CHEMI Nutra

WHITE PAPER

PHOSPHOLIPID CHOLINE

a natural, brain and body beneficial choline source for all stages of life

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CHOLINE

Choline is the newest vitamin recognized as an essential nutrient and assigned a daily value of 550 mg/day—and deservedly so. Choline plays vital roles in human health and wellness and is needed in all stages of life. Furthermore, the phospholipid form of choline known as α-Glyceryl Phosphoryl Choline (α-GPC) is the demonstrably preferred form of this nutrient. This white paper will review the research on α-GPC, but first let us examine the fundamentals of choline.

Introduction to Choline

Choline is an essential nutrient related to the water-soluble B-complex vitamins, folate, pyridoxine, and B12, and to the essential amino acid methionine. This essential nutrient is present in some foods and is also available as a dietary supplement. Choline is a primary source of methyl groups which are needed for various metabolic processes. The body needs choline to synthesize phosphatidylcholine and sphingomyelin, two major phospholipids vital for cell membranes. Therefore, all plant and animal cells need choline to preserve their structural integrity. In addition, choline is needed to produce the following effects in the body:

ANTI-INFLAMMATORY

Choline selectively activates alpha7 nicotinic acetylcholine receptors, which leads to decreases in the production of inflammatory cytokines including tumor necrosis factor (TNF). Choline also reduce asthma-associated inflammation by lowering lipophosphatidylcholine levels.

MEMORY NEURONS

Choline intake during pregnancy and lactation probably affects the birth, death, and migration of cells in the hippocampus during fetal brain development and possibly changes the distribution and morphology of neurons responsible for memory function in the brain, and seems to play a role in preventing neural tube defects. Additionally, choline appears to improve hippocampal growth in the infant, possibly through the fourth year of life.

COGNITIVE

Choline can increase the density of alpha7 nicotinic acetylcholine receptors, which play a role in normal cognitive processes including attention, working memory, and executive function. Through activation of alpha7 nicotinic acetylcholine receptors, choline can improve cognitive function.

ANTIOXIDANT

Clinical research suggests that choline may decrease oxidative stress.

CARDIOVASCULAR

Dietary intake of choline may decrease homocysteine levels, a risk factor for cardiovascular disease. The effect of dietary choline intake may be greatest on those with lower folate levels.

NEUROLOGIC/CNS

Choline concentrates in nervous tissue as a component of cell membranes. It is required for phospholipid synthesis and is involved in brain development, neurotransmission, and signaling.

Choline adequacy

Humans can produce choline in the liver (typically as phosphatidylcholine), but the amount that the body naturally synthesizes is not sufficient to meet human needs. As a result, humans must obtain some choline from the diet. Premenopausal women might need less choline from the diet than children or other adults because estrogen induces the gene that catalyzes the biosynthesis of choline. When a diet is deficient in folate, a B-vitamin that is also a methyl donor, the need for dietary choline rises because choline becomes the primary methyl donor.

Choline metabolism

When choline-containing compounds are ingested, pancreatic and mucosal enzymes liberate free choline from about half of the fat-soluble forms (e.g. phosphocholine, glycerolphosphocholine, and free choline) and some water-soluble forms (e.g. phosphatidylcholine and sphingomyelin). Free choline, phosphocholine, and glycerophosphocholine are absorbed in the small intestine, enter the portal circulation, and are stored in the liver, where they are subsequently phosphorylated and distributed throughout the body to make cell membranes. The remaining fat-soluble phospholipids (phosphatidylcholine and sphingomyelin) are absorbed intact, incorporated into chylomicrons, and secreted into the lymphatic circulation, where they are distributed to tissues and other organs, including the brain and placenta.

Recommended Intakes

Intake recommendations for choline and other nutrients are provided in the Dietary Reference Intakes (DRIs) developed by the Food and Nutrition Board (FNB) of the Institute of Medicine (IOM). Since insufficient data were available to establish an Estimated Average Requirement (EAR) for choline, the FNB instead established Adequate Intakes (AIs) for all ages that are based on the prevention of liver damage as measured by serum alanine aminostransferase levels. The amount of choline that individuals need is influenced by the amount of methionine, betaine, and folate in the diet, as well as gender, pregnancy, lactation, stage of development, ability to produce choline endogenously, and genetic mutations that affect choline needs. Additionally, plasma choline levels are reduced following prolonged or high-intensity exercise. The table below lists the current AIs for choline.

Adequate Intakes (AIs) for Choline

Age	Male	Female	Pregnancy	Lactation
Birth - 6mo.	125mg/day	125mg/day		
7-12mo.	150mg/day	150mg/day		
1-3yr.	200mg/day	200mg/day		
4-8yr.	250mg/day	250mg/day		
9-13yr.	375mg/day	375mg/day		
14-18yr.	550mg/day	400mg/day	450mg/day	550mg/day
+19yr.	550mg/day	425mg/day	450mg/day	550mg/day

The daily value for choline (the one-size-fits-all dose for adults and children over 4 years of age) is 550 mg/day.

Choline salts vs. α-GPC

CHOLINE SALTS

Choline's importance to human health is well established and widely recognized. It is also well established—but not as widely recognized—that common choline salts are not especially bioavailable or effective for cognitive function. For example, a 1998 study found that adults receiving supplements with 2450-5100 mg of choline bitartrate failed to significantly increase cerebral choline levels. Likewise, a 1999 study also demonstrated that neither short-term nor long-term choline supplementation (as lecithin) altered choline metabolites in the human brain. This is consistent with a more recent 2016 study showing that daily supplementation with 2000-2500 mg/day choline bitartrate failed to improve memory performance.

a-GPC

By contrast, a 1993 study found that supplementation with radiolabeled α-GPC resulted in wide distribution the liver, kidney, lung, spleen and brain. Likewise, other researchers have suggested that demonstrated improvements from α-GPC supplementation may be due to increased bioavailability of choline for the synthesis of acetylcholine. Essentially, α-GPC is a rapidly absorbed source of choline which does not carry the electrical charge of regular choline, making it easier to cross the blood brain barrier, thereby raising free plasma choline more rapidly than other sources and allowing it to be incorporated into brain phospholipids within 24 hours from absorption.

Citicoline vs. α-GPC

In other research, the bioavailability of intramuscular administration of citicoline was compared to that of α-GPC. The results were that choline values with α-GPC were significantly higher than those with citicoline, likely due to the lower choline concentration with citicoline. In a study with mild to moderate vascular dementia patients, intramuscular treatment with either 1000 mg/day citicoline or α-GPC produced symptomatic improvement, but α-GPC possessed statistically higher efficacy and overall greater activity assessed by both patients and investigators, compared to citicoline. Similar results were found in two other studies comparing intramuscular treatment with 1000 mg/day citicoline or α-GPC in patients with mild to moderate multi-infarct dementia, and in a study of in patients with senile mental decline.

Cognition

Due to its ability to increase production of acetylcholine and phosphatidylcholine, α-GPC has demonstrated value for improving memory in healthy adults, as well as adults with dementia. First of all, α-GPC is also a precursor to membrane phospholipids. Therefore, it might improve neuronal functioning by improving neuronal membrane fluidity. In addition, levels of α-GPC-derived choline in the brain are thought to increase much more slowly, but also remain elevated for longer period of time.

COGNITIVE FUNCTION IN HEALTHY YOUNG ADULTS

The purpose of this double-blind, placebo-controlled study was to examine the effect of an acute ingestion of an α-GPC supplement (including CholineAid® α-GPC, Chemi Nutra, Austin, TX, USA) designed to improve reaction time and subjective measures of alertness, energy, fatigue, and focus compared to placebo. Nineteen physically active subjects (17 male, 2 female) were randomly assigned to a group that either consumed α-GPC supplement (21.1 ± 0.6 years) or placebo (21.3 ± 0.8 years). Subjects reported to the Human Performance Laboratory and were provided with one serving α-GPC supplement or a placebo (rice flour, PL). Subjects ingested the capsules with 12 ounces of bottled water. Following consumption, rested quietly for 10-minutes prior to completing a 9-question survey and commencing exercise (PRE). The survey consisted of questions describing subjective feelings of energy, fatigue, alertness and focus at that moment. Following the completion of the questionnaire subjects performed a 4-minute quickness and reaction test on the Makoto testing device. Subjects then performed a 10-min bout of exhaustive exercise that included a 30-second Wingate Anaerobic Power test, and the maximal number of push-ups and sit-ups performed in one minute. Subjects then repeated the questionnaire and reaction testing sequence (POST).

CONT. →

The results were that subjects consuming α-GPC supplement maintained reaction time performance between PRE and POST measures, while a significant decline between PRE and POST measures were observed in subjects consuming PL. Acute use of α-GPC supplement resulted in an ability to maintain focus and alertness following an acute bout of exhaustion. Subjects consuming PL realized significant declines in both focus and alertness. In conclusion, ingestion of α-GPC supplement maintained reaction performance to both visual and auditory stimuli following a high-intensity bout of exhaustive exercise, while subjects consuming a placebo experienced significant reductions in performance. α-GPC supplement might be an effective supplement to improve brain functions in young healthy college students during times of increased stress.

A prior study was carried out to test the effects of α-GPC on memory impairment induced by scopolamine in thirty-two healthy young volunteers, randomly allocated to four different groups. They were given a ten-day pretreatment with either α-GPC or placebo, p.o., and on the eleventh day either scopolamine or placebo, intra-muscular. Before and 0.5, 1, 2, 3, and 6 h after injection the subjects were given attention and mnemonic tests. The findings of this study indicate that α-GPC is able to antagonize impairment of attention and memory induced by scopolamine.

COGNITIVE FUNCTION IN OLDER SUBJECTS

A comparative study was conducted to assess the effects of oxiracetam (a nootropic drug) versus α-GPC in brain aging. Forty male patients (aged 55-65 years) with senile organic brain syndrome (an older term used to describe a neurocognitive disorder) were randomly assigned to 1 g/day of either therapy for 12 weeks.

The clinical efficacy of the two agents was assessed by neuropsychological and clinical parameters, including measurements of reaction time. Treatment with oxiracetam was associated with early improvement that was maintained for the duration of therapy, but whose effects ceased following discontinuation of therapy. In contrast, the response to α-GPC was slower but more sustained. Eight weeks after discontinuation of therapy, the clinical effects were as apparent as during the eighth week of treatment ($P<0.001$). Both agents were well tolerated and can be expected to be particularly effective in long-term patient management.

COGNITIVE FUNCTION IN ALZHEIMER'S PATIENTS

In an open, uncontrolled clinical study, 817 elderly patients with the diagnoses of primary degenerative dementia of the Alzheimer's type (PDD), multi-infarct dementia (MID) or mixed dementia (MD) were treated with 1,200 mg/day of α-GPC. Although an open label design makes it hard to reach final conclusions, results showed that both the dimensional changes in rating scale scores and the proportional changes in terms of number of patients showing improvement are well beyond the 20% range which have been reported with an inactive placebo. Furthermore, unlike temporary benefits seen with placebos, the changes improvement with A-GPC continued throughout the study period and were not restricted to alleviation of psychopathology and improvement of performance but extended to areas intimately linked to social behavior. There were also indications that patients with MID responded more favorably than patients with PDD, and patients with moderate and moderately severe cognitive decline responded more favorably than patients with very mild and mild cognitive decline.

To assess the efficacy and tolerability of α-GPC in the treatment of cognitive impairment due to mild to moderate Alzheimer's Disease (AD), a multicenter, double-blind, randomized, placebo-controlled trial was conducted. A total of 261 patients (age range, 60-80 years) were treated with 400 mg α-GPC or placebo capsules, 3 times daily for 180 days. Outcome measures that were assessed at the beginning of the investigation and after 90 and 180 days of treatment, included scores of the Alzheimer's Disease Assessment Scale-Cognitive Subscale (ADAS-Cog), the Mini-Mental State Examination (MMSE), the Global Deterioration Scale (GDS), the Alzheimer's Disease Assessment Scale-Behavioral Subscale (ADAS-Behav), all items of the Alzheimer's Disease Assessment Scale (ADAS-Total), and the Clinical Global Impression (CG) scale. The Global Improvement Scale (GIS) score was assessed after 90 and 180 days of treatment. Results were that the average decrease in ADAS-Cog score in patients treated with A-GPC was 2.42 points after 90 days of treatment and 3.20 points at the end of the study (the lower the score, the better) ($P < 0.001$). However, in patients receiving placebo there was an average increase in ADAS-Cog score of 0.36 points ≤1 after 90 days of treatment and 2.90 points after 180 days of treatment. All other assessed parameters consistently improved after 90 and 180 days versus baseline in the α-GPC group, whereas in the placebo group they remained unchanged or worsened. In conclusion, this study suggests that α-GPC is clinical useful and tolerable in the treatment Alzheimer's Disease.

Cholinesterase inhibitors (ChE-Is), drugs that reduce the breakdown of acetylcholine in the body, are commonly used for the treatment of mild-to-moderate symptoms of Alzheimer's disease (AD). However, the long-term effects of ChE-Is on the cognitive, functional, and behavioral symptoms of the disease are small and not always apparent in practice. To assess if the ChE-I donepezil (10 mg/day) with α-GPC (1,200 mg/day) has a more favorable clinical profile than therapy with donepezil alone (+placebo), a 12-month, double-blind multi center trial was conducted, with data from 91 AD patients (aged 56-91 years) who also had ischemic cerebrovascular disease. Patients cognitive functions, daily activities and behavioral symptoms were assessed by the Mini-Mental State Evaluation (MMSE), Alzheimer's Disease Assessment Scale Cognitive subscale (ADAS-cog), Basic Activities of Daily Living (BADL), Instrumental Activities of Daily Living (IADL) and Neuropsychiatric Inventory (NPI), of severity and of caregiver distress measures (NPI-F and NPI-D). The results were that patients receiving donepezil+placebo showed a slight worsening of MMSE, ADAS-cog, IADL and NPI-D scores and no changes in the BADL and NPI-F scores. By contrast, donepezil+ α-GPC showed improvements in all scores ($P<0.05$) (except the BADL) to donepezil+placebo.

COGNITIVE FUNCTION IN STROKE

In addition, stroke and transient ischemic attack (TIA) patients who receive α-GPC within 10 days post-stroke might have improved outcomes. Preliminary evidence suggests that these patients who get α-GPC 1200 mg/day intramuscularly for 28 days, followed by 400 mg three times daily (1200 mg/day) orally for 6 months, have improved recovery including improved cognitive and behavioral function.

EXERCISE PERFORMANCE

The purpose of this randomized, double-blind, placebo-controlled, crossover study was to measure the acute effects of α-GPC supplementation in comparison to caffeine or placebo on mood, cognitive function, and physiological performance. Twenty participants (10 male, 10 female; 22.0 ± 3.4 years of age) consumed 200 mg of α-GPC (CholineAid® α-GPC, Chemi Nutra, α-GPC-L), 400 mg of α-GPC (α-GPC-H), 200 mg of caffeine (CA), and a placebo (PL). Participants performed the following measurements 30 minutes after supplementation: visual analog scales (VAS) for six different moods, a serial subtraction test (SST), and tests for reaction time, hand-eye coordination, power, speed, and agility. SST scores were 18.1% and 10.5% faster in the α-GPC-L (6.19 ± 2.21 s) group compared to CA (7.32 ± 5.67 s) and PL (6.85 ± 2.52 s), respectively. Vertical Jump Peak Power was 8.5% higher in the α-GPC -L (2,041.3 ± 547.2 W), 7.5% higher in the α-GPC -H (2,023.1 ± 942.8 W) and 2.0% higher in the CA group (1,920.4 ± 689.6 W) in comparison to PL (1,881.9 ± 576.9 W). The group consuming CA had significantly higher scores on the VAS for jitteriness compared to α-GPC-H ($p = 0.019$). There were no other statistically significant differences between supplement groups for any of the dependent variables.

The present randomized, dose-comparison, placebo-controlled study was designed to assess the efficacy of two doses of α-GPC (CholineAid® α-GPC, Chemi Nutra) in comparison to placebo and caffeine for increasing countermovement jump performance, isometric strength, and psychomotor function. Forty-eight healthy, college aged males volunteered for the present study and underwent baseline assessment of various performance tests. Following this assessment participants were randomly assigned to groups consisting of 500 mg α-GPC, 250 mg α-GPC, 200 mg Caffeine or Placebo taken daily. Blood samples were collected 1 h and 2 h post initial dose to quantify serum free choline and thyroid stimulating hormone then subjects returned after 7 days of supplementation to repeat performance assessments. Results were that serum free choline was found to be elevated in the two A-GPC groups as compared to placebo (132% and 59% respectively). Serum TSH was found to be significantly depressed in the 500 mg A-GPC group compared to other treatments ($p < 0.04$). Group differences were noted for maximum velocity and maximum mechanical power on the countermovement jump ($p < 0.05$) with the 250 mg A-GPC group demonstrating the greatest improvements in result. In conclusion, this and previous evidence indicates that α-GPC should be considered as an emerging ergogenic supplement.

A double-blind, placebo-controlled, crossover study, with a one-week wash-out period (two testing sessions), was conducted with 17 subjects (mean age 21.18 years) to examine the acute effects of α-GPC ingestion on anaerobic performance. One hour prior to the testing, subjects ingested a solution—placebo or 300 mg α-GPC. Subjects completed three assessments of anaerobic performance: the counter movement jump (CMJ), 40-yd dash, and 30-second Wingate anaerobic test (WAnT). The best of three attempts of the CMJ and 40-yd were used in analysis. They completed only one trial on the 30-second WAnT. Results showed a significant difference between placebo (68.5±11.5 cm) and α-GPC (69.8±11.5 cm) for the CMJ performance. A trend towards significance ($p=.069$) was found for the 30-second WAnT minimum power under the α-GPC condition. No other differences were noted. α-GPC demonstrated properties of an ergogenic aid.

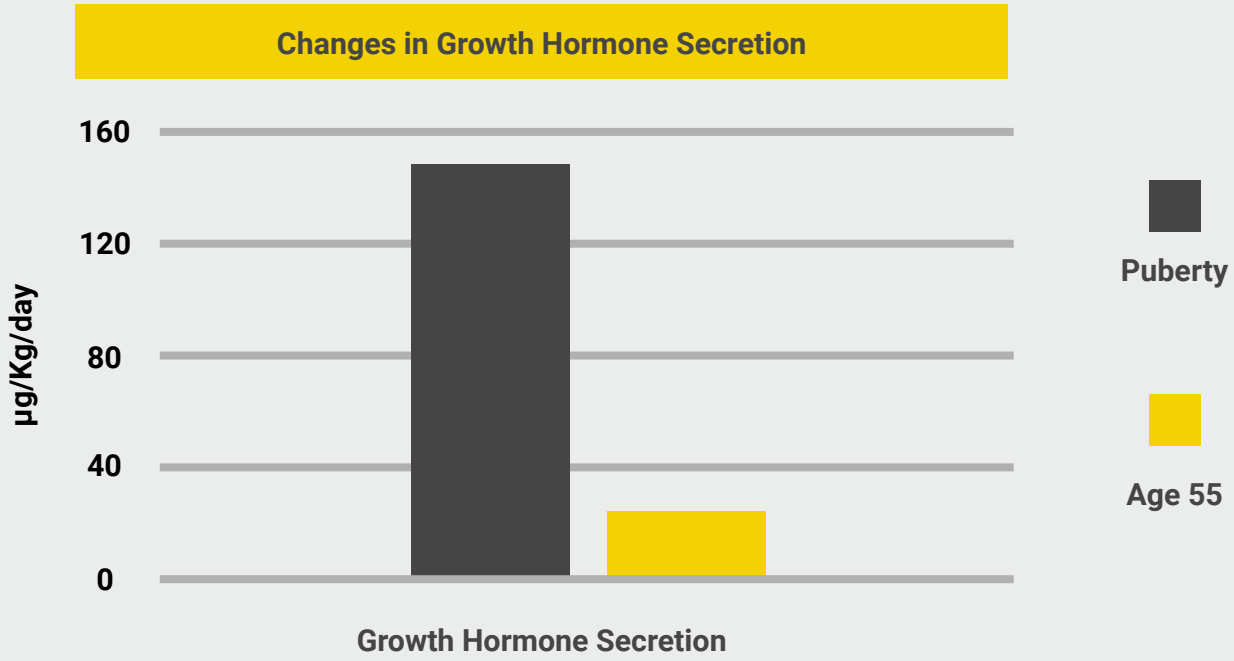
To investigate and identify the acute effects of α-GPC ingestion on performance testing (hand-grip strength, jump height, power output/rate of force development, mood, & reaction time), a randomized, double-blind, crossover study was conducted with 27 college-aged recreationally trained individuals (15 male, 12 female; mean 21.66 ±SD 1.88 years). Subjects ingested a placebo (dextrose, Group 1) or α-GPC (6 mg·kg^{-1} body mass, Group 2). Baseline measurements were obtained on the initial visit. On supplemental visits, immediately following ingestion, subjects were required to sit in a rested state for 25-minutes.

Subjects first completed the 4-item mood questionnaire to assess subjective feelings of energy, fatigue, alertness and focus for tasking, followed by the pre reaction-time (RT) test. Body composition was then measured via the Bod Pod, followed by a standardized questionnaire indicating age and training experience. Each test consisted of four stations assessing physical task performance. Subjects participated on three different occasions separated by 2-14 days, and there were no significant differences between any anthropometric variables, heart rate or blood pressure pre to post testing. Likewise, there were no differences between males and females for any test variable. Results were that there were statistical significances for the following the dependent variables (fatigue, plyometric push-up, mood change, and reaction-time test). Participants reported lower levels of fatigue following α-GPC ingestion, yielding a significant difference of when compared to baseline (p=0.019). The (p=0.019) group showed increased levels of peak force production during the plyometric push-up when compared to placebo of (p=0.014). Mood change increased with α-GPC ingestion compared to placebo was significant (p=0.023); and subjects scored more accurate (less incorrect answers) during the reaction-time test post α-GPC ingestion compared to placebo (p=0.014). In conclusion, α-GPC supplementation showed a 12% increase in upper body power output, and 12% improvement in accuracy during the reaction-time test. This investigation supports α-GPC as an ergogenic aid for physical or cognitive stimulation.

The purpose of the present double-blind, placebo controlled, cross-over study was to determine if 6 days of supplementation with 600 mg/day α-GPC (CholineAid® α-GPC, Chemi Nutra) would augment isometric force production compared to a placebo in 13 college aged males (Age: 21.9 ± 2.2 yrs). Baseline measurements were taken for the participants in the isometric mid-thigh pull in a custom squat cage on a force platform and upper body isometric test against a high frequency load cell. The participants then consumed either α-GPC or placebo and at the end of 6 days performed isometric mid-thigh pull and an upper body isometric test. A one-week washout period was used before the participants baseline was re-measured and crossed over to the other treatment. Results were that the α-GPC treatment resulted in significantly greater isometric mid-thigh pull peak force change from baseline (p=0.044) compared with placebo (α-GPC: 98.8 vs Placebo: -39.0). For the upper body test the α-GPC treatment trended towards greater change from baseline force production (α-GPC: 50.9 vs. Placebo: -14.9) but failed to obtain statistical significance. Researchers concluded that α-GPC is effective at increasing lower body force production after 6 days of supplementation, and sport performance coaches can consider adding α-GPC to the diet of speed and power athletes to enhance muscle performance.

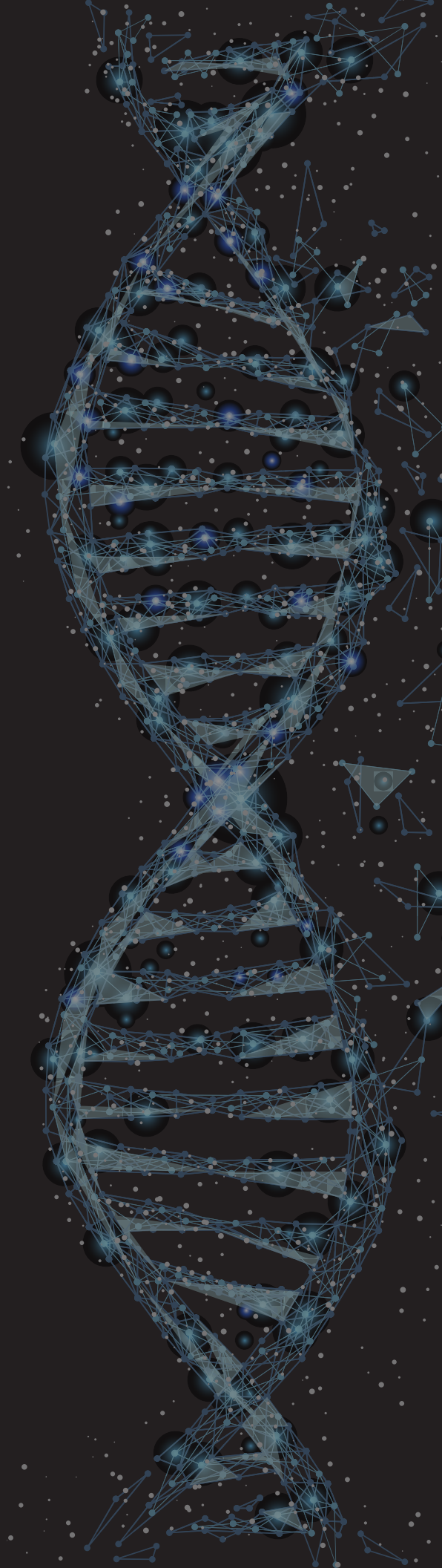
Growth hormone

Growth hormone (GH) is released from the pituitary gland. As the name suggests, in childhood it has It has growth-promoting effects—but it also has beneficial effects in adulthood. Specifically, it stimulates protein synthesis in muscle, it stimulates the release of fatty acids from adipose tissue (aka, fat), it helps maintain bone health, and it inhibits the uptake of glucose by muscle while stimulating uptake of amino acids, which can then be used in the synthesis of proteins and encourages muscle to shift to using fatty acids as a source of energy. In adults, GH is secreted during sleep and in response to exercise.



However, GH secretion rates decline with age. In fact, compared to puberty there is an 83% decline in GH by age 55. This decline in GH production is associated with alterations in body composition, hormonal status, and functional capacity that mimic the changes seen in adult GH deficiency or partial hypogonadism (diminished functional activity of the gonads—the testes in males or the ovaries in females—that may result in diminished sex hormone biosynthesis). In addition to deteriorating memory and cognitive function, the changes in body composition that are most pronounced in normal aging include a reduction in bone density and in muscle mass and strength, an increase in body fat, and adverse changes in lipoprotein profiles. These facts have led to the following statement by researchers from the VA Puget Sound Health Care System and University of Washington, School of Medicine: “Aging is not a disease. Rather, it is a physiological state of relative GH deficiency.”

13 *Phospholipid choline*



It turns out that α-GPC may help to promote GH release. Here's how it works. A hormone called growth hormone releasing hormone (GHRH) is released from the hypothalamus, which in turn stimulates GH secretion from the pituitary. On the other hand, the hypothalamic hormone somatostatin inhibits GH release. With aging, the imbalance between stimulatory (GHRH) and inhibitory (somatostatin) activities on GH secretion leads to an enhanced somatostatin action and decreased GH release. Part of the reason for this imbalance is a concurrent decrease in levels of acetylcholine, since this neurotransmitter has been shown to play an important role in the control of GH secretion. Anticholinergic drugs (or drugs that decrease acetylcholine activity) have been shown to diminish GH response, while drugs able to increase acetylcholine transmission are able to potentiate the stimulatory effect of GHRH through a decrease of somatostatin release. Increasing GHRH, by decreasing somatostatin release is a novel approach to increasing GH levels.

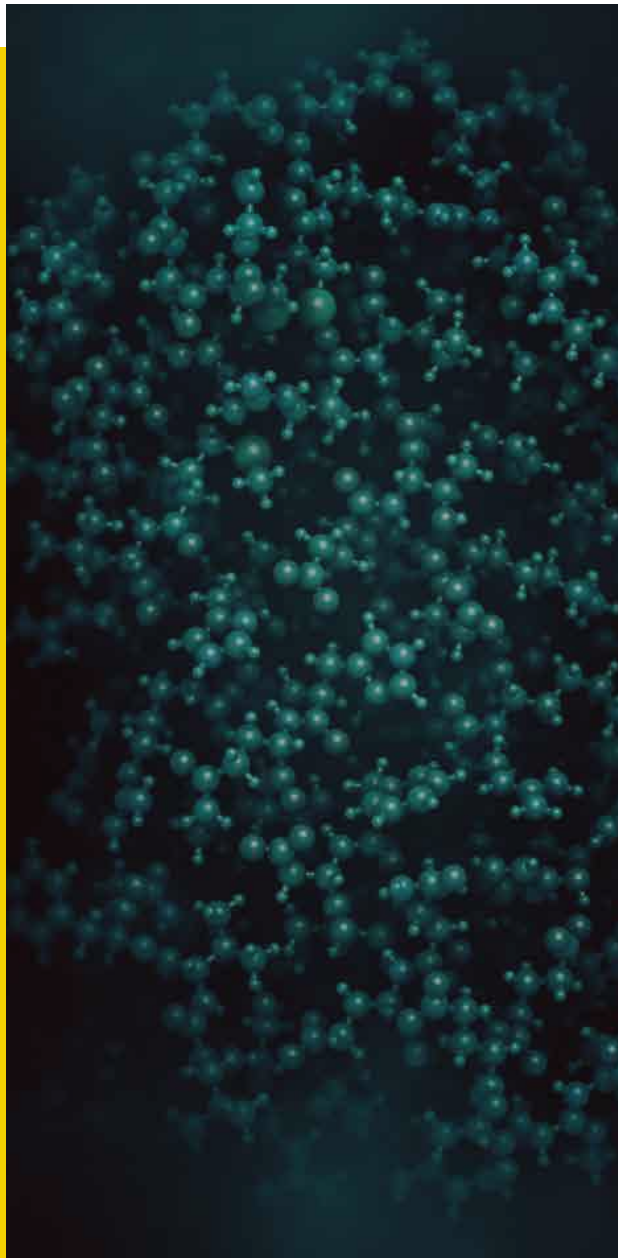
α-GPC fits into the GH picture because, research has demonstrated that it increases acetylcholine release. Research suggests that both older and younger people can benefit from α-GPC to promote GH secretion, but the difference will simply be more profound in older people since they have naturally lower levels of GH. In one study, GHRH was given to eight young and seven old human volunteers, with or without the addition of 1 gram of α-GPC. The results were that the groups given the α-GPC experienced a greater stimulatory effect of GHRH on HGH secretion in both young and old subjects—about 130% greater HGH in the case of the older subjects, and about 35% greater in the case of younger subjects.

In a follow-up study, α-GPC was tested in 10 normal elderly subjects (aged 79.4 ± 1.7 years). The subjects were given GHRH with 2 grams of α-GPC. The results demonstrated that when the 2 grams of A-GPC was administered with GHRH, the HGH responses were significantly higher than those found after GHRH alone, but not significantly different from those seen in the elderly subjects who received 1 gram of α-GPC. This suggests that a maximal potentiation of GHRH-induced GH responses could be achieved by the lower dose.

The amounts of HGH released from the anterior pituitary are summarized in the table below:

Substance(s) given	Young Subjects (1g)	Old Subjects (1g)	Old Subjects (2g)
GHRH	1375 + 263	415 + 119	191 + 57
GHRH + A-GPC	1849 + 215	977 + 273	804 + 182

A later randomized, placebo-controlled, cross-over study examined the effects of a supplement containing primarily 600 mg/day α-GPC (CholineAid® α-GPC, Chemi Nutra) or placebo on serum GH levels, explosive performance, and post-exercise substrate oxidation in seven men with at least two years of resistance training experience. The supplement was taken 90-minutes prior to completing 6 sets × 10 repetitions of squats at 70% of their pre-determined 1-repetition maximum. Results were that, compared to baseline (pre) values, peak GH increased 44-fold during A-GPC (from 0.19 ± 0.06 to 8.4 ± 2.1 ng/mL) vs. 2.6-fold during placebo (from 1.9 ± 0.8 to 5.0 ± 4.8 ng/mL, $P < 0.03$). These data indicate that a single 600 mg dose of α-GPC, when administered 90 minutes prior to resistance exercise, increases post-exercise serum GH.



Autism

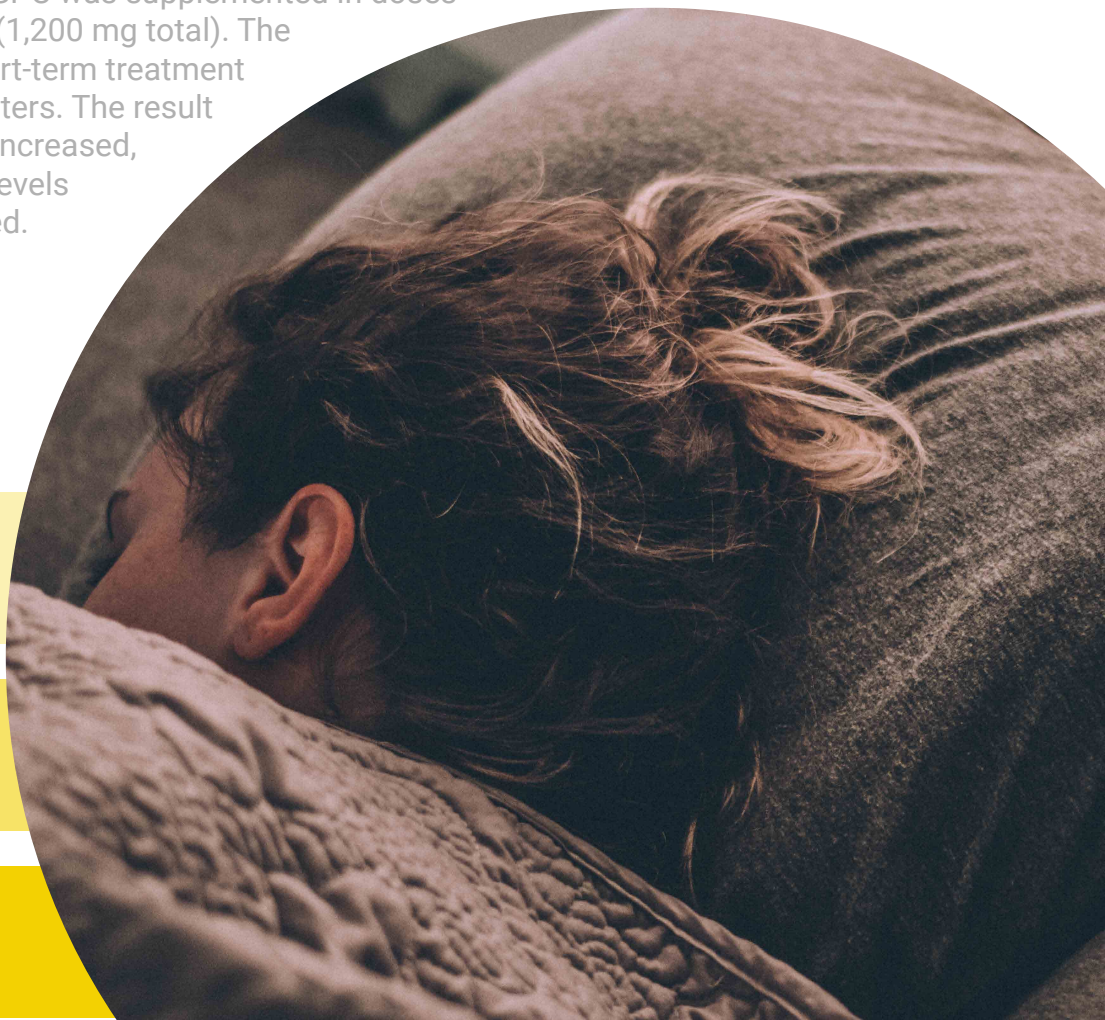
In addition to behavioral issues, mental developmental delay and cognitive dysfunction are usually diagnosed in childhood autism patients. Children who receive only neuroleptic therapy (i.e. major tranquilizers) generally do not demonstrate significant improvement of cognitive functions. Consequently, it is important to include neuroprotective therapy as part of a strategy for treating autism. Since the cholinergic system plays an important role in the functioning of those parts of the brain affected in autism, and since research suggests that α-GPC helps improve cognitive functions and behavioral reactions, a study was conducted to evaluate the therapeutic effects of α-GPC during a 8-week course of supplementation using 400 mg/day in 20 children (aged 3–8 years) with autism. There were ten patients with mild autism, three with moderate autism, and seven with moderate/severe autism. The patients were assessed twice: before (0 day) and after the therapeutic course (56th day). Results showed positive effects observed at the end of therapeutic course in 89 % of the patients: significant improvement in 61 % and minimal improvement in 28 %. Statistically significant positive changes in the patients' state were observed in the general improvement of behavior ($p < 0.001$), development of social and communicative skills, as well as self-service, reduction of marked speech disturbances ($p < 0.001$) and motor sphere dysfunction ($p < 0.001$), enhancement of learning activity and productivity ($p < 0.05$). Improvement was also seen in concentration, imitation, social play activity, speech understanding, thinking, emotional response. The authors concluded that, overall, α-GPC may be recommended for combined therapy with neuroleptics as an effective and safe medicine for the treatment of cognitive and behavioral disorders in patients with childhood autism.

Sleep

Rapid eye movement (REM) sleep is a unique phase of sleep during which the sleeper is likely to dream more vividly. This phase is characterized most notably by an abundance of the acetylcholine. The different theories about REM sleep's function are that it helps in forming new memories, stimulates the central nervous system, and restores brain chemistry to a normal balance. A lack of REM sleep can result in mild psychological disturbances, such as anxiety, irritability, hallucinations, and difficulty concentrating may develop and appetite may increase.

It is now well known that rapid eye movement (REM) density, which is a measure of the frequency of REMs during REM sleep, increases over the course of the sleep episode in successive REM sleep episodes. Protocols utilizing extended sleep episodes have suggested that REM density increases with prolonged sleep duration and eventually saturates at a maximal level It has even been proposed that REM density is an index of sleep satiety—that is, when you get good quality sleep, you don't need as much sleep. Accordingly, an inverse relationship between the depth of sleep and REM density has been observed. Consequently, there would seem to be an advantage to increasing REM density. Such an increase was seen with α-GPC supplementation.

A single-blind controlled study was conducted in eight healthy young male subjects to assess the effect of α-GPC, on sleep. α-GPC was supplemented in doses of 400 mg, three times daily (1,200 mg total). The study examined whether short-term treatment modified REM sleep parameters. The result were that REM density was increased, which returned to pre-study levels once supplementation ceased.



CONCLUSION

Choline is a vital nutrient for human health and wellness and is needed in all stages of life. The phospholipid form of choline known as α-Glyceryl Phosphoryl Choline (α-GPC) is the demonstrably preferred form of this nutrient. Research demonstrates that α-GPC provides benefits for cognition (young and older people), exercise performance, promoting growth hormone release, behavioral disorders in autism, and supporting REM in sleep.

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