sayón-Orea et al. (2013) found higher odds of obesity onset in shorter-sleeping adult men—but not women.

Actigraphically measured short sleep. When sleep was measured with actigraphy, shorter sleep was associated with higher BMI in cross-sectional studies of older adults (Patel et al., 2008). In middle-aged and younger adults, the results were mixed (Baron et al., 2011; Lauderdale et al., 2009). Shorter actigraphically measured sleep was associated with higher odds of being obese in children (Spruyt et al., 2011) and older adults (Patel et al., 2008), but not adolescents (A. P. Kong et al., 2011). Nixon et al. (2008) reported higher body fat percentage in children with shorter actigraphic sleep duration, although Gallagher, Cron, and Meininger (2010) and Sung et al. (2011) did not confirm this finding. Prospective studies did not reveal any relationship between shorter sleep and BMI gain in adults (Appelhans et al., 2013).

Polysomnography. Cross-sectional studies that used polysomnography to measure total sleep duration produced mixed results. Higher BMI was found to be related to shorter sleep in adult women, but this trend disappeared when the analyses

were adjusted for age (Sahlin et al., 2009). Some researchers found that shorter sleep was associated with higher age-adjusted BMI and higher odds of obesity in children and adolescents (X. Liu et al., 2008). Others found the odds of obesity to be *lower* in short sleepers compared to people with the average sleep duration (Wojnar et al., 2010). Still others found no association (Drescher, Goodwin, Silva, & Quan, 2011). A longitudinal study (G. E. Silva et al., 2011) found that shorter polysomnographic sleep in children was associated with higher odds of obesity at 5-year follow-up.

In summary, researchers conducting epidemiological studies (cross sectional and longitudinal) found shorter sleep to be generally associated with higher adiposity markers across all age groups, although some studies did not confirm this association. A number of experimental sleep-extension interventions designed for short-sleepers were ongoing at the time of this review (Cizza et al., 2010; Logue et al., 2012; Taylor et al., 2011), but the results of these studies have not yet been published.

Long Sleep Duration and Obesity

Self-report. Compared to a large number of studies on short sleep, there has been a relative paucity of research in long sleep. In cross-sectional studies, longer self- and parent-reported sleep has been associated with higher age-adjusted BMI in children (Chaput, Lambert, et al., 2011; Danielsen, Pallesen, Stormark, Nordhus, & Bjorvatn, 2010) and adolescent boys, but not girls (Guo et al., 2013; Yu et al., 2007). In adults, the results have been mixed; some study findings suggested that higher BMI is associated with longer sleep (Bjorvatn et al., 2007; Kronholm, Laatikainen, Peltonen, Sippola, & Partonen, 2011; Tu et al., 2012); others found no significant difference in BMI between

average and long sleepers (Ford et al., 2013; Kronholm, Laatikainen, et al., 2011; Theorell-Haglöw et al., 2012; Watson, Buchwald, Vitiello, Noonan, & Goldberg, 2010).

Researchers who have categorized participants as obese, overweight, or normal weight (rather than using BMI as a continuous variable) have also found mixed results. Calamaro et al. (2010) found higher odds of obesity in adolescents reporting longer sleep hours; Yu et al. (2007) found elevated odds only in boys, and Knutson and Lauderdale (2007) found reduced odds in both sexes. Compared to median sleepers, adults sleeping longer were found to have higher odds of being overweight or obese (Buxton & Marcelli, 2010; Magee, Caputi, et al., 2010). Chaput, Després, et al. (2007) found elevated odds only in women. Magee, Iverson, and Caputi (2010), Meyer et al. (2012), and Tuomilehto et al. (2008) found this association only in men, and Anic et al. (2010), Krueger and Friedman (2009), and Stranges, Dorn, et al. (2008) did not find this relationship in either men or women. At the same time, some epidemiological studies found an association between longer sleep and higher odds of clinical *underweight*, which was attributed to a morbidity causing both weight loss and sleep extension (Krueger & Friedman, 2009; S. Park et al., 2010; Shankar, Koh, Yuan, Lee, & Yu, 2008).

Researchers have found an association between longer self- and parent-reported sleep and higher waist circumference in children (Chaput, Lambert, et al., 2011) and adolescent boys, but not girls (Yu et al., 2007). In adults, the association of longer sleep and higher waist circumference was seen mostly in women (Theorell-Haglöw et al., 2012; Tu et al., 2012), although Tuomilehto et al. (2008) found higher waist circumference in men, rather than women, and López-García et al. (2008) found it in both

men and women over 60. Other researchers found no association between sleep and waist circumference (Chaput et al., 2009; Fogelholm et al., 2007; Ford et al., 2013; Stranges, Dorn, et al., 2008). Cross-sectional associations between longer self- and parent-reported sleep and higher body fat percentage has been noted in some studies of children (Chaput, Lambert, et al., 2011), but not adolescents (Yu et al., 2007) or adults (Chaput et al., 2009).

Researchers have not found associations between longer parent- or self-reported sleep and subsequent BMI gain in longitudinal studies of children (Landhuis et al., 2008; Lumeng et al., 2007) and adolescents (Calamaro et al., 2010; Lytle et al., 2013). Lytle et al. (2013) found no longitudinal association between sleep duration and body fat percentage in adolescents. However, Hairston et al. (2010) and Yiengprugsawan et al. (2012) found BMI and fat volume gain in younger adults, although not in adults older than 40 (Hairston et al., 2010). Other researchers found BMI gain only in adult women (López-García et al., 2008; Lyytikäinen et al., 2011), particularly postpartum women (Gunderson et al., 2008), although Appelhans et al. (2013) found no sleep-related weight gain in women. On the other hand, Watanabe, Kikuchi, Tanaka, and Takahashi (2010) found a relationship between long sleep and weight gain only in men. In contrast, Hasler et al. (2004) found longitudinal BMI *loss* to be associated with longer self-reported sleep in adults under 40 years of age.

Actigraphy and polysomnography. Researchers measuring sleep duration with actigraphy have found no cross-sectional relationship between long sleep duration and BMI (Gallagher et al., 2010; van den Berg et al., 2008). Polysomnographic studies found

elevated BMI to be associated with longer sleep in both children and adolescents (Wojnar et al., 2010), but not in adults (Sahlin et al., 2009).

To summarize, longer sleep has been associated with higher markers of adiposity, although the findings have been even less consistent than those for shorter sleep. Overall, recent research has suggested the existence of a U-shaped relationship between sleep duration and obesity, with both shorter and longer sleep being associated with higher adiposity, while an average sleep duration—usually between 7 and 8 hours in the case of adults (Buxton & Marcelli, 2010)—associated with the population-level adiposity minimum. Such a curvilinear relationship has implied an interaction of several physiological processes, some of which were activated with short sleep, and some, with long sleep. The following section includes a review of possible energy-balance mechanisms that may contribute to this relationship at different durations of habitual sleep.

Energy Balance and Obesity Across the Sleep-Duration Spectrum

At short sleep durations, the relationship between sleep duration and adiposity must be dominated by the mechanisms that result in a negative correlation. Conversely, on the long-sleep-duration part of the U-curve, the sleep–adiposity relationship is dominated by the mechanisms that promote energy accumulation with sleep extension, thus producing a positive correlation between sleep and adiposity. The discussion of explanatory theories will start with the mechanisms that imply a negative correlation, followed by those that imply a positive correlation. This section will be concluded by a

review of alternative interpretations of the observed relationship between sleep and adiposity.

More Sleep, Less Fat

On the negative-slope part of the sleep–adiposity curve, four mechanisms have been theorized to cause adiposity gain in response to sleep restriction. These included increased fatigue, altered resting energy expenditure, increased hunger, and more time and opportunity to eat (McNeil, Doucet, & Chaput, 2013; Patel & Hu, 2008).

Fatigue. People who feel more fatigued during the day as a result of inadequate sleep at night may try to avoid high levels of physical activity, resulting in lower overall energy expenditure (Wells & Siervo, 2011). Indeed, some studies showed that short sleepers were less active and spent more time in sedentary activities like watching TV (Garaulet et al., 2011). However, other studies reported that short sleepers might in fact be more active during the day than long sleepers (Wells et al., 2008).

Total energy expenditure might be affected by involuntary movements (e.g., fidgeting) and posture (standing vs. sitting), all of which may be inhibited due to fatigue (Patel, Malhotra, White, Gottlieb, & Hu, 2006). Fatigue might also influence individuals' adherence to the physical activity regimen resulting in longitudinal weight gain (Chaput et al., 2012).

Resting energy expenditure. People getting inadequate amount of sleep at night—and feeling more fatigued during the day—may experience a subjective energy insufficiency (Garaulet et al., 2011). A possible physiological approach that the organism might be using to deal with a perceived energy insufficiency, is reducing

resting energy expenditure and thermoregulation energy expenditure, all of which contribute to energy imbalance (Patel et al., 2006). At present, however, there has been little evidence to support this conjecture (McNeil et al., 2013).

Hunger. Another related theory predicted that short sleep somehow altered the people's preference for calorie-dense foods and snacks (Chapman et al., 2013; Knutson, 2012). Indeed, experimental sleep restriction was found to increase appetite while not affecting total energy expenditure (St-Onge et al., 2011). This may be explained by the compensation of subjective energy insufficiency with consumption of more calories (Garaulet et al., 2011), more frequent or irregular meals (Haghighatdoost, Karimi, Esmaillzadeh, & Azadbakht, 2012), night-time snacking (S. Kim, DeRoo, & Sandler, 2011), and fast-food consumption (Baron et al., 2011); each of these results in positive energy balance.

On the other hand, increased consumption of calories in response to shorter sleep may be due to a dietary shift towards more calorie-dense carbohydrates (Kjeldsen et al., 2013; Nedeltcheva, Kilkus, Imperial, Schoeller, & Penev, 2010) and fats (St-Onge et al., 2011). Gangwisch (2009) theorized that sleep restriction might affect glucose and fat metabolism because of sleep-entrained ancestral adaptations to seasonality. Gangwisch (2009) proposed that shortening night duration—and, thus, shortening sleep—coincided with appearance of sweet fruit in the hunter-gatherer diet, which could have been used as a cue for early humans to adjust their metabolism for effective assimilation of energy from plant sources. This theory might explain the apparent interaction of short sleep and glucose metabolism in predicting diabetes and obesity (Knutson, 2012).

However, it has been speculated that during the last glacial maximum, the highest latitude of human colonization was not much higher than the present-day Lyon, France (J. R. Stewart & Stringer, 2012). At this latitude, the period of complete darkness is never shorter than 7 hours (Morrissey, 2012). This could not qualify for obesity-promoting short sleep duration as per current understanding (Buxton & Marcelli, 2010), which seemed to undermine the plausibility of Gangwisch's (2009) theory.

One thing that might compel early humans to lose sleep in complete darkness could be an environmental threat that was stressful enough to prevent an individual from naturally falling asleep, for example, nocturnal predators. Psychological stress has been believed to cause adiposity gain, because a stressed individual was likely to have less time to forage—and so must have absorbed and stored whatever nutritional energy was available (Elder et al., 2012). Sleep restriction itself may have been a behavioral marker of environmental stress (McNeil et al., 2013). Because environmental stress might result in energy insufficiency, a thrifty gene must have been designed in such a way that it was expressed in the presence of relevant stress markers, like sleep restriction. Such expression of the thrifty gene must have resulted in more active energy acquisition (Bouchard, 2007). This logic could provide an evolutionary explanation why sleep restriction beyond a certain minimum might cause adiposity gain.

More opportunity to eat. Finally, people sleeping shorter hours may consume more calories per day simply because they have more waking hours to do so (Yiengprugsawan et al., 2012). Chaput, Després, et al. (2011a) noted an interaction between sleep duration and disinhibition in eating, suggesting that people who exercise

less control over their eating habits will eat more in proportion to the number of hours they stay awake (Killgore et al., 2013). Indeed, O'Keeffe, Roberts, Kelleman, Roychoudhury, and St-Onge (2013) showed that when energy intake was controlled for, short-term sleep restriction had no direct effect on lipid levels in non-obese adults. Food intake has been found to be mediated by social and leisure activities; these activities, in turn, are a function of the time available to engage in them (Ulijaszek & Lofink, 2006). A growing consensus among researchers has pointed towards non-homeostatic motivation to eat—that is, food consumption due to social, hedonic, or cognitive reasons, rather than motivated by hunger—as a culprit behind short-sleep related weight gain (McNeil et al., 2013). Thus, there have been a number of pathways that might explain a negative correlation between sleep duration and adiposity gain on the shorter-sleep-duration side of the sleep–adiposity curve.

More Sleep, More Fat

The association between long sleep and obesity has not been equally well researched (Knutson & Leproult, 2010). Although some studies found a positive correlation between sleep duration and adiposity, very few theories have been proposed to explain it (Yiengprugsawan et al., 2012).

It has been proposed that, from the point of view of the 24-hour energy budget, the expenditure of energy due to movement during wakefulness is higher than that during sleep (Nielsen et al., 2011). This must result in a positive correlation between sleep duration and change in energy reserves (Wells et al., 2008). The same logic applies to

the energy consumption of the brain, which is higher during wakefulness (Schmid, Hallschmid, Jauch-Chara, Born, & Schultes, 2008).

By following this logic further, it has been argued that the function of sleep is energy conservation. A certain degree of similarity has been noted between sleep and annual hibernation of seasonal animals (Cizza et al., 2011). Indeed, a correlation between resting metabolism and sleep duration has been observed in a wide range of mammal species (Hitze et al., 2009; Wells & Siervo, 2011). Thus, the more energy an organism expends at rest, the higher must be the pressure for energy conservation and so, perhaps, to extend sleep. It may be assumed that once sleep is extended beyond a certain threshold, it may cause insufficient energy expenditure and weight gain (Wells & Siervo, 2011). However, the conceptualization of sleep and an energy-saving mechanism has not been uncontroversial (Siegel, 2009). A better understanding of the adaptive value of sleep (which has still been a mystery in many respects; Siegel, 2009) might help explain sleep's contribution to the energy balance equation.

In summary, prior studies proposed a variety of possible explanations for the observed U-shaped relationship between sleep duration and adiposity. Variation in sleep duration has seemed to result in a complex interaction of many factors in the energy expenditure–accumulation equation, an interaction that has still not been fully understood. There has been a notable gap in theoretical explanation of the observed positive relationship between sleep and adiposity at longer sleep durations.

An Evolutionary Theory of Sleep-Induced Adiposity Gain in Descendants of European Hunter-Gatherers

In order to address this explanatory gap, the present work proposed an evolutionary theory of adiposity gain induced by long sleep. This section will discuss the main tenets of this theory.

After migrating from Africa to Europe about 50,000 years ago (Carbonell et al., 2010), early humans faced a novel set of survival challenges resulting from warm/cold European seasonality (J. R. Stewart & Stringer, 2012). Researchers have found a sharp reduction in diversity of mitochondrial DNA in populations living in colder climates (Balloux, Handley, Jombart, Liu, & Manica, 2009). This led many researchers to conclude that adaptation to cold seasonality resulted from strong natural selection (Nishimura et al., 2012).

During the Upper-Paleolithic period (approximately 50,000 – 10,000 years ago), anatomically modern humans were hunter-gatherers (Dunbar et al., 2009). They did not keep livestock or long-term stores of grain, which could help them survive periods of food scarcity (Pond, 1998): the first signs of farming and animal domestication in Europe have been dated at approximately 10,000 years ago (Gignoux et al., 2011).

Instead, upper-Paleolithic Europeans had to rely on internal adiposity stores to buffer energetic stresses of a cold winter (Wells, 2009a). Given the circumstances, the most efficient adiposity gain could be achieved in late summer and early fall, times during which both animal and plant food were still abundant (Gangwisch, 2013). An

evolved method of adiposity gain in anticipation of the approaching winter or, in other words, a season-sensing thrifty gene, would provide a clear survival advantage.

A sensory activation mechanism for the expression of this thrifty gene could be provided by the variation in the photoperiod, which is the average duration of a light day. In Europe, late summer and early fall are associated with a rapid contraction of the photoperiod. Take, for example, the present-day Lyon, France, which is believed to have been continuously inhabited by humans throughout the Upper-Paleolithic period, when advancing glaciers covered most of Northern Europe (J. R. Stewart & Stringer, 2012). In Lyon, the night lengthens from about 7 hours in late June to 9.5 hours in late August (Broomberg, 2008; Morrissey, 2012). Such change in the photoperiod has been an early environmental sign of the approaching cold season.

A change in photoperiod has been known to affect human behavior, in particular, the duration of habitual sleep (Peixoto et al., 2009). It has been shown that pineal melatonin production in response to gradual onset of darkness (Dumont, Lanctôt, Cadieux-Viau, & Paquet, 2012) is the main factor in the association between the duration of environmental darkness and the duration of sleep (Diethelm et al., 2010). It has thus been possible to conjecture that the European Upper-Paleolithic humans extended their habitual sleep with seasonal contraction of photoperiod.

A possible co-occurrence of photoperiod-entrained sleep extension and serendipitous adiposity gain in late summer, which resulted in improved survival during winter, could eventually produce a thrifty gene, the expression of which—primed by sleep extension—stimulated adiposity gain. The main theoretical contribution of this

work has been the conjecture that sleep extension, rather than mere change in environmental light exposure (Danilenko et al., 2013), resulted in adaptive seasonal adiposity gain in European Upper-Paleolithic hunter-gatherers.

An important moderating factor for the phenotypic expression of this sleep-sensing thrifty gene must have been biological sex. Adiposity has been known to play an important role in reproductive function and sexual selection (Wells, 2006); thus, adiposity-related survival strategies cannot be taken out of context of reproductive strategies. Therefore, a postulation of a circannual variation in adiposity must be logically linked with a concomitant variation in reproductive activity.

There are three adiposity-related factors that have a direct relationship with reproductive success. First is the energy required for the production of a gamete; second factor is access to sexual partners, which is closely related to individual's physical mobility; and third is the energy required for gestation and rearing of offspring (Bribiescas, 2011). A relative degree of similarity can be observed between sexes in the evolutionary tradeoffs related to the production of gametes: here, goals of reproduction compete for the same set of energy resources that are needed for individual survival (Wells, 2009a).

There is, however, a clear difference between the sexes in the adiposity strategies related to the other two factors. For males, the overall reproductive success is directly dependent on access to a maximum number of mates; for females, a larger number of mates does not translate into more surviving offspring (Bribiescas, 2011).

Two distinct adiposity modes must be present in males: a reproductive mode and a survival mode. In reproductive mode, a high muscle-to-fat ratio is needed to migrate for a large distance in search of receptive mates, and to defeat and deter competing males once the new mate is found (Heyer et al., 2012). Any remaining energy must be invested in sperm production rather than adiposity accumulation (Wells, 2009a). At the completion of this reproductive cycle, males must switch to the survival mode, which, towards the end of the summer, must result in suppression of reproductive activity (Eriksson et al., 2008) and adiposity gain in anticipation of winter. This strategy is in line with the seasonal adiposity gain induced by sleep extension, as postulated in this work.

For females, on the other hand, the reproductive mode should not be associated with reduced adiposity—quite the opposite: female reproductive cycle involves a substantial energy expenditure over several years (Jasienska, 2011), and so preparation for conception must be associated with adiposity gain and maintenance—rather than loss (Pike, 2011). In fact, the reproductive cycle is so energetically costly for women that the only way to keep it manageable is to try to distribute the cost of reproduction over a longer period of time. This is the reason why, in comparison to other primates, for example, human milk is less energetically dense, human babies have to rely on mother's milk much longer and, as a consequence, become mature and nutritionally self-sufficient much later (Valeggia & Ellison, 2011). It is possible that menstrual, rather than circannual cycle of female sexual receptivity evolved in response to a multi-year cycle of reproduction (Jasienska, 2011). In the context of this discussion, it has been possible to argue that the lack of seasonal variation in female sexual receptivity might explain the

observed lack of seasonal variation is female adiposity (Davis et al., 2010). This, in turn, must lead to a conclusion that the adaptive adiposity gain induced by long sleep, as theorized here, must not be observed in women.

In summary, the present work postulated that a trait of seasonal adiposity gain primed by sleep extension evolved in the European Upper-Paleolithic human hunter-gatherers. By logical conclusion, this trait was phenotypically expressed only in males. It was further theorized that the present-day ubiquitous use of electric lighting must have suppressed light-entrained seasonal variation in sleep duration (Johnsen, Wynn, Allebrandt, & Bratlid, 2013). As a result, habitually long sleep duration must have resulted in the chronic expression of the theorized trait of sleep-induced adiposity gain in the present-day descendants of European hunter-gatherers. Thus, habitual sleep extension in White males was expected to lead to gradual adiposity gain.

One of the key factors in the logical derivation of the proposed theory was the observed seasonal variation of photoperiod in Europe. The lack of circannual photoperiod variation in Sub-Saharan Africa must imply that the theorized trait of sleep-induced adiposity gain was absent in Blacks, who are believed to be descendants of African Upper-Paleolithic hunter-gatherers (McEvoy et al., 2011). This theorized population difference has been used in derivation of the research hypothesis that was tested as part of this study.

Tests of Interaction Between Race and Sleep Duration

The present work attempted to empirically verify the proposed theory of sleep-induced adiposity gain, by testing a statistical interaction between race and self-reported

sleep duration in the model predicting the age-adjusted fat-mass index (FMI). This section will review prior studies that collected relevant data and attempted to test this interaction before.

Systematic differences between Blacks and Whites in both sleep habits and obesity prevalence have been well known (Lauderdale et al., 2009). However, very few studies had adequately-sized samples of Black participants to test the interaction of race and sleep in adiposity-prediction models (Appelhans et al., 2013). In most studies that collected data on race/ethnicity, there was on average one Black participant for ten Whites, thus making it difficult to draw reasonable comparisons (Thomson et al., 2012). Other studies have not reported attempts to analyze the interaction (Storfer-Isser et al., 2012), even if they had enough power (Wilsmore et al., 2012). The race was simply used as a covariate to adjust the analyses (Appelhans et al., 2013).

Nevertheless, Meyer et al. (2012) found a significant interaction between race and sleep duration in a multivariable-adjusted model for mean BMI. Other studies, however, found no interaction between race and sleep duration in models predicting BMI (G. E. Silva et al., 2011), waist circumference (St-Onge et al., 2010), and longitudinal weight gain (Lauderdale et al., 2009). A comparison between Blacks and Hispanics in prediction of fat volume accumulation as a function of sleep duration produced no significant interaction (Hairston et al., 2010). No studies were found to test the focal interaction in a model predicting FMI.

To summarize, there have been very few adequately-powered studies testing the interaction between race and sleep duration in models predicting adiposity indices. The findings of these studies were mixed.

Lactase Persistence: Another Adaptation to Energy Insufficiency

The theory proposed here is novel, and so very few studies have been found to analyze the relationship between all relevant variables. However, a large degree of similarity can be noted between the present theory and the evolutionary theory of lactase persistence (Ingram, Mulcare, Itan, Thomas, & Swallow, 2009) in the sense that both theories predict a population difference in metabolic mechanisms, based on the evolutionary histories of these two populations.

Lactase is the enzyme needed for digestion of milk sugar lactose. In most mammals, the production of lactase is reduced after weaning (Gerbault et al., 2011). In humans, some ethnic groups have been shown to have a high prevalence of adult lactase persistence, or the ability to produce lactase—and thus metabolize fresh milk—well into adulthood (Mattar et al., 2009). This phenomenon has been explained by an evolutionary rationale: an ability to digest milk from domesticated animals provided additional survival advantage under conditions of nutritional stress (Gerbault et al., 2011), a rationale similar to the one used in the present work to justify the hypothesized trait of sleep-induced adiposity gain.

The second similarity between lactase persistence and the theorized trait of sleep-induced obesity is the different prevalence in Blacks and Whites. For example, Mattar et al. (2009) found that about 43% of Brazilian Whites and only 20% of Brazilian Blacks

had lactose persistence (see also Itan et al., 2010). It has to be noted, however, that the evolution of lactase persistence has been associated with cattle herding as a response to nutritional stress, irrespective of a specific type of nutritional seasonality, which explains why, even among Blacks, different ethnic groups have had different levels of prevalence: this variation reflected differential proliferation of cattle herding in pre-historic Africa (Ingram, Raga, et al., 2009; Itan et al., 2010).

Thus, it has been stated that the evolutionary theory of lactase persistence could be used as an exemplar in the analysis of the logical consistency of the theory of sleep-induced adiposity gain proposed here.

Summary and Conclusions

Obesity has been associated with many negative health outcomes, increased healthcare costs, impaired psychological functioning, lower subjective quality of life, and higher mortality. On the basis of the energy-balance theory, researchers have defined obesity as a consequence of a disruption of energy homeostasis in the body.

The energy-balance theory has been the main theoretical framework behind a large number of interventions aimed at alteration of energy intake, energy expenditure, or both. However, only bariatric surgery has had lasting weight-reduction effects; less radical interventions have not been as effective and often resulted in weight regain.

The modern obesity research has concentrated on identifying factors that moderated processes of energy homeostasis. A large number of factors have been considered, including various dimensions of sleep behavior. Among these dimensions, habitual sleep duration has been studied extensively in the last several years, involving

different methods of sleep-duration assessment (self-report, actigraphy, and polysomnography), adiposity assessment (both body-mass- and fat-mass-based), as well as different research designs. Both short and long sleep have been found to be associated with increased adiposity, despite a certain degree of inconsistency in selection of cut-offs for the definition of short and long durations. Although several theories have been advanced to explain the short-sleep relationship, there has been a relative paucity of explanatory theories dealing with the association of long sleep and adiposity gain. The present work has attempted to fill this gap.

The present study is situated in the evolutionary tradition of obesity research. This tradition has been grounded in the assumption that the human energy homeostasis is an evolved property. A better understanding of the adaptive value of adiposity throughout the entire history of human evolution might shed more light on the functioning of various evolved mechanisms that moderate energy homeostasis. Understanding these mechanisms, in their turn, might help explain how they may become maladaptive in the present-day environment. In this context, the present work postulated a theory of sleep-induced adiposity gain that evolved as an adaptation to European seasonality in early humans who migrated out of Africa. The separation was theorized to have produced different sleep-related adiposity strategies in two resulting groups: African and European Upper-Paleolithic hunter-gatherers. The proposed theory was tested by analyzing a statistical interaction between race and sleep duration within a regression model predicting degree of adiposity in the descendants of these two groups.

Chapter 3: Research Method

Introduction

The purpose of this study was to test an evolutionary theory that has postulated that for descendants of Upper-Paleolithic European hunter-gatherers, extension of sleep provided a behavioral cue indicating an approaching winter, which instigated adaptive fat accumulation. The test of this theory was effected by a comparison of two populations: Blacks and Whites. The theorized trait of sleep-induced adiposity gain was expected to be present in the former, and absent in the latter, a disparity that should have resulted in a racial difference in the relationship between habitual sleep duration and adiposity.

The present chapter will discuss the research design that was used in the study and the proposed source of empirical data, the National Health and Nutrition Examination Survey (NHANES). The methodology, sampling design, as well as recruitment and measurement procedures used in NHANES will be discussed. Further, the data preparation and data analysis plan will be outlined and the intended statistical tests will be discussed. The chapter will conclude with the discussion of threats to validity of the findings, as well as the ethical procedures used in the NHANES study.

Research Design

The approach of this dissertation has been theory testing; therefore, a quantitative method was chosen (Creswell, 2009). Because the present work advanced a hypothesis that related to evolutionary traits in humans, the only feasible design was correlational.

In this study, I analyzed the interaction of race and sleep in a model predicting age-adjusted fat mass index. I conducted secondary analysis of the data obtained as part

of the National Health and Nutrition Examination Survey (NHANES; CDC, National Center for Health Statistics, 2013b). The discovery of a specific evolutionary trait, similar to the one theorized here, can only rely on large epidemiological surveys, like NHANES, in order to obtain necessary power.

Methodology

This dissertation included secondary analysis of data from the National Health and Nutrition Examination Study (NHANES) conducted by the National Center for Health Statistics of the Centers for Disease Control and Prevention (CDC, National Center for Health Statistics, 2013a).

Population

The target population of the NHANES survey was the civilian, noninstitutionalized U.S. population (CDC, National Center for Health Statistics, 2011b). The overall goal of NHANES sampling was to match the US population proportions in accordance to the census data. However, in order to improve precision, certain population subdomains, including, for example, people with low income, were oversampled (that is, sampled with probabilities that were set higher than the respective population proportions; CDC, National Center for Health Statistics, 2011b). The target population of the present study is Black and White males 16 year or older.

Sampling Procedure

The NHANES sampling design used a complex, four-stage, cluster probability sample, the definition of which started with a selection of primary sampling units (PSU), which were whole counties or a number of several smaller contiguous counties (CDC,

National Center for Health Statistics, 2011b). The sampling frame for PSUs consisted of all counties in the United States as per the 2000 Census data (CDC, National Center for Health Statistics, 2006a). In the first stage, PSUs were selected randomly with the probability of selection proportional to the size of the PSU, as defined according to the Census data (CDC, National Center for Health Statistics, 2012a). Each year, 15 PSUs were surveyed (CDC, National Center for Health Statistics, 2012a).

In the second stage, within each PSU, segments were selected, which consisted of one or several blocks of houses, with the probability of selection proportional to the total population of the segment (CDC, National Center for Health Statistics, 2012a). In the third stage, within each segment, a number of households were selected. Finally, in the fourth stage, within each household, one or several members of the household were invited to participate in the study (CDC, National Center for Health Statistics, 2011b). At the third and the fourth stages, the probability of selection of a household in the segment—and a given person within the household—was matched to the actual population distributions for age, sex, and race according to the 2000 United States Census (CDC, National Center for Health Statistics, 2012a). In order to improve the precision of prevalence estimates in specific population groups, some groups were oversampled (i.e. proportionally more representatives were drawn from these groups than would be required for a nationally-representative sample). For example, in the 2004–2005 data cycle, adolescents (12–19 years of age) were oversampled (CDC, National Center for Health Statistics, 2006b). In all data cycles used in the present study, Blacks and persons over 60 years were oversampled as well (CDC, National Center for Health Statistics,

2011b). On average 1.6 persons were invited to participate from one household (CDC, National Center for Health Statistics, 2012a). Sampling weights provided in the datasets were calculated taking into account complex sampling design, unequal probability of selection, and non-response bias (CDC, National Center for Health Statistics, 2012a).

Data Collection

Power analysis. The trait of sleep-induced obesity theorized in the present work was believed to have a complex behavioral element, so it was very unlikely that it could be attributed to a single nucleotide polymorphism, which would be immediately apparent in an individual (Hirschhorn, 2005). Further, a similarity with the trait of lactase persistence, which was discussed above, predicted that no large difference in prevalence between Blacks and Whites could be expected (Mattar et al., 2009). Therefore a small effect size was predicted in this study ($f^2 = 0.02$; Aiken & West, 1991).

Calculation of the sample size. Software program G*Power (Faul, Erdfelder, Buchner, & Lang, 2009; 2012) was used to calculate the required sample size. Assuming a small effect size ($f^2 = 0.02$), significance level $\alpha = 0.05$, and power $1 - \beta = 0.80$ (Aiken & West, 1991) and simple random sampling (SRS) from an infinite population, finding the target interaction as the eighth predictor in the model containing seven covariates would require 725 participants.

Because NHANES used a complex multi-stage probability sampling design, rather than SRS, an adjustment had to be made to reflect the non-random nature of the obtained sample. This adjustment was achieved by multiplying the sample size under the SRS assumption by the design effect (Lohr, 2010). The actual design effect has usually

been dependent of the particular population sub-domain and the variable in question (CDC, National Center for Health Statistics, 2012a). Kato et al. (2009) cited unpublished recommendations from CDC staff for a conservative estimate of the design effect in NHANES to be between 3.5 and 10. A design effect of 5 resulted in the required sample size of 3,625 participants.

Procedures for recruitment, participation, and data collection. NHANES data collection was conducted under contractual arrangement (CDC, National Center for Health Statistics, 2011b). In the location that has been included in the cluster sample, local government and healthcare officials were notified of the upcoming survey; local media were contacted by CDC with a request to feature stories about the survey (CDC, National Center for Health Statistics, 2013a). Households included in the sample received a letter signed by the Director of the National Center for Health Statistics telling about the survey and asking the recipients to participate in the survey.

The households included in the sample were then visited by the NHANES interviewers to conduct initial introduction, screening, and provide a formal invitation to participate in the study (CDC, National Center for Health Statistics, 2011b). Participant consent was obtained in accordance with ethics procedures described below.

Demographic information (including age and race) were collected during an interview in the participant's home. Body measurements were obtained in specially equipped mobile examination centers. Most survey operations were computer-enabled: interview data were collected using touch-screen computers; body measurements were collected using network-connected scales and stadiometers (CDC, National Center for

Health Statistics, 2005). Waist circumference and skinfolds were measured by a specially trained technician using steel tape and caliper respectively, and measurements were entered into the computer manually (CDC, National Center for Health Statistics, 2005).

Procedure for gaining access to the dataset. The data for NHANES have been collected continuously but released in batches covering 2 years of data collection. The data files were available for free download from the website of the CDC/National Center for Health Statistics (2013b). No additional permissions were required for the use of the datasets.

Variable Operationalization

This section will discuss the way the variables were operationalized within the NHANES survey during the period of data collection for the dataset used in the present study. In all cases, the NHANES variable name is provided.

Age. Participant age in years (variable RIDAGEYR) was calculated by computer from the birth-date reported by the participant during the initial home interview.

Race. Data on participant race was acquired during the initial home interview. The data in the NHANES demographic file (variable RIDRETH) has been coded on the basis of the answers to two questions: "Do you consider yourself Hispanic or Latino" (CDC, National Center for Health Statistics, 2006d, p. 8) and "What race do you consider yourself to be? Please select one or more" (CDC, National Center for Health Statistics, 2006d, p. 9) with possible options of American Indian or Alaskan Native, Asian, Black or African American, Native Hawaiian or Pacific Islander, White, Other, Don't know, and

Refused to answer. In the present study, the nominal values of RIDRETH used in the analysis were 'Non-Hispanic Black' and 'Non-Hispanic White'.

Habitual sleep duration. Data on habitual sleep duration was acquired from the interview with a participant at the mobile examination center, as provided in response to the question "How much sleep do you usually get at night on weekdays or workdays?" (CDC, National Center for Health Statistics, 2008, p. 1). The response was recorded in variable SLD010H in whole hours (CDC, National Center for Health Statistics, 2008). In case the participant reported that he or she slept during very short periods of time, the participant was asked to estimate the total hours he or she usually slept at night (CDC, National Center for Health Statistics, 2008). These data were collected only from participants aged 16 years or older (CDC, National Center for Health Statistics, 2008), which set a restriction on the target population to which the findings could be generalized.

Sleep disorders. As part of the interview, each participant was also asked whether he or she had been diagnosed with a sleep disorder; data from participants who responded in the affirmative (SLQ060 = 1), were excluded from the present analysis.

Anthropometric measurements. Height, weight, waist circumference, and skinfolds were measured according to the guidelines outlined in the *NHANES Procedures Manual* (CDC, National Center for Health Statistics, 2005). Height (BMXHT) was measured in centimeters using Seca electronic stadiometer to the nearest 0.1 cm; weight (BMXWT) was measured in kilogram using Toledo electronic scale to the nearest 0.1 kg; waist circumference (BMXWAIST) was measured in centimeters using steel tape to the

nearest 0.1 cm; and triceps (BMXTRI) and subscapular (BMXSUB) skinfolds were measured in millimeters using Holtain caliper to the nearest 0.1 mm (CDC, National Center for Health Statistics, 2005).

Usually height and weight were not measured in participants who arrived to the mobile examination center in a wheelchair. For confidentiality, weight was not reported in persons who had had amputations (CDC, National Center for Health Statistics, 2007).

Preparation of the Analytic Dataset

According to the recommendations of CDC/National Center for Health Statistics (2006a, 2006b), several datasets were merged in order to obtain an analytic dataset with necessary statistical power. In order to do that, the following procedure was implemented.

For the 2-year data cycles of 2005–2006, 2007–2008, and 2009–2010, the following files were downloaded from the CDC/National Center for Health Statistics website: DEMO_D.XPT, DEMO_E.XPT, and DEMO_F.XPT, containing demographic and sampling-design information for the respective data cycles above; BMX_D.XPT, BMX_E.XPT, and BMX_F.XPT, containing body-measurement information; and SLQ_D.XPT, SLQ_E.XPT, and SLQ_F.XPT, containing responses to sleep questionnaire.

IBM Statistical Package for the Social Sciences (SPSS) (Version 20) was used for all data preparation and analysis. The files for each 2-year data cycle were imported into IBM SPSS, sorted by respondent sequence number (SEQN) in ascending order and saved on the local machine. The three files for each 2-year cycle were merged using SEQN as a

key variable, in order to obtain a 2-year analytic dataset with all required variables (CDC, National Center for Health Statistics, 2012a). The three resulting 2-year analytic datasets were then appended to each other in historical order to obtain a single 6-year analytic dataset (CDC, National Center for Health Statistics, 2012a). A new sampling weight was calculated for the 6-year dataset: MEC6YR = WTMEC2YR / 3 (CDC, National Center for Health Statistics, 2012a).

Because NHANES had a complex, multi-stage probability cluster design, calculation of variance and any inferential analysis had to take into account the non-random nature of sampling used in the study (CDC, National Center for Health Statistics, 2012a; Lohr, 2010). In order to prevent the disclosure of the actual primary and secondary sampling units (i.e., US counties, blocks, and households), which had been used for data collection, NHANES created masked variance units, clusters, and weights in such a manner that the calculated variances closely approximated those calculated with the knowledge of the actual sampling plan (CDC, National Center for Health Statistics, 2012a). As part of the preparation of the dataset for the analysis, a data analysis plan was generated using the IBM SPSS Statistics (Version 21) Analyze | Complex Samples | Prepare for Analysis procedure with masked variance pseudo-stratum (SDMVSTRA) used as a stratum identifier, masked variance pseudo-PSU (SDMVPSU) as a cluster identifier, and the calculated 6-year weight (MEC6YR) as sample weight.

The CDC team conducted thorough quality analysis for the datasets as part of the release preparation (CDC, National Center for Health Statistics, 2012a). As part of this preparation, masked-variance-unit sampling weights were calculated with adjustment for

non-response (CDC, National Center for Health Statistics, 2012a). Therefore, records with missing data were excluded from the analysis with minimum expected bias from non-response (Lohr, 2010). No further data cleaning was conducted as part of this analysis.

Preliminary manipulation of variables. Habitual sleep duration represented by variable SLD010H in the NHANES dataset was recoded as a continuous variable. A dummy variable was created for race (RACE) and assigned '0' for non-Hispanic Blacks (RIDRETH = 4), and '1' for non-Hispanic Whites (RIDRETH = 3). For correct calculation of sample weights, the entire dataset was put into analysis, with a subpopulation variable specifically generated to filter out data for female participants, those reporting their race as neither Black nor White, and those indicating presence of a sleep disorder (SLQ060 = 1; CDC, National Center for Health Statistics, 2012a).

Fat-mass index (FMI) was calculated from body-fat percentage (BF%) by multiplying BF% by the total body weight in kilograms (NHANES variable BMXWT) and then dividing it by the squared standing height in meters (BMXHT divided by 100).

Body fat percentage was calculated from the sum of triceps and subscapular skinfolds, age, total body weight, standing height, and waist circumference according to the formulas obtained from Davidson et al. (2011). It has been established that body composition varies not only by age and sex, but also by race (Deurenberg & Deurenberg-Yap, 2007), so Davidson et al. (2011) developed sex- and race-specific formulas by regressing BF% (obtained from dual-energy x-ray absorptiometry) on different

combinations of skinfold measurements (biceps, triceps, subscapular, and suprailiac), waist circumference, body weight and height, as well as age.

Because NHANES datasets contained only triceps and subscapular skinfolds measurements, the following formulas for these two skinfolds were used. For White males, the formula proposed by Davidson et al. (2011) was:

$$p_W = 22.7753 \log_{10}(s + t) + 0.0492\, a + 0.07267\, m - 0.134\, h + 0.1843\, c - 15.8901,$$

where p_W – body fat percentage for White males, s – subscapular skinfold in mm (values for this variable obtained from the NHANES dataset variable BMXSUB), t – triceps skinfold in mm (BMXTRI), a – age in years (RIDAGEYR), m – body weight in kg (BMXWT), h – standing height in cm (BMXHT), and c – waist circumference in cm (BMXWAIST).

For Black males, the following formula was used (Davidson et al., 2011):

$$p_B = 24.7198 \log_{10}(s + t) + 0.07\, a + 0.02821\, m - 0.0705\, h + 0.1758\, c - 27.43365,$$

where p_B is the body fat percentage for Black males.

In addition to the above two formulas, Davidson et al. (2011) developed formulas that allow to calculate BF% from just one of the skinfolds. These formulas were used in case data on either triceps or subscapular skinfold had been missing. For records with missing subscapular skinfold, the following body-fat percentage formulas for triceps skinfold were used (Davidson et al., 2011):

$$p_W = 16.6485 \log_{10} t + 0.0396\, a + 0.15113\, m - 0.2187\, h + 0.2136\, c + 4.9926332,$$

$$p_B = 18.911 \log_{10} t + 0.0847\, a + 0.12217\, m - 0.1513\, h + 0.1409\, c - 2.507321.$$

For records with missing triceps skinfold, the following formulas for subscapular skinfold were used (Davidson et al., 2011):

$$p_W = 18.4103 \log_{10} s + 0.0588\, a + 0.06238\, m - 0.121\, h + 0.236\, c - 11.72966,$$

$$p_B = 12.646 \log_{10} s + 0.0765\, a + 0.12004\, m - 0.1683\, h + 0.2789\, c - 6.960515.$$

For quality control, a random sample of records was entered manually into the body-fat calculation spreadsheet developed by Davidson et al. (2011, online supplement), to verify correct entry of the formulas. No further data imputation was conducted.

Main Analysis

The research hypothesis was: In Whites, compared to Blacks, there would be a stronger increase in the age-adjusted fat mass index (FMI) per unit of sleep duration as measured in male participants of NHANES aged 16 years and older. In terms of the multiple regression model, the above difference in the increase of age-adjusted FMI per unit of sleep duration was statistically represented as a significant interaction of race by sleep duration in the prediction of age-adjusted FMI. Conversely, a null hypothesis predicted no significant interaction between race and sleep duration in the above model.

A significant interaction of race and sleep would imply a difference in the impact of sleep duration on fat accumulation between Whites and Blacks. The proposed theory predicted that the race-by-sleep interaction term to be positive, since a higher adjusted sleep-induced fat accumulation was expected in Whites, when fat accumulation in Blacks was taken as a baseline.

Covariates. A number of known covariates were included in the model to avoid specification error (Berry, 1993). Participant age was included because adiposity levels

have been known to vary by age (Wells, 2009a). Body fat percentage, and hence fat-mass index, has been known to increase with age in a linear fashion (Flegal et al., 2009); thus, no higher-order terms were used. Age moderated the relationship between skinfold thickness and total body fat mass (Davidson et al., 2011). The level of this moderation was known to be stronger in Blacks compared to Whites (Davidson et al., 2011); thus, the age-by-race interaction was included.

Sleep has been shown to be related to FMI in a complex curvilinear fashion (Chaput et al., 2008). This relationship was controlled in the model. A linear and quadratic terms for sleep duration were included in the model. Age has been known to moderate the relationship between sleep duration and fat accumulation in such a way that the relationship disappeared in participants after approximately 40 years of age (Hairston et al., 2010). This was the rationale for inclusion of age-by-sleep interaction that would compensate the linear and quadratic terms for sleep duration, as the age increased. To achieve this, the age-by-sleep and age-by-sleep-squared terms were required.

The analysis of the data justified inclusion of three additional terms: race-by-sleep-squared, age-by-race-by sleep, and age-by-race-by-sleep-squared in order to test more complex, higher-degree interactions (Aiken & West, 1991). To summarize, the regression model that was tested was represented as follows:

$$\begin{aligned} \text{FMI} = {} & b_0 + b_1 \cdot \text{race} + b_2 \cdot \text{age} + b_3 \cdot \text{sleep} + b_4 \cdot \text{sleep}^2 + b_5 \cdot \text{age} \cdot \text{race} + \\ & + b_6 \cdot \text{age} \cdot \text{sleep} + b_7 \cdot \text{age} \cdot \text{sleep}^2 + b_8 \cdot \text{race} \cdot \text{sleep} + b_9 \cdot \text{race} \cdot \text{sleep}^2 + \\ & + b_{10} \cdot \text{age} \cdot \text{race} \cdot \text{sleep} + b_{11} \cdot \text{age} \cdot \text{race} \cdot \text{sleep}^2 + \varepsilon. \end{aligned} \quad (1)$$

The focal statistical test in this study was for positive b_8 significant at $\alpha = 0.05$.

Threats to Validity

External validity. The use of the United States sample of Blacks and Whites may limit generalization to all descendants of African and European Upper-Paleolithic hunter-gatherers, because ancestral genotypes of the American Blacks and Whites have been intermixing in the course of the recent history (McEvoy et al., 2011). This may cause the observed pattern of sleep-related adiposity to be specific to American Blacks and Whites in such a way that this pattern may not generalize to African Blacks or European Whites. Also, the use of participants aged 16 years and above does not allow generalization of study findings to children and adolescents.

Internal validity. Self-report data collected via interview were found to be prone to a number of non-sampling error factors such as recall bias, misunderstanding of the question, and social desirability in responses (CDC, National Center for Health Statistics, 2006a). In particular, self-reported sleep has been known to be poorly correlated with objectively-measured durations, especially in Black participants (Lauderdale et al., 2008). Examination data are liable to measurement error and examiner effects (CDC, National Center for Health Statistics, 2006a), although CDC attempted to minimize these effects by rigorous data quality control, instrument calibration, examiner training, and external validation of the protocols by public health specialists and independent researchers (CDC, National Center for Health Statistics, 2006a).

Construct and statistical conclusion validity. The self-reported race is both a social and a biological construct (Andreasen, 2000), which may or may not be related to the ancestral genotype the individual is carrying. Because systematic cultural differences

have been found between Blacks and Whites in both attitudes to obesity (Ulijaszek & Lofink, 2006) and normative sleep practices (Nielsen et al., 2011), a significant adiposity-related interaction between race and sleep might be a marker of a cultural—rather than ancestral genotype—difference. Thus, a significant finding in this study might be a sign of differential cultural background, rather than differential ancestral survival. For this reason, it would not be possible to use a significant finding in this study to support the proposed theory, if a racial difference in sleep-related adiposity emerged as a cultural, rather than physiological phenomenon.

The NHANES's sleep questionnaire queried participants about the duration of their sleep during weeknights, but not during weekends (CDC, National Center for Health Statistics, 2008). Yet, compensation of short sleep during the week with longer sleep during weekend has been shown to moderate the impact of short sleep on adiposity gain (Wing et al., 2009). This restriction of the analysis to weeknight sleep might impair the construct validity of 'habitual sleep' and limit the generalizability of the findings.

Ethical Procedures

The collection of the NHANES data used in the present study was approved by the National Center for Health Statistics Research Ethics Review Board (Protocol No. 2005-06; CDC, National Center for Health Statistics, 2012c). The demographic data on age and race of participants used in the present study was obtained as part of the home-interview element of NHANES. Participants were briefed about their participation in interviews and examinations, and signed consent forms described below (CDC, National Center for Health Statistics, 2013a).

The initial *Home Interview Consent* form (CDC, National Center for Health Statistics, 2011a) outlined the purpose of the NHANES study, stated that the person was free to decline participation, and did not have to answer all of the questions, even if he or she agreed to participate; the form requested specific permission to conduct additional health research by linking the data obtained in the interview with other data on health and nutrition obtained about the same person as part of other NHANES surveys and examinations (CDC, National Center for Health Statistics, 2011a). The form provided name and phone number of the person to contact about the survey, stated that all personally identifiable information will be treated in strict confidence, and outlined legal sanctions that would be used against representatives of the National Center of Health Statistics for willful disclosure of such information (CDC, National Center for Health Statistics, 2011a).

Body measurements and responses to the sleep questionnaire were obtained from participants taking part in a medical-examination element of NHANES conducted in mobile examinations centers (CDC, National Center for Health Statistics, 2012b). When necessary, free transportation was provided to and from mobile examination center (CDC, National Center for Health Statistics, 2013a). Prior to beginning of the medical examination, all participants received a full list of examinations they would undergo, with the indication of the examinations for which they would receive full results (CDC, National Center for Health Statistics, 2010a). The examination consent brochure received by participants (CDC, National Center for Health Statistics, 2010b) described the purpose and the content of the examination, stated that participants would receive free

health-test results and a small gift in compensation for their time and effort; the brochure further discussed safety implications of the examinations, stated that the participant was free to decline any or all parts of the examination, described how the data would be used, asserted that any personally identifiable data would be treated as strictly confidential, and provided the name, phone number, and the address of the person they may contact with their questions about the examination. The examiner then read the consent form (CDC, National Center for Health Statistics, 2009) to the participant and asked him/her to sign it.

All data in public-release files were anonymous, including geographical stratification information, which had been regrouped and recoded into masked strata and clusters to maintain confidentiality (CDC, National Center for Health Statistics, 2012a). The CDC regulated and monitored secure storage and transmission of any personally identifiable information collected as part of NHANES (CDC, National Center for Health Statistics, 2006c).

Summary

The present study tested a directional hypothesis about an adjusted interaction of race and sleep duration in the model predicting fat-mass index (FMI). The analysis used the archival data collected between 2005 and 2010 as part of a National Health and Nutrition Examination Survey (NHANES). FMI was calculated from skinfold data according to Davidson et al. (2011). After adjustment for the complex multi-stage clustered probability sampling design of NHANES, the number of participant records required for the analytic dataset was at least 3,625. IBM SPSS Statistics Complex

Samples Version 20 was used to analyze NHANES's stratified clustered analytic dataset obtained from CDC's National Center for Health Statistics.

Chapter 4: Results

Introduction

The purpose of the study was to test the evolutionary theory of sleep-induced adiposity gain in modern descendants of European hunter-gatherers. The Institutional Review Board approved the study (approval #06-26-13-0086499). A directional hypothesis tested a prediction that for Whites, compared to Blacks, there would be a greater increase in the age-adjusted fat mass index (FMI) per unit of sleep duration as measured in male participants of NHANES aged 16 years and older. The statistical model was tested using multiple-regression in which the dependent variable (DV) was age-adjusted FMI; independent variables (IV) included, among other terms, the one measuring the interaction of race by sleep duration. This chapter describes the characteristics of the sample, provides the details of conducted data manipulations, and reports the results of the main and post-hoc analyses.

The main analysis consisted of two parts. First, a regression model described by the formula (1) in Chapter 3 was tested. The second analysis involved retaining statistically significant parameters in an analytical model, the analysis of which involved calculating the first derivative of FMI by age for Blacks and for Whites, and then comparing the resulting slopes. Given that the rate of FMI gain was found to be dependent on sleep duration, but in the opposite direction to what was predicted, a third analysis was performed. The final part of the results contains a summary of the analysis of the annual rates of FMI change individually for Blacks and Whites.

Sample Demographics

The dataset for this study was downloaded from the website of the CDC/National Center for Health Statistics (2013b). Data were collected in three 2-year cycles between 2005 and 2010. For the present study, the data were restricted to Black and White male participants 16 years and older, who did not report a sleep disorder and for whom the available anthropometric data allowed to calculate the FMI. The final analysis dataset contained 5313 records.

Table 1 contains the sample demographics. The cluster sample weights were used to calculate the total US populations of Black and White males represented by the present complex probability sample (Lohr, 2010, p. 288).

Table 1

*Demographic Characteristics of the Study Sample (*N = *5,313)*

	Black Males	White Males
Number of participants	1,668	3,645
Population represented by the sample	10,071,071	66,274,806
Age, years	40.1 [38.9, 41.3]	45.0 [44.0, 46.0]
Body weight, kg	86.0 [84.9, 87.0]	87.5 [86.8, 88.3]
BMI, kg / m^2	27.5 [27.1, 27.8]	27.8 [27.5, 28.0]
FMI, kg / m^2	5.58 [5.37, 5.79]	6.38 [6.21, 6.56]
Weeknight sleep duration, hours	6.51 [6.43, 6.60]	6.93 [6.87, 6.98]

Note. Figures in brackets are 95% confidence intervals. BMI = body mass index, FMI = fat mass index.

Black participants were on average younger than White participants ($p < .001$) and had significantly lower fat mass index (FMI; $p < .001$), although there were no significant differences in body weight and body mass index (BMI) between the two

groups. Blacks reported significantly lower average weeknight sleep duration (6.5 hours, compared to Whites' 6.9 hours, $p < .001$).

Main Analysis

In order to conduct multivariate regression analysis of moderation effects, IVs were centered (Aiken & West, 1991, p. 9); prior to that, original variables were transformed as needed to minimize skew and kurtosis. Three variables required such transformation: participant age (NHANES variable RIDAGEYR), sleep duration (SLD010H) and FMI.

Because NHANES oversampled adolescents and adults over 80, the sample distribution by age strongly deviated from normality. To resolve this issue, age variable RIDAGEYR was subjected to a cubic transformation: the sample median age (47 years) was subtracted from RIDAGEYR and the result was taken to the third power; this resulted in heavily over-sampled tails of the distribution to be spread out more thinly, thus closer approximating normal distribution. The resulting distribution was then centered according to Aiken and West (1991, p. 9) by subtracting its mean (which, as a result of prior transformation, was negative: −1130). Sleep duration (SLD010H) was close to normal distribution and did not require non-linear transformation; it was centered according to Aiken and West (1991, p. 9) by subtracting the combined-sample mean of 6.87 hours. In contrast, FMI required non-linear transformation due to a heavy skew in the distribution. Also, for some participants, the FMI calculation according to Davidson et al. (2011) produced small negative values; these records were retained in the dataset to avoid violating regression assumptions of range restriction (Berry, 1993). However, in

order to ensure that all FMI values were larger than 1 kg/m^2 before entering a logarithmic transformation, 1.5 was added to all values. Because FMI was the DV, it was not centered (Aiken & West, 1991, p. 35). The dummy variable RACE was neither transformed nor centered (Aiken & West, 1991, p. 130). Thus, the following transformed variables (henceforth denoted in italics) were obtained:

$$age = (\text{RIDAGEYR} - 47)^3 + 1130, \quad (2)$$

$$sleep = \text{SLD010H} - 6.8725, \quad (3)$$

$$fmi = \log_{10}(\text{FMI} + 1.5), \quad (4)$$

where RIDAGEYR is self-reported age in years in the NHANES dataset, SLD010H – self-reported habitual weeknight sleep in hours, and FMI – fat-mass index calculated according to Davidson et al. (2011).

Table 2 contains correlations among the transformed and centered variables. These correlations justify the inclusion of predictor variables, in particular, the squared sleep duration.

Table 2

Correlations Between Variables in the Regression Model

	fmi	*age*	*sleep*
age	.347**		
sleep	−.073**	.021	
$sleep^2$	−.030*	.020	−.039**

Note. * $p < .05$. ** $p < .01$.

As provided in formula (1) in Chapter 3, the following regression model was tested:

$$fmi = b_0 + b_1 \cdot race + b_2 \cdot age + b_3 \cdot sleep + b_4 \cdot sleep^2 + b_5 \cdot age \cdot race +$$

$$+ b_6 \cdot age \cdot sleep + b_7 \cdot age \cdot sleep^2 + b_8 \cdot race \cdot sleep + b_9 \cdot race \cdot sleep^2 +$$

$$+ b_{10} \cdot age \cdot race \cdot sleep + b_{11} \cdot age \cdot race \cdot sleep^2 + \varepsilon.$$

Table 3 contains the results of the multivariate regression model; Table 4 contains the variance – covariance matrix for significant parameters used in further analyses. Exclusion of three multivariate outliers (two persons reported sleeping on average 1 hour per night, and one person reported sleeping 12 hours and had body-mass index of 49 kg/m^2) did not result in a substantial change of the model.

Table 3

Multiple Regression Analysis Predicting Fat Mass Index in Male Participants of NHANES

Parameter	*B*	SE *B*	β	*t*(47)	*p*	ΔR^2
Constant (b_0)	$8.076 \cdot 10^{-1}$	$5.75 \cdot 10^{-3}$		140.48	.000	
race (b_1)	$5.182 \cdot 10^{-2}$	$7.05 \cdot 10^{-3}$	.117	7.35	.000	.011
age (b_2)	$6.445 \cdot 10^{-6}$	$4.60 \cdot 10^{-7}$	.380	14.01	.000	.093
sleep (b_3)	$-1.352 \cdot 10^{-2}$	$3.41 \cdot 10^{-3}$	–.067	–3.97	.000	.005
sleep^2 (b_4)	$4.947 \cdot 10^{-4}$	$1.27 \cdot 10^{-3}$	.006	.39	.698	.002
age × *race* (b_5)	$-1.700 \cdot 10^{-6}$	$5.35 \cdot 10^{-7}$	–.095	–3.18	.003	.001
age × *sleep* (b_6)	$4.846 \cdot 10^{-7}$	$1.97 \cdot 10^{-7}$	.032	2.46	.018	.001
age × *sleep*^{2} (b_7)	$-1.474 \cdot 10^{-7}$	$1.11 \cdot 10^{-7}$	–.028	–1.33	.190	.001
race × *sleep* (b_8)	$3.109 \cdot 10^{-3}$	$4.39 \cdot 10^{-3}$	.015	.71	.482	.000
race × *sleep*^{2} (b_9)	$-4.063 \cdot 10^{-3}$	$1.66 \cdot 10^{-3}$	–.044	–2.46	.018	.000
age × *race* × *sleep* (b_{10})	$-6.833 \cdot 10^{-7}$	$2.56 \cdot 10^{-7}$	–.047	–2.68	.010	.001
age × *race* × *sleep*^{2} (b_{11})	$3.408 \cdot 10^{-8}$	$1.30 \cdot 10^{-7}$	.006	.26	.794	.000

Note. $R^2 = .115$ ($N = 5310$, $p < .001$). SE = standard error of the estimate.

Table 4

Variance – Covariance Matrix for the Significant Regression Coefficients.

	b_0	b_1	b_2	b_3
b_0	$3.305 \cdot 10^{-5}$			
b_1	$-2.848 \cdot 10^{-5}$	$4.967 \cdot 10^{-5}$		
b_2	$-8.501 \cdot 10^{-11}$	$-1.166 \cdot 10^{-11}$	$2.117 \cdot 10^{-13}$	
b_3	$1.182 \cdot 10^{-6}$	$-6.203 \cdot 10^{-6}$	$-2.890 \cdot 10^{-10}$	$1.162 \cdot 10^{-5}$
b_5	$-2.426 \cdot 10^{-10}$	$-1.507 \cdot 10^{-10}$	$-2.165 \cdot 10^{-13}$	$6.442 \cdot 10^{-10}$
b_6	$-2.705 \cdot 10^{-10}$	$4.095 \cdot 10^{-10}$	$6.059 \cdot 10^{-15}$	$-5.470 \cdot 10^{-11}$
b_9	$1.227 \cdot 10^{-6}$	$-3.125 \cdot 10^{-6}$	$2.092 \cdot 10^{-10}$	$1.251 \cdot 10^{-6}$
b_{10}	$3.530 \cdot 10^{-10}$	$-4.185 \cdot 10^{-10}$	$-2.058 \cdot 10^{-14}$	$1.736 \cdot 10^{-11}$

	b_5	b_6	b_9	b_{10}
b_5	$2.862 \cdot 10^{-13}$			
b_6	$-9.402 \cdot 10^{-15}$	$3.890 \cdot 10^{-14}$		
b_9	$-1.118 \cdot 10^{-10}$	$-4.970 \cdot 10^{-11}$	$2.740 \cdot 10^{-6}$	
b_{10}	$2.006 \cdot 10^{-14}$	$-3.914 \cdot 10^{-14}$	$4.148 \cdot 10^{-11}$	$6.527 \cdot 10^{-14}$

The resulting mathematical model, which was used for further analyses is presented in formula (5) (terms with non-significant parameters were excluded from the multivariate model, since by definition, they are not statistically different from zero).

$$fmi = b_1 \cdot race + b_2 \cdot age + b_3 \cdot sleep + b_5 \cdot age \cdot race + b_6 \cdot age \cdot sleep +$$

$$+ b_9 \cdot race \cdot sleep^2 + b_{10} \cdot age \cdot race \cdot sleep. \quad (5)$$

The research hypothesis stated that in Whites, compared to Blacks, there would be a stronger increase in the age-adjusted FMI per unit of sleep duration. The a-priori model represented by formula (1) postulated that a simple interaction between sleep and race would be found in the form of a significant positive parameter b_8. However, in the regression model, this parameter was not statistically significant; instead, two complex

interactions involved race and sleep (race × sleep^2 and age × race × sleep). Participant age was present in several terms in mathematical model (5); this suggested complex longitudinal effects on FMI. In particular, sleep duration interacted with age, and the effect of sleep on FMI was different for Blacks and Whites, as evidenced by the significant moderating effect of race. Note that the three-way interaction of race by age by sleep (b_{10}) could be interpreted from two different points of view: age moderation of race by sleep interaction and race moderation of age by sleep interaction; mathematically, both interpretations are equivalent (Aiken & West, 1991, p. 52).

In order to test the research hypothesis with the help of the mathematical model in (5), the change in the age-adjusted FMI was represented by the rate of annual gain in FMI, which was obtained as a partial derivative of the FMI by age. Substituting *race* = 0 for Blacks and *race* = 1 for Whites in (5), the partial derivatives of FMI by age for Blacks and Whites were obtained as (Aiken & West, 1991, p. 75):

$$\partial\, fmi_{\text{BLACKS}} / \partial\, age = b_2 + b_6 \cdot sleep \qquad (6)$$

$$\partial\, fmi_{\text{WHITES}} / \partial\, age = b_2 + b_5 + b_6 \cdot sleep + b_{10} \cdot sleep \qquad (7)$$

In post-hoc analyses (Aiken & West, 1991, p. 77), the difference between the FMI rates was estimated by subtracting formula (6) from (7); cancellation of terms resulted in the formula:

$$Diff = \partial\, fmi_{\text{WHITES}} / \partial\, \text{age} - \partial\, fmi_{\text{BLACKS}} / \partial\, \text{age} = b_5 + b_{10} \cdot sleep. \qquad (8)$$

The test of statistical significance of the difference in the FMI rates was conducted by calculating the *t* statistic (*df* = 47 according to the NHANES dataset

complex sampling design). The standard error of the difference *Diff* in (8) was (Aiken & West, 1991, p. 26, formula 2.10):

$$SE_{DIFF} = (s_{5/5} + 2s_{5/10} \cdot sleep + s_{10/10} \cdot sleep^2)^{1/2},$$

where $s_{5/5}$ and $s_{10/10}$ are variances of parameter estimates b_5 and b_{10}, and $s_{5/10}$ is their covariance (from Table 4). Table 5 reports the differences in the rates of FMI gain between Whites and Blacks for different sleep durations.

Table 5

Difference in Annual Rates of FMI Gain between White and Black Male Participants of NHANES

	Habitual sleep duration				
	5 hours	6 hours	7 hours	8 hours	9 hours
Diff	$-4.21 \cdot 10^{-7}$	$-1.10 \cdot 10^{-6*}$	$-1.79 \cdot 10^{-6**}$	$-2.47 \cdot 10^{-6***}$	$-3.15 \cdot 10^{-6***}$

Note. $Diff = \partial\, fmi_{\text{WHITES}} / \partial\, age - \partial\, fmi_{\text{BLACKS}} / \partial\, age$. * $p < .05$ (two-tailed). ** $p < .01$ (two-tailed). *** $p < .001$ (two-tailed).

Black males had, compared to Whites, a higher rate of FMI gain for longer sleep durations; this result was opposite to the prediction of the research hypothesis. Thus, additional post-hoc analyses were conducted in order to understand the effect of different habitual sleep duration on the age-related FMI gain separately for Blacks and Whites.

Additional Analyses

Formulas (6) and (7) were used to describe the respective rates of FMI gain. The standard errors for the rates of FMI change were calculated in order to conduct significance tests (Aiken & West, 1991, p. 26). Table 6 contains the annual rates of FMI gain in Blacks and Whites.

Table 6

Post-Hoc Analysis of the Annual Rates of FMI Gain in Black and White Male Participants of NHANES

	Habitual sleep duration					
	5 hours	6 hours	7 hours	8 hours	9 hours	*p* for trend
Blacks	$5.54 \cdot 10^{-6}$	$6.02 \cdot 10^{-6}$	$6.51 \cdot 10^{-6}$	$6.99 \cdot 10^{-6}$	$7.48 \cdot 10^{-6}$	.000
Whites	$5.12 \cdot 10^{-6}$	$4.92 \cdot 10^{-6}$	$4.72 \cdot 10^{-6}$	$4.52 \cdot 10^{-6}$	$4.32 \cdot 10^{-6}$	.223

Note. All FMI rates significantly different from zero, $p < .001$ (two-tailed).

The results in Table 6 show that both Blacks and Whites were found to experience significant age-related gain in fat mass across the lifespan. However, Blacks and Whites differed in the relationship between annual rate of adiposity gain and habitual sleep duration. In Whites, the rate of FMI gain for long sleepers was slightly lower than for short sleepers, but the difference was not significant (comparison of the rates for those reporting 5 and 8 hours of sleep, $t(47) = 1.997$, $p = .052$). In contrast, Black long sleepers experienced a faster lifespan adiposity gain compared to White long sleepers (comparison of the rates of FMI gain in Blacks and Whites sleeping 8 hours, $t(47) = 3.838$, $p < .001$) and to Black short sleepers (comparison of the rates in those sleeping 5 and 8 hours, $t(47) = 2.773$, $p = .008$).

Following from equation (6), the sleep gradient of the annual rate of FMI gain in Blacks (partial derivative by sleep) equals $b_6 = 4.846 \cdot 10^{-7}$; it was positive and significant, $t(47) = 2.457$, $p < .001$. This result can also be seen in Table 6: the rate of the FMI gain in Blacks increased as the sleep duration rises from 5 hours to 9 hours, meaning

that the longer-sleeping Blacks faced a steeper lifetime adiposity gain. In Whites, the sleep gradient of the annual rate of FMI gain was received as a partial derivative by sleep from equation (7) as $b_6 + b_{10} = -1.987 \cdot 10^{-7}$, $t(47) = -1.234$, $p = .223$; in this model, the sleep gradient was negative, but not significant, meaning that no substantial difference in the annual rate of adiposity gain was seen between short- and long-sleeping White males.

Summary

Results showed that FMI had a complex interaction with age, race, and sleep duration in Black and White male participants of NHANES aged 16 years and older. Analyses showed: (a) the higher order interactions race × sleep^2 and age × race × sleep were significant predictors in the model for fat mass index; (b) Black males had, compared to Whites, a higher rate of FMI gain for longer sleep durations; and (c) in Whites, the rate of FMI gain for long sleepers and short sleepers was not statistically different, while in Blacks, long sleepers experienced a faster lifespan adiposity gain compared to both White long sleepers and to Black short sleepers. Taken together, these findings meant that the null hypothesis was retained and no support was found for the proposed evolutionary theory of sleep-induced obesity. In addition, it was found that long sleep was a significant risk factor for age-related adiposity gain in Black males aged 16 years and older, while in Whites, sleep duration had no significant effect on adiposity gain.

Chapter 5: Discussion, Conclusions, and Recommendations

Introduction

The purpose of this study was to test the evolutionary theory of sleep-induced adiposity gain in descendants of European Upper-Paleolithic hunter-gatherers; the hypothesis predicted a faster sleep-related increase in age-adjusted fat-mass index (FMI) in White men, as compared to Black men. The research hypothesis was tested with the use of a mathematical model of the effect of sleep, race, and age on adiposity, which was developed on the basis of archival cross-sectional data obtained as part of National Health and Nutrition Examination Survey (NHANES).

The results of the analysis of the model did not provide support for the research hypothesis. Additional analyses indicated a race-specific pattern of association between sleep duration and adiposity gain. Overall, the results showed that longer sleep was associated with a higher adiposity gain in Black men aged 16 years and older, while no such association was found in White men. This chapter includes a more detailed interpretation of the obtained results in the context of prior research, a discussion of the limitations of the present study, and recommendations for further research and practice.

Interpretation of the Findings

The main empirical contribution of this work is a comprehensive mathematical model of the effect of sleep duration on adiposity of Black and White males aged 16 years and older. Two kinds of results were obtained by analyzing the obtained mathematical model predicting FMI from age, race, and sleep duration. First, the model analysis falsified the research hypothesis developed in this work. Second, the analysis

described the relationship between habitual sleep duration and the annual rate of adiposity gain in Black and White men; these results are described in order.

The Falsified Research Hypothesis

As shown by the results of the model analysis, longer sleep did not result in a significant increase in adiposity in Whites, either in general, or in a specific comparison to Blacks. In fact, a serendipitous finding related to the age-related adiposity gain showed exactly the opposite outcome of the comparison between Blacks and Whites. Thus, the present findings did not provide support for the idea of sleep-induced adiposity gain in Whites: longer sleep in White men did not result in any change in rates of adiposity gain as they age, whereas for Blacks, the results showed that adiposity gain increases with age (Table 6).

From an evolutionary point of view, the results obtained here lend more support to the contention of Gangwisch (2013) that sleep restriction, rather than sleep extension, leads to a specific change in metabolism and, thus, obesity (although this trend is insignificant in the present findings). Gangwisch (2013) hypothesized that shortening nights in the spring acted as a cue for increased consumption of carbohydrates (as seen in experimental sleep restriction studies, e.g., Nedeltcheva et al., 2009) and induced Upper-Paleolithic Europeans to shift from a predominantly fat/protein diet they relied upon during winter, to a more carbohydrate-oriented diet in spring. Thus, a less calorie-dense carbohydrate diet obtained from plants resulted in a springtime fat loss in early human males, which agrees with the logic of the seasonal adiposity variation discussed in this work. Then, it is possible to speculate that the modern rise in obesity results from the

fact that today's high-carbohydrate snacks are much more calorie-dense than 50 thousand years ago (Wells, 2013), leading to a runaway adiposity gain, instead of seasonal adiposity loss.

It has been observed that sleep restriction leads to important behavioral changes in humans. Galli et al. (2013) reported that both calorie and alcohol consumption are inversely related to sleep duration in short sleepers, which agreed with the finding of Chaput, Després, et al. (2011a) who suggested that weight gain in modern humans is further exacerbated by behavioral disinhibition induced by short sleep. Combined with the observation that short sleep increases the expression of aggressive impulses and violent behavior (Kamphuis, Meerlo, Koolhaas, & Lancel, 2012), these studies seem to support the evolutionary logic proposed in this work arguing that seasonal variations in sleep and adiposity were intricately related with behavioral and reproductive strategies of male hunter-gatherers, including springtime migration and competition for mates (Heyer et al., 2012).

However, the evolutionary explanation suggested above still has substantial problem areas. For example, Brondel, Romer, Nougues, Touyarou, and Davenne (2010) found that an experimental sleep restriction induced dietary shift towards fats, rather than carbohydrates, so the sleep – diet relationship seems to be even more complicated than suggested above. Further, while the above discussion supports the idea of seasonal weight loss in Upper-Paleolithic males, the lack of evidence for seasonal weight gain observed in this work resonates with the voices that question the general idea of adaptive value of human obesity (Speakman, 2006). Neel's (1962) theory of the thrifty genotype

that equated obesity to a survival strategy assumed that early humans had to deal with constant hardship and malnutrition. Yet, Shostak (1981) writing about !Kung, one of the last remaining hunter-gatherer tribes in Kalahari desert in Africa, marveled at the apparent "stability and security of the !Kung subsistence base, [which] seems to indicate that gathering and hunting, even in this marginal environment, is not a terribly arduous way of life" (Shostak, 1981, p. 96).

Admittedly, the environmental challenges faced by European hunter-gatherers throughout the ice age might have been quite different, but the evolutionary framework proposed in this work have not yet provided a compelling explanation of the association between sleep and adiposity gain in Whites. Further, the proposed evolutionary framework clearly fails to account for the sleep-related variation in the annual rate of adiposity gain in Blacks found in this study—unless one postulates a yet-unknown sleep-related metabolic imbalance in Blacks that this study failed to control for. To summarize, the evolutionary theory is still unable to provide a coherent picture of an adaptive relationship between sleep duration and adiposity gain, if one indeed exists.

The Effect of Sleep Duration on Adiposity Gain

The present work used cross-sectional epidemiological data to model the long-term adiposity effect of maintenance of a certain habitual sleep duration. Several studies developed similar models of this effect from cross-sectional relationships observed between habitual sleep duration and various markers of adiposity. The most recent study of Ford et al. (2013) analyzing the same NHANES dataset as the present work, found that longer sleep was associated with BMI and waist circumference relationships in both

Black and White males. However, Ford et al. (2013) did not discover a complex set of age, race, and sleep interactions reported here, because the models of BMI and waist circumference in Ford et al. (2013) did not include any third-order interactions (in the present work, the difference between Blacks and Whites in the way the rate of adiposity gain varied with sleep was discovered because the model included the three-way interaction of age × race × sleep duration). Another cross-sectional study (Yi et al., 2013) found an inverse relationship between self-reported sleep duration and subcutaneous fat measured with computed tomography in Japanese men aged 30 years and older, most of whom slept less than 7 hours. At the same time, R. Liu et al. (2012) found no relationship between sleep duration and FMI in Chinese male twins. Garaulet et al. (2011) found no correlation between sleep and FMI in predominantly White European adolescent males 13−17 years old, but Chaput et al. (2009) found higher fat mass in shorter sleeping White adults. Overall, cross-sectional studies produced mixed results for the relationship between sleep and adiposity indices.

Most cross-sectional studies did not find racial-group differences in models predicting BMI (G. E. Silva et al., 2011) or waist circumference (St-Onge et al., 2010); however, in comparison to the BMI of average sleepers, Meyer et al. (2012) found longer sleep to be associated with lower BMI in Whites, no substantial difference in Blacks or Hispanics—and significantly higher BMI in the mixed/other ethnic group.

An important methodological difference of the present study from earlier cross-sectional studies was the use of the age trend of FMI as the basis for comparison of adiposity among groups. One could argue that the analysis of the derivative of FMI by

age, rather than average FMI, provides a more accurate model of the long-term effect of sleep duration on adiposity when examining cross-sectional data: because lifespan adiposity gain is observed in many modern human populations (Flegal et al., 2009), the specific effect of sleep on adiposity is better represented by a difference in the annual rate of change in FMI (i.e. derivative of FMI by age) rather than a difference in static FMI values.

Due to this methodological difference, the results of this study seem to agree much better with longitudinal, rather than cross-sectional empirical studies. For example, Hasler et al. (2004) analyzed the annual rates of BMI gain in White European adults who were followed between the ages of 27 and 40, and found positive annual BMI-gain rates for the sleep durations between 5 and 9 hours; these rates were not different among White men, in agreement with the present finding for FMI. Similarly, there was no significant sleep-related variation in the rate of BMI or waist-circumference change in a 5-year prospective study of predominantly male sample of British civil servants (Stranges, Cappuccio, et al., 2008). Likewise, Lyytikäinen et al. (2011) found no significant sleep-related difference in 5–7-year weight gain in 40–60-year-old men—but not women—in Helsinki Health Study in Finland. Finally, López-García et al. (2008) found no sleep-related difference in the 2-year BMI change in White Spanish men 60 years and older. Findings of the present study are consistent with most of these studies that indicate no variation in the rate of FMI gain in White males as a function of sleep duration.

Some longitudinal studies found a statistically significant inverse relationship between sleep duration and the change in adiposity indices, unlike the present model's

prediction of non-significant relationship in Whites. However, these studies usually reported sex-adjusted outcomes for combined samples, rather than separate results by sex (e.g., Landhuis et al., 2008). In addition, several longitudinal studies found a U-shaped relationship between sleep duration and the change of various adiposity indices. Chaput et al. (2008) found a higher rate of a 6-year increase in the body fat percentage in Canadian Whites aged 21–64 years reporting sleep durations of less than 6 or more than 9 hours, compared to those reporting sleep of 7 or 8 hours. Similarly, Watanabe et al. (2010) reported a positive rate of BMI gain for Japanese men sleeping 5 hours or less and 9 hours or more, whereas the BMI gain rates for sleep durations from 6 to 9 hours were not significantly different from zero. Results from the present study do not indicate such protective effect in median sleep durations in either White of Black men.

Finally, the longitudinal study of Hairston et al. (2010) followed a minority sample that combined Blacks and Hispanics. Although no separate analyses were reported for individual sexes and races, in the combined sample, Hairston et al. (2010) found a U-shaped relationship between sleep and the rate of gain of subcutaneous fat, with sleep durations of both 5 hours or less and 8 hours or more associated with significantly higher rates of gain, compared to sleep durations of 6–7 hours. The analysis of Hairston et al. (2010), however, only adjusted for age, race, and sex, without analyzing more complex interactions, which, in this study, uncovered a sleep-related increase in the annual rate of FMI gain in Blacks.

Similarly to all previous studies, the present model was adjusted for age, race, and sleep duration. However, a racial difference in the sleep-related adiposity gain was only

discovered when both age × sleep (b_6) and race × age × sleep (b_{10}) interactions were included in the model (Table 3). Previous researchers (from a review of the literature) have not included these interactions in their models.

Limitations of the Study

The main limitation of the study is the use of self-reported, rather than objectively measured, sleep duration. It is possible that this study's significant difference between Black and White men was an artifact of a racial-group difference in the precision of sleep reports. Lauderdale et al. (2008) found that the correlations between self-reported and actigraphically measured sleep durations were around 0.56 (95% CI: 0.44, 0.68) in Whites, and 0.29 (95% CI: 0.10, 0.48) in Blacks. Indeed, when Lauderdale et al. (2009) measured sleep actigraphically, no significant difference was found between 5-year BMI gain in Black and White males (although, admittedly, the sample size was so small that no Black males were found to have sleep durations longer than 7.5 hours).

Another limitation of this study was impossibility to control for cultural, as opposed to the hypothesized genetic, differences between Blacks and Whites. Recent findings of Hicken, Lee, Ailshire, Burgard, and Williams (2013) suggested that a phenomenon of racism-related vigilance, i.e., mental anticipation of racial discrimination, might reduce quality and duration of sleep in Blacks compared to Whites, thus introducing a possible interaction between race and sleep duration in various health outcomes, including obesity. The findings of Hicken et al. (2013) allow to speculate that racism vigilance may contribute to stress-related low quality of sleep (Akerstedt, Kecklund, & Axelsson, 2007) and stress-related adiposity gain (Elder et al., 2012; Yang

et al., 2013), both of which may be present in American Blacks, but absent in American Whites. Although this logic would predict higher adiposity gain in shorter-, rather than longer-sleeping Blacks, it provides an example of how lack of control for culturally relevant variables may introduce unexpected race-group interactions. Unfortunately, the lack of such culturally relevant variables in the NHANES dataset did not allow to control for their influence on adiposity.

The other important limitation of this study was its cross-sectional design. Only well-controlled trials, like, for example, the ongoing study of Cizza et al. (2010), would allow to demonstrate the actual impact of sleep duration on long-term adiposity gain.

Another limitation of this study was the lack of control for clinical conditions that could lead to both under-nutrition and extended sleep (Krueger & Friedman, 2009). This may have resulted in confounding of mean adiposity estimates for longer sleep durations. A more robust design was used by Ford et al. (2013) who controlled for a larger number of clinical conditions reported in NHANES, which, however, precluded Ford et al. (2013) from testing a large number of interactions, due to loss of statistical power.

Recommendations

The main scientific contribution of this work is the mathematical model of the effect of sleep extension on fat-mass index in Black and White males aged 16 years or older. Although the evolutionary theory of obesity proposed in this work was not supported by the empirically derived model, the model itself can be used to inform the design of future behavioral interventions.

Reliable conclusions about adiposity effects of sleep extension or restriction may only be obtained from rigorous clinical trials (such as those reported in Cizza et al., 2010). However, these clinical trials will benefit from more precise hypotheses derived from mathematical models similar to the one proposed here. Sleep extension interventions aimed at other health outcomes, such as hypertension (Haack et al., 2013), may also use the proposed mathematical model to predict mediating effects of adiposity loss within the targeted effect.

Implications

Very few epidemiological studies attempt to conduct separate analyses of the interaction of race with various health-related interventions. This is mostly due to small statistical power available for racial-group comparisons in representative samples (Wilsmore et al., 2012), which, however, may lead to inattention to important racial differences in health outcomes. Such inattention may result in blind application of findings from White populations to racial minorities.

An implication for practice, which can be derived from this work, lies in the development of behavioral interventions that will be both culturally-specific to Black populations (e.g., Corsino et al., 2012) and will factor in the patterns of sleep – adiposity relationship that are specific to Blacks. In particular, the findings of this study argue against the inclusion of sleep-extension protocols in behavioral interventions designed for Black men (contrary to Cizza et al., 2010).

Longer habitual sleep has been associated with depression (Krueger & Friedman, 2009). Therefore, psychology practitioners designing and implementing specific anti-

obesity interventions for Black men have to consider whether depression-related sleep extension threatens their clients weight-management goals. Practitioners should likewise monitor whether habitual sleep extension in Black men increases the likelihood of behavioral relapses—and whether depression may be a contributing factor here (Krueger & Friedman, 2009).

In the modern world, budgeting of time required for physical exercise emerges as an important factor in behavioral management of obesity (Maniwa et al., 2012). Similarly, more time is required to buy and prepare healthy food, compared to procuring calorie-dense snacks. Because longer sleep duration is, as follows from the present results, a specific obesity-risk factor for Black males, self-monitoring of sleep duration (via a diary or actigraphically) should be included in comprehensive programs of behavioral management of obesity. (When actigraphy is used to measure daily energy expenditure in an obesity intervention, the measurement protocol should be extended to include sleep.) Information on sleep duration can then be used in problem-solving analysis with the client, in order to understand how extended sleep may limit the amount of time available for exercise and healthy food preparation.

Conclusion

Although the results of this study did not provide support for the evolutionary theory of sleep-related adiposity gain in descendants of European Upper-Paleolithic hunter-gatherers proposed in this work, an important racial-group interaction was discovered in the additional analysis. According to this finding, longer sleep in Black males aged 16 years and older may potentially lead to adverse long-term adiposity gain.

The implications of this finding include a potentially deleterious effect of anti-obesity sleep extension interventions in Black males (as well as possibly negligible effect of such interventions in White males). Sleep duration monitoring should be recommended in Black males to improve time budgeting in behavioral interventions to achieve better results in planning and implementing daily physical exercise regimen.

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Curriculum Vitae

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Work Experience

Aug 2013 – Present. National Technical University of Ukraine KPI, Faculty of Electronics. Lecturer (Probabilistic Methods of Data Analysis, Measurement in Electronic Systems).

Oct 2012 – Aug 2013. Intervale Group. Senior Vice President, Global Business Development.

Oct 2008 – June 2013. RM-Techno. Organizational Psychologist (Part-time)

Jan 1999 – Mar 2012. Oberthur Technologies. Regional Business Director.

Sep 2010 – Jan 2011. National Technical University of Ukraine KPI, Faculty of Electronics. Lecturer (Part-time; Mathematical Statistics)

Aug 2008 – Dec 2010. Intervale Group. Independent Board Director.

Apr 1997 – Jan 1999. De La Rue International. Country Manager.

June 1996 – Apr 1997. Oracle Corporation. Sales Manager.

May 1994 – June 1996. Unisys Corporation. Sales Manager.

Community Work

Sep 2009 – Present. Kyiv International School. Advisory Board Member

Peer-Reviewed Publications

Chadyuk, A. V., & Chadyuk, V. A. (2009). Teaching students to work in a distributed research team. *Electronics & Communications, 6*(53), 76-80. Available from http://www.nbuv.gov.ua/Portal/natural/eis/2009_6/11_Chaduyk.pdf