



VITAMIN D AND MOOD DISORDERS

PEGGY NEPPS, PSY.D.

Clinical Psychologist

Coordinator, Behavioral Science

Assistant Director, Dept. of Family & Community Medicine, Lancaster General Hospital

The incidence of depression has been increasing throughout the last century. Although this may be due in part to improved diagnosis and treatment, it has also been suggested that reduced circulating levels of vitamin D brought on by the sunlight-deprivation of industrialized lifestyles may play a role.¹ Interest in a possible connection between vitamin D and mood disorders arose from the observation of an increased likelihood of low bone density among women with a history of depression. Michelson and coworkers studied 24 women with past or current major depressive disorder.² In comparison with controls matched for age, body-mass index, race and menopausal status, the patients with depressive disorders had a significantly lower mean bone density as measured at the femoral neck, Ward's triangle and the trochanter, even though their vitamin D levels were similar to those of the control patients.

Most of the research on vitamin D and mood disorders has been done in patients diagnosed with Seasonal Affective Disorder (SAD). SAD refers to a recurrent major depressive disorder characterized by lethargy, hypersomnia, hyperphagia, and carbohydrate craving that has its onset and remission at specific times of the year. Episodes most often begin in the fall or winter, a time of year when daily exposure to natural light (photoperiod) is shortest and body stores of vitamin D are typically lowest. SAD can be successfully treated with artificial, broad-spectrum light therapy at an intensity of 10,000 lux for 30 minutes a day. The mechanism by which phototherapy is effective is unknown. It has been suggested that seasonal changes in sunlight might affect circulating levels of vitamin D₃, which in turn could alter serotonin levels in the brain, and thus alter mood. If so, phototherapy (which would include enough of the 280-320 nanometer wavelengths of light needed to trigger vitamin D production) might work by improving vitamin D levels.

Research on the role of vitamin D in the etiology and treatment of mood disorders has yielded conflicting results. Harris & Dawson-Hughes³ conducted a 1-year prospective study of seasonal mood changes in women

who received 400 mg a day of Vitamin D supplements. Mood states in fall and winter were significantly worse than in spring and summer, but overall, mood was no better in supplemented patients than in unsupplemented controls, and mood scores showed no correlation with plasma levels of vitamin D. This study and others have been criticized, however, on the basis of the type of vitamin D used for supplementation (ergocalciferol vs. cholecalciferol) and the lack of direct measurements of vitamin D levels.

In contrast to the negative findings of the above study, Lansdowne & Provost⁴ found that, compared with a placebo control group, healthy subjects who received either 400 or 800 I.U. of vitamin D₃ daily for five days in winter had a significant increase in positive affect. In a prospective, randomized study of SAD patients, Gloth and coworkers⁵ compared light treatment (one week of daily full-spectrum light exposure) with a single dose of 100,000 I.U. of vitamin D₂ (ergocalciferol). Vitamin D supplementation was superior to light therapy in reducing symptoms of SAD, and in addition, vitamin D levels in both groups correlated with improvements in mood.

There is, however, an important difference between Gloth's study, with its finding that vitamin D has beneficial effects, and Harris & Dawson-Hughes' negative study; a difference that may both explain the differing results and provide support for the role of vitamin D in understanding SAD. While the study with negative results used healthy volunteers whose plasma levels of vitamin D were normal, Gloth found a beneficial effect for vitamin D in SAD patients whose vitamin D levels were low. It may be that supplementation above the normal level of vitamin D does not enhance mood in the way that correcting a deficient level does.

Vitamin D deficits, however, are not specific to SAD, even among psychiatric patients. Levels of some forms of vitamin D (25-hydroxyvitamin D₃ and 1,25-hydroxyvitamin D₃) have been shown to be lower not only in patients with mood disorders, but in patients with schizophrenia

and alcoholism as well. Schneider and coworkers⁶ posit that such deficits may be caused by dietary and lifestyle factors; patients with chronic psychiatric and substance abuse problems may have less-nutritious diets and fewer opportunities for exposure to natural light. Alternatively, given vitamin D's probable role in the synthesis of neurotrophic factors and neurotransmitters,⁷ a more direct effect is possible. Interestingly, SAD can also be successfully treated with serotonin reuptake inhibitors, which increase the availability of serotonin in the synapses of the brain. If vitamin D is involved in serotonin synthesis, that could provide an explanation for the role of vitamin D in the maintenance of normal mood.

More and better research is clearly needed to clarify the relationships among seasonal changes in mood, light exposure, and vitamin D levels. There is a need to study, among other things, the possible efficacy of combining light therapy with vitamin D supplementation for SAD. If vitamin D deficiency does prove to play a role in mood regulation, supplementation may offer a safe and inexpensive treatment option which also provides additional health benefits. Although it would be premature to recommend vitamin D as an intervention for depression, even the early evidence available thus far provides additional support for encouraging adequate levels of vitamin D year-round.

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Peggy Nepps, Psy.D.
 Clinical Psychologist
 Coordinator, Behavioral Science
 Assistant Director, Dept. of Family & Community Medicine
 Lancaster General Hospital
 555 N. Duke Street
 Lancaster, PA 17604
 717-544-1880
 mmnepps@lancastergeneral.org