Why Can’t We Fall Asleep? by The New Yorker, by Maria Konnikova 07/07/15

This is the first piece in a three-part series on sleep. Read part two, on sleeping and dreaming, and part three, on wakefulness.

Here’s what’s supposed to happen when you fall asleep. Your body temperature falls, even as your feet and hands warm up—the temperature changes likely help the circadian clocks throughout your body to synchronize. Melatonin courses through your system—that tells your brain it’s time to quiet down. Your blood pressure falls and your heart rate slows. Your breathing evens out. You drift off to sleep.

That, at least, is the ideal. But going to sleep isn’t always a simple process, and it seems to have grown more problematic in recent years, as I learned through a series of conversations this May, when some of the world’s leading sleep experts met with me to share their ongoing research into the nature of sleeping. (The meetings were facilitated by a Harvard Medical School Media Fellowship.) According to Charles Czeisler, the chief of the Division of Sleep and Circadian Disorders at Brigham and Women’s Hospital, over the past five decades our average sleep duration on work nights has decreased by an hour and a half, down from eight and a half to just under seven. Thirty-one percent of us sleep fewer than six hours a night, and sixty-nine per cent report insufficient sleep. When Lisa Matricciani, a sleep researcher at the University of South Australia, looked at available sleep data for children from 1905 to 2008, she found that they'd lost nearly a minute of sleep a year. It's not just a trend for the adult world. We are, as a population, sleeping less now than we ever have.

The problem, on the whole, isn't that we're waking up earlier. Much of the change has to do with when we choose to go to bed—and with how we decide to do so. Elizabeth Klerman is the head of the Analytic and Modeling Unit, also in the Sleep and Circadian Disorders division at Brigham and Women's Hospital. Her research tracks how multiple individual differences in our environment affect our circadian rhythms and our ability to fall asleep easily and soundly. “When you go to bed affects how long you can sleep, no matter how tired you are,” she told me.

A wide array of factors can determine just how quickly you’ll be able to drift off to sleep when you choose to do so. To determine their relative importance—and separate the things that truly make a difference from the ones that aren’t all that essential—Klerman first assesses her subjects’ habits and histories: What is their sleep-wake history—that is, what are their usual routines, and what are the problems they’ve encountered in the past? What about pharmaceuticals: Which do they use, and are any targeted as sleeping aids? Once this information has been gathered, she brings them to the lab, monitors their sleep in a controlled environment, and determines how each of the various factors affects the ability to fall asleep.

Part of how easily we go to sleep is genetic: many sleep disturbances, ranging from insomnia to circadian disruption, have a large genetic component. (Elizabeth Kolbert wrote about insomnia for this magazine, in 2013.) If you’re “out of phase” from typical bedtimes due to circadian disruption, for example, your melatonin levels are off: the hormone that should be telling you to fall asleep isn’t produced in enough quantity, or the requisite receptors are missing. While we are still a long way off from fully understanding the precise ways in which genes affect sleep in humans, the neurobiologist Dragana Rogulja, who studies the transition from wakefulness to sleep in Drosophila melanogaster, the fruit fly, has begun to answer that question for other animals. Many sleep genes, she points out, are conserved across species. And the
sleep patterns in the flies, she told me, are remarkably similar to those in humans. One specific mutation in the flies’ genes can lead to a “sleep initiation deficit.” Isolating that gene and tracking its mechanism of action through the flies’ bodies and brains may bring us a step closer to understanding how similar deficits operate in human sleepers.

Even so, however, our genes haven’t really changed in the past century. Genetic predisposition can’t explain why so many of us have started to have more trouble falling asleep. The vast majority of the story has to do with our environment. Good “sleep hygiene,” many researchers have found, is essential when it comes to falling asleep; it can even overcome some unfortunate genetic predispositions. Conversely, bad sleep hygiene can equal, in its effects, some of the most problematic genetic disorders.

Some of the elements of sleep hygiene are basically the same as good health practices. Nicotine, caffeine, and alcohol all negatively impact sleep, the more so the closer they’re consumed to bedtime. We fall asleep faster when we exercise and have regular mealtimes. Eat too late or too much and sleep becomes more elusive. (The effect is reciprocal: sleep disturbance is associated with weight gain.) Go to bed hungry, and sleep likewise escapes your grasp. Any schedule variability, in fact, may detract from sleep ability: in some preliminary results, Rosalind Picard, the director of the Affective Computing Research Group at M.I.T.'s Media Lab and the co-director of the Advancing Wellbeing Initiative, found that sleep variability was one of the most important factors in determining how well someone slept: it was better to go to bed at a consistent time than to try to pull an all-nighter tonight and “catch up” tomorrow. Regular sleep schedules also predict better G.P.A. and mood.

But it may be that the most important aspect of sleep hygiene has to do with light—which, of course, has gotten more pervasive during the past century, especially at night. Humans have evolved to be exquisitely sensitive to the most minute changes in the light around us. In fact, there are specific photoreceptors in the eye that only respond to changes in light and dark, and which are used almost exclusively to regulate our circadian rhythms. These melanopsin receptors connect directly to the part of the brain that regulates our internal body clocks. They work even in many people who are blind: though they can’t see anything else, their bodies still know how to adjust their circadian clocks to stay on schedule. Light helps the body predict the future: it’s a sign of how our environment will change in the coming hours and days, and our bodies prepare themselves accordingly. As the Harvard circadian neuroscientist Steven Lockley told me, “Our clocks have evolved to anticipate tomorrow.”

Now, however, that natural prediction system is being constantly wrong-footed. The problem isn't just artificial light in general. Increasingly, we are surrounded by light on the short-wave, or “blue light,” spectrum—light which our circadian systems interpret as daylight. Blue light emanates from our computers, our televisions, our phones, and our e-readers; ninety per cent of Americans use electronic devices that emit it. When we spend time with a blue-light-emitting device, we are, in essence, postponing the signal to our brain that tells it that it’s time to go to sleep. (“What have we done with our dusk?” Charles Czeisler asks.) When “dusk” gets pushed progressively later because of these false light cues, we get a surge of energy rather than the intended melatonin release.

Czeisler has found that artificial light can shift our internal clocks by four or even six time zones, depending on when we’re exposed to it. In one study, out earlier this year in the journal PNAS, Czeisler and his colleagues asked people to read either a printed book or a light-emitting e-book about four hours before bed, for five evenings in a row. The effects were profound. Those who'd read an e-book released less melatonin and were less sleepy than those who'd read a regular book; their melatonin release was delayed by more than an hour and a half, and their circadian clocks were time-shifted. It took them longer to fall asleep. The next morning, they were less alert. These resetting effects can result not just from prolonged reading but from a single exposure. In his sleep lab, Lockley has seen it happen after exposing
subjects to short-wavelength light for less than twelve minutes.

Many people who can't get to sleep turn to sleeping aids. Unfortunately, existing pharmaceuticals may not be enough to significantly counteract the effects of environmental overstimulation. The sleep we get from existing medications is different from regular sleep, and may not be as effective as we think. Matt Bianchi, the chief of the Sleep Medicine Division and head of the Sleep Informatics Laboratory at the Massachusetts General Hospital, says that people using sleep aids only sleep, on average, thirty to forty minutes more than people who don't use them. And no drugs on the market mimic exactly the natural progression of sleep. Instead, many of them suppress REM and slow-wave sleep and thus, Bianchi has written, may “impair the restorative value of sleep.” Some sleeping aids also come with the risk of parasomnia—behaviors such as eating or leaving the house while asleep, with no subsequent memory of the event in question. And none of these drugs are typically recommended for more than a week at a time. (Ian Parker wrote about Ambien and its possible successors in 2013.)

But there are non-pharmaceutical ways to facilitate sleep as well. Lockley and Czeisler have been developing a system of lights, to be used by NASA, to help us become sleepy at the right moment. They use the same wavelengths that prevent us from sleeping to make us more awake early in the day. Then they slowly transition to longer, “warmer” wavelengths to prepare our bodies for sleep. (They'll be installed on the International Space Station in October, 2016; for consumers, there are blue-light filters that can be placed on most any electronic device.) Other approaches are more behavioral. Susan Redline, a sleep researcher at Harvard Medical School, recommends cognitive-behavioral therapy. Other “mind-body therapies,” she said, like tai chi, yoga, and meditation, may also be helpful. She is currently developing a yoga intervention for low-income Boston residents who report sleep problems. The work builds on previous studies suggesting that people sleep better after doing yoga. A Brazilian intervention, led by Pedro Hallal, a researcher at the Federal University of Pelotas, provided free public gyms in low-income areas; this also had promising effects on sleep.

The search for solutions matters: as a society, we're becoming worse at going to sleep, and we're doing little to counteract that decline. But why should we care? Why is sleeping less so problematic? What useful purpose does sleep even serve? As researchers learn more about what happens while we sleep, they're understanding, more and more, how vital it really is.

Possible response options:

- What further research does this article make you want to do? Do it, and write about what you found and how it relates to the article.
- Do any of the sleep problems the author describes relate to your experience? Be sure to reference exactly which parts of Konnikova's passage you're responding to through the use of in-text citations.