pathogenetic mechanisms are genetically determined and environmentally patterned. In retinal cell biology, evidence is accumulating for genetic dependence of major components in photoreceptor renewal, metabolism, and function. Given the strong latitude dependence of SAD incidence and the rapid reversibility of symptoms by traveling south or by light therapy, the syndrome must be understood as strongly environmentally dependent and not as the inevitable consequence of a genetic defect. A genetic analysis of SAD, therefore, might appropriately focus on these photoreceptor renewal factors in contrast to a gene for depression per se.

How does light therapy provide its fast corrective influence? We postulate that, first, photoreceptor synaptic activity suppresses degradation after repeated exposure to bright light and, second, that subsensitive visual cells do not respond with light-elicited disk shedding (as is the case for other species). Therefore, photoreceptor outer segments would elongate and increase visual pigment. Although the existence of effect of light stimulation on the outer segment disk assembly rate in mammals is controversial, such effects have been shown in the amphibian. In addition, light stimulation of inner segment metabolism has been demonstrated in several species. We would thus interpret the therapeutic action of light to be mediated by a parametric effect of light in stimulating retinal synthetic activity and normalizing light sensitivity (and, as a consequence, triggering central nervous system mechanisms). Since the postulated metabolic changes are not vision related, far longer exposure times are required than those necessary to elicit a visual signal.


Do Winter Depressives Experience Summer Nights in Winter?

To the Editor.—The discovery of the antidepressive effects of light in seasonal affective disorder, winter type, has inspired explanations of pathogenesis of and therapy for this disorder in terms of circadian (dys)regulations. The original hypothesis of day length being the crucial variable was rapidly rejected. Subsequently, circadian phase and the daily total number of photons received by the patient were proposed as relevant factors. Unfortunately, neither of these proposals appeared to be compatible with all experimental data. In the present study, aspects of the previous hypotheses are put together into a new hypothesis, the outlines of which will be discussed.

The Circadian Pacemaker.—Functions of the circadian pacemaker are twofold. Apart from regulating the timing of processes within the circadian range, the pacemaker controls the annual timing of certain behaviors. To survive, many species must migrate, molt, hibernate, and reproduce at sharply defined intervals in the year. Whereas the extreme precision of the circadian pacemaker is highly needed for accurate estimation of the time of year, circadian processes do not seem to need this accuracy. Perhaps the primary function of the circadian pacemaker is to measure annual time, while the presence of such an accurate timing device is merely convenient to secondarily regulate circadian processes. If so, circadian dysregulations should be considered in their annual context.

To measure day length adequately the pacemaker must sharply discriminate between light and darkness. Moonlight at night and weather conditions during the day must not interfere. Hence, the threshold for discriminating light and darkness in humans must be somewhere between 10 and 500 lux. If the pacemaker is not to be misled by the application of artificial light, the threshold should even surpass about 100 lux.

Are Winter Depressives Supersensitive to Light?—There are some indications of increased sensitivity to light in winter depressives, both on the level of visual sensitivity and on the level of

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melatonin reactions to light. Although these indications need further empirical confirmation, it is tempting to speculate on their consequences. As is schematically explained in the Figure, there are important consequences given increased pacemaker sensitivity to light. Whereas artificial room light is below the detection level of the normal pacemaker, the supersensitive pacemaker does detect it. Consequently, darkness is detected at a much later time, leading to a circadian phase delay of the pacemaker. This is consistent with the data. Furthermore, the pacemaker will signal a long summer day instead of a short winter day. The resulting discrepancies between internal milieu and external circumstances may provide the condition for the development of winter depression.

**Light Therapy.**—The application of bright light is an effective treatment of winter depression. What does light do to the visual system and the circadian pacemaker? Changes in light intensity lead to adaptation, both on a short-term basis and in the long run. Adaptation to bright light is achieved by reducing light sensitivity. Eventual supersensitivity to light in winter depressives will therefore partially or totally be normalized by bright-light treatment. As a consequence, the pacemaker will no longer detect artificial room light, and winter days will be signaled instead of summer days.

Slight differences have been reported for the effectiveness of light treatment as a function of the time of application, with morning light being somewhat more effective than evening light. Within the context of the present hypothesis, these differences may be explained in two ways. First, differences in response may result from differences in visual sensitivity as a function of the time of day: the dark-adapted eye in the morning is more sensitive to light than the light-adapted eye in the evening. Second, it must be noted that evening light coincides with the interval of preferred darkness, thereby extending day length and reducing the effect.

**Comment.**—The present hypothesis attributes a major role to the seasonal function of the pacemaker in the pathogenesis and cure of winter depression. It is suggested that supersensitivity to light makes the pacemaker detect summer days in winter. Bright light might be therapeutic because it lowers light sensitivity and consequently normalizes pacemaker output. In this way, the present hypothesis integrates the original day length hypothesis and the earlier proposed photon-catch hypothesis. The circadian phase position of the pacemaker is not relevant to the present hypothesis, but the observed circadian phase shifts underlying the phase shift hypothesis are fully compatible with increased pacemaker sensitivity to light.

**Sleep Architecture in Eating Disorders**

*To the Editor.*—In a recent report in the Archives Berger et al. indicated that there is a contradiction between their findings and an earlier report from our group. In fact, we believe that in all major respects the data by Berger et al are consistent with the conclusions we reached.

As noted by Berger et al, their report on the induction of rapid eye movement (REM) sleep by the cholinergic agonist RS-86 contains data relevant not only to depressive illness, but also to eating disorders. In the last several years it has been suggested that patients with anorexia nervosa and bulimia have a predisposition to major depressive illness. In their study, Berger et al found that, compared with controls, 12 patients with eating disorders (6 with anorexia nervosa, 6 with bulimia) did not exhibit the more rapid induction of REM sleep observed in patients with major depressive illness. They concluded that “the results do not support the assumption of a close biological association between eating disorders and primary depressive disorders at the level of sleep EEG [electroencephalographic] patterns.”

In our earlier report, we reported on EEG monitored sleep in 8 women with anorexia nervosa and 14 women with bulimia. We reached a virtually identical conclusion: “Most patients with anorexia nervosa and bulimia do not exhibit the type of sleep disturbances characteristic of patients with major depressive illness.”

In our study of 22 patients, we found that a few individuals with current depression had short REM latencies. Therefore, while we concluded that the majority of patients with eating disorders do not exhibit the EEG sleep abnormalities typically seen in major depressive illness, we left open the possibility that a subgroup of patients, particularly those who were also depressed, might do so. Among their 12 patients, Berger et al did not observe sleep abnormalities in those with concurrent depression. This appears to be the only “contradiction” between the two studies, and, in our view, it is a very minor one.

In short, we commend Berger et al for an interesting and important study. We feel that their conclusions concerning patients with eating disorders are, in all major ways, a confirmation and extension, not a contradiction, of our previous work.

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