

VIEWPOINT

Electronic glow may disrupt sleep for people on the spectrum

BY CHRISTOPHER COLWELL

13 NOVEMBER 2017

Many of us, and many children and adolescents, regularly use electronic devices such as laptops, cell phones and tablets at night. Exposure to the light of these devices can make it harder both to fall asleep and, if we wake up, to get back to sleep.

For example, people who use e-readers before sleep take longer to fall asleep and are less alert the next morning than those who read a printed book, according to a 2015 study¹. Conversely, young people who spend one week exposed to a natural light-dark cycle go to bed earlier and are less tired the next day².

These effects only exacerbate sleep problems among people with autism. Children with autism are often particularly drawn to **electronic devices**. And for those who have difficulty sleeping, sleep disruption may boost the use of electronic devices at night, contributing to the problem.

Between 40 and 80 percent of children with autism experience problems with sleep, compared with about 20 percent of their neurotypical peers. Children with autism may stay up too late and wake up often during the night. These and other **sleep problems** are associated with a worsening of some of their autism features, such as **repetitive behaviors** and social impairments³.

Sleep disruption can have numerous effects on health and behavior. And its impact can spread to caretakers and even an entire family. For this reason, improving sleep should be considered a critical therapeutic goal of autism research.

The causes of the sleep problems of individuals with autism vary. They are likely to include genetic, physiological and environmental factors. For example, one physiological change needed for a good night's sleep is the raising of the threshold for response to sensory stimulation. So sensory hypersensitivity may present a significant barrier to sleep in some people with autism, as every noise, light or other disturbance can awaken them.

Night light:

In the brain, light from electronic devices, among other sources, not only stimulates sight, but also activates a brain circuit that governs our 'circadian rhythms' — our daily cycles of arousal, mood and hormone secretion, among other body functions⁴.

There is a set of photoreceptors in the eye that are not part of the visual system, but instead connect to other brain regions, including the suprachiasmatic nucleus in the hypothalamus⁴. This nucleus is our body's master clock, regulating the rhythms of the other body systems; light can shift or reset this clock.

Specifically, light exposure in the early night will delay the clock such that it is harder to wake up the next morning and go to sleep the subsequent night. So nightly exposure to even **dim light** can disrupt our circadian cycle and have broad effects on our biology⁵.

The cells in the eye that connect to the clock are most sensitive to light in the blue-green part of the electromagnetic spectrum. These wavelengths are abundant in the light that shines from electronic devices.

When adolescents can't sleep, they may turn to these devices to pass the time. We don't yet know, however, whether people with autism who have severe sleep problems are exposed to more nocturnal light exposure than those who have mild or no sleep issues.

One study published last year indicates that electronic media use at night negatively affects sleep in children with autism⁶. In that study, parents of 101 children with autism completed questionnaires assessing their children's sleep habits, media access in the bedroom — including television, video game devices, and computers — and the timing of media use at night.

The parent responses suggest that children with autism who use electronic media before bed take twice as long to fall asleep as those who do not.

Battling the blues:

We believe that nocturnal blue-green light exposure can exacerbate sleep-wake disturbances in people with autism, and may worsen behavior the following day. We only have hints of this effect in mice.

In ongoing work, we exposed mice missing **CNTNAP2**, a gene tied to autism, to low levels of light at night. We found that mice exposed to the light show disruptions in their sleep-wake cycle and increased inflammation in the brain, as well as diminished social interactions and more repetitive behaviors. The light we used (5 lux) has about the same intensity as light generated by electronic

media or even a night light.

Light-triggered perturbations of the circadian system could affect the immune system. Body rhythms govern various immune processes, including the expression of genes for immune chemicals called cytokines and the proliferation of immune cells. Several studies have found evidence that exposure to light at night alters inflammatory responses in the nervous system in mice⁷. Similarly, in people, even a few days of circadian misalignment increases inflammatory markers^{8,9}.

People with autism may be more vulnerable to the impact of these circadian perturbations than typical individuals are. We don't yet have evidence to support this conjecture, but a growing body of preclinical research and clinical observations suggests it is correct.

In the meantime, we can try to ameliorate this disruption by increasing light exposure — specifically exposure to natural light, which is dominated by blue-green wavelengths — during the day and avoiding light at night. If you need a night light, look for red ones. These are widely available commercially. The circadian system can still detect these red-light signals, but it is much less sensitive to them than to the shorter, blue-green wavelengths.

Nighttime light is just one of the features in our modern world that make it challenging to get a good night's sleep. When we eat, exercise and even sleep also affects our endogenous timing system. And these factors may be particularly important in people with autism.

Christopher Colwell is professor of psychiatry and biobehavioral sciences at the University of California, Los Angeles.

REFERENCES:

1. Chang A.M. *et al. Proc. Natl. Acad. Sci. USA* **112**, 1232-1237 (2015) [PubMed](#)
2. Wright K.P. Jr *et al. Curr. Biol.* **23**, 1554-1558 (2013) [PubMed](#)
3. Hundley R.J. *et al. J. Autism Dev. Disord.* **46**, 3448-3457 (2016) [PubMed](#)
4. Colwell C.S. (2015) *Circadian medicine*. Hoboken, NJ: Wiley-Blackwell.
5. Lazzerini Ospri L. *et al. Annu. Rev. Neurosci.* **40**, 539-556 (2017) [PubMed](#)
6. Mazurek M.O. *et al. J. Dev. Behav. Pediatr.* **37**, 525-531 (2016) [PubMed](#)
7. Bedrosian T.A. *et al. Annu. Rev. Physiol.* **78**, 109-131 (2016) [PubMed](#)
8. Morris CJ *et al. Proc. Natl. Acad. Sci. U.S.A.* **113**, E1402-E1411 (2016) [PubMed](#)
9. Wright KP Jr *et al. Brain Behav. Immun.* **47**, 24-34 (2015) [PubMed](#)