

- Branca D, Scutari G, Siliprandi N. Pantethine and pantothenate effect on the CoA content of rat liver. *Internat J Vit Nutr Res.* 1984; 54:211-216.
- Carrara P, Matturri L, Galbussera M, et al. Pantethine reduces plasma cholesterol and the severity of arterial lesions in experimental hypercholesterolemic rabbits. *Atherosclerosis.* 1984; 53:255-264.
- Cighetti G, Del Puppo M, Paroni R, et al. Pantethine inhibits cholesterol and fatty acid syntheses and stimulates carbon dioxide formation in isolated rat hepatocytes. *J Lipid Res.* 1987; 28:152-161.
- Clark JJ, Livesey JC, Steele JE. Delay or inhibition of rat lens opacification using pantethine and WR-77913. *Exp Eye Res.* 1996; 62:75-84.
- Congdon NT, West ST, Duncan DT, et al. The effect of pantethine and ultraviolet-B radiation on the development of lenticular opacity in the emory mouse. *Curr Eye Res.* 2000; 20:17-24.
- Donati C, Barbi G, Cairo G, et al. Pantethine improves the lipid abnormalities of chronic hemodialysis patients: results of a multicenter clinical trial. *Clin Nephrol.* 1986; 25:70-74.
- Donati C, Bertieri RS, Barbi G. [Pantethine, diabetes mellitus and atherosclerosis. Clinical study of 1045 patients]. [Article in Italian]. *Clin Ter.* 1989; 128:411-422.
- Friberg G, Pande J, Ogun O, Benedek GB. Pantethine inhibits the formation of high-Tc protein aggregates in gamma B crystallin solutions. *Curr Eye Res.* 1996; 15:1182-1190.
- Gaddi A, Descovich GC, Nosedà G, et al. Controlled evaluation of pantethine, a natural hypolipidemic compound, in patients with different forms of hyperlipoproteinemia. *Atherosclerosis.* 1984; 50:73-83.
- Galeone F, Scalabrino A, Giuntoli F, et al. The lipid-lowering effect of pantethine in hyperlipidemic patients: a clinical investigation. *Curr Ther Res.* 1983; 34:383-390.
- Gensini GF, Prisco D, Rogasi PG, et al. Changes in fatty acid composition of the single platelet phospholipids induced by pantethine treatment. *Int J Clin Pharm Res.* 1985; 5:309-318.
- Hayashi H, Kobayashi A, Terada H, et al. Effect of pantethine on action potential of canine papillary muscle during hypoxic perfusion. *Jap Heart J.* 1985; 26:289-296.
- Hiramatsu N, Kishida T, Hamano T, Nataka M. Effects of dietary pantethine levels on contents of fatty acids and thiobarbituric acid reactive substances in the liver of rats orally administered varying amounts of autoxidized linoleate. *J Nutr Sci Vitaminol (Tokyo).* 1991; 37:73-87.
- Hoffman B, Lang A, Ostermann G, et al. Effect of pantethine on platelet functions *in vitro*. *Curr Ther Res.* 1987; 41:791-801.
- Iida J, Nishimura K, Ueki S, Azuma I. Macrophage activation with pantethine and pantetheine-4'-phosphate. *Int J Vitam Nutr Res.* 1985; 55:405-411.
- Kumerova AO, Silova AA, Utno Lia. [Effect of pantethine on post-heparin lipolytic activity and lipid peroxidation in the myocardium]. [Article in Russian]. *Biull Eksp Biol Med.* 1991; 111:33-35.
- Maggi GC, Donati C, Criscuoli G. Pantethine: a physiological lipomodulating agent in the treatment of hyperlipidemias. *Curr Ther Res.* 1982; 32:380-386.
- Nagi-Ostaszewski I, Lau-Cam CA. Protection by pantethine, pantothenic acid and cystamine against carbon tetrachloride-induced hepatotoxicity in the rat. *Res Commun Chem Pathol Pharmacol.* 1990; 67:289-292.
- Prisco D, Rogasi PG, Matucci M, et al. Effect of pantethine treatment on platelet aggregation and thromboxane A<sub>2</sub> production. *Curr Ther Res.* 1984; 35:700-706.
- Vecsei L, Alling C, Widerlov E. Comparative studies of intracerebroventricularly administered cysteamine and pantethine in different behavioral tests and on brain catechols in rats. *Arch Int Pharmacodyn Ther.* 1990; 305:140-151.
- Watanabe A, Hobara BS, Kobayashi M, et al. Lowering of blood acetaldehyde but not ethanol concentrations by pantethine following alcohol ingestion: different effects in flushing and nonflushing subjects. *Alcoholism: Clin Exper Res.* 1985; 9:272-276.
- Webster MJ. Physiological and performance responses to supplementation with thiamin and pantothenic acid derivatives. *Eur J Appl Physiol.* 1998; 77:486-491.
- Wittwer CT, Graves CP, Peterson MA, et al. Pantethine lipomodulation: evidence for cysteamine mediation *in vitro* and *in vivo*. *Atherosclerosis.* 1987; 68:41-49.
- Yoon SB, Kajiyama K, Ogura R. [Effect of pantethine on adriamycin-induced cardiotoxicity]. [Article in Japanese]. *Kurume Igakkai Zasshi.* 1982; 45:598-606.

## Pantothenic Acid

### TRADE NAMES

Pantothenic acid is available generically from numerous manufacturers. Branded products include: Panto-250 (Bio-Tech Pharmacal).

### DESCRIPTION

Pantothenic acid, a member of the B-vitamin family, is an essential nutrient in human nutrition. It is sometimes referred to as vitamin B<sub>5</sub>. Pantothenic acid is involved in a number of biological reactions, including the production of energy, the catabolism of fatty acids and amino acids, the synthesis of fatty acids, phospholipids, sphingolipids, cholesterol and steroid hormones, and the synthesis of heme and the neurotransmitter acetylcholine. It also appears to be involved in the regulation of gene expression and in signal transduction. Roger J. Williams, the discoverer of pantothenic acid and a scientist who pioneered the use of nutrients for the prevention and treatment of disease, thought that pantothenic

acid might be helpful in the management of certain medical disorders, such as rheumatoid arthritis.

The term pantothenic acid is derived from the Greek word *pantos*, meaning everywhere. Pantothenic acid is widely distributed in plant and animal food sources, where it occurs in both bound and free forms. Rich sources of the vitamin, include organ meats (liver, kidney), egg yolk, avocados, cashew nuts and peanuts, brown rice, soya, lentils, broccoli and milk. Royal jelly and brewer's yeast, both of which are used as nutritional supplements, are two of the richest sources of pantothenic acid. The richest sources of the vitamin are the ovaries of cod and tuna. Pantothenic acid is synthesized by intestinal microflora and this may also contribute to the body's pantothenic acid requirements.

Pantothenic acid deficiency in humans is rare. Symptoms of pantothenic acid deficiency, which has occurred under conditions of severe malnutrition, include numbness in the toes and painful burning in the feet (melalgia). Experimentally-induced pantothenic acid deficiency in humans, produced headache, fatigue, insomnia, intestinal disturbances, paresthesias of the hands and feet, impaired antibody production, an elevated sedimentation rate and an impaired eosinopenic response to ACTH. Most of these symptoms and signs resolved with administration of pantothenic acid.

The principal biologically active forms of pantothenic acid are coenzyme A (CoA) and acyl carrier protein (ACP). In both CoA and ACP, the business center of the molecule is the pantothenic acid metabolite 4'-phosphopantetheine. Coenzyme A is comprised of 4'-phosphopantetheine linked by an anhydride bond to the nucleotide adenosine 5'-monophosphate. 4'-Phosphopantetheine itself is comprised of pantothenic acid linked at one end, via an amide bond, to beta-mercaptoethylamine, derived from L-cysteine, and at the other end to a phosphate group. The sulfhydryl group of 4'-phosphopantetheine, which is the business end of the coenzyme, forms thioesters with acyl groups producing acyl-CoA derivatives, including acetyl-CoA.

Acetyl-CoA is produced via beta-oxidation of fatty acids, via the metabolism of carbohydrates—glucose 6-phosphate to pyruvate to acetyl-CoA—and via the catabolism of amino acids. Acetyl-CoA has a number of metabolic opportunities. It is metabolized in the tricarboxylic acid cycle to produce carbon dioxide, water and energy. It can also be metabolized to fatty acids, cholesterol and steroid hormones. Acetyl-CoA also participates in a number of acetylation reactions, including the formation of acetylcholine, melatonin, N-acetylglucosamine, N-acetylgalactosamine and N-acetylneuraminic acid. Finally, acetyl-CoA is involved in the acetylation of proteins and peptides. Histone acetylation is an epigenetic mechanism of gene regulation. In general, chro-

matin fractions enriched in actively transcribed genes are also enriched in highly acetylated core histones, whereas silent genes are associated with nucleosomes with a low level of acetylation. Nucleosomes are the fundamental units of chromosomes.

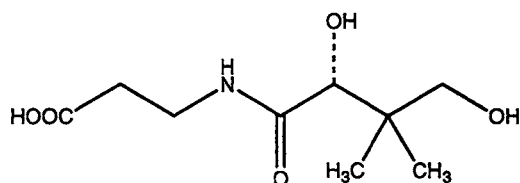
The other major form of pantothenic acid is acyl carrier protein or ACP. Acyl carrier protein (ACP) functions as a coenzyme in the fatty acid synthetase complex which is central to the *de novo* synthesis of fatty acids. The prosthetic group of acyl carrier protein is again, 4'-phosphopantetheine. 4'-Phosphopantetheine binds to acyl carrier protein via a phosphodiester linkage to a serine residue of acyl carrier protein. The function of ACP in fatty acid synthesis is analogous to that of coenzyme A in the beta-oxidation of fatty acids. ACP serves as an anchor to which the acyl intermediates are esterified. The acyl intermediates are esterified to the sulfhydryl group of 4'-phosphopantetheine. 4'-Phosphopantetheine is added to ACP in a posttranslational modification reaction which is catalyzed by a transferase enzyme acting on CoA. In the case of coenzyme A, the sulfhydryl groups are also esterified to acyl groups, such as the acetyl group. However, the 4'-phosphopantetheine part of the structure is not esterified to a serine residue in a protein (ACP), but is bound to the nucleotide adenosine 5'-monophosphate.

In addition to acetyl-CoA, other forms of CoA also play important biological roles. Malonyl-CoA supplies two-carbon units for the synthesis of fatty acids up until palmitic acid, and succinyl-CoA reacts with delta-aminolevulinic acid in the first reaction of heme biosynthesis. Myristoyl-CoA, derived from the 14-carbon saturated fatty acid myristic acid, is involved in the myristoylation of proteins; palmitoyl-CoA is involved in the palmitoylation of proteins, and farnesyl-CoA and geranylgeranyl-CoA are involved in protein isoprenylation. Protein isoprenylation, myristoylation and palmitoylation appear to play roles in signal transduction, among other biological activities.

The principal supplemental form of pantothenic acid is calcium D-pantothenate (D-calcium pantothenate). This marketed supplement is usually made synthetically. Dexpantenol, the corresponding alcohol of pantothenic acid is also available. Dexpantenol is a synthetic form which is not found naturally. Dexpantenol is converted to pantothenic acid in the body, and therefore can be considered a provitamin form of pantothenic acid. Dexpantenol is used topically to promote wound healing. It is also used in various cosmetic products.

In addition to being known as vitamin B<sub>5</sub>, pantothenic acid is also known as D(+)-pantothenic acid, D-pantothenic acid, D(+)-N-(2,4-dihydroxy-3,3-dimethylbutyryl)-beta-alanine

and (*R*)-*N*-(2,4-dihydroxy-3,3-dimethyl-1-oxobutyl)-beta-alanine. D-pantothenic acid is the biologically active enantiomer of the vitamin and is comprised of beta-alanine and a dihydroxy acid called pantoic acid. Its molecular formula is  $C_9H_{17}NO_5$ , its molecular weight is 219.24 daltons and its structural formula is as follows:



Pantothenic Acid

Dexpanthenol, the corresponding alcohol of pantothenic acid, is also known as pantothenol, provitamin B<sub>5</sub> and (*R*)-2,4-dihydroxy-*N*-(3-hydroxypropyl)-3,3-dimethylbutyramide. Its molecular formula is  $C_9H_{19}NO_4$  and its molecular weight is 205.3 daltons.

#### ACTIONS AND PHARMACOLOGY

##### ACTIONS

Pantothenic acid may have antioxidant and radioprotective activities. It has putative anti-inflammatory, wound healing and antiviral activities. It also has putative activity in the management of rheumatoid arthritis.

##### MECHANISM OF ACTION

Pantothenic acid and its derivatives, 4'-phosphopantothenic acid, pantothenol and pantethine, have been shown, *in vitro*, to protect cells against lipid peroxidation. This protective effect does not appear to be due to the scavenging of the reactive oxygen species by pantothenic acid. It is thought that the antioxidant effect of pantothenic acid is due to its stimulation of increased cellular levels of coenzyme A. Coenzyme A may facilitate removal of lipid peroxides by increasing mobilization of fatty acids, and promote repair of plasma membranes by activating phospholipid synthesis. Pantothenic acid has also been shown to increase levels of cellular reduced glutathione. The mechanism by which pantothenic acid increases glutathione levels is unknown. However, increased levels of glutathione may play a large role in the protective effect of pantothenic acid against peroxidative damage of cell membranes.

Pantothenol has been demonstrated to protect rats against some of the deleterious effects of gamma radiation. The deleterious effects of gamma radiation occur via the generation of reactive oxygen species resulting in the peroxidation of lipid membranes. It is thought that the protective effects of pantothenol, which is a precursor of pantothenic acid, is due, in part, to its promotion of coenzyme A biosynthesis and to

its increasing cellular levels of reduced glutathione (see above).

There is some evidence that pantothenic acid may be helpful in the management of some with rheumatoid arthritis. The mechanism of this putative effect is unclear. Activated granulocytes play a role in the inflammatory response by production of reactive oxygen species. Pantothenic acid, in the form of calcium D-pantothenate, was found to significantly inhibit the release of myeloperoxidase from granulocytes *in vitro*, as well as to inhibit the production of reactive oxygen species by these cells. This effect of pantothenic acid as well as the antioxidant effect of the vitamin discussed above, may account, in part, for the putative action of pantothenic acid in rheumatoid arthritis.

Pantothenic acid has been shown to accelerate wound healing in experimental animals. The mechanism of the putative wound healing effect of pantothenic acid is unclear. In human dermal fibroblasts in culture, calcium D-pantothenate was demonstrated to accelerate the wound healing process by increasing the number of cells migrating into a wounded area, as well as their mean migration speed. Dexpanthenol (pantothenol), the corresponding alcohol of pantothenic acid, is used topically for the treatment of various minor skin disorders and to promote wound healing. Topical dexpanthenol has been found to improve stratum corneum hydration, reduce transepidermal water loss and to stabilize the epidermal barrier function. The putative wound healing activity of pantothenic acid, may also be accounted for, in part, by its possible antiinflammatory activity.

##### PHARMACOKINETICS

Dietary sources of pantothenic acid include bound and unbound forms. The principal bound form of the vitamin is coenzyme A. The principal supplementary form of the vitamin is calcium D-pantothenate. Dietary coenzyme A is hydrolyzed in the intestine to dephospho-CoA, phosphopantetheine and pantetheine. Pantetheine, in turn, is hydrolyzed to pantothenic acid. Pantothenic acid is efficiently absorbed from the small intestine. Absorption at low intakes occurs via a sodium-dependent carrier-mediated active transport process, and at higher intakes, by passive diffusion. Pantothenic acid is transported via the portal circulation to the liver and via the systemic circulation to other tissues of the body. Uptake of pantothenic acid by most cells of the body is by a sodium-dependent process.

Pantothenic acid is metabolized to coenzyme A via a sequence of steps. Coenzyme A is a precursor of acyl carrier protein. Approximately 95% of CoA is found in the mitochondria. Coenzyme A is catabolized by a number of hydrolytic steps resulting in pantothenate and cysteamine. Unchanged pantothenate is the major urinary excretion

product of pantothenic acid. In dogs, a glycosylated catabolite of the vitamin, pantothenyl-4'-beta-glucoside, has been identified in the urine. Glycosylated catabolites of pantothenic acid have not, to date, been reported in humans.

#### INDICATIONS AND USAGE

There is some dated evidence that pantothenic acid may be of some benefit in some with rheumatoid arthritis. Research results are mixed, but overall not encouraging, with respect to claims that pantothenic acid enhances exercise performance. There is some animal and *in vitro* evidence that pantothenic acid may aid in wound healing. However, one human clinical study did not show a wound healing effect for oral pantothenic acid. A topical form of the provitamin, dexpantenol (pantothanol) is used for the treatment of minor skin disorders, including for the promotion of wound healing. There is preliminary evidence that pantothenic acid may be helpful in treating those with hepatitis A and a suggestion from animal studies that it may be helpful in some with Duchenne muscular dystrophy. There is no evidence that it prevents loss of hair and graying of hair.

#### RESEARCH SUMMARY

Decades ago there were reports that supplemental pantothenic acid could ameliorate some bone and cartilage disorders in acutely pantothenic acid-deficient young rats. Years later, it was noted that blood levels of pantothenic acid are significantly reduced in rheumatoid arthritis (RA) patients. A clinical trial tested 50 milligrams daily of injected calcium pantothenate. Blood levels rose to normal, and significant symptomatic relief was achieved in many of the test subjects. When the pantothenate was withdrawn, symptoms returned. The best results were achieved in a subgroup of vegetarians, and still better results were reported in vegetarians who were given a combination of pantothenic acid and royal jelly. (See Royal Jelly). This study was conducted in 1963.

In 1980, a double-blind, placebo-controlled study followed-up on the initial report. Subjects with various forms of arthritis were randomized to receive oral calcium pantothenate or placebo. Dosage was as follows: one tablet (500 milligrams) daily for two days, then one twice a day for three days, then one three times a day for four days and, finally, one four times a day thereafter.

Calcium pantothenate was no better than placebo in treating all forms of arthritis—except RA. In the subset of subjects with RA, pantothenic acid produced significant results. The researchers noted, in particular, that “highly significant effects were recorded for calcium pantothenate in reducing the duration of morning stiffness, degree of disability and severity of pain, whereas placebo produced no effects on these symptoms.” As these researchers noted, further investigation is warranted.

There have been reports that pantothenic acid speeds wound healing in animal models. However, recent, double-blind, prospective, randomized trial found “no major improvement” of the human skin wound healing process. This 21-day study tested a combination of 1 gram of ascorbic acid and 0.2 grams of pantothenic acid daily on 49 subjects. Further investigation is needed to determine if oral pantothenic acid has any role in wound healing. A topical form of the provitamin, pantothanol, appears to play some role in the management of minor skin disorders.

Claims that pantothenic acid enhances exercise performance rest, primarily, in the results of one study in which experienced distance runners received 2 grams of pantothenic acid daily for 14 days. Their performance was significantly better than that of equally well-trained distance runners who received placebo.

In another study, however, 1 gram of pantothenic acid daily for two weeks did not enhance the performance of distance runners compared with distance runners given placebo.

More recently, highly trained cyclists performed no better with a combination of 1 gram of allithiamin and 1.8 grams of a 55%/45% pantethine/pantothenic acid mixture than they did with placebo. The substances were administered for seven days prior to each exercise performance test. The researchers concluded: “we found the oral administration of these compounds to have no effect on any physiological or performance parameters during steady-state or high-intensity exercise.”

There is preliminary evidence that pantothenic acid, in combination with pantetheine, may be helpful in the treatment of hepatitis A. Pantetheine is thought to be more active in this respect than pantothenic acid.

Administration of pantothenic acid increased skeletal muscle energy metabolism in the murine model of Duchenne muscular dystrophy, the mdx mouse. Reduced energy metabolism in slow- and fast-twitch skeletal muscle fibers has been reported in the mdx mouse. It is thought that this is due to a decreased oxidative utilization of glucose and free fatty acids. It is speculated that inefficiency of coenzyme A transport in the mitochondria might account for the reduced energy metabolism. Administration of pantothenic acid to mdx mice increased the cytoplasmic synthesis of CoA, and increased the thermogenic response to glucose, more in the muscles of mdx mice than in control muscles. Human studies are warranted.

#### CONTRAINDICATIONS, PRECAUTIONS, ADVERSE REACTIONS

##### CONTRAINDICATIONS

Pantothenic acid is contraindicated in those hypersensitive to any component of a pantothenic acid-containing product.

Dexpanthenol (pantothenol), the alcohol analog of pantothenic acid, is contraindicated in those hypersensitive to any component of a dexpanthenol-containing product. Oral and parenteral dexpanthenol are also contraindicated in those with ileus due to mechanical obstruction and those with hemophilia.

**PRECAUTIONS**

Pregnant women and nursing mothers should avoid doses of pantothenic acid greater than the U.S. RDA (10 milligrams/day) unless a higher dosage is prescribed by their physicians.

Those who have developed contact dermatitis from use of dexpanthenol may develop eczema from the use of oral pantothenic acid. Dexpanthenol, also known as pantothenol and provitamin B<sub>5</sub>, in addition to being used for the treatment of various minor skin disorders, is an ingredient in many cosmetic products.

The use of pantothenic acid for any medical condition must be medically supervised.

**ADVERSE REACTIONS**

Contact dermatitis has been reported with topical use of dexpanthenol.

**INTERACTIONS**

**NUTRITIONAL SUPPLEMENTS**

**Biotin:** High doses of pantothenic acid may inhibit the absorption of biotin produced by the microflora in the large intestine. Pantothenic acid and biotin appear to use the same uptake carrier in colonocytes.

**OVERDOSAGE**

There are no reports of pantothenic acid overdosage in the literature.

**DOSAGE AND ADMINISTRATION**

The principal form of supplementary pantothenic acid is calcium D-pantothenate. Calcium D-pantothenate is available in multivitamin, multivitamin/multimineral and B-complex products, as well as single ingredient products. Typical doses of pantothenic acid range from 10-50 milligrams/day. Single ingredient tablets and capsules of pantothenic acid are available in doses ranging from 100 to 500 milligrams.

The Food and Nutrition Board of the Institute of Medicine of the U.S. National Academy of Sciences has recommended the following Dietary Reference Intakes (RDI) for pantothenic acid:

Infants	Adequate Intakes (AI)
0 through 6 months	1.7 milligrams/day ≈ 0.2 mg/Kg
7 through 12 months	1.8 milligrams/day ≈ 0.2 mg/Kg
Children	
1 through 3 years	2 milligrams/day

4 through 8 years	3 milligrams/day
Boys	
9 through 13 years	4 milligrams/day
14 through 18 years	5 milligrams/day
Girls	
9 through 13 years	4 milligrams/day
14 through 18 years	5 milligrams/day
Men	
19 years and older	5 milligrams/day
Women	
19 years and older	5 milligrams/day
Pregnancy	
14 through 50 years	6 milligrams/day
Lactation	
14 through 50 years	7 milligrams/day

There is no evidence of toxicity associated with the ingestion of pantothenic acid. Therefore, a lowest-observed-adverse-effect level (LOAEL) and an associated no-observed-adverse-effect level (NOAEL) cannot be determined.

The optimal intakes of pantothenic acid are not known.

The U.S. RDA for pantothenic acid, which is used for determining percent daily values on nutritional supplement and food labels, is 10 milligrams/day.

**HOW SUPPLIED**

*Capsules* — 100 mg, 250 mg, 500 mg

*Capsules, Extended Release* — 1000 mg

*Liquid* — 200 mg/5 mL

*Tablets* — 100 mg, 200 mg, 250 mg, 500 mg, 1000 mg

*Tablets, Extended Release* — 500 mg, 1000 mg

**LITERATURE**

Anon. Calcium pantothenate in arthritic conditions. A report from the General Practitioner Research Group. *Practitioner*. 1980; 224:208-211.

Aprahamian M, Dentinger A, Stock-Damge C, et al. Effects of supplemental pantothenic acid on wound healing: experimental study in rabbit. *Am J Clin Nutr*. 1985; 41:578-589.

Barton-Wright EC, Elliot WA. The pantothenic acid metabolism of rheumatoid arthritis. *Lancet*. 1963; 2:862-863.

*Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B<sub>6</sub>, Folate, Vitamin B<sub>12</sub>, Pantothenic Acid, Biotin, and Choline*. Washington, DC: National Academy Press; 1998.

Even PC, Decrouy A, Chinet A. Defective regulation of energy metabolism in mdx-mouse skeletal muscles. *Biochem J*. 1994; 304:649-654.

Gehring W, Gloor M. Effect of topically applied dexpanthenol on epidermal barrier function and stratum corneum hydration. Results of a human *in vivo* study. *Arzneimittelforschung*. 2000; 50:659-663.

Hahn C, Roseler S, Fritzsche R, et al. Allergic contact reaction to dexpanthenol: lymphocyte transformation test and evidence for microsomal-dependent metabolism of the allergen. *Contact Dermatitis*. 1993; 28:81-83.

Kapp A, Zeak-Kapp G. Effect of Ca-pantothenate on human granulocyte oxidative metabolism. *Allerg Immunol (Leipzig)*. 1991; 37:145-150.

Kehrl W, Sonnemann U. [Dexpanthenol nasal spray as an effective therapeutic principle for treatment of rhinitis sicca anterior]. [Article in German]. *Laryngorhinootologie*. 1998; 77:506-512.

Komar VI. [The use of pantothenic acid preparations in treating patients with viral hepatitis A]. [Article in Russian]. *Ter Arkh*. 1991; 63:58-60.

Kumerova AO, Utmo LIa, Lipsberga ZE, Shkestere IIa. [Study of pantothenic acid derivatives as cardiac protectors in a model of experimental ischemia and reperfusion of the isolated heart]. [Article in Russian]. *Biull Eksp Biol Med*. 1992; 113:373-375.

Litoff D, Scherzer H, Harrison J. Effects of pantothenic acid supplementation on human exercise. *Med Sci Sports Exercise*. 1985; 17:287(Abstract 17).

Loftus EV Jr, Tremaine WJ, Nelson RA, et al. Dexpanthenol enemas in ulcerative colitis: a pilot study. *Mayo Clin Proc*. 1997; 72:616-620.

Moiseenok AG, Dorofeev BF, Omel'ianchik SN. [The protective effect of pantothenic acid derivatives and changes in the system of acetyl CoA metabolism in acute ethanol poisoning]. [Article in Russian]. *Farmakol Toksikol*. 1988; 51:82-86.

Nice C, Reeves AG, Brinck-Johnsen T, Noll W. The effects of pantothenic acid on human exercise capacity. *J Sports Med*. 1984; 24:26-29.

Plesofsky-Vig N. Pantothenic acid. In: Shils, ME, Olson JA, Shike M, Ross AC, eds. *Modern Nutrition in Health and Disease*. 9th ed. Baltimore MD: Williams and Wilkins; 1999:423-432.

Rychlik M. Quantification of free and bound pantothenic acid in foods and blood plasma by a stable isotope dilution assay. *J Agric Food Chem*. 2000; 48:1175-1181.

Sachs M, Asskali F, Lanaros C, et al. [The metabolism of panthenol in patients with postoperative intestinal atony]. [Article in German]. *Z Ernahrungswiss*. 1990; 29:270-283.

Slyshenkov VS, Moiseenok AG, Wojtczak L. Noxious effects of oxygen reactive species on energy-coupling processes in Ehrlich ascites tumor mitochondria and the protection by pantothenic acid. *Free Rad Biol Med*. 1996; 20:793-800.

Slyshenkov VS, Omelyanchik SN, Moiseenok AG, et al. Panthenol protects rats against some deleterious effects of gamma radiation. *Free Rad Biol Med*. 1998; 24:894-899.

Slyshenkov VS, Rakowska M, Moiseenok AG, Wojtczak L. Pantothenic acid and its derivatives protect Ehrlich ascites tumor cells against lipid peroxidation. *Free Rad Biol Med*. 1995; 19:767-772.

Slyshenkov VS, Rakowska M, Wojtczak L. Protective effect of pantothenic acid and related compounds against permeabilization of Ehrlich ascites tumor cells by digitonin. *Acta Biochim Pol*. 1996; 43:407-410.

Vaxman F, Olender S, Lambert A, et al. Effect of pantothenic acid and ascorbic acid supplementation on human skin wound healing process. A double-blind, prospective and randomized trial. *Eur Surg Res*. 1995; 27:158-166.

Webster MJ. Physiological and performance responses to supplementation with thiamin and pantothenic acid derivatives. *Eur J Appl Physiol*. 1998; 77:486-491.

Weimann BI, Hermann D. Studies on wound healing: effects of calcium-D-pantothenate on the migration, proliferation and protein synthesis of human dermal fibroblasts in culture. *Int J Vitam Nutr Res*. 1999; 69:113-119.

Williams RJ. *Biochemical Individuality*. New York, NY: John Wiley and Sons, Inc; 1963.

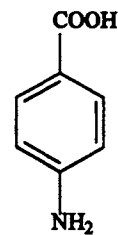
## Para-Aminobenzoic Acid (PABA)

### TRADE NAMES

PABA and para-aminobenzoate potassium are available from numerous manufacturers generically. Branded products for para-aminobenzoate potassium include M2 Potassium (Miller Pharmacal) and Potaba (Glenwood).

### DESCRIPTION

Para-aminobenzoic acid or PABA is a non-protein amino acid that is widely distributed in nature. It is sometimes referred to as vitamin Bx, but it is neither a vitamin nor an essential nutrient for humans. PABA is an intermediate in the synthesis of folic acid in bacteria. The sulfonamide antibiotics are structurally similar to PABA and interfere with the synthesis of nucleic acids in sensitive microorganisms by blocking the conversion of PABA to the coenzyme dihydrofolic acid, a reduced form of folic acid. In humans, dihydrofolic acid is obtained from dietary folic acid; thus sulfonamides do not affect human cells. PABA is also known as 4-aminobenzoic acid. It is a solid substance with slight solubility in water. Its chemical structure is:



PABA (p-Aminobenzoic acid)