

# USANA Technical Bulletin

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## Depression

### Description

- Mental depression is characterized by altered mood. There is loss of interest in all usually pleasurable activities such as food, sex, work, friends, hobbies, or entertainment.<sup>1</sup> Because short periods of disinterest are a normal part of life, this pattern must last for longer than two weeks for a person to be diagnosed with clinical depression.

### Causes

- The multiple causes of depression are not well understood. Currently, researchers believe that causes include genetic, familial, biochemical, physical, and social causes.

### Types

- Primary depression describes unipolar with no known cause other than family history.
- Secondary depression refers to symptoms that occur in response to a specific event or that have a recognizable organic basis.<sup>1</sup> For example Cushing's disease, cancer and many other diseases may prompt depression. Prescription drugs or drug abuse may also lead to depression. Hypercalcemia (excess blood levels of calcium) caused by hyperthyroidism may lead to depression.<sup>2</sup>

### At Risk

- People with a family history of depression have a higher risk, as do people with a history of drug and alcohol abuse.

### Prevention and Management

- Eating disorders are often associated with severe depression. It is important to recognize the potential for inadequate nutrition during a depressive stage. Some anti-depressive drugs are monoamine oxidase (MAO) inhibitors. These drugs increase levels of norepinephrine and serotonin.<sup>3</sup> Some foods (see below) also increase levels of these neurotransmitters and it may be beneficial to ensure that they are included in the diet.
- There is evidence that folate deficiency and depression are related.<sup>4,5</sup>
- Pyridoxine is necessary for the conversion of tryptophan to serotonin. Serotonin is a neurotransmitter. A deficiency of serotonin may cause depression.<sup>4</sup>

- A recent observational study indicated that vitamin B<sub>12</sub> levels were low in hospitalized psychiatric patients. The same study indicated that those patients with the lowest levels had more affective disorders (such as depression) than those with normal levels.<sup>6</sup>
- One of the first clinical symptoms of experimental scurvy in humans is depression.<sup>7</sup> Other observational studies also indicate low levels of vitamin C may contribute to depression in some cases.<sup>7</sup>
- Inositol may be helpful in reducing depression.<sup>8</sup>
- Magnesium may be helpful in reducing the symptoms of depression.<sup>9</sup>
- St. John's Wort contains hypericin which may increase norepinephrine, a neurotransmitter in the brain, that is an important mood regulator.<sup>10</sup>

## Sources of Additional Information

- <http://www.mhsource.com/edu/psytimes/title.html>

## Abstracts

**Fava M, Borus JS, Alpert JE, Nierenberg AA, Rosenbaum JF, Bottiglieri T. Folate, vitamin B12, and homocysteine in major depressive disorder. *Am J Psychiatry* 1997 Mar;154(3):426-8.** OBJECTIVE: The authors examined the relationships between levels of three metabolites (folate, vitamin B12, and homocysteine) and both depressive subtype and response to fluoxetine treatment in depressed patients. METHOD: Fluoxetine, 20 mg/day for 8 weeks, was given to 213 outpatients with major depressive disorder. At baseline, depressive subtypes were assessed, and a blood sample was collected from each patient. Serum metabolite levels were assayed. Response to treatment was determined by percentage change in score on the 17-item Hamilton Depression Rating Scale. RESULTS: Subjects with low folate levels were more likely to have melancholic depression and were significantly less likely to respond to fluoxetine. Homocysteine and B12 levels were not associated with depressive subtype or treatment response. CONCLUSIONS: Overall, the results are consistent with findings linking low folate levels to poorer response to antidepressant treatment. Folate levels might be considered in the evaluation of depressed patients who do not respond to antidepressant treatment.

## References

- <sup>1</sup> Taber's Cyclopedic Medical Dictionary. 16<sup>th</sup> ed. Philadelphia:F.A. Davis Company; 1985. p 48.
- <sup>2</sup> Joborn C et al. Psychiatric symptomatology in patients with primary hyperparathyroidism. *Ups J Med Sci* 1986;91(1):77-87.
- <sup>3</sup> Diseases. Springhouse (PA):Springhouse Corporation; 1993 p 50.
- <sup>4</sup> Werbach M. Nutritional Influences on Mental Illness. Tarzana (CA)Third Line Press; 1991 p 125.
- <sup>5</sup> Bailey L. Folate in Health and Disease. New York:Marcel Dekker; 1995 p 436.
- <sup>6</sup> Bell I et al. Vitamin B<sub>12</sub> and folate status in acute geropsychiatric inpatients: Affective and cognitive characteristics of a vitamin nondeficient population. *Biol Psychiatry* 1990;27(2):125-37.
- <sup>7</sup> Hodges RE et al. Clinical manifestations of ascorbic acid deficiency in man. *Am J Clin Nutr* 1971;24:432-43.
- <sup>8</sup> Levine J, Barak Y, Kofman O, Belmaker RH. Follow-up and relapse analysis of an inositol study of depression. *Isr J Psychiatry Relat Sci* 1995;32(1):14-21.
- <sup>9</sup> Hall RCW, Joffe JR. Hypomagnesemia: physical and psychiatric symptoms. *JAMA* 1973;224(13):1749-52.
- <sup>10</sup> Werbach M. Healing with Food. New York:Harper Perennial; 1993 p 105-106.