

# Alcoholism and Seasonal Affective Disorder

Leo Sher

**Seasonal changes in mood and behavior (seasonality) may be closely related to alcoholism. Some patients with alcoholism have a seasonal pattern to their alcohol misuse. They may be self-medicating an underlying seasonal affective disorder (SAD) with alcohol or manifesting a seasonal pattern to alcohol-induced depression. Both genetic and environmental factors play a role in the etiology and pathogenesis of alcoholism and SAD, operating, at least in part, through the brain serotonergic system. Family and molecular genetic studies suggest that there may be a genetic link be-**

**tween seasonality and alcoholism. Certain environmental and social factors may contribute to the development of seasonality in patients with alcoholism. The fact that SAD and alcoholism may be comorbid shows the importance of a thorough diagnostic interview. Both mental health and drug and alcohol professionals should be provided with education to assist with appropriate identification, management, and referral of patients presenting with comorbid alcoholism and SAD.**

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**A**LCOHOL USE and alcohol-related problems are very common in Western societies.<sup>1,2</sup> For example, 90% of people in the United States drink alcohol.<sup>3</sup> Thirty percent or more drinkers develop temporary alcohol-related problems. Severe alcohol-related impairment (alcohol dependence) is observed at some time during their lives of approximately 10% of men and 3% to 5% of women, with an additional 5% to 10% of each sex developing persistent but less intense alcohol-related problems that are diagnosed as abuse.<sup>1</sup>

Alcoholism and depression are a prevalent combination of psychiatric disorders among individuals seeking treatment.<sup>4</sup> The Epidemiologic Catchment Area study reported a high concordance for alcoholism and mood disorders.<sup>5</sup> Among subjects with a lifetime history of alcoholism, 13.4% had a history of mood disorder. These disorders clustered together at a rate approximately two times higher (odds ratio = 1.9) than would be expected relative to the prevalence of each disorder in the general population. Among patients with a history of mood disorder, 21.8% met criteria for alcohol abuse or dependence at some points of their lives (odds ratio = 1.9); and among patients with bipolar disorder, the comorbidity for alcoholism was 81.6% (odds ratio = 3.7). Hasin and Grant<sup>6</sup> using data from the National Longitudinal Alcohol Epidemiologic Survey demonstrated that prior alcohol dependence increased the risk of current major depressive disorder more than fourfold.

## SEASONALITY AND SEASONAL AFFECTIVE DISORDER

Seasonal affective disorder (SAD), a condition where depressions in fall and winter alternate with nondepressed periods in the spring and summer, can be considered a distinct subtype of major de-

pression.<sup>7-10</sup> In the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)<sup>11</sup> SAD is listed as a specifier of either bipolar or recurrent major depressive disorder, with a seasonal pattern of major depressive episodes. The tendency to experience seasonal changes in mood and behavior, known as seasonality, is manifested to various degrees in a large segment of the population. Seasonality can be viewed as dimension ranging from absence of seasonal changes to extreme changes with the seasons. People with SAD are at the extreme end of this spectrum. Healthy individuals may have seasonal variations in mood and behavior. A telephone survey in the Washington area found that 92% of the survey subjects noticed seasonality to varying degrees.<sup>12</sup> For 27% of the sample seasonal changes were a problem, and 4.3% to 10% of subjects, depending on the case-finding definition, rated a degree of seasonal impairment equivalent to that of patients with SAD. A mail survey in New York, NY found about 6% with potential clinical severity, 18% reporting milder symptoms considered bothersome, and 35% noting symptoms but without complaint.<sup>13</sup> Most mail and telephone surveys to study the prevalence of SAD report much higher rates than face-to-face interviews.<sup>8</sup> For example, the combined prevalence of SAD and subsyndromal SAD was 3.1% over 2 consecutive years and 2.4% over 3 consecutive

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*From the Division of Neuroscience, Department of Psychiatry, Columbia University, New York, NY.*

*Address reprint requests to Leo Sher, M.D., Division of Neuroscience, Department of Psychiatry, Columbia University, 1051 Riverside Dr, Suite 2917, Box 42, New York, NY 10032.*

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years in a sample of 417 people interviewed twice during follow-up.<sup>14</sup>

Genetic predisposition may play an important role in the etiology of SAD.<sup>8,9,15</sup> Most studies of pathophysiology of SAD have focused on circadian rhythm and serotonergic abnormalities.<sup>7-10,15,16</sup> There is likely substantial heterogeneity in the etiology and pathophysiology of SAD. Light therapy has been the mainstay of treatment for SAD. The efficacy of light treatment has been demonstrated in many studies around the world.<sup>7-10</sup>

#### SEASONALITY AND ALCOHOLISM

Recent data suggest that seasonality may be closely related to alcoholism. Some patients with alcoholism have a seasonal pattern to their alcohol abuse.<sup>17</sup> Avery et al.<sup>18</sup> reported that during their research on SAD, they have had to exclude many potential subjects from their studies because those subjects were abusing alcohol or had a history of alcohol abuse. Anderson et al.<sup>19</sup> reported that, in an alcohol/substance abuse program, 23% of the patients had SAD. Patients with alcoholism may be self-medicating an underlying depression with alcohol, especially given the carbohydrate craving associated with SAD, or manifesting a seasonal pattern to alcohol-induced depression.

Bright light therapy has been reported to be more effective than a dim control in depressed detoxified alcoholics.<sup>20</sup> Avery et al.<sup>18</sup> suggested if some alcoholics attempt to self-medicate SAD with alcohol, or if SAD predisposes this population to alcohol relapse, then treatment of SAD with light therapy may be beneficial in preventing relapse into alcoholism in this population. Light therapy might be attractive to many abstinent alcoholics who are skeptical of drug therapy. Avery et al.<sup>18</sup> performed a controlled study to examine the effectiveness of dawn simulation in abstinent alcoholics with SAD and found that dawn simulation was helpful in decreasing depression in SAD patients with a history of alcoholism.

Family studies suggest that there is a link between alcoholism and SAD.<sup>18,21</sup> Using the family history method, Allen et al.<sup>21</sup> found that 41% of SAD patients had first-degree relatives with alcoholism compared with only 18% of non-SAD patients. However, alcoholism was not significantly overrepresented in the patients with SAD. Other authors reported that between 8% and 38% of patients with SAD had a family history of alcohol-

ism.<sup>13,22-26</sup> Avery et al.<sup>18</sup> have found that the incidence of alcoholism among first-degree relatives of patients with SAD (even those without a personal history of alcohol abuse) is greater than the incidence among blood relatives of controls (20% *v* 7.2%).

Both genetic and environmental factors play a role in the etiology and pathogenesis of alcoholism and SAD, operating, at least in part, through the brain serotonergic system.<sup>7,15,27-29</sup> Molecular genetic studies of seasonality and SAD have focused on serotonin and, especially, on the role of the serotonin transporter promoter repeat length polymorphism (5-HTTLPR) in the etiology of seasonality and SAD.<sup>15,28,29,30-34</sup> Serotonin transporter affects presynaptic reuptake of serotonin, terminating serotonergic neurotransmission and recycling supplies of serotonin. The serotonin transporter gene-linked polymorphic region (5-HTTLPR) is a common deletion/insertion polymorphism of the human serotonin transporter gene.

Rosenthal et al.<sup>30</sup> reported an association between the 5-HTTLPR and SAD. There was also an association between the 5-HTTLPR and seasonality in SAD patients. Sher et al.<sup>32,33</sup> reported that the 5-HTTLPR was associated with seasonality in a general population sample. It has been shown that the influence of the serotonin transporter gene on seasonality is largely independent of its effects on neuroticism.<sup>33</sup> Johansson et al.<sup>34</sup> found no association between the 5-HTTLPR and SAD or seasonality. The results of the studies by Rosenthal et al.<sup>30</sup> and by Johansson et al.<sup>34</sup> cannot be compared because of a considerable difference in the methodology. Likely, the serotonin transporter gene is involved in the biological mechanisms of seasonality.

Several research groups studied the possible association of the 5-HTTLPR (the same polymorphism as in seasonality studies) with alcoholism. Schmidt et al.,<sup>35</sup> Sander et al.,<sup>36,37</sup> and Hallikainen et al.<sup>38</sup> found that the frequency of the short allele is significantly increased in alcoholic patients with severe dependence as compared with nonalcoholic control subjects. Similar results were reported by Hammoumi et al.<sup>39</sup> and Lichtermann et al.<sup>40</sup> Thompson et al.<sup>41</sup> found a trend toward increased frequency of the short allele in alcohol-dependent subjects. Turker et al.<sup>42</sup> reported the existence of a significant association between the short allele of

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