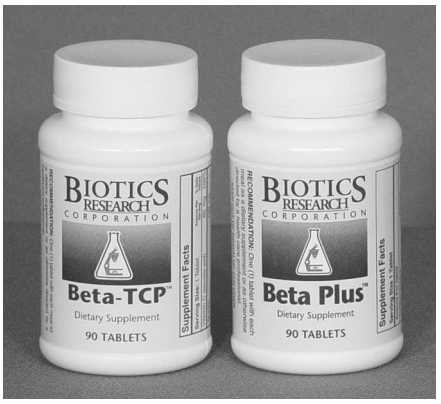
Biotics Research Corporation

Technical Support

Products #1210 & 1215

Beta Plus™ - Beta-TCP™

Nutritional Support for Bile Production and Biliary Stasis



In the 16th century, Paracelsus introduced the concept of the tartaric diseases to explain how stones are formed in the human body by the precipitation of substances from body fluids, analogous to the deposition of tartar in wine casks. Today we know that in industrialized countries more than 80% of gallstones consist mainly of cholesterol, the prevalence of gallstones is about 10%, and in people between 40 and 50 years of age the 5-year incidence is about 3%.

Bile, Defined

Bile functions as the body's emulsifying agent, critical for fat digestion and assimilation. Bile is produced by the liver, and is temporarily stored in the gall bladder. Bile is released into the small intestine in response to hormones, such as cholecystokinin, when fat enters the intestine.

Bile consists of a mixture of bile salts and bile acids, cholesterol, bilirubin and phospholipids, (chiefly phosphatidylcholine). Electroplytes are present at levels found in serum. The ratios of individual lipids are critical to maintain a stable micellar liquid. The molar ratios are typically 5:15:80 for cholesterol/phosphatidylcholine/bile salts. If the bile concentration becomes too high, cholesterol will precipitate and gallstones will form in the gall bladder, a condition known as cholelithiasis.[1]

Bile Formation

Bile salts and acids represent oxidized derivatives of cholesterol. About 80% of the cholesterol in the body will eventually be disposed of as cholic acid. The primary bile acids, cholic acid and chenodeoxycholic acid, possess a carboxylic acid side chain, which confers hydrophilic properties to the lipophilic sterol ring and creates detergent-like molecules. The liver attaches taurine and glycine to bile acids to create bile salts (taurocholate or taurochenodeoxycholate, and glycocholate or glycodeoxycholate, respectively). Bacterial enzymes in the colon can convert these to secondary bile acids, deoxycholate and lithocholate.

Bile and Digestion

Bile is needed for efficient uptake of oily nutrients. When bile acids and bile salts first encounter ingested fats, they act as emulsifiers to create suspensions, which can be broken down enzymatically. The process involves several important steps:

1. The combined action of bile salts and pancreatic lipase initiates hydrolysis of triglycerides to free fatty acids and diglycerides, resulting in the formation of emulsions containing other lipid-soluble nutrients, including vitamins and carotenoids. The particle size of these emulsions ranges from 200 to 5000 nm in diameter.

2. Lipase is then able to hydrolyze both di- and triglycerides to monoglycerides and free fatty acids. Lipase requires a smaller protein called colipase, another pancreatic product, in order to bind to triglycerides and activate the lipase.

3. Upon further release of bile salts, the lipid aggregates become smaller, from 3 to 10 nm in diameter. The normal endpoint of triglyceride digestion is a product containing 70% free fatty acid anions, 25% beta monoglycerides, and 5% cholesterol. The micelles are then taken up by the epithelial cells of the brush border membrane via passive diffusion. After absorption, the fate of fatty acids depends upon their sizes. Medium chain fatty acids, with less than 10-12 carbons, pass directly from the mucosal cells into the portal blood and bind to serum albumin. Longer chain fatty acid anions are re-esterified with beta monoglycerides in the smooth endoplasmic reticulum to reform triglycerides. The newly synthesized triglycerides are then complexed with apoproteins, cholesterol and phospholipids, to produce particles called chylomicrons. Chylomicrons are released from mucosal cells by exocytosis and enter the lymph, rather than entering the bloodstream directly.

Enterohepatic Circulation

Bile salts do not cross the mucosal barrier into the lymphatic system, but rather they are reabsorbed as micelles in the lower region of the small intestine. Most of the bile salts released into the intestine are reabsorbed in the lower ileum where bacteria can reduce free bile acids to lithocholate and deoxycholate. The absorbed bile acids and salts are transported via the portal vein to the liver as complexes with serum albumin. The liver efficiently extracts them, conjugates them with amino acids, and again secretes them as bile, which is returned to the gall bladder to continue to aid digestion. Bile salts are recirculated 2-3 times through the liver with each meal.

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Betaine

Beets contain high amounts of Betaine which is used to add a methyl group to Homocysteine, and thus form Methionine and Dimethylglycine. Dimethylglycines function as a methyl-donor which helps in both detoxification and the immune system pathways. In one study, exposure to carbon tetrachloride (CCE4), revealed a reduction in liver necrosis and a significant reduction in liver damage following oral treatment of betaine.[2] Another study showed that after injection of CCE4 into test animals, supplemental betaine reduced triglyceride in the liver and centrilobular hepatic lipidosis.[3]

Fiber and the Binding of Bile Components

Certain kinds of dietary fiber bind bile salts. Examples include pectin (found in fruits and berries), hemicelluloses (found in cereal grains), and certain types of fiber that occur in legumes. When the diet is rich in partially soluble fiber, more bile is excreted (not reabsorbed). As a consequence, blood cholesterol levels may be reduced to account for more bile salt formation, possibly slowing the development of atherosclerosis.

Nutritional Support of Bile Formation

Bile. When potentially pathogenic enteric bacteria were cultured in the presence of bile, then incubated with cultured human intestinal epithelial cells, fewer bacteria were internalized by intestinal cells. Therefore, exposure to bile salts during bacterial growth apparently decreases the tissue invasiveness.[4]

Pancrelipase (pancreatic lipase). This enzyme hydrolyzes triglycerides micellized with bile to monoglycerides, containing an acetyl link to position two of glycerol. Thus resulting free fatty acids and monoglycerides are absorbed by the gut mucosa and are reassembled as triglycerides. Preparations of lipase have also been shown to reduce fecal fat.[5]

Taurine. This derivative of cysteine is a highly charged compound. When it is conjugated to bile acids, it creates an even more polar region of the bile acid, thereby increasing its amphipathic (detergent-like) properties. As the substrate of Phase II detoxification enzymes, taurine may become depleted in the liver when the supply is inadequate to meet metabolic needs. In genetically stroke-prone, hypertensive rats, supplementation with taurine prevented increases in serum cholesterol levels.[6] Taurine increased the activity of cholesterol-7-hydroxylase and stimulated bile production. Additionally, dietary intake of taurine resulted in compromised absorption of vitamin D in preterm infants.[7]

Vitamin C. The enzyme responsible for the first step in converting cholesterol to bile acids, cholesterol-7-alpha hydroxylase, is vitamin C dependent. In guinea pigs with vitamin C deficiency, cholesterol accumulates in the liver and blood, resulting in decreased bile acid ratios in bile and an increased incidence of gallstones[8]. In hyper-cholesterolemic humans with low vitamin C status, additional vitamin C lowered blood cholesterol.

References

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PRODUCT INFORMATION

Beta Plus™ is available in bottles of 90 and 180 tablets.

Beta-TCP™ is available in bottles of 90 and 180 tablets.

Product Adjuncts: **MCS®**, **Mg-Zyme™**, **B_6 Phosphate**, **Phosphatidlylcholine**, **Livotrit Plus®** for **Beta-TCP™**

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