pii: jc-18-00718 https://dx.doi.org/10.5664/jcsm.7696

JCSM
Journal of Clinical Sleep Medicine

LETTERS TO THE EDITOR

Obstructive Sleep Apnea, Insomnia, and Snoring: Evolutionary Adaptations to Increase Alertness During Sleep?

Liam Holley, BE

Bella Vista, NSW, Australia

The nature of sleep differs greatly among species. It has been hypothesized that evolutionary adaptation to ecological factors contributes significantly to these differences.[1] In endotherms, the risk of predation, energy conservation, and wakeful functioning may be conflicting influences in shaping sleep. Take the examples of the koala and giraffe. Two leaf-eating mammals with very different patterns. The koala sleeps safe in a tree for 14 hours a day. The giraffe's habitat on the plains is rich with predators. It usually sleeps upright for around half an hour a day, in short segments, and with ears twitching, sensing danger. The giraffe is an extreme example. A study of 39 mammal species found the amount of REM sleep correlated inversely with predatory danger.[2]

Communal living offers added safety. Such is the situation for human evolution. Sleeping times and durations vary between individuals. A relatively small group increases the likelihood that individuals will be awake to notice danger. Arousability to stimulus also varies. This could mean that while the group as a whole gains benefits of deeper sleep in some, it also profits from the increased alertness of light sleepers.

Many conditions associated with stress are also linked to obstructive sleep apnea (eg, posttraumatic stress disorder, stroke, hypertension). Could upper airway resistance be a mechanism for increasing arousability that dynamically responds to stress as an indicator for danger? Sériès et al.[3] found repeated waking with auditory stimuli leads to increased breathing abnormalities, and concluded that sleep fragmentation contributes to sleep apnea. However, perhaps stress caused by the alarms triggered an airway response as a mechanism for increasing sensitivity during sleep. Sleep fragmentation may be a byproduct, along with the undesirable comorbidities of obstructive sleep apnea. If stress correlates with danger, airway resistance increasing arousability could be a beneficial survival mechanism. In this way, one trades the increased risk of chronic disease later in life for immediate protection from danger.

Snoring seems like an evolutionary mistake—an announcement of vulnerability. However, with the present reasoning perhaps there is a selfish advantage. The snorer provides auditory stimulus to those around them, fragmenting sleep and increasing alertness of their group, allowing themselves both sleep and protection.

Suppose airway resistance is a mechanical means of increasing alertness, could insomnia be the manifestation of a mental one? It is interesting to hypothesize the pros and cons for each, although they often coexist and comorbidities are largely the same. Perhaps insomnia is more discerning and rapid in response, but with greater psychological cost?

It is hoped that these theories will contribute to the scientific inquiry into the pathogenesis of obstructive sleep apnea, lead to better understanding of the links between comorbidities, and aid the identification of patients who would or would not benefit from therapy.

In light of these proposed theories one wonders if it is coincidental the giraffe draws its breath through such a long tube.

CITATION

Holley L. Obstructive sleep apnea, insomnia, and snoring: evolutionary adaptations to increase alertness during sleep? *J Clin Sleep Med*. 2019;15(3):523.

REFERENCES

1. Siegel JM. Sleep viewed as a state of adaptive inactivity. *Nate Rev Neurosci*. 2009;10(10):747–753.
2. Allison T, Cicchetti DV. Sleep in mammals: ecological and constitutional correlates. *Science*. 1976;194(4266):732–734
3. Sériès F, Roy N, Marc I. Effects of sleep deprivation and sleep fragmentation on upper airway collapsibility in normal subjects. *Am J Respir Crit Care Med*. 1994;150(2):481–485.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication November 20, 2018
Submitted in final revised form November 20, 2018
Accepted for publication January 14, 2019
Address correspondence to: Liam Holley, 1 Elizabeth MacArthur Drive, Bella Vista, NSW, Australia, 2153.

DISCLOSURE STATEMENT

The author is employed full time as a Lead Research Engineer by ResMed Ltd.