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Sleep: a serious contender for the prevention of obesity and non-communicable diseases

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Sleep — how seriously do we need to take it?

There is a perception that time spent asleep is time wasted. Anecdotally one hears of people sleeping no more than 3–4 h per night with no apparent ill effects. As tempting as a short sleep regime sounds, we know that sleep is critical for survival. In 1983 Rechtschaffen and colleagues showed that rats developed pathology and died within 14 to 21 days of total sleep deprivation.¹ Data accumulated over the past 40 years from prospective cohort studies indicate higher all-cause mortality rates among people who sleep either less than 6 h or more than 9 h per night.² We also know that sleep loss affects neurobehavioural performance, metabolism and obesity, and psychological health.³

How much sleep do we need?

The latest guidelines published by the National Sleep Foundation in the US recommend that adults (18–64 y) obtain 7–9 h of sleep per night, teenagers (14–17 y) 8–10 h per night and school-aged children (6–13 y) 9–11 h per night.⁴ Recognising the inter-individual variability in sleep need, the authors suggest that for some adults as little as 6 h may suffice, while others may require 10–11 h. Genetic factors are significant determinants of inter-individual sleep requirements.⁵ Age and gender are also important. For example, sleep duration tends to decrease with age, and females tend to sleep longer and suffer more from lack of sleep than males.⁶ Technically, 'short sleep' is defined as < 6 h per night, but it is important to understand that short sleep is not the same as 'sleep insufficiency'.³ While for some people 5–6 h per night might suffice, it may be insufficient for others, resulting in the accumulation of sleep debt, which may in turn lead to chronic health and behavioural problems. Identifying people at risk for or suffering from sleep insufficiency is key.

How much sleep are we getting?

Youngstedt et al.⁷ reviewed data from 168 studies around the world, representing 6 052 individuals, in whom sleep was measured objectively, i.e. with polysomnography or actigraphy. These collective data from studies conducted between 2010 and 2015 found total sleep time to range between 5.8 and 7.8 h per night.⁷ While this is no more or less than that reported between 1970 and 2010,⁷ it is clear that the majority of these individuals would not meet the current sleep guidelines.⁴

Sleep data for South Africans specifically are limited. One study has objectively measured sleep in 9-year-old to 11-year-old South African children from a range of ethnic and socioeconomic backgrounds using a hip-worn accelerometer. Total sleep time of the girls was 9.2 ± 0.8 h per night ($n = 262$) and 9.1 ± 0.7 h for the boys ($n = 162$).⁸ As a group these children are at the lower end of

the recommended range for school-aged children. Concerning, though, is that one third sleep < 9 h per night and as such do not meet the guidelines.⁴ In a recent survey,⁹ 825 South African teenagers reported an average sleep time of 7.2 ± 1.2 h — again less than the recommended amount of nocturnal sleep for adolescents.

While there are not yet any objectively measured sleep data on South African adults, self-report sleep time from 1 311 black Africans (of whom one-third were unemployed) logged total average sleep time as 8.8 ± 1.7 h.¹⁰ While this certainly appears to be sufficient, it is interesting to note that shorter sleep was associated with a higher BMI in females older than 40 years.¹⁰ Another study described the sleep habits of older South African adults (> 50 y), and found that on average 11% sleep < 6 h per night.¹¹ The fact that > 20% Indian, Asian and white respondents sleep < 6 h per night, while almost 30% of the black respondents sleep > 10 h per night, highlights the need to account for and understand ethnic differences in sleep needs and beliefs. Furthermore, given the U-shaped relationship between sleep duration and mortality,² understanding the potential health effects of longer sleep duration observed in the black population may be important.

The link between short sleep and non-communicable diseases

Recent data from the 2014 South African National Health and Nutrition Examination Survey indicate that non-communicable diseases (NCDs) such as cardiovascular disease, cancer, chronic respiratory diseases and diabetes mellitus type 2 (T2DM) are increasing in prevalence, and now account for more than a third (37%) of all deaths in South Africa.¹² Given that one of the key risk factors for T2DM and cardiovascular disease is obesity, it is alarming that 25% of South African females are overweight and 39% are obese.¹² Lifestyle modification is one of the ways in which prevention of obesity and NCDs is currently addressed. Specifically, sedentary behaviour, smoking, poor diet and alcohol abuse are targeted as modifiable behaviours likely to reduce obesity and the risk for NCDs. We propose that good sleep hygiene be added to this approach.

The reason for concern around insufficient, poor quality sleep is that there appears to be a vicious cycle between reduced sleep duration, a disrupted circadian rhythm and adverse cardiometabolic consequences. There is now compelling evidence suggesting that both short and long sleep are associated with obesity, T2DM, hypertension and cardiovascular disease.¹³ One study exploring the mechanisms of metabolic changes resulting from sleep curtailment observed that when

individuals were restricted to 5.7 h of sleep per night for one week, the expression of more than 700 genes was altered, including those associated with circadian rhythms, metabolism, immune and stress responses.¹⁴

Knutson et al.¹⁵ proposed a model to explain how sleep loss may lead to the development of obesity and T2DM. Sleep loss results in increased sympathetic nervous system activity as well as increased levels of evening cortisol, night-time growth hormone and pro-inflammatory cytokines, all of which contribute to increased insulin resistance and decreased glucose tolerance. Concomitantly, sleep loss increases orexin and ghrelin levels (hunger hormones) and decreases leptin levels (satiety hormone). Thus with an increased appetite and more time to eat, energy intake may increase above energy demands, contributing to weight gain.¹⁵

Conclusions

There is a significant body of evidence linking short sleep with the development of obesity and T2DM. We note, however, that much of this research is based on individuals living in industrialised societies. In order to determine whether this is relevant in the South African context, there is an urgent need for objectively measured sleep data in South Africans from a range of cultural and socioeconomic backgrounds. These data then need to be interrogated for relationships with obesity and markers of NCDs such as BMI, glucose tolerance, insulin sensitivity, blood pressure and blood lipid profile. This in turn may be used to inform guidelines and intervention strategies aimed at optimising the sleep duration and quality with the overall goal of reducing the prevalence of obesity and NCDs in South Africans.

References

1. Rechtschaffen A, Gilliland MA, Bergmann BM, et al. Physiological correlates of prolonged sleep deprivation in rats. *Science*. 1983;221:182–184.
2. Grandner MA, Hale L, Moore M, et al. Mortality associated with short sleep duration: the evidence, the possible mechanisms, and the future. *Sleep Med Rev*. 2010;14:191–203.
3. Grandner MA, Patel NP, Gehrman PR, et al. Problems associated with short sleep: bridging the gap between laboratory and epidemiological studies. *Sleep Med Rev*. 2010;14:239–247.
4. Hirshkowitz M, Whiton K, Albert SM, et al. National sleep foundation's sleep time duration recommendations: methodology and results summary. *Sleep Health*. 2015;1:40–43.
5. Gottlieb DJ, O'Connor GT, Wilk JB. Genome-wide association of sleep and circadian phenotypes. *BMC Med Genet*. 2007;8:S9.
6. Porkka-Heiskanen T, Zitting KM, Wignen HK. Sleep, its regulation and possible mechanisms of sleep disturbances. *Acta Physiol*. 2013;208:311–328.
7. Youngstedt SD, Goff EE, Reynolds AM, et al. Has adult sleep duration declined over the last 50+ years? *Sleep Med Rev*. 2015;28:65–81.
8. Katzmarzyk PT, Barreira TV, Broyles ST, et al. Relationship between lifestyle behaviors and obesity in children ages 9–11: results from a 12-country study. *Obesity*. 2015;23:1696–1702.
9. Reid A, Maldonado CC, Baker FC. Sleep behavior of South African adolescents. *Sleep*. 2002;25:423–427.
10. Pretorius S, Stewart S, Carrington MJ, et al. Is there an association between sleeping patterns and other environmental factors with obesity and blood pressure in an urban African population? In: M Buchowski (editor). *PLoS ONE* 2015;10:e0131081.
11. Peltzer K. Sociodemographic and health correlates of sleep problems and duration in older adults in South Africa. *S Afr J Psych*. 2012;18:150–156.
12. Shisana O, Labadrios D, Rehle T, et al. The South African National Health and Nutrition Examination Survey (SANHANES-1). Cape Town: HSRC Press; 2013. p. 1–423.
13. Buxton OM, Marcelli E. Short and long sleep are positively associated with obesity, diabetes, hypertension, and cardiovascular disease among adults in the United States. *Soc Sci Med*. 2010;71:1027–1036.
14. Moller-Levet CS, Archer SN, Bucca G, et al. Effects of insufficient sleep on circadian rhythmicity and expression amplitude of the human blood transcriptome. *Proc Natl Acad Sci USA*. 2013;110:E1132–E1141.
15. Knutson KL, Van Cauter E. Associations between sleep loss and increased risk of obesity and diabetes. *Ann NY Acad Sci*. 2008;1129:287–304.

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