

SAD—help arrives with the dawn?

At temperate latitudes, much the most common type of seasonal affective disorder (SAD) is recurrent winter depression, which affects individuals in autumn and winter and remits in the spring and summer. Common winter symptoms include low mood, anergia, irritability, anxiety, poor concentration, reduced libido, and social withdrawal. Unlike people with non-seasonal depression, those with SAD generally have hypersomnia (often with prominent daytime somnolence) and a tendency to eat more (predominantly carbohydrates and chocolate) and thus to gain weight. The fact that many people experience some of these symptoms to some extent supports the idea that winter depression is not a discrete entity but is a continuum within the population. Recent studies in the UK suggest that about 3% of the adult population are affected to a clinically significant degree.^{1,2}

Winter depression is often treated successfully with non-sedative antidepressants, such as the specific serotonin-reuptake inhibitors, although the evidence for their efficacy is disappointingly sparse.³ Light therapy, which consists of exposure to bright artificial light, at least 2500 lux, has been much more widely investigated. In these studies, bright light has generally proved effective. However, these findings have rightly been viewed with some scepticism given the difficulties in finding an appropriate comparator. Placebo treatments have included dim red light (500 lux) and sham negative-ion generators, and the use of such comparators, especially among participants who are often knowledgeable and self-diagnosed, may not be ideal. In fact, the best evidence for the efficacy of bright-light treatment in SAD derives from the finding that morning light is more effective than light administered later in the day.⁴

This efficacy of early morning light gives credence to the "phase-shift hypothesis" in the pathogenesis of SAD, as proposed by Lewy and co-workers⁵ in 1987. This hypothesis followed from the observation that the onset of melatonin secretion was delayed in depressed patients with SAD and that morning exposure to bright light was not only effective, but advanced the onset of melatonin secretion. In short, exposure to morning light phase-advanced SAD patients who had phase-delay, and was deemed to be therapeutic for that reason.

David Avery and colleagues,⁶ building on his group's previous studies, have added important support for the phase-shift hypothesis in SAD and have probably moved forward the treatment of winter depression. These investigators compared 30 min of bright light (10 000 lux) at 0600 h against dawn simulation and against placebo in 95 patients with SAD, all of whom had hypersomnia. Dawn simulation, first used by Terman and colleagues,⁷ consisted of exposure to white light of gradually increasing brightness (peaking at 250 lux after 90 min), which started at 0430 h while the patient was asleep. The placebo treatment consisted of a dim red light, peaking at 0.5 lux, over the same period as the active dawn simulation. The investigators took care to limit exposure to other light sources that might exert a therapeutic or a phase-shifting effect. Patients had very dark bedrooms, and if they went to the toilet during the night they did so with minimum exposure to light. If they had to go outside when the sun was shining, they were instructed to wear dark glasses. Despite the third precaution, patients' well-being during the trial correlated positively with hours of sunshine during the preceding week, which confirms the sensitivity of SAD symptoms to ambient light levels. The main finding of

the trial was that dawn simulation was associated with a significantly higher rate of remission and a greater reduction in symptoms than either bright light or placebo.

This superiority of dawn simulation over bright light and a credible placebo is impressive. However, these results differ from a previous study of dawn simulation by another group,⁸ and it is puzzling that bright light was not better than placebo. Avery and colleagues⁶ suspect that part of the efficacy of dawn simulation may be due to the method of delivery, which helped patients adhere to a waking-up time of 0600 h and thus a regular sleep schedule. Adherence in the bright-light group may have been less than complete since depressed patients with hypersomnia may not always reliably use a lightbox at 0600 h.

It is widely accepted that bright light, used while eyes are open is necessary for light therapy to be effective. The effectiveness of a relatively dim light (250 lux), used during sleep in dawn simulation,⁶ is then puzzling. Furthermore, light therapy presumably operates through its effects on photopigments in the eye. The single photopigment in the human eye that is involved in melatonin suppression and thus for advancing circadian phase, is sensitive to light towards the blue end of the spectrum,⁹ whereas the eyelids of sleeping people transmit much more red than blue or green light.¹⁰ These apparently conflicting findings may be resolved by the hypothesis of Avery and colleagues that the low light in dawn simulation falls on a particularly sensitive part of the light-phase response curve. The dawn signal may thus be able to phase-advance circadian rhythms, even though it is of low illuminance. Certainly, from an evolutionary perspective, it makes sense that human physiology would be entrained to awaken at fairly low ambient light levels.

Dawn simulation merits further exploration. Currently, it constitutes a further piece in the intriguing jigsaw of the pathogenesis and management of seasonal affective disorder.

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