The Neuroscience of Bedtime

by Dirk Hanson

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What’s your chronotype? Are you a morning lark or a night owl? The manner in which your brain processes light, an activity known as phototransduction, is responsible for whether you find yourself nodding off in the chair every evening about 8:30, or whether you regularly stay up until the wee hours and sleep as late as you can. If you are such an owl that you have trouble getting to work or school on time and feel groggy most of the day, you might have delayed sleep phase disorder (DSPD). Too early, and it’s advanced sleep phase disorder (ASPD), which is less common. Circadian misalignment of this kind can affect everything from your risk of clinical depression to your chances of cardiovascular disease. It can alter your BMI and play a role in whether or not you smoke cigarettes.

A 2012 study in Annals of Neurology found that variations in the PER1 and PER2 genes, members of a gene group responsible for circadian rhythms, were strongly involved in determining who rises early and who rises late. Genome-wide association studies undertaken using data from the UK Biobank and from the genetics company 23andMe also support the conclusion of a genetic origin for delayed and advanced sleep phase disorders.

Our intrinsic 24-hour clock mechanism resides in the brain’s suprachiasmatic nucleus (SCN), a set of neuron clusters in the hypothalamus that receive input from the retina via the optic nerve. Via phototransduction, the amount of light captured by the brain is translated into electrical signals. This system evolved to allow early life forms to accurately peg light and dark cycles caused by the rising and setting of the sun. The SCN also controls the synthesis of melatonin in the pineal gland, a crucial component of wake/sleep cycles. Both larks and owls inherit variations of these genetically determined behaviors that produce something very much like jet lag without the plane ride. Clock-dependent alertness pathways in the brain stay activated later at night in the owls, and give out earlier in the larks. Peak performance times, body temperature cycles, and hormonal rhythms are all canted away from the norm in people with sleep phase disorders.

In a commentary for Proceedings of the National Academy of Sciences (PNAS), Joseph Bass of Northwestern University’s department of medicine wrote that “alignment between behavioral cycles and the light-
The body clock also helps establish our eating cycles, body weight, and glucose levels. Circadian misalignment results in “a decrease in leptin, increase in glucose and insulin, increase in mean arterial blood pressure, and reduced sleep efficiency” according to Frank Scheer and coworkers who authored the PNAS study. Scheer adds that people with disordered body clocks also show mild hypertension, “indicating that over the long term, cumulative cardiovascular risk may increase as a result of circadian misalignment.”

Thus it is not surprising that scientists have raised the possibility that DPSD and depression share common genetic underpinnings. In 2014, a survey in BMC Psychiatry suggested that delayed sleep phase disturbances were twice as common among depressed young people in Australia compared with the population at large. Higher levels of tobacco use were also associated with this group (38 percent compared with the national smoking rate of roughly 17 percent). The researchers called smoking “the only independent predictor of delayed sleep onset.” They also suggest that the onset of DSPD may represent a marker signifying “a circadian pathophysiological profile of depression that may have a different prognosis and response to treatment.”

Indeed, there is additional evidence of a link between delayed sleep phase disorder and seasonal affective disorder (SAD). Night owls are more than three times as likely to report seasonal affective disorder during winter months than normal sleepers, Heon-Jeong Lee and colleagues report in the Journal of Affective Disorders: “DSPD might share similar pathophysiological mechanisms” with seasonal affective disorder (SAD), “since both manifest problems of delayed circadian rhythm phase and both are treated by morning light therapy and melatonin before bedtime.”

This treatment protocol, used for both delayed and advanced sleep disorders as well as for SAD, is a form of chronotherapy, or the deliberate shifting of sleep-wake cycles. Full-spectrum light therapy for two hours in the morning and melatonin treatment in the early evening help advance
the body clock in night owls. Similarly, with early risers, bright light in the evening and melatonin supplements in the morning serve to delay the circadian cycle to align it with normal day-night cycles.

As for morning people, the overall profile is less worrisome, though those with ASPD often have an impaired social life because evening activities conflict with their desire to sleep. In addition, according to work published in *Nature Communications*, genetic correlations suggest that a morning chronotype may share underlying biology with a slightly higher BMI.

Nonetheless, it seems that morning larks present a healthier profile than the owls.

“There is little if any evidence that early morning risers have more depression than the average person,” says Daniel F. Kripke, a sleep researcher and emeritus professor of psychiatry at the University of California, San Diego. “At our sleep clinic, we probably see ten cases of delayed sleep phase for every case with advanced sleep phase.” Sleep patterns of morning people, Kripke notes, resemble those of “an agricultural society without electric light.”

In sum, says Kripke, “people in the ‘morningness’ half of the population do not complain of it much except in unusual or extreme cases.”