

USANA Technical Bulletin

Disclaimer: The information provided in this Technical Bulletin is strictly educational. It may not be used to promote USANA products nor is it intended as medical advice. For diagnosis and treatment of medical disorders, consult your health care professional. When there are references to third party websites, addresses and/or phone numbers, USANA, Inc. makes no claim, actual or implied, regarding the content or validity of the information obtained from these outside sources. This Technical Bulletin may be copied and freely distributed only if all text remains intact and unchanged.

Diabetes Mellitus

Description

- Diabetes mellitus is a chronic metabolic disorder that affects carbohydrate, protein, and fat metabolism. Carbohydrates are normally broken down in the body to glucose, the body's main source of energy. Insulin, a hormone produced in the pancreas, is essential for the transport of glucose into the cells for energy and also for glycogen storage. It also stimulates protein synthesis and free fatty acid storage in the adipose (fat) tissues. In diabetes, insufficient production of insulin or insensitivity to insulin impairs the body's ability to convert glucose to energy, and compromises the body cells' access to essential nutrients for fuel and storage.¹

Types

- Type I (juvenile) or insulin dependent diabetes mellitus is caused by the destruction of the insulin-producing beta cells in the pancreas. By the time the disease becomes apparent, up to 80% of the beta cells have been destroyed. This process is theorized to be the result of an autoimmune disorder.
- Type II (adult onset) or non-insulin dependent diabetes mellitus is a defect in insulin utilization. Normal amounts of insulin are made, but cannot be properly utilized by the body.¹

Complications

- Diabetics have a higher risk for developing various chronic illnesses that affect virtually all the body systems. The most common complications include kidney failure (diabetic nephropathy), blindness (diabetic retinopathy), peripheral nerve damage (diabetic neuropathy), heart attack, high blood pressure, arteriosclerosis (cardiovascular disease), gangrene, and foot ulcers.²

At Risk

- The tendency to develop diabetes can be hereditary, other factors that can contribute to the development of type II diabetes include diet and lifestyle, pregnancy, surgery, physical and emotional stress, and obesity.

Prevention and Management

- Diet and lifestyle are the most important factors for the prevention of non-insulin dependent diabetes and in management of insulin-dependent diabetes. Regular aerobic exercise, such as brisk walking, jogging, swimming, or bicycling, will improve how the body uses insulin and aids in the regulation of blood sugar and lipid levels.³
- A high fiber diet is associated with an improved ability to handle blood sugar.⁴ When the diet is high in fiber, cells are more sensitive to insulin and increase the number of insulin receptor sites for burning glucose.⁵
- Antioxidants, including vitamin E, vitamin C, beta-carotene, bioflavonoids, and B-complex vitamins can help protect against free radical damage.⁶
- Vitamin E levels in the blood of diabetics are lower than levels found in the blood of subjects without diabetes. Poor dietary intake of vitamin E may alter blood sugar levels, while an adequate intake may help to modulate blood sugar levels.⁷
- Vitamin C metabolism and tissue levels are altered in diabetes.⁸ Optimal vitamin C intake may help to regulate blood sugar and aid in the prevention of diabetes.⁹
- Minerals may also play a role in protecting against the damaging effects of diabetes. Diabetics tend to lose magnesium through their kidneys more than non-diabetics. Type I diabetics who get at least 450 mg of magnesium are able to improve insulin production and maintain better control of blood sugar levels.¹⁰
- Trace minerals such as chromium are essential for insulin and glucose metabolism.¹¹

Abstracts

Liu V, Abernathy R. Chromium insulin in young subjects with normal glucose tolerance. *AM J Clin Nutr* 1982;35:661-7. This study investigated the chromium-insulin relationship in young subjects who had normal glucose tolerance at different levels of insulin secretion during their glucose loading. Their findings were consistent with the hypothesis that chromium may facilitate sensitivity to insulin. When chromium was found to be low, there was an increase in abnormal glucose tolerance, which the authors felt could be reversed in some cases with chromium supplementation.

References

- ¹ Diseases. 2nd ed. Springhouse (PA):Springhouse Corporation; 1997 p 1036.
- ² Golan R. Optimal Wellness. New York:Ballantine Books; 1995 p 359-40.
- ³ O'Dea K. Marked improvement in carbohydrate and lipid metabolism in diabetic Australian aborigines after temporary reversion to traditional lifestyle. *Diabetes* 1984;33(6):596-603.
- ⁴ Jenkins DJA et al. Dietary fibers, fiber analogues, and glucose tolerance: importance of viscosity. *British Medical Journal* 1978;1:1392.
- ⁵ Fukagawa NK et al. High-carbohydrate, high fiber diets increase peripheral insulin sensitivity in healthy young and old adults. *American Journal of Clinical Nutrition* 1990 Sep;52(3):524-8.
- ⁶ Packer L. The Role of Anti-Oxidative Treatment in Diabetes. *Diabetologia* [Unvi. Of California, Berkley] 1993;36(11):1212-1213.
- ⁷ Bierenbaum M, Noon F, Machlin L et al. The effect of supplemental vitamin E on serum parameters in diabetic, post coronary and normal subjects. *Nutr. Rep. In.* 1985;31:1171-1180.
- ⁸ Stankova L. Plasma ascorbate concentrations and blood cell dehydroascorbate transport in patients with diabetes mellitus. *Metabol* 1984;33:347-353.
- ⁹ Chen L, Thacker R. Effects of dietary vitamin E and high supplementation of vitamin C on plasma glucose and cholesterol levels. *Nutr Res* 1985;5:527-534.

¹⁰ Paolisso G et al. Daily magnesium supplements improve glucose handling in elderly subjects. American Journal of Clinical Nutrition 1992;55:1161-1167.

¹¹ Liu V, Abernathy R. Chromium insulin in young subjects with normal glucose tolerance. AM J Clin Nutr 1982;35:661-7.