CME ACTIVITY

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CME Objectives

After completing this CME activity, the psychiatrist will be able to:

- Diagnose winter depression (seasonal affective disorder)
- Describe dawn simulation
- Discuss the possible relationship between seasonal affective disorder and alcoholism

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Faculty Disclosure

In the spirit of full disclosure and in compliance with all Accreditation Council for Continuing Medical Education Essentials, Standards, and Guidelines, all faculty for this CME activity were asked to complete a full disclosure statement. The information received is as follows:

Dr. Avery received the dawn simulators for this study from Pi Square, Inc., Seattle, Washington.

Neither Drs. Bolte nor Ries has significant relationships with entities that may have influenced their presentation in any way.

Discussion of Investigational Information

During the course of their talks and discussions in this article, faculty may be presenting investigational information about pharmaceutical agents that is outside Food and Drug Administration–approved labeling. This information is intended solely as continuing medical education and is not intended to promote off-label use of any of these medications. Please refer to page 42 for a list of indications of off-label usage describing any medication discussed in this enduring material that, in the authors' clinical estimation, is outside the manufacturer's current recommendations for standard prescribing practices.

Dawn Simulation Treatment of Abstinent Alcoholics With Winter Depression

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Background: Recent data suggest that winter depression (seasonal affective disorder [SAD]) may be a subtype of affective disorder that is closely related to alcoholism. Dawn simulation has been shown in controlled trials to be effective in SAD. The present study examined the effectiveness of dawn simulation in abstinent alcoholics who met DSM-III-R criteria for major depression, or bipolar disorder, depressed with seasonal pattern.

Method: All 12 subjects with winter depression had a history of either alcohol dependence or alcohol abuse according to DSM-III-R and had been abstinent from alcohol for at least 6 months. They also fulfilled criteria for SAD according to Rosenthal and were hypersomnic and drug free. After a 1-week baseline period, the subjects were randomly assigned to a 1-week treatment period at home with either a white 1.5-hour dawn from 4:30 a.m. to 6:00 a.m. peaking at 250 lux or a red 1.5-hour dawn from 4:30 a.m. to 6:00 a.m. peaking at 2 lux. The subjects were told that they would receive daily either a red or a white dawn reaching the same illuminance, an illuminance that would be much dimmer than standard bright light treatment. At the end of each week, the subjects were blindly assessed by a psychiatrist using the Structured Interview Guide for the Hamilton Depression Rating Scale–Seasonal Affective Disorder version (SIGH-SAD).

Results: For the 6 subjects completing the white dawn treatment, the mean SIGH-SAD score decreased from 33.0 at baseline to 15.8 after treatment. For the 6 subjects completing the dim red dawn treatment, the mean SIGH-SAD score decreased from 34.3 to 32.7. The mean post-dawn SIGH-SAD score was significantly lower after the white dawn treatment than after the dim red dawn treatment (ANCOVA with baseline SIGH-SAD as the covariate, $F = 12.95$, $p < .01$). Superiority of the white dawn was also found by analogous analyses for the Hamilton Rating Scale for Depression (HAM-D) ($p < .01$) and the SAD Subscale ($p < .05$).

Conclusion: The present study suggests that dawn simulation may be helpful in decreasing depression in abstinent alcoholics with SAD. Further study is necessary to confirm these preliminary findings and to determine whether dawn simulation might be helpful in preventing relapse in abstinent alcoholics who have SAD.

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Recent data suggest that winter depression (seasonal affective disorder [SAD]) may be a subtype of affective disorder that is closely related to alcoholism. For example, Allen et al.[1] found that SAD patients were much more likely to have a family history of alcoholism than were nonseasonal depressed patients. In addition, some alcoholics have a seasonal pattern to their alcohol abuse.[2,3] Certain alcoholics may be self-medicating an underlying depression with alcohol or manifesting a seasonal pattern to alcohol-induced depression.[4–6] Yahia et al.[7] found that bright light therapy was more effective than a dim light control in depressed detoxified alcoholics.

Although bright light therapy has been shown to be effective in treating SAD,[8] dawn simulation has also been shown, in controlled trials, to be effective in treating SAD.[9,10] This newer treatment is a low-level light that gradually increases in intensity just before a patient awakens.[11] If some alcoholics attempt to self-medicate SAD with alcohol, or if SAD predisposes this population to alcohol relapse, then treatment of SAD with a light therapy may be beneficial in preventing relapse into alcoholism in this population. Before a large prospective study of light therapy for the prevention of relapse is undertaken, it is reasonable to document that light therapy is effective in treating the depressive symptoms in abstinent alcoholics with SAD. The present study examines

the effectiveness of dawn simulation compared with a placebo condition in abstinent alcoholics who have SAD.

METHOD

Subjects were recruited through advertisements and through publicity concerning our program. Individuals were initially screened by telephone interview to include subjects who had a history of alcohol abuse or alcohol dependence according to DSM-III-R criteria,[12] had abstained from alcohol for at least 6 months, and fulfilled DSM-III-R criteria for major depression or bipolar disorder, depressed and seasonal pattern during that abstinence. The subjects also met Rosenthal criteria for SAD[13] during that period of abstinence; subjects reported regularly occurring fall-winter depressions (with at least two occurring during consecutive fall-winters) that remitted during the spring or summer. Subjects gave their informed consent after the procedures and possible side effects were fully explained.

Those who met the initial criteria were interviewed in person and given a physical examination to rule out medical or psychiatric problems other than depression. They were administered the Structured Interview Guide for the Hamilton Depression Rating Scale–Seasonal Affective Disorders version (SIGH-SAD).[14] Subjects had to have a SIGH-SAD score of at least 20. The SIGH-SAD comprises the 21-item Hamilton Rating Scale for Depression (HAM-D)[15] and 8 supplementary items (SAD Subscale) concerning the atypical symptoms commonly seen in winter depression such as hypersomnia, increased appetite, and weight gain. Those with significant medical problems such as cardiac, hepatic, renal, respiratory, endocrinologic, neurologic, or hematologic disease were excluded. Persons undergoing a major psychosocial stress such as bereavement were also excluded. Subjects were considered for study entry if they had been free from psychiatric medication for 1 or more months before the study. We excluded persons who routinely took antihistamines, decongestants, aspirin, appetite suppressants, or sleeping medication.

Since hypersomnia is present in 83% of winter depressives[16] and may be relevant to the appropriate timing of light therapy,[17,18] only hypersomnic winter depressives were studied to increase homogeneity. Hypersomnia was defined as sleep duration of at least an hour more per night during an episode of winter depression than euthymic summer sleep duration; this is a broader definition of hypersomnia than is given in the DSM-IV criteria for atypical features, ≥ 10 hours of sleep per day or ≥ 2-hour increment over the duration when in remission.[12] In studying a morning light treatment such as dawn simulation, a hypersomnic SAD sample is most appropriate and also representative of the great majority of SAD patients. Persons who were extremely hypersomnic and awakened after 9:00 a.m. were excluded to minimize the possible sleep deprivation imposed by the protocol.

The subjects were informed that the purpose of the study was to compare dawn treatments of different colors. They were told that the dawn simulation treatments would be much dimmer than bright light treatment, the more commonly used treatment of SAD, which utilizes a 2500- to 10,000-lux light box. The difference in light intensity of the two dawns was not revealed until the end of the 2-week study.

Expectations of the response to both light treatments were assessed by each subject at baseline before any subject had seen the actual dawn signals. Subjects rated their expected response on a global scale (1 = worse, 2 = no change, 3 = slight improvement, 4 = much improvement, 5 = very much improvement). At the end of each week's light treatment, subjects rated their own response to light treatment on the same global scale. At the end of both the baseline week and the treatment week, the subjects were blindly assessed by a psychiatrist using the SIGH-SAD.

Subjects were instructed to sleep only between the hours of 9:00 p.m. and 6:00 a.m. and keep a 14-day log of their sleep during the entire 2-week study. The study took place in the Seattle area (latitude 47°), where the 6:00 a.m. time of awakening is always before sunrise during the fall and winter. The subjects were instructed not to drink alcohol or use drugs of abuse or other potentially psychoactive medications such as antihistamines, sleeping medication, and appetite suppressants. The subjects' bedrooms were required to be dark. If a street light or security light did shine through the bedroom windows, subjects were given a sheet of black plastic to cover the windows. Subjects were asked to turn off any nightlights or hall lights that might shed light into the bedroom. They were instructed to avoid any outdoor light before 8:00 a.m. and any direct sunlight during the day. If there was no cloud cover during the daytime, the subjects were to either stay indoors or use sunglasses if they had to go outside. Outdoor light even on a cloudy day may be as bright as a bright light box (2500 to 10,000 lux), and this intensity of light, especially early in the morning, could have had a therapeutic effect independent of our intervention. Direct sunlight (50,000–100,000 lux) during the day could be therapeutic.

Table 1. Mean ± SD Depression Ratings at Baseline and After Treatment With Either Dawn Simulation or a Dim Red Signal*

	Dawn Simulation (N = 6)		Dim Red Signal (N = 6)		ANCOVA	
Rating Scale	Baseline	Posttreatment	Baseline	Posttreatment	F	p
SIGH-SAD (29 items)	33.0 ± 6.0	15.8[a] ± 11.8	34.3 ± 8.2	32.7 ± 11.6	13.0	<.01
HAM-D[a] (21 items)	18.3 ± 3.3	7.7[a] ± 6.9	20.5 ± 5.0	19.5 ± 7.6	12.7	<.01
SAD Subscale (8 items)	14.7 ± 3.4	8.2[a] ± 5.0	13.8 ± 3.9	13.2 ± 5.1	6.0	<.05

*Abbreviations: SIGH-SAD = Structured Interview for the Hamilton Depression Rating Scale–Seasonal Affective Disorder version, HAM-D = Hamilton Rating Scale for Depression.
[a]p < .01 vs. score for dim red signal treatment by two-tailed paired t tests.

Subjects whose SIGH-SAD scores at the end of the baseline week were greater than 20 were randomized into one of two 1-week treatment groups:

1. The dawn simulation consisted of a white light with a gradually increasing (approximately 3 $\log_{10}$ lux/h) illuminance (during sleep) from 4:30 a.m. to 6:00 a.m. peaking at 250 lux (similar to average room light level) in the bedroom while the subject was asleep.
2. The placebo dawn simulation consisted of a dim red light with a gradually increasing illuminance (during sleep) from 4:30 a.m. to 6:00 a.m. peaking at 2 lux (similar to civil twilight and about 10 times brighter than moonlight).

In both dawn simulation conditions, a Remcraft model 1051 fixture (Remcraft, Miami, Fla.) was used with a Juno filter holder (#T570 $4^3/_4$-inch size, Juno Lighting, Inc., Des Plaines, Ill.) positioned 4 feet from the pillow. The end of a string attached to the base of each fixture identified a point 4 feet from the light. The subjects were instructed to place the end of the string on their pillow when positioning the fixtures on the wall in back of the bed and to point the lamp at the pillow. The fixture was placed on the wall so that direct light from the fixture would strike the eyelids whether the patient was sleeping on his left or right side.

In the white dawn condition, a 100-watt R30 Phillips flood lamp was used with a Roscolux gel filter #102 Light Tough Frost (Rosco, Hollywood, Calif.). In the dim red dawn condition, a 30-watt R20 Phillips flood lamp was used with a Roscolux lux Medium Red (#27) gel filter, two Tough Silk (#104) filters, and a Neutral Density (#3404) filter (Rosco, Hollywood, Calif.). The final illuminances of both signals at 4 feet were confirmed before being given to the subjects using a sensitive Vactec Photometer (Model #3107) (Vactec, Maryland Heights, Mo.). The incandescent light was plugged into a dawn simulator device (SunUp, Pi Square, Inc., Seattle, Wash.), which provides a gradually increasing voltage to the incandescent light starting at a time that can be specified. Subjects were asked to sleep until 6:00 a.m., at which time they were awakened by an alarm. If they awakened before 6:00 a.m., they were asked to avoid looking at the lights, close their eyes, and try to go back to sleep. Subjects used these lights daily during the treatment week.

Side effects were assessed by systematic questioning of subjects. Subjects were asked to rate each effect as absent, mild, moderate, or severe. Slight early morning awakening was defined as any awakening during the dawn signal after which the subjects were easily able to go back to sleep.

Statistical analyses included analysis of covariance (ANCOVA), two-way repeated measures analysis of variance (ANOVA), paired and unpaired Student's t tests, Spearman's rank-order correlation coefficient, and Fisher's exact test.

RESULTS

Of the 12 subjects, 6 (5 women and 1 man) were randomly assigned to the white, 1.5-hour, 250-lux dawn, and 6 (4 women and 2 men) were randomly assigned to the red, 1.5-hour, 2-lux signal. Nine subjects had a history of alcohol abuse; 3 subjects (2 who received the white dawn and 1 who received the red dawn) had histories of alcohol dependence. The mean ± SD ages were similar in the white dawn (33.0 ± 10.0 years) and red dawn (34.3 ± 14.6 years) groups (t = 0.18, p = N.S.). Six subjects had major depression, recurrent, and 6 (4 in the white dawn group and 2 in the red signal group) had bipolar II disorder because of their histories of hypomania. The results include the original data from 5 subjects (3 randomly assigned to white, and 2 to red) from a previous study.[10] These subjects met the same criteria as the others in this study. All the patients were studied between November 17 and February 28.

The SIGH-SAD scores, HAM-D scores, and the SAD Subscale scores are summarized in Table 1. The

Table 2. Times of Sleep Onset and Awakening and Sleep Duration (± SD)

	White 1.5-h 250-lux Dawn (N = 6)		Red 1.5-h 2-lux Dawn (N = 5)[a]	
	Baseline	Treatment	Baseline	Treatment
Sleep onset	11:17 pm ± 75 min	10:42 pm ± 49 min	11:23 pm ± 39 min	11:30 pm ± 49 min
Wake-up	6:13 am ± 42 min	5:55 am ± 27 min	6:18 am ± 33 min	6:24 am ± 42 min
Sleep duration (h)	6.9 ± 0.6	7.2 ± 0.4	6.9 ± 0.4	6.9 ± 1.0

[a]Sleep log was not completed by 1 subject in the red dawn group.

ANCOVA analyses, using the baseline scores as the covariate, showed greater efficacy for the white dawn compared with the red signal. Three of the 6 subjects receiving the white dawn had posttreatment SIGH-SAD scores of less than 8; none of the 6 receiving the red dawn fulfilled those criteria.

At baseline, the expectation for the white dawn was significantly greater than for the red dawn for the entire sample (3.2 ± 0.4 vs. 2.7 ± 0.5 two-tailed $t = 3.63$, $p < .005$). There was a nonsignificant trend for the baseline expectations for the signal actually received to be greater for the white dawn group compared with the red signal group (3.3 ± 0.5 vs. 2.8 ± 0.4, two-tailed $t = 2.15$, $p = .06$). However, the global improvement score after treatment was significantly greater for subjects who received the white dawn signal than for those who received the dim red signal when the baseline expectation score for the signal received was used as a covariate (ANCOVA, $F = 9.0$, $p < .05$, 3.9 ± 0.7 vs. 2.1 ± 1.2).

There were no significant correlations within the white dawn group between the expectation for response to the white dawn and the actual self-rated improvement ($r = .66$, p = N.S.) or between the expectations for response and the change in SIGH-SAD score ($r = .41$, p = N.S.). Within the red signal group, there were significant negative correlations between the expectations to the red dawn and the self-rated improvement ($r = –.87$, $p < .05$) and between the expectations for response and the change in the SIGH-SAD score ($r = –.89$, $p < .05$).

The sleep logs were completed by all subjects except 1 (who received the red dawn). The estimated times of sleep onset and awakening and sleep duration are presented in Table 2 for 5 subjects who received the red dawn and 6 subjects who received the white dawn. A two-way repeated measures ANOVA revealed no differences in sleep onset time during the baseline week and treatment week ($F = 0.95$, $df = 1,9$; p = N.S.), no differences in sleep onset time between groups ($F = 0.91$, $df = 1,9$; p = N.S.), and no significant interaction for sleep onset time between group and order ($F = 2.19$, $df = 1,9$; p = N.S.) A two-way repeated measures ANOVA revealed no differences in the time of awakening during the baseline week and treatment week ($F = 0.46$, $df = 1,9$; p = N.S.), no differences in time of awakening between groups ($F = 0.59$, $df = 1,9$; p = N.S.), and no significant interaction for time of awakening between group and order ($F = 1.85$, $df = 1,9$; p = N.S.) A two-way repeated measures ANOVA revealed no differences in sleep duration during the baseline week and treatment week ($F = 0.35$, $df = 1,9$; p = N.S.), no differences in sleep duration between groups ($F = 0.21$, $df = 1,9$; p = N.S.), and no significant interaction for sleep duration between group and order ($F = 0.63$, $df = 1,9$; p = N.S.).

Slight early morning awakening was experienced by 5 of the 6 subjects using the white dawn and by none of the 6 using the dim red signal (Fisher's exact test, $p < .05$). One subject experienced a mild headache with the white dawn.

DISCUSSION

Although the sample size is small, the results suggest that dawn simulation is effective in decreasing the level of depression in a group of abstinent alcoholics who have SAD. The presence of a history of alcoholism does not seem to lessen the degree of response to dawn simulation; the decreases in the SIGH-SAD scores for our subjects are similar to the decreases seen in studies of SAD patients with no history of alcoholism.[9,10]

Given the greater expectations for the white dawn compared with the red dawn, a significant placebo effect is possible. When the expectations were statistically controlled for as a covariate, the white dawn was still superior to the red dawn. Within the group receiving the white dawn, the correlation between expectation and improvement was large ($r = .66$) but not statistically significant. Interestingly, within the red dawn group, those who had high expectations were more likely to have a poor response ($r = –.89$, $p < .05$). The small sample size limits our interpretation of the role of expectations.

Although the time of sleep onset, the time of awakening, and sleep duration did not statistically differ between the two groups, the sleep onset tended to be earlier after the white dawn, the time of awakening tended to be later after the red dawn, and the sleep duration was slightly greater with the white dawn. It is possible that the brighter dawn signal worked as a more effective alarm clock and made the sleep-wake cycle more regular. The question of the mechanism of action of dawn simulation is beyond the scope of this paper and is discussed elsewhere.[10]

The other study of light therapy in detoxified alcoholics, by Yahia et al.,[7] differed from our study in a number of ways. In their study, the alcoholics had been admitted to a VA hospital and had been detoxified from alcohol for only a 2-week period. None reported any seasonal pattern of depression. The detoxified alcoholics were then treated with either bright light therapy (2500 lux) or a dim light (< 400 lux) control condition for 2 hours from 9:00 a.m. to 11:00 a.m. for 5 consecutive days. Blind global ratings showed that 11 (50%) of 22 of the bright light group had much or very much improvement compared with only 5 (26%) of 19 of the dim light group.

The results of the present study and the study by Yahia et al.[7] take on importance because data suggest that SAD is a subtype of affective disorder that is closely related to alcoholism. Regier et al.[19] found that 37% of alcohol disorders were comorbid with mental disorders. Alcoholism and depression often coexist, overlap in their presentations, and create diagnostic confusion.[4,6] Alcoholics are frequently depressed during intoxication and even when abstaining.[4,5] Patients with major depression and bipolar depression may develop secondary alcoholism. In subjects with coexisting alcoholism and depression, the alcohol abuse usually precedes the depression (primary alcoholism with secondary depression); patients who have a clear diagnosis of depression that precedes the alcoholism are much less common.[20] However, studies of alcoholism and depression have in general focused on the endogenous-melancholic subtype and may have missed the "atypical" symptoms common in SAD.

Alcohol transiently elevates the mood of alcoholics for minutes or hours, but then the mood worsens with either discontinuance or more sustained alcohol use.[21] Alcohol also transiently elevates the mood in depressed patients who have no history of alcoholism.[22] These findings raise the possibility that some alcoholics may be attempting to deal with depression/dysphoria by drinking. Relapse is more common among alcoholics who have other psychopathology.[20,23]

Although some studies have shown that blood relatives of alcoholics have a high incidence of depression and that blood relatives of depressed patients have a high incidence of alcoholism, other studies have not.[24] The risk of alcoholism may be especially great among depressed patients with a history of hypomania (bipolar II).[25] The incidence of alcoholism among male relatives is higher in females with depression; the incidence of depression in female relatives is higher in males with alcoholism.[26]

Winokur[26] has identified a subcategory of unipolar depression, depressive spectrum disease, which has a positive family history of alcoholism among first-degree blood relatives. Depressive spectrum disease differs from unipolar depression without a family history of alcoholism in a number of respects: an earlier onset, an increased proportion of females, a lack of endogenous symptoms of depression (early morning awakening, decreased appetite, weight loss), and a lack of biological markers of endogenous depression such as abnormal dexamethasone suppression and short REM latency. These data suggest the possibility that a single disorder manifests itself as depressive spectrum disorder in women and as alcoholism in men.

SAD is a type of depression that recurs every fall-winter and remits in the summer. Unlike classic endogenous-melancholic depression, which is associated with early morning awakening, decreased appetite, and weight loss, SAD is associated with "atypical symptoms," hypersomnia, increased appetite (especially carbohydrate craving), and weight gain.[27] These atypical symptoms are predictive of response to light in SAD patients.[28] SAD subjects often experience hypomania in the summer and thus are diagnosed as bipolar II. SAD subjects resemble depressive spectrum disorder subjects in a number of ways: a higher proportion of women, an early onset, a lack of endogenous symptoms, normal dexamethasone suppression,[29] and normal REM latency.[30]

Family studies suggest a relationship between alcoholism and SAD. We found that the incidence of alcoholism among first-degree blood relatives of winter depressives (even those without a personal history of alcohol abuse) is greater than the incidence among blood relatives of controls (33/165, 20% vs. 5/69, 7.2%; $p < .05$) (D.H.A., M.A.B., R.R. Unpublished data. 1989). Using the Family History Method, Allen et al.[1] found that 41% of SAD patients had first-degree relatives with alcoholism compared with only 18% of non-SAD patients. During our past research on SAD, we have had to eliminate many potential subjects from our SAD

studies because of their concurrent or past alcohol abuse. Anderson et al.[3] found that, in an alcohol/substance abuse program, 23% of the patients had SAD and 40% were subsyndromal; among the female patients, 50% had SAD. McGrath and Yahia[2] found that among hospitalized VA alcoholics, 6 of 108 had SAD with clear seasonal alcohol use preceded by depressive symptoms. In this severely addicted population, the true incidence of SAD may have been underestimated; subjects who had early undetected SAD may have developed nonseasonal alcohol addiction that obscured the SAD.

The incidence of SAD increases with latitude; in a four-center study, the incidence ranged from 1% in Florida to 5% in Maryland and 10% in New Hampshire.[31] Alcohol consumption also may increase with latitude in the United States.[32]

SAD subjects experience carbohydrate craving, which is thought to reflect an underlying serotonin deficiency.[33,34] Carbohydrates,[35] including alcohol,[36] are effective in acutely increasing brain serotonin levels. Antidepressant medications that increase serotonergic function may be effective in treating SAD.[37,38]

Taken together, these data suggest a relationship between SAD and alcohol abuse/dependence and raise the possibility that some persons use alcohol to treat their underlying SAD, or that SAD predisposes to the development of alcohol abuse/dependence. It is likely that abstinent alcoholics who have SAD are at greater risk for relapse into alcoholism if their SAD remains untreated. The present study suggests that dawn simulation may be helpful in decreasing depression in abstinent alcoholics with SAD. This type of treatment may be more acceptable to many recovering persons who would rather not use medications. Further study is necessary to confirm these preliminary findings and to determine whether dawn simulation might be helpful in preventing relapse in abstinent alcoholics who have SAD.

Drug name: dexamethasone (Decadron and others).

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DISCLOSURE OF OFF-LABEL USAGE

At the present time, neither bright light therapy nor dawn simulation has been approved by the Food and Drug Administration for the treatment of any medical condition. However, light therapy has been recommended in the treatment of major depression with seasonal pattern by the U.S. Department of Health and Human Services in the following publication: Public Health Service, Agency for Health Care Policy and Research. Clinical Practice Guideline Number 5: Depression in Primary Care, vol 2: Treatment of Major Depression. Washington, DC: US Government Printing Office; 1993. AHCPR Publication No. 93-0551.

Instructions

Psychiatrists may receive 1 hour of Category 1 credit toward the American Medical Association Physician's Recognition Award by reading the article starting on page 36 and correctly answering at least 70% of the questions in the quiz that follows.

1. Read each question carefully and circle the correct corresponding answer on the Registration form.
2. Type or print your full name, address, phone number, and fax number in the spaces provided.
3. Mail the Registration form along with a check, money order, or credit card payment in the amount of $10 to: Physicians Postgraduate Press, Office of CME, P.O. Box 752870, Memphis, TN 38175-2870.
4. For credit to be received, answers must be postmarked by the deadline shown on the CME Registration form. After that date, correct answers to the quiz will be printed in the next issue of the *Journal*.

All replies and results are confidential. Answer sheets, once graded, will not be returned. Unanswered questions will be considered incorrect and so scored. Your exact score can be ascertained by comparing your answers with the correct answers to the quiz, which will be printed in the *Journal* issue after the submission deadline. The Physicians Postgraduate Press Office of Continuing Medical Education will keep only a record of participation, which indicates the completion of the activity and the designated number of Category 1 credit hours that have been awarded.

1. One of the most common symptoms of winter depression is:
a. Early morning awakening
b. Hypersomnia
c. Decreased appetite
d. Weight loss
e. None of the above

2. Dawn simulation used in this study reached a maximum intensity of:
a. 25 lux
b. 250 lux
c. 1000 lux
d. 2500 lux
e. 10,000 lux

3. The light intensity of a typical light box used for treatment for winter depression is:
a. 2–10 lux
b. 25–100 lux
c. 250–1000 lux
d. 2500–10,000 lux
e. 50,000–100,000 lux

4. Compared with nonseasonal major depression, seasonal affective disorder is more likely associated with:
a. Endogenous depressive symptoms
b. Abnormal dexamethasone suppression
c. Short REM latency
d. A family history of alcoholism
e. None of the above

5. Mental disorders are comorbid with alcoholism in what percentage of alcoholics?
a. 6%
b. 11%
c. 37%
d. 49%
e. 73%

6. Depressive spectrum disease is defined by:
a. Family history of alcoholism
b. Family history of depression
c. Symptoms
d. Course of illness
e. None of the above

7. Seasonal affective disorder and depressive spectrum disease have which of the following in common?
a. Higher proportion of women
b. An early onset
c. A lack of endogenous symptoms
d. All of the above
e. None of the above

Answers to the July 1997 CME quiz

1. e
2. b
3. e

Circle the one correct answer for each question.

1.	a	b	c	d	e
2.	a	b	c	d	e
3.	a	b	c	d	e
4.	a	b	c	d	e
5.	a	b	c	d	e
6.	a	b	c	d	e
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Deadline for mailing

For credit to be received, the envelope must be postmarked no later than July 1998 (outside the continental United States, September 1998).

Keeping a copy for your files

Retain a copy of your answers and compare them with the correct answers, which will be published after the submission deadline.

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Please evaluate the effectiveness of this CME activity on a scale of 1 to 5 (1 being poor, 5 being excellent).

1. Overall quality of this CME activity ____
2. Content ____
3. Format ____
4. Faculty ____
5. Achievement of educational objectives:
   - A. Enabled the reader to diagnose winter depression (seasonal affective disorder). ____
   - B. Enabled the reader to describe dawn simulation. ____
   - C. Enabled the reader to discuss the possible relationship between seasonal affective disorder and alcoholism. ____
6. This CME activity provided a balanced, scientifically rigorous presentation of therapeutic options related to the topic, without commercial bias. ____
7. Please comment on the impact that this CME activity might have on your management of patients.

_____

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8. Please offer additional comments and/or suggested topics for future CME activities.

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