Souvenaid® Evidence

This overview contains a selection of the scientific evidence behind Souvenaid's carefully chosen ingredients. Souvenaid contains **Fortasyn Connect**™, which is a well-researched, proprietary blend of key nutrients that are understood to support synapses in the brain.* This blend includes docosahexaenoic acid (DHA), eicosapentaenoic acid (EPA), choline, uridine monophosphate (UMP), folic acid, pyridoxine (vitamin B_6), vitamin B_{12}, vitamin C, vitamin E, selenium and phospholipids. Souvenaid helps to strengthen synapses to support memory function* in people with normal age-related memory concerns.

Publication	Title	Study Purpose	Methodology	Result/Conclusion
Wurtman RJ, Ulus IH, Cansev M, Watkins CJ, Wang L, Marzloff G. Brain Res. 2006;1088(1): 83-92.[1]	Synaptic Proteins and Phospholipids Are Increased in Gerbil Brain by Administering Uridine Plus DHA Orally.	To evaluate the impact of choline, uridine & DHA consumption on membrane phosphatide and synaptic protein levels in gerbil brains.	Animals were fed the following diets for 4 weeks: • Control group consumed a standard choline-containing (0.1%) diet (n=8) • Group 1 was fed a diet supplemented with UMP (0.5%) (n=8) • Group 2 was fed an un-supplemented diet plus DHA (300 mg/kg) (n=8) • Group 3 was fed both the UMP-supplemented diet and DHA (n=8)	This study demonstrated that diets supplemented with uridine and DHA significantly increased membrane phosphatide levels that are known to support synapses in the brain.
Cansev, M and Wurtman RJ. Neuroscience. 2007;148:421-31.[2]	Chronic Administration of DHA or EPA, But Not ARA, Alone or in Combination With Uridine, Increases Brain Phosphatide and Synaptic Protein Levels in Gerbils.	To investigate the effects of providing polyunsaturated fatty acids alone, or in combination with a uridine supplemented diet.	Gerbils were fed the following diets for 28 days: a control choline-containing diet (0.1%) with linoleic acid (LA; 23 mg/kg) and alpha-linolenic acid (ALA; 1.5 mg/kg) or a UMP (0.5%) supplemented diet while also receiving gavage feedings of DHA, EPA, ARA (300 mg/kg) or a placebo. Following the study period, their brains were analyzed for phosphatides.	This study demonstrated that compared to uridine-rich diets alone, uridine supplemented diets with the addition of omega-3 polyunsaturated fatty acids, specifically DHA and EPA, significantly increased levels of brain phasphatides, in the gerbils, known to support synaptic membranes.
Sakamoto T, Cansev M, Wurtman RJ. Brain Res. 2007;1182:50-59.[3]	Oral Supplementation with DHA and UMP Increases Dendritic Spine Density in Adult Gerbil Hippocampus.	To examine the effects of supplemental DHA and UMP on the number of dendritic spines in gerbils.	Adult male gerbils were fed a control diet or UMP-supplemented diet (0.5% UMP) with gavage feedings of various doses of DHA (0, 50, 100 or 300 mg/kg/day) or a placebo daily. After 4 weeks, brain tissue was analyzed, and neuroimaging was performed to assess dendritic spine density.	Diets with DHA or DHA and UMP resulted in a significant increase in the number of dendritic spines and membrane phosphatides in the hippocampus. This study demonstrated that supplementation of DHA and a source of uridine may increase the number of brain synapses.
Wurtman RJ, Cansev M, Sakamoto T, Ulus IH. Annu Rev Nutr. 2009;29:59-87.[4]	Use of Phosphatide Precursors to Promote Synaptogenesis.	To review the biochemical mechanisms of how consumption of specific nutrients may play a role in increasing the quantity of synaptic membranes and number of brain synapses.	Comprehensive narrative review of DHA, uridine, and choline consumption and synaptogenesis.	It has been well-studied that combined supplementation of DHA, uridine and choline in animals increased phosphatide levels, synaptic proteins, and dendritic spines of hippocampal neurons in animals.

ARA = arachidonic acid; DHA = docosahexaenoic acid; EPA = eicosapentaenoic acid; FC = Fortasyn Connect™; UMP = uridine-5'-monophosphate

Publication	Title	Study Purpose	Methodology	Result/Conclusion
Wurtman RJ, Cansev M, Sakamoto T, Ulus I. Nutr Rev. 2010;68 Suppl 2(Suppl 2):S88-S101.[5]	Nutritional Modifiers of Aging Brain Function: Use of Uridine and Other Phosphatide Precursors to Increase Formation of Brain Synapses.	To review the effects of oral supplementation of phosphatide precursors, specifically DHA, uridine and choline, on synapse formation.	Comprehensive narrative review of phosphatide precursors (DHA, uridine and choline) consumption and impact on brain synapse formation.	Many studies have demonstrated that oral administration of DHA, uridine, and choline can boost brain phosphatide levels, increase synapse formation and ultimately improve cognition and neurotransmitter release in animals.
Cansev M, van Wijk N, Turkyilmaz M, Orhan F, Sijben JW, Broersen LM. Neurobiol Aging. 2015;36(1):344-351.[6]	Specific Multi-nutrient Enriched Diet Enhances Hippocampal Cholinergic Transmission in Aged Rats.	To investigate the mechanism of improved memory through FC supplementation in aged rats and assessment of acetylcholine release and synaptic membrane formation.	Rats were randomized to consume a regular control diet (n=4) for 4 weeks, or a FC supplemented diet for either 4 weeks (n=5) or 6 weeks (n=5). Following the dietary intervention, brain tissue was analyzed for acetylcholine release, tissue acetylcholine and choline content, phospholipid levels and synaptic protein levels.	Diets supplemented with FC significantly increased acetylcholine levels and release, increased levels of phospholipids and synaptic proteins in aged rats. This study demonstrated the impact of the specific FC nutrient combination of precursors and cofactors to support learning and memory function.
Van Deijk AF, Broersen LM, Verkuyl JM, Smit AB, Verheijen MHG. Front Neurosci. 2017;11:440. Published 2017 Aug 4.[7]	High Content Analysis of Hippocampal Neuron-Astrocyte Co-cultures Shows a Positive Effect of Fortasyn Connect on Neuronal Survival and Postsynaptic Maturation.	To study the effects of FC on synaptogenesis through supplementation in a novel neuron-astrocyte co-culture cell model that mimics brain metabolic pathways.	Cell models were developed from rodent cells and inoculated with a control stock solution and varying dilutions of FC (1:20 of the stock solution, 1:10 and 1:5) after 5 days *in vitro* and were analyzed on day 14 *in vitro*.	This study demonstrated that the use of stronger dilutions of FC can increase neuronal survival and strengthen postsynaptic terminals. The use of FC also positively impacted synapse maturation. Improved synaptic function has been shown to improve memory and cognitive function.

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Lifestyle changes, including dietary changes, have been well-studied[8-12] and have shown to have a positive impact on memory function. But, until now, there hasn't been a dietary supplement with this unique combination and concentration of key nutrients to strengthen synapses to help support memory function. Souvenaid was designed[1-7, 13-27] to include essential building blocks and cofactors for maintaining and strengthening brain synapses involved in memory function. Research has also demonstrated that Souvenaid is safe and well tolerated.[28-32] Nutricia remains committed to research in the area of age-related memory loss.

NUTRICIA
Souvenaid®
Helps support memory function*

Available at souvenaid.com and amazon.com

Souvenaid is a dietary supplement for people with normal age-related memory concerns.

1. Wurtman, et al. 2006;1088:83-92. **2.** Cansev, et al. 2007;148:421-31. **3.** Sakamoto, et al. 2007;1182:50-9. **4.** Wurtman, et al. 2009;29:59-87. **5.** Wurtman, et al. 2010;68 Suppl 2:S88-101. **6.** Cansev, et al. 2015;36:344-51. **7.** van Deijk, et al. 2017;11:440. **8.** Jia, et al. 2023;380:e072691. **9.** Bettio, et al. 2017;79:66-86. **10.** Monti, et al. 2014;5:337s-43s. **11.** Ngandu, et al. 2015;385:2255-63. **12.** Valls-Pedret, et al. 2015;175:1094-103. **13.** van Wijk, et al. 2012;9:49. **14**. Cansev, et al. 2005;1058:101-8. **15.** Ulus, et al. 2006;26:561-75. **16**. Wang, et al. 2005;27:137-45. **17**. Pooler, et al. 2005;134:207-14. **18**. Wurtman, et al. 2009;29:59-87. **19**. Farkas, et al. 2002;954:32-41. **20.** Wang, et al. 2007;1133:42-8. **21.** Savelkoul, et al. 2012;120:631-40. **22.** Teather, et al. 2006;136:2834-7. **23**. de Wilde, et al. 2003;988:9-19. **24**. de Wilde, et al. 2002;947:166-73. **25**. De Bruin, et al. 2003;80:63-79. **26.** Holguin, et al. 2008;191:11-6. **27.** de Wilde, et al. 2011;27:327-39. **28.** Olde Rikkert, et al. 2015;44:471-80. **29.** Scheltens, et al. 2012;31:225-36. **30.** Shah, et al. 2013;5:59. **31.** Soininen, et al. 2017;16:965-75. **32**. Soininen, et al. 2021;17:29-40.6

*These statements have not been evaluated by the Food and Drug Administration. This product is not intended to diagnose, treat, cure or prevent any disease.

For Healthcare Professionals Only