BioPC Pro





CLINICAL APPLICATIONS

- Supports Healthy Cellular Structure, Function and Signaling
- Supports Mitochondrial and Immune Function
- Supports GI Barrier Health and Integrity
- Helps Maintain Normal Inflammatory Balance

IMMUNE HEALTH[†]

Phospholipids are an integral part of biological membranes, and they tend to decrease as we age. They are critical for optimal cell and mitochondrial membrane function including their growth, shape and repair. Optimal cell membrane function facilitates healthy cell signaling, which is important for energy production, immune function, and regulation of inflammatory cascades. Additionally, BioPC Pro leverages the benefits of a full-spectrum phosphatide blend to help balance the nervous system. Together, phosphatidylcholine (PC), phosphatidylethanolamine (PE), phosphatidylinositol (PI), and phosphatidylserine (PS) help achieve optimal nervous system function when it is overstimulated. BioPC Pro also helps digest and process fats and supports better focus, cognitive function, and overall brain health. This rapidly absorbed blend provides 5840 mg* of naturally bioavailable phosphatides per serving, making it one of the most concentrated preparations available.

Overview

Phosphatides make up the major structural lipids in cellular membranes.¹ PC was one of the first biological amphiphiles (a molecule having both hydrophilic and hydrophobic parts) to be discovered, and it is the most abundant phospholipid in cellular membranes.^{2, 3} With this initial research conducted at the same time as the fluid mosaic cell surface model developed, it led to a widespread view of phosphatides as cellular building blocks.¹

But emerging evidence is making the case that in addition to its structural role, phosphatides also play a significant biological role in cell signaling, maintaining mitochondrial function, and transforming cellular membranes, enzymes, and receptors to be more functional. ^{1, 4, 5, 6, 7, 8, 9, 10} While phosphatides are sometimes used individually, the use of more complex phosphatide blends

containing PC, PE, PI, and PS are considered more useful.^{1, 11, 12, 13} Research shows that oral phospholipids are processed efficiently by the body, with more than 90% absorbed within hours of ingestion.^{1, 14, 15} For these reasons and more, restoring phosphatide levels through supplementation can be highly beneficial.

Phosphatides and the Mitochondrial-Immune Interface

In addition to cellular energy production, mitochondria perform several critical functions in the cell including the modulation of calcium signaling, regulation of cell death, maintenance of cellular redox balance, and innate immune signaling, making phospholipid movement to and from the mitochondrion essential for cellular integrity.^{1, 2, 16}

While phosphatides make up a large portion of the lipids comprising cellular and intracellular membranes, it is their metabolism that creates a tightly controlled cell signaling network essential for a healthy innate immune response.¹⁷ Phosphatides and their metabolites influence multiple aspects of innate immunity including cell shape, stickiness and degranulation.³ Phospholipids themselves are also recognized by innate-like T cells, which are considered essential for recognizing foreign organisms, as well as self-antigens.³

When various cellular stressors including reactive oxygen species (ROS) and danger signals are released in the presence of cellular or mitochondrial damage, the formation of inflammasomes are induced, which triggers innate immune defenses.^{1, 18, 19, 20} Inflammasomes such as NLRP3 induce cytokine release which increases oxidative stress and inhibits autophagy and mitophagy.^{1, 21, 22} The NLRP3 inflammasome is,



itself, activated by mitochondrial ROS, suggesting that healthy mitochondria are essential for a balanced immune response.¹

Studies show that stealth immune challenges are also associated with elevated mitochondrial oxidative stress.²³ These results suggest an interruption in cellular communication may be at the core of continued mitochondrial stress and danger signals in stealth immune challenges.²³ Oral phosphatides can be used to help modify mitochondrial signals and promote healthy inner mitochondrial membrane potential, thereby improving electron transport chain function and ATP synthesis (including perceived energy, mood and cognition) and overall immune-health balance.^{1, 24, 25}

Phosphatides and the Gut-Immune Interface

In a healthy gastrointestinal tract, the presence of PC in the mucosal secretion mucus creates a hydrophobic surface layer that safeguards the intestinal tissues and modulates mucosal signaling.^{1, 26, 27} PC represents more than 90% of the phospholipids comprising intestinal mucus, which serves as a primary component of the intestinal barrier creating a protectant shield against GI-related immune challenges by binding foreign matter.²⁸ Poor nutrition, stress insults, and alterations in the gut microbiome all have a negative impact on the health of intestinal mucus.²⁹ In the presence of GI challenges, the PC content of intestinal mucus may decrease by up to 70%, leaving the distal GI tract especially vulnerable. However, several studies show that oral PC supplementation replenishes the phosphatide content of intestinal mucus, reinforcing the body's first, and perhaps most important, immune defense mechanism.3,4,5

Directions

1 scoop (10 grams) per day with a meal or as recommended by your health care professional. May be mixed in juice or shakes, or sprinkled on food.

Does Not Contain

Gluten, corn, yeast, artificial colors and flavors.

Cautions

If you are pregnant or nursing, consult your health care professional before taking this product.

Supplement Facts Serving Size 1 Scoop (10 Grams) Servings Per Container About 30		
	Amount Per Serving	% Daily Value
Calories	60	
Total Fat	5 g	6% *
Saturated Fat	1 g	5% *
Polyunsaturated Fat	3 g	
Monounsaturated Fat	1 g	
Total Carbohydrate	3 g	1% *
Protein	<1 g	
Phosphorus	130 mg	10% *
Sunflower Lecithin Powder	10 g	**

* Percent Daily Values are based on a 2,000 calorie diet.
** Daily Value not established.

ID# 173030 10.6 oz (300 Grams)

Typical naturally occuring phospholipid profile (per 10 g serving)[‡]:

Phosphatidylcholine 2.5 g (2,500 mg)
Phosphatidylinositol 2 g (2,000 mg)
Phosphatidylethanolamine 1 g (1,000 mg)
Phosphatidic acid 0.4 g (400 mg)

*subject to natural variability

References

- 1. Nicolson, G. L., & Ash, M. E. (2014). Lipid Replacement Therapy: a natural medicine approach to replacing damaged lipids in cellular membranes and organelles and restoring function. *Biochimica et biophysica acta*, 1838(6), 1657–1679. https://doi.org/10.1016/j.bbamem.2013.11.010
- 2. Furse, S., & de Kroon, A. I. (2015). Phosphatidylcholine's functions beyond that of a membrane brick. *Molecular Membrane Biology*, 32(4), 117–119. https://doi.org/10.310 9/09687688.2015.1066894
- 3. Acoba, M. G., Senoo, N., & Claypool, S. M. (2020). Phospholipid ebb and flow makes mitochondria go. *The Journal of Cell Biology*, 219(8), e202003131. https://doi.org/10.1083/jcb.202003131
- 4. Blaton, V., Vandamme, D., & Peeters, H. (1974). Activation of lipoprotein lipase in vitro by unsaturated phospholipids. *FEBS letters*, 44(2), 185-188.



- 5. Horsch, A. K., Hudson, K., & Day, A. J. (1977). Uptake and metabolism of 3H-fatty acid labelled lecithin by normal and atherosclerotic intima in vivo and in vitro. *Atherosclerosis*, 26(4), 493-504.
- 6. Howard, A. N., & Patelski, J. (1974). Hydrolysis and synthesis of aortic cholesterol esters in atherosclerotic baboons: Effect of polyunsaturated phosphatidyl choline on enzyme activities. *Atherosclerosis*, 20(2), 225-232.
- 7. Waligora, Z., Patelski, J., Brown, B. D., & Howard, A. N. (1975). Effect of a hypercholesterolaemic diet and a single injection of polyunsaturated phosphatidyl choline solution on the activities of lipolytic enzymes, acyl-CoA synthetase and acyl-CoA cholesterol acyl-transferase in rabbit tissues. *Biochemical Pharmacology*, 24(24), 2263-2267.
- Karaman, A., Demirbilek, S., Sezgin, N., Gürbüz, N., & Gürses, I. (2003). Protective effect of polyunsaturated phosphatidylcholine on liver damage induced by biliary obstruction in rats. *Journal of Pediatric Surgery*, 38(9), 1341-1347.
- 9. Olbrich, K., Rawicz, W., Needham, D., & Evans, E. (2000). Water permeability and mechanical strength of polyunsaturated lipid bilayers. *Biophysical Journal*, 79(1), 321-327.
- Buko, V., Artsukevich, A., Maltsev, A., Nikitin, V., Ignatenko, K., Gundermann, K. J., & Schumacher, R. (1994). Effect of polyunsaturated phosphatidylcholine on lipid structure and cAMP-dependent signal transduction in the liver of rats chronically intoxicated with ethanol. *Experimental and Toxicologic Pathology*, 46(4-5), 375-382.
- 11. Nicolson, G. (2003). Lipid replacement as an adjunct to therapy for chronic fatigue, anti-aging and restoration of mitochondrial function. *The Journal of the American Nutraceutical Association Vol.6*, No. 3, Summer 2003, 4-10
- 12. Nicolson, G. L., & Ellithorpe, R. (2006). Lipid replacement and antioxidant nutritional therapy for restoring mitochondrial function and reducing fatigue in chronic fatigue syndrome and other fatiguing illnesses. *Journal of Chronic Fatigue Syndrome*, 13(1), 57-68.
- 13. Nicolson, G. L. (2005). Lipid replacement/antioxidant therapy as an adjunct supplement to reduce the adverse effects of cancer therapy and restore mitochondrial function. *Pathology & Oncology Research*, 11(3), 139-144.
- 14. Zierenberg, O., & Grundy, S. M. (1982). Intestinal absorption of polyenephosphatidylcholine in man. *Journal of Lipid Research*, 23(8), 1136–1142.

- 15. O. Zierenberg. (1983). Clinical and biochemical studies of the transport of polyenephosphatidylcholine in human serum and its physiological impact on cholesterol distribution between serum lipoproteins. In Avogaro, P., & Catapano, A. L. *Phospholipids and atherosclerosis*. (pp. 175-189). New York: Raven Press.
- Cloonan, S. M., & Choi, A. M. (2013). Mitochondria: sensors and mediators of innate immune receptor signaling. *Current Opinion in Microbiology*, 16(3), 327–338. https://doi.org/10.1016/j.mib.2013.05.005
- 17. O'Donnell, V. B., Rossjohn, J., & Wakelam, M. J. (2018). Phospholipid signaling in innate immune cells. *The Journal of Clinical investigation*, 128(7), 2670–2679. https://doi.org/10.1172/JCI97944
- Brookes, P. S., Yoon, Y., Robotham, J. L., Anders, M. W., & Sheu, S. S. (2004). Calcium, ATP, and ROS: a mitochondrial love-hate triangle. *American Journal of Physiology Cell Physiology*, 287(4), C817–C833. https://doi.org/10.1152/ajpcell.00139.2004
- 19. Latz, E., Xiao, T. S., & Stutz, A. (2013). Activation and regulation of the inflammasomes. *Nature Reviews Immunology*, 13(6), 397–411. https://doi.org/10.1038/nri3452
- 20. Dostert, C., Pétrilli, V., Van Bruggen, R., Steele, C., Mossman, B. T., & Tschopp, J. (2008). Innate immune activation through Nalp3 inflammasome sensing of asbestos and silica. *Science*, 320(5876), 674–677. https://doi.org/10.1126/science.1156995
- 21. Franceschi, C., Bonafe, M., Valensin, S., Olivieri, F., De Luca, M., Ottaviani, E., & De Benedictis, G. (2000). Inflamm-aging: an evolutionary perspective on immunosenescence. *Annals of the New York Academy of Sciences*, 908(1), 244-254.
- 22. Rodgers, M. A., Bowman, J. W., Liang, Q., & Jung, J. U. (2014). Regulation where autophagy intersects the inflammasome. *Antioxidants & Redox Signaling*, 20(3), 495-506.
- 23, Peacock, B. N., Gherezghiher, T. B., Hilario, J. D., & Kellermann, G. H. (2015). New insights into Lyme disease. *Redox biology*, 5, 66–70. https://doi.org/10.1016/j.redox.2015.03.002
- 24. Agadjanyan, M., Vasilevko, V., Ghochikyan, A., Berns, P., Kesslak, P., Settineri, R. A., & Nicolson, G. L. (2003). Nutritional supplement (NT Factor™) restores mitochondrial function and reduces moderately severe fatigue in aged subjects. *Journal of Chronic Fatigue Syndrome*, 11(3), 23-36.



- 25. Seidman, M. D., Khan, M. J., Tang, W. X., & Quirk, W. S. (2002). Influence of lecithin on mitochondrial DNA and age-related hearing loss. *Otolaryngology—Head and Neck Surgery*, 127(3), 138-144.
- 26. Dial, E. J., Zayat, M., Lopez-Storey, M., Tran, D., & Lichtenberger, L. (2008). Oral phosphatidylcholine preserves the gastrointestinal mucosal barrier during LPS-induced inflammation. *Shock (Augusta, Ga.)*, 30(6), 729–733. https://doi.org/10.1097/SHK.0b013e318173e8d4
- 27. Ehehalt, R., Braun, A., Karner, M., Füllekrug, J., & Stremmel, W. (2010). Phosphatidylcholine as a constituent in the colonic mucosal barrier—physiological and clinical relevance. *Biochimica et Biophysica Acta*, 1801(9), 983-993.
- 28. Stremmel, W., Ehehalt, R., Staffer, S., Stoffels, S., Mohr, A., Karner, M., & Braun, A. (2012). Mucosal protection by phosphatidylcholine. *Digestive Diseases*, 30 Suppl 3, 85–91. https://doi.org/10.1159/000342729
- 29. Diebel, L. N., & Liberati, D. M. (2014). Reinforcement of the intestinal mucus layer protects against Clostridium difficile intestinal injury in vitro. *Journal of the American College of Surgeons*, 219(3), 460–468. https://doi.org/10.1016/j.jamcollsurg.2014.05.005

