

The more sleep – the better the physical state?

Benedikt Gasser^{a,*}

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^a Swiss Health & Performance Lab, Institute of Anatomy, University of Bern, Hochschulstrasse 6, 3012 Bern, Switzerland.

*Corresponding Author: Ph: +41 31 631 84 68; Email: benedikt.gasser@yahoo.com

Abstract: Studies focusing on effects of sleep on physical performance respectively running are sparse. In the younger past knowledge evoked that sleep debt has many effects on dietary intake and predisposes adiposity or diabetes. It is suggested that the current obesity epidemic is also caused by sleep debt which influences endocrine homeostasis (Leptin, Ghrelin, Neuropeptide Y, galanin, orexin, and insulin homeostasis) and affects eating behavior in consequence. Especially glucose homeostasis is strongly coupled with endurance capacity respectively running performance. Sleep debt goes in with a dysregulation of the Melatonin system affecting ACTH-Cortisol Homeostasis while having effects on running performance. Running has positive effects on sleep quality but the counter mechanism that good sleep improves running performance is only partly elucidated. However, sufficient sleep is of highest importance for improving personal best times and hard training days are only possible with adequate sleep. When looking forward newest findings indicate that hard training days go in with a reduced sleep efficiency underlying the complex mechanism remaining to be elucidated.

Key Words: Running, Performance, Sleep, Obesity



Benedikt Gasser is a Medical Doctor and Biostatistician mainly interested in endurance sports especially running and orienteering and alpinism. His recent research focused mainly of requirements of cardiovascular system during mountaineering. Besides, he focused on skeletal muscle

especially eccentric contraction and their respective usage in cardio vascular rehabilitation.

Introduction

Humans have exceptional capabilities to run long distances in hot, arid conditions [1, 2]. These abilities, unique among primates and rare among mammals, derive from a suite of specialized features that permit running humans to store and release energy effectively in the lower limb, help keep the body's center of mass stable and overcome the

thermoregulatory challenges of long distance running. Human endurance running performance capabilities compare favorably with those of other mammals and probably emerged sometime around 2 million years ago in order to help meat-eating hominids compete with other carnivores [1,2]. From these evolutionary developments may results hints that exercise is really the best medicine while having lots of protective effects on different organ systems [3-6]. Having the other side of the coin of well-trained runner's obesity can be mentioned. An impressive and still growing number of studies have noted the concomitant increased incidence of obesity with decreased amount of sleep in the population over the last 40 years [7]. Evidence suggests that biological mediators of appetite and energy homeostasis may be affected by sleep duration [7]. One principle mechanism of sleep regulation is over glandula pinealis by the Hormone Melatonin, which is secreted in darkness [8]. Besides that the CRH-ACTH System with the Cortisol Awakening response

is to mention [8]. Further, hormones such as Leptin, Ghrelin, Neuropeptide Y, Galanin and Orexin must be considered. It's a broad elucidated fact, that Cortisol affects glucose homeostasis having potential effects on habitus and therefore endurance performance [9-11]. Furthermore, the current obesity epidemic is also caused by sleep debt which influences endocrine homeostasis (Leptin, Ghrelin, Neuropeptide Y, insulin homeostasis) and affects eating behavior in consequence. Especially glucose homeostasis is strongly coupled with the endurance capacity respectively running performance.

Evidence Acquisition

There is emerging evidence that the effects of sleep deprivation on feeding may be related, in part, to changes in appetite and energy-regulating peptides, though the evidence is still slender and sometimes contradictory [7]. Focusing on the relationship of sleep debt and physical activity obesity as a result of a lack of physical activity can be mentioned. There is emerging evidence that the effects of sleep deprivation on feeding may be related partly to changes in appetite and energy-regulating peptides, however evidence is still slender and sometimes contradictory [7]. In principle, sleep is regulated by different Hormones: e.g. the Cortisol wakening response is to mention, which yields to rising Cortisol levels in the morning mediated via CRH on Hypothalamus and ACTH on pituitary gland level. [8] Another important branch of endocrine regulation of sleep is Glandula pinealis [8, 12]. Glandula pinealis synthesizes MSH and Melatonin from Serotonin [8,12]. The Production and Release of Melatonin is inhibited by light via retinal stimuli Neuronal connection to the vegetative nerve system is over Nervus opticus, Ncl. Suprachiasmaticus, the thoracic cornu laterale and ganglion cervicale superius [8,12]. The circadian changes of hormone concentration in blood influences the Hypothalamus pituitary Releasing hormones, whereby Melatonin is a strong inhibitor on Gonadoliberin. [8,12] On Hypothalamus level GnRH stimulates FSH and LH on pituitary gland and production of especially

Testosterone in men and Estrogen and Progesterone in female in testes respectively ovary implying broader effects on steroid hormone levels [8,12]. Concerning other parameters regulating appetite different endocrine factors can be mentioned. Relatively clear evidence exists concerning a relationship between Ghrelin, [7,13,14] Leptin [14-16] Galanin [7,17,18] and Orexin [19] Ghrelin is a gut-derived hormone released prior to feeding that stimulates hunger, feeding, and fat storage [7,13]. In rats deprived of sleep for 5 hours, plasma ghrelin levels and food intake were increased in comparison to non-sleep deprived controls [7,14]. Beside Ghrelin Leptin is to mention. Leptin is another metabolic hormone that appears to be affected by sleep deprivation, which is secreted from adipose cells, attenuates food intake and increases energy expenditure [7,15]. Although leptin levels did not change significantly in the aforementioned 5-hour deprivation study in rats [14], another study reported that leptin concentrations were significantly suppressed and feeding was increased after chronic sleep deprivation for 16 days [7,16]. Taken together, these basic studies reveal a potential link between sleep deprivation and systems involved in feeding regulation; sleep deprivation is consistently associated with changes in levels of hormones and peptides that lead to increased feeding that are reversed by sleep [7]. Interestingly, experimental Evidence in Humans Sleep debt exerts profound effects on metabolic hormones. These changes favor not only increased food intake and energy storage, but also potentially the development of diabetes and heart disease. [7,20-24] Young men restricted to 4 hours of sleep a night for 6 days had a 40% decrease in the rate of glucose clearance and a 30% reduction in insulin response and glucose effectiveness, a measurement of the ability of glucose to mediate its own clearance independent of insulin [7,24] . Taken together, these basic studies reveal a potential link between sleep deprivation and systems involved in feeding regulation. To sum up, sleep deprivation is consistently associated with changes in levels of hormones and peptides that lead to

increased feeding that are reversed by sleep. However it must be considered that effects on metabolism in not trained state are different compared to trained athletes [8-10]. Moreover, a dampened diurnal rhythm of leptin effect was also described in a study involving 10 male subjects who were sleep deprived for 88 hours [7, 25]. During partial sleep deprivation, thyroid stimulating hormone levels and the duration of its secretion have also been found to be blunted even when energy intake and activity levels were hold constant [20, 22]. Another explanation proposed to explain the association between sleep debt and obesity relates to general motor activity [7]. It has been suggested that sleep debt causes tiredness, tiredness decreases activity, and decreased activity contributes to weight gain [7, 26]. Sleep apnea and other sleep disorders of breathing produce sleep time restriction and have also been linked to increased occurrence of hypertension, insulin resistance, and recently to increased plasma neuropeptide Y levels, an appetite-stimulating peptide, all independently of body weight [7,27,28].

It appears, however, that the link between sleep debt and obesity may be independent of physical activity [7]. In a sample of 7641 participants of the Finnish 2000 Health Examination Survey, controlling for the level of physical activity did not affect the significant association between sleep duration and obesity [7,27]. Moreover, a recent study reported that women that reported being short sleepers were more likely to have gained weight than those than self-reported to be longer sleepers, independent of levels of physical activity [7,30]. From animal models some astonishing hints exist. The most robust effect of sleep deprivation in rodents is an increase in food intake without a corresponding increase in body weight [7,31]. Other effects were shown also on biochemical level acute sleep deprivation reduced insulin sensitivity in skeletal muscle in overexpressed male mice [32]. Weight loss is likely an effect of chronic and extensive sleep deprivation and/or the methods used to produce it [7] One exception to this pattern of

weight loss was reported in a study of short-term sleep restriction in which rats had access to a carbohydrate-rich diet; following 72 hours of rapid eye movement (REM) sleep deprivation, rats gained weight [7, 33]. Sleep deprivation for a period of 72 hrs produced an increase in day, night and 24 hrs intakes of Carbohydrate Rich diet and Total diet and consequently Body weight was also increased [33]. Sleep disturbances are associated with various metabolic diseases such as hypertension and diabetes [34]. Glucose intolerance with hyperglycemia resulted, although plasma insulin levels and body weight increases were identical between control and chronic sleep debt mice. Findings from animal models suggest that Chronic Sleep Deprivation impair glucose tolerance by inducing gluconeogenesis and suppressing glycolysis.

As initially mentioned, effects of physical activity on sleep quality are proven [35], but the contradictory is hardly elucidated. Interestingly, for a large sample of 17,184 participants aged 18 years or older from rural areas of China at baseline from 2007 to 2008 and followed up from 2013 to 2014 were analyzed concerning their sleep pattern. During 6-year follow-up, Liu et al. (2006) identified 1,101 deaths, whereby the multivariable-adjusted mortality risk was higher with short-duration sleepers (< 6.5 hours) and long-duration sleepers (\geq 9.5 hours) versus 6.5-7.5 hours. The multiplicative interaction of long sleep duration with some lifestyle risk factors and health statuses increased the mortality risk in men and women (low level of physical activity; hypertension; type 2 diabetes mellitus). Sleep duration could be a predictor of all-cause mortality and its interaction with physical activity, hypertension, and T2DM [36].

Further studies investigate the relationship between sleep duration or sleep quality and the risk of type 2 diabetes [37]. Total 563 patients without diabetes who received \geq 1 year follow-up examination were included in the analysis. The median follow-up period was 2.5 years. Poor sleep quality was associated with a higher risk of diabetes after adjusting for age, sex, body mass index, income,

physical activity, and family history of diabetes [37]. As a risk factor for the development of diabetes, poor sleep quality may independently increase the incidence of diabetes. To sum up, Obesity and diabetes are becoming a huge medical problem, whereby a relationship with sleep debt seems to exist [7, 38-42] Reasons for this increase are incompletely understood [7, 43-45].

McAllister et al. (2009) showed a relationship between fewer hours of sleep per day (sleep debt) and the incidence of obesity from 1960 to present. As average daily sleep times have decreased, the incidence of obese adults has increased [7] Hints can be derived by different studies [7, 46-52].

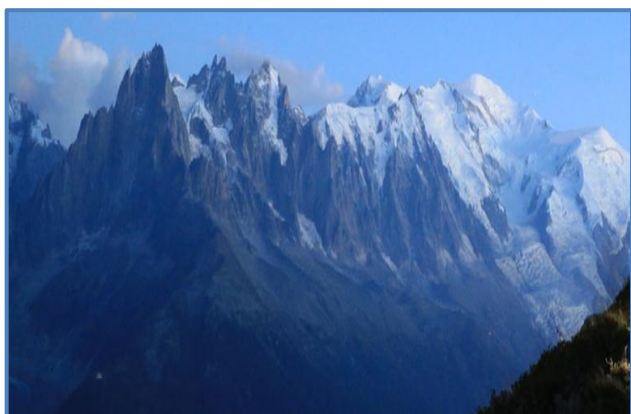


Figure 1. The Ultra-Trail du Mont-Blanc with 170km distance and 10000 meters to climb requires a special sleep management in order to successfully complete the race.

Interestingly from Ultramarathon races some hints exist. The relationship between sleep strategies and performance during the Ultra-Trail du Mont-Blanc 2013 was analyzed, to test the hypothesis that sleep management can influence athletic performance [54]. Almost all runners specifically adopted sleep management strategies before the race [54]. Among the finishers 72% didn't sleep at all during the race and 28% took at least one break for sleep [54]. Non-sleepers completed the race significantly faster than the sleepers. Race time was significantly positively correlated with drowsiness and significantly negatively correlated with the number participations in this race. Runners who adopted a sleep management strategy based on

increased sleep time before the race completed the race faster. Most finishers seemed to be aware of the importance of developing sleep management strategies and increasing sleep time some nights before the race appeared to be the most relevant strategy to improve performance.

Finally, also the effect that athletes know from their personal experience respectively their body sense that they are after a hard training or a competition too exhausted to sleep, was recently elucidated. Sleep efficiency in Normal Training was higher while during intensified training [55] Percentage sleep time fell during intensified training despite an increase in time in bed [55]. To sum up, 9-days of intensified training in highly-trained cyclists resulted in significant and progressive declines in sleep quality, mood state and maximal exercise performance.

Results and Conclusions

As Cicero once wonderful mentioned it is exercise alone that supports the spirits and keeps the mind in vigor (Cicero 106 BC to 43 BC) from a more modern perspective it is also adequate recovery and especially sleep that supports [56]. As initially mentioned exercise is really medicine [1-6] But the other side of the coin seems also to be true: adequate and sufficient sleep supports positive effects of physical activity and even alone adequate sleep and therefore regeneration is important for mind and other organ systems [56].

Taken the information together, evidence concerning effects of sleep on running performance have to be taxed as sparse or just don't exist. What has been analyzed are the effects of physical activity on sleep. Analyses reveal that acute exercise has small beneficial effects on total sleep time, sleep onset latency, sleep efficiency, stage 1 sleep, and slow wave sleep, a moderate beneficial effect on wake time after sleep onset, and a small effect on rapid eye movement sleep [57]. Regular exercise has small beneficial effects on total sleep time and sleep efficiency, small-to-medium beneficial effects on

sleep onset latency, and moderate beneficial effects on sleep quality. Effects were moderated by sex, age, baseline physical activity level of participants, as well as exercise type, time of day, duration, and adherence [57]. Significant moderation was not found for exercise intensity, aerobic/anaerobic classification, or publication date.

However, evidence concerning effects of sleep debt on metabolic diseases are relatively broadly discussed implying an effect on the risk to develop diabetes or hypertonia or in general increasing risk of developing chronic diseases the counter mechanism of sleep on running performance is hardly elucidated [7, 58, 59]. In principle, sleep follows a pattern of first four phases of in sleep followed by phases of REM sleep, whereby the first is getting shorter and the last are getting longer during the night. Different hormones regulate the circadian rhythmic such as Melatonin or Cortisol [8]. Especially Cortisol affects Glucose metabolism and as a consequence nutritional intake. These effects interact with several Hormones regulating food intake such as Ghrelin, Leptin, Galanin, Orexin or Neuropeptid Y. These Hormones regulate food intake, whereby effects concerning the interaction process of sleep debt, food intake and physical activity are somehow controversy discussed and not finally elucidated. What has to be mentioned for the relationship for sleep and physical activity is even truer concerning the relationship of sleep and running performance. Well conducted studies analyzing the effects of running hardly exist, in contrast to general recommendations clearly stating the important aspect of sleep for e.g. marathon performance [8-10]. Especially, it was mentioned that during high training volume more sleep is needed allowing the body to recover faster [8-10] However, these effects remain vague due to lack of serious evidence. The interactions of the different partners influencing performance on different organ systems and its interaction with the different signaling pathway probably influenced by sleep debt remain to be elucidated. Thereby, a mechanistic understanding of all network players influencing human physical

activity performance is out of view, whereby only aspects e.g. of some main players (sleep debt on cortisol homeostasis) are described. We understand sleep on a descriptive level with our ability to analyze EEG, EOG or EMG patterns during sleep a complex understanding of all network players with its topologic impact is out of view. Future research with a strong focus on modeling interactions will allow understanding the mechanism better allowing deepening our knowledge of sleep on performance.

Practical Implications

From a practical point of view, e.g. runners that want to crack the 3-hour limit in marathon racing normally have to consider, that besides other important aspects of preparation more sleep is required [9-11]. If you are constantly short in sleep, you cannot well perform the marathon. - Elite training of more than 130 km per week are only possible with adequate nutrition and more sleep. - It sounds trivial but you recover while sleeping. Overnight energy system, enzymes, neurotransmitter and musculature is restored and repaired. - A lot of elite runners also take a nap after lunch being well rested for the afternoon training. - To sum up, try to sleep right, sufficient and adequate assuring not being disturbed during the night.

References

- [1] Lieberman, E. Daniel, and Dennis M. Bramble, The evolution of marathon running: Capabilities in humans, *Sports Medicine*, 37 (2007) 288290.
- [2] D.E. Lieberman, 2015, Human Locomotion and Heat Loss: An Evolutionary Perspective, *Comprehensive Physiology*, 5, Pp. 99-117
- [3] H.H. Hoppeler, Molekulare Mechanismen der Anpassungsfähigkeit der Skelettmuskulatur. *Schweizerische Zeitschrift für «Sportmedizin und Sporttraumatologie»* 59 (2011) 6–13.
- [4] P. Hofmann, Cancer and Exercise: Warburg Hypothesis, Tumour Metabolism and High-Intensity Anaerobic Exercise, *Sports (Basel)*,6 (2018) E10. doi: 10.3390/sports6010010 .

- [5] B.K. Pedersen, B. Saltin, Evidence for prescribing exercise as therapy in chronic disease, *Scandinavian Journal of Medicine & Science in Sports*, 16 (2006) 3-63.
- [6] M.V. Chakravarthy, F.W. Booth, Eating, exercise, and "thrifty" genotypes: connecting the dots toward an evolutionary understanding of modern chronic diseases, *Journal of Applied Physiology*, 2004; 96 (2004) 3-10.
- [7] E.J. McAllister, N.V. Dhurandhar, S.W. Keith, L.J. Aronne, J. Barger, M. Baskin, R.M. Benca, J. Biggio, M.M. Boggiano, J.C. Eisenmann, M. Eloheid, K.R. Fontaine, P. Gluckman, E.C. Hanlon, P. Katzmarzyk, A. Pietrobelli, D.T. Redden, D.M. Ruden, C. Wang, R.A. Waterland, S.M. Wright, D.B. Allison, Ten Putative Contributors to the Obesity Epidemic, *Critical Reviews in Food Science and Nutrition*, 49 (2009) 868-913.
- [8] Schmidt & Thews. *Physiologie des Menschen*. Springer: Heidelberg, Berlin.
- [9] Steffny H. *Optimales Lauftraining*. München: Südwestverlag; 2010
- [10] Steffny H. *Das grosse Laufbuch*. München: Südwestverlag; 2008
- [11] Zintl F. (1997). *Ausdauertraining: Grundlagen, Methoden, Trainingssteuerung*. (4. Aufl.). Wien; Zürich: BLV.
- [12] Sidler, D. (2001). *Endocrinology*. Sidlerscripts. University of Berne.
- [13] A.J. Van der Lely, M. Tschöp, M.L. Heiman, E. Ghigo, Biological, physiological, pathophysiological, and pharmacological aspects of ghrelin, *Endocrine Reviews*, 25 (2004) 426-457.
- [14] B. Bodosi, J. Gardi, I. Hajdu, E. Szentirmai, Obal F Jr, J.M. Krueger, Rhythms of ghrelin, leptin, and sleep in rats: effects of the normal diurnal cycle, restricted feeding, and sleep deprivation, *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, 287 (2004) R1071-1079.
- [15] J.L. Halaas, K.S. Gajiwala, M. Maffei, S.L. Cohen, B.T. Chait, D. Rabinowitz, R.L. Lallone, S.K. Burley, J.M. Friedman, Weight-reducing effects of the plasma protein encoded by the obese gene, *Science* 269 (1995) 543-546.
- [16] C.A. Everson, W.R. Crowley, Reductions in circulating anabolic hormones induced by sustained sleep deprivation in rats, *American Journal of Physiology-Endocrinology and Metabolism*, 286 (2004) E1060-1070.
- [17] S.E. Kyrkouli, B.G. Stanley, S.F. Leibowitz, Galanin: stimulation of feeding induced by medial hypothalamic injection of this novel peptide, *European Journal of Pharmacology*, 122 (1986) 159-160
- [18] X. Lu, A.M. Barr, J.W. Kinney, P. Sanna, B. Conti, M.M. Behrens, T. Bartfai, A role for galanin in antidepressant actions with a focus on the dorsal raphe nucleus, *Proceedings of the National Academy of Sciences of the United States of America*, 102 (2005) 874-879.
- [19] M. Pedrazzoli, V. D'Almeida, P.J. Martins, R.B. Machado, L. Ling, S. Nishino, S. Tufik, E. Mignot, Increased hypocretin-1 levels in cerebrospinal fluid after REM sleep deprivation, *Brain Research*, 995 (2004) 1-6.
- [20] K. Spiegel, R. Leproult, M. L'hermite-Baleriaux, G. Copinschi, P.D. Penev, E. Van Cauter, Leptin levels are dependent on sleep duration: relationships with sympathovagal balance, carbohydrate regulation, cortisol, and thyrotropin, *The Journal of Clinical Endocrinology and Metabolism*, 89 (2004) 5762-5771.
- [21] B. Bjorvatn, I.M. Sagen, N. Oyane, S. Waage, A. Fetveit, S. Pallesen, R. Ursin, The association between sleep duration, body mass index and metabolic measures in the Hordaland Health Study, *Journal of Sleep Research*, 16 (2007) 66-76.
- [22] J. Flint, S.V. Kothare, M. Zihlif, E. Suarez, R. Adams, A. Legido, F. De Luca, Association between inadequate sleep and insulin resistance in obese children, *The Journal of Pediatrics*, 150 (2007) 364-369.

- [23] K.L. Knutson, K. Spiegel, P. Penev, E. Van Cauter, The metabolic consequences of sleep deprivation, *Sleep Medicine Reviews*, 11 (2007) 163–178.
- [24] M.I. Trenell, N.S. Marshall, N.L. Rogers, Sleep and metabolic control: waking to a problem?, *Clinical and Experimental Pharmacology and Physiology*, 34 (2007) 1–9.
- [25] E. Van Cauter, U. Holmback, K. Knutson, R. Leproult, A. Miller, A. Nedeltcheva, S. Pannain, P. Penev, E. Tasali, K. Spiegel, Impact of sleep and sleep loss on neuroendocrine and metabolic function, *Hormone Research*, 67(Suppl 1) (2007) 2–9.
- [26] J.M. Mullington, J.L. Chan, H.P. Van Dongen, M.P. Szuba, J. Samaras, N.J. Price, H.K. Meier-Ewert, D.F. Dinges, C.S. Mantzoros, Sleep loss reduces diurnal rhythm amplitude of leptin in healthy men, *Journal of Neuroendocrinology*, 15 (2003) 851–854.
- [27] S. Taheri, Sleep and metabolism: bringing pieces of the jigsaw together, *Sleep Medicine Reviews*, 11 (2007) 159– 162.
- [28] E. Tasali, E. Van Cauter, Sleep-disordered breathing and the current epidemic of obesity: consequence or contributing factor?, *American Journal of Respiratory and Critical Care Medicine*, 165 (2002) 562–563.
- [29] A. Barcelo, F. Barbe, E. Llompart, M. de la Pena, J. Duran-Cantolla, A. Ladaria, M. Bosch, L. Guerra, A.G. Agusti, Neuropeptide Y and leptin in patients with obstructive sleep apnea syndrome: Role of obesity, *American Journal of Respiratory and Critical Care Medicine*, 171 (2004) 183-187.
- [30] M. Fogelholm, E. Kronholm, K. Kukkonen - Harjula, T. Partonen, M. Partinen, M. Härmä, Sleep-related disturbances and physical inactivity are independently associated with obesity in adults, *International Journal of Obesity*. 2007 Jun 19
- [31] S.R. Patel, A. Malhotra, D.P. White, D.J. Gottlieb, F.B. Hu, Association between reduced sleep and weight gain in women, *American Journal of Epidemiology*, 164 (2006) 947–954.
- [32] A. Rechtschaffen, B.M. Bergmann, C.A. Everson, C.A. Kushida, M.A. Gilliland, Sleep deprivation in the rat: X. Integration and discussion of the findings, *Sleep* 25 (1989) 68–87.
- [33] A.J. Brager, L. Heemstra, R. Bhambra, J.C. Ehlen, K.A. Esser, K.N. Paul, C.M. Novak, Homeostatic effects of exercise and sleep on metabolic processes in mice with an overexpressed skeletal muscle clock, *Biochimie.* 2017 Jan;132 (2017) 161-165.
- [34] J.L. Bhanot, G.S. Chhina, B. Singh, U. Sachdeva, V.M. Kumar, REM sleep deprivation and food intake, *Indian Journal of Physiology and Pharmacology*, 33 (1989) 139–145.
- [35] K. Oishi, S. Ohyama, S. Higo-Yamamoto, Chronic sleep disorder induced by psychophysiological stress induces glucose intolerance without adipose inflammation in mice, *Biochemical and Biophysical Research Communications*, 495 (2018) 2616-2621.
- [36] I. Hartescu, K. Morgan, C.D. Stevinson, Increased physical activity improves sleep and mood outcomes in inactive people with insomnia: a randomized controlled trial, *Journal of Sleep Research*, 24(2015) 526-534.
- [37] F. Liu, H. Zhang, Y. Liu, X. Sun, Z. Yin, H. Li, K. Deng, Y. Zhao, B. Wang, Y. Ren, L. Zhang, J. Zhou, C. Han, X. Liu, D. Zhang, G. Chen, S. Hong, C. Wang, D. Hu, M. Zhang, Sleep Duration Interacts With Lifestyle Risk Factors and Health Status to Alter Risk of All-Cause Mortality: The Rural Chinese Cohort Study, *Journal of Clinical Sleep Medicine*, 2018 Apr 30. pii: jc-17-00515.
- [38] J.A. Lee, S. Sunwoo, Y.S. Kim, B.Y. Yu, H.K. Park, T.H. Jeon, B.W. Yoo, The Effect of Sleep Quality on the Development of Type 2 Diabetes in Primary Care Patients, *Journal of Korean Medical Science*, 31 (2016) 240-246.
- [39] M.L. Baskin, J. Ard, F. Franklin, D.B. Allison, Prevalence of obesity in the United States, *Obesity Reviews*, 6 (2005) 5– 7.

- [40] C. Gubelmann, R. Heinzer, J. Haba-Rubio, P. Vollenweider, P. Marques-Vidal, Physical Activity is Associated with Higher Sleep Efficiency in the General Population: The CoLaus Study, *Sleep*, 2018 Mar 30. doi: 10.1093/sleep/zsy070.
- [41] C.L. Ogden, M.D. Carroll, K.M. Flegal, High body mass index for age among US children and adolescents, 2003–2006, *JAMA* 299 (2008) 2401–2405.
- [42] LA. Helmchen, R.M. Henderson, Changes in the distribution of body mass index of white US men, 1890– 2000, *Annals of Human Biology*, 31 (2004) 174–181.
- [43] B. Caballero, The global epidemic of obesity: an overview, *Epidemiologic Reviews*, 29 (2007) 1–5.
- [44] A.V. Astrup, S. Rossner, T.I. Sorensen, Alternative causes of obesity, *Ugeskr Laeger*, 168 (2006) 135–137.
- [45] G.A. Bray, C.M. Champagne, Beyond energy balance: there is more to obesity than kilocalories, *Journal of The American Dietetic Association*, 105 (2005) S17–23.
- [46] J.C. Eisenmann, Insight into the causes of the recent secular trend in pediatric obesity: Common sense does not always prevail for complex, multi-factorial phenotypes, *Preventive Medicine*, 42 (2006) 329–335.
- [47] L. Terman, A. Hocking, The sleep of school children, its distribution according to age, and its relationship to physical and mental efficiency, *Journal of Educational Psychology*, 4 (1913) 269–282.
- [48] A. McGhie, S.M. Russell, The Subjective Assessment of Normal Sleep Patterns, *Journal of Mental Science*, 108 (1962) 642– 654
- [49] E.C. Hammond Some preliminary findings on physical complaints from a prospective study of 1,064,004 men and women. *American Journal of Public Health Nations Health*, 54 (1964) 11–23.
- [50] G. Tune, Sleep and wakefulness in normal human adults. *British Medical Journal*, 2 (1968) 269–271.
- [51] C.D. Palmer, G.A. Harrison, R.W. Hiorns, Sleep patterns and life style in Oxfordshire villages, *Journal of Biosocial Science*, 12 (1980) 437–467.
- [52] R.A. Hicks, C. Fernandez, R.J. Pellegrini, Self-reported sleep durations of college students: normative data for 1978–79, 1988–89, and 2000–01, *Perceptual and Motor Skills*, 93 (2001) 139–140.
- [53] N.M. Punjabi, K. Bandeen-Roche, T. Young, Predictors of objective sleep tendency in the general population, *Sleep* 26 (2003) 678–683.
- [54] Mont Blanc ultrarace, Download on 1. June 2018 from <http://utmbmontblanc.com/en/page/.html>
- [55] M. Pousset, J. Laroppe, R. Hurdiel, J. Girard, L. Poletti, C. Thil, A. Didelot, B. Chenuel, Sleep Management Strategy and Performance in an Extreme Mountain Ultra-marathon, *Research in Sports Medicine*, 23 (2015) 330-336.
- [56] S.C. Killer, I.S. Svendsen, A.E. Jeukendrup, M. Gleeson, Evidence of disturbed sleep and mood state in well-trained athletes during short-term intensified training with and without a high carbohydrate nutritional intervention, *Journal of Sports Sciences*, 35 (2017) 1402-1410.
- [57] D.K. Ehlers, J. Fanning, E.A. Salerno, S. Aguiñaga, J. Cosman, J. Severson, A.F. Kramer, E. McAuley, Replacing sedentary time with physical activity or sleep: effects on cancer-related cognitive impairment in breast cancer survivors, *BMC Cancer*, 18 (2018) 685.
- [58] M.A. Kredlow, M.C. Capozzoli, B.A. Hearon, A.W. Calkins, M.W. Otto, The effects of physical activity on sleep: a meta-analytic review, *Journal of Behavioral Medicine*, 38 (2015) 427-449.
- [59] N. Kalak, M. Gerber, R. Kirov, T. Mikoteit, J. Yordanova, U. Pühse, E. Holsboer-Trachsler, S. Brand, Daily morning running for 3 weeks improved sleep and psychological functioning

in healthy adolescents compared with controls,
Journal of Adolescent Health, 51 (2012) 615-
622.

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Competing Interests

The authors declare that they have no competing interests.

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