Electroretinography in patients with winter seasonal affective disorder

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Abstract

A retinal sensitivity abnormality has been hypothesized in seasonal affective disorder (SAD). To explore this hypothesis, the electroretinogram (ERG) was used to assess retinal sensitivity at the level of the rod photoreceptor system. We examined 27 depressed patients who met DSM-III-R criteria for major depression, recurrent, with a seasonal (winter) pattern and 23 normal control subjects who were age-paired and sex-matched as much as possible with the SAD patients. ERG testing was performed in dark-adapted, dilated eyes in winter between 10:00 and 15:00 h. Retinal sensitivity was based on the light stimulus intensity necessary to reach a 50-mV amplitude threshold. We found that retinal sensitivity was significantly lower (0.21 log units) in SAD patients compared with normal control subjects and that 55% of the patients had a retinal sensitivity value one standard deviation lower than the mean value of the control subjects. These results are consistent with a retinal hyposensitivity hypothesis for SAD, but the explanation for lower rod photoreceptor sensitivity in SAD is not known. We hypothesize that brain neurotransmitter dysregulation may be at the origin of both the mood disorder and retinal sensitivity change.

1. Introduction

Winter seasonal affective disorder (SAD) is a subtype of depressive disorder characterized by recurrent major depressive episodes that occur in autumn/winter, with full remission of symptoms in spring/summer (Rosenthal et al., 1984). The pathophysiology of winter SAD is unknown, but ocular light sensitivity hypotheses have been the focus of several investigations in seasonal affective disorder (SAD) with mixed results. The link between SAD and the retina arose from the fact that the effect of light therapy appears to be mediated through the eyes (Wehr et al., 1987). In 1990, two contrasting hypotheses were proposed.
to account for the role of the retina in SAD (Beersma, 1990; Remé et al., 1990). Beersma (1990) proposed SAD patients have retinal hyper-
sensitivity to light. This would lead to perception
of evening room light as an extension of the
photoperiod yielding a depressogenic phase delay
of circadian rhythms sometimes reported in SAD
(Lewy et al., 1987). Therefore, the efficacy of
morning light therapy results from a corrective
realignment of circadian rhythms through resyn-
chronisation of the biological clock with the nat-
ural light/dark cycle. In contrast, Remé and
colleagues (1990) proposed that SAD patients
have retinal hyposensitivity to light. This hypoth-
esis is based on the proposition that in the normal
population, the wintertime decrease of natural light
exposure may lead to a compensatory mechanism
of increased retinal light sensitivity, an ocular
physiological adaptation termed photostasis that
has been observed in some animal studies (Penn
and Williams, 1986; Parker and Williams, 1995;
Schremser and Williams, 1995a,b). They argued
that if this process were absent or weakened in
patients with SAD, they would be hyposensitive
to light compared with normal subjects. Conse-
quently, they would not be able to absorb enough
light to maintain a euthymic state, explaining the
need for light therapy.

There is support for both retinal hypersensitivity
and hyposensitivity hypotheses, but the supporting
data are highly dependent on the techniques used
to measure light sensitivity. Studies using the dark
adaptation threshold test (DAT) found no differ-
ence in the rod photoreceptor threshold obtained
in patients with SAD and normal controls, either
in summer or winter (Oren et al., 1993; Terman
and Terman, 1999), but cone photoreceptor sensi-
tivity was higher in both winter and summer in
SAD patients than in controls (Terman and Ter-
man, 1999). The latter result was interpreted as
supportive of the hypersensitivity hypothesis in
SAD, but for the cone photoreceptors only.

In contrast, using the electrooculogram (EOG)
technique, EOG ratios have been found to be low
in SAD patients relative to controls in winter (Lam
et al., 1991; Ozaki et al., 1993), supporting the
hyposensitivity hypothesis. Normal subjects were
found to have increased EOG ratios in winter
compared with summer, whereas no changes were
observed in SAD patients (Ozaki et al., 1995).
Interestingly, no change in the EOG ratio was
observed after 1 week of light therapy even when
light therapy was clinically effective. Also, sup-
porting the hyposensitivity hypothesis were find-
ings with the full flash electoretinogram (ERG)
technique. Women with SAD compared with
matched controls, but not men, had lower b-wave
amplitudes (Lam et al., 1992). Because the light
stimulus used in that study triggered a mixed rod-
cone response, it was not possible to clearly
specify the origin of the change. However, a small-
sample, mixed-sex study using the pattern ERG
(PERG), which elicits an electrical response from
the retina (mostly the macular region), did not
find any differences between SAD patients and
controls (Oren et al., 1993). Recently, a more
specific ERG technique in which various dim blue-
green light intensities generate a detailed scotopic
luminance response function (Hébert et al., 1996)
was used to investigate seasonal change in rod
sensitivity in subsyndromal affective disorder (S-
SAD), that is, people who experience a non-
clinical, milder form of winter depression (Kasper
et al., 1989). S-SAD subjects showed a wintertime
decrease in rod sensitivity, but normal subjects had
no seasonal change (Hébert et al., 2002).

In the present study, we used flash ERG to
assess retinal sensitivity in winter in a large group
of well-diagnosed SAD patients and normal con-
trol subjects. In this study, patients and controls
were tested in winter, and a short version of the
rod luminance-response function was obtained
with only four intensities. Retinal sensitivity was
defined as the intensity necessary to reach a fixed
b-wave amplitude criterion of 50 µV. We hypothe-
sized that if the SAD patients were indeed signifi-
cantly less sensitive to light than the controls, the
intensity of light needed to reach the criterion
during a depressive episode would be higher than
in the control subjects.

2. Methods

2.1. Subjects

Twenty-seven depressed SAD patients who had
been psychototropic drug-free for at least 5 weeks
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