Choline: needed for normal development of memory.
Choline: needed for normal development of memory.

Zeisel SH.

Source
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Abstract
Choline is a dietary component essential for normal function of all cells. It, or its metabolites, assures the structural integrity and signaling functions of cell membranes; it is the major source of methyl-groups in the diet (one of choline's metabolites, betaine, participates in the methylation of homocysteine to form methionine); and it directly affects nerve signaling, cell signaling and lipid transport/metabolism. In 1998, the National Academy of Sciences, USA, issued a report identifying choline as a required nutrient for humans and recommended daily intake amounts. Eggs are an excellent dietary source of choline. Pregnancy and lactation are periods when maternal reserves of choline are depleted. At the same time, the availability of choline for normal development of the brain is critical. When rat pups received choline supplements (in utero or during the second week of life), their brain function changed, resulting in the lifelong memory enhancement. This change in memory function appears to be due to changes in the development of the memory center (hippocampus) in the brain. The mother's dietary choline during a critical period in brain development of her infant influences the rate of birth and death of nerve cells in this center. These changes are so important that we can pick out the groups of animals whose mothers had extra choline even when these animals are elderly. Thus, memory function in the aged rat is, in part, determined by what the mother ate. This is not the first example of a critical nutrient that must be present at a specific time in brain development. If folate isn't available in the first few weeks of pregnancy, the brain does not form normally. Thus, we suggest that pregnancy is a period when special attention has to be paid to dietary intake.

PMID: 11023003    Free full text
„Choline: an important micronutrient for maximal endurance-exercise performance?“

Cholin: ein wichtiger Mikronährstoff für maximale Leistung bei Ausdauersport?

Χολίνη: ένα σημαντικό μικροθρεπτικό συστατικό για μέγιστες επιδόσεις ασκήσεων αντοχής;

Choline: un important micronutriment pour une performance maximale à l'exercice d'endurance?

Cholin: důležitá mikroživina pro maximální vytrvalostní výkon?
Choline: an important micronutrient for maximal endurance-exercise performance?

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Abstract

Choline plays a central role in many physiological pathways, including neurotransmitter synthesis (acetylcholine), cell-membrane signaling (phospholipids), lipid transport (lipoproteins), and methyl-group metabolism (homocysteine reduction). Endurance exercise might stress several of these pathways, increasing the demand for choline as a metabolic substrate. This review examines the current literature linking endurance exercise and choline demand in the human body. Also reviewed are the mechanisms by which exercise might affect blood choline levels, and the links between methyl metabolism and the availability of free choline are highlighted. Finally, the ability of oral choline supplements to augment endurance performance is assessed. Most individuals consume adequate amounts of choline, although there is evidence that current recommendations might be insufficient for some adult men. Only strenuous and prolonged physical activity appears sufficient to significantly decrease circulating choline stores. Moreover, oral choline supplementation might only increase endurance performance in activities that reduce circulating choline levels below normal.

PMID: 18458362
Exercise and neuromodulators: choline and acetylcholine in marathon runners.

Sport und Neuromodulatoren: Cholin und Acetylcholin bei Marathonläufern.

Άσκηση και νευροτροποποιητές: χολίνη και ακετυλοχολίνη σε μαραθωνοδρόμους.

Exercice et neuromodulateurs : la choline et l’acétylcholine chez les marathoniens.

Cvičení a neuromodulátory: cholin a acetylcholin u maratonových běžců.
Exercise and neuromodulators: choline and acetylcholine in marathon runners.

Conlay LA, Sabounjian LA, Wurtman RJ.

Source Laboratory of Neuroendocrine Regulation, Massachusetts Institute of Technology.

Abstract

Certain neurotransmitters (i.e., acetylcholine, catecholamines, and serotonin) are formed from dietary constituents (i.e., choline, tyrosine and tryptophan). Changing the consumption of these precursors alters release of their respective neurotransmitter products. The neurotransmitter acetylcholine is released from the neuromuscular junction and from brain. It is formed from choline, a common constituent in fish, liver, and eggs. Choline is also incorporated into cell membranes; membranes may likewise serve as an alternative choline source for acetylcholine synthesis. In trained athletes, running a 26 km marathon reduced plasma choline by approximately 40%, from 14.1 to 8.4 μM. Changes of similar magnitude have been shown to reduce acetylcholine release from the neuromuscular junction in vivo. Thus, the reductions in plasma choline associated with strenuous exercise may reduce acetylcholine release, and could thereby affect endurance or performance.

PMID: 1483754
Effects on the diet on brain neurotransmitters.

Wirkung der Ernährung auf Neurotransmitter im Gehirn.

Αποτελέσματα της δίαιτας σε νευροδιαβιβαστές του εγκεφάλου.

Effets de la nutrition sur les neurotransmetteurs.

Vliv potravy na neurotransmitery v mozku.
Effects on the diet on brain neurotransmitters.

Fernstrom JD.

Abstract

The synthesis of neurotransmitters in mammalian brain responds rapidly to changes in precursor availability. Serotonin synthesis depends largely on the brain concentrations of L-tryptophan, its precursor amino acid. This relationship appears to be physiologic: when brain tryptophan levels vary because of insulin secretion or meal ingestion, corresponding alterations occur in the rate of serotonin formation. The ability of any food to modify brain tryptophan (and serotonin) depends on how its ingestion changes the serum concentration of not only tryptophan, but also several other large neutral amino acids that compete with tryptophan for uptake into the brain. Such precursor-induced changes in brain serotonin appear to be functionally important: animals having a reduced level of brain serotonin (caused by the chronic ingestion of a naturally tryptophan-poor diet, such as corn) demonstrate a heightened sensitivity to painful stimuli; this pain sensitivity can be acutely restored to normal values by a single injection of L-tryptophan, which rapidly elevates brain serotonin. The synthesis of catecholamines (e.g., dopamine, norepinephrine) in the brain also varies with the availability of the precursor amino acid L-tyrosine. Single injections of this amino acid increase brain tyrosine levels and accelerate brain catechol synthesis, while injections of a competing neutral amino acid (e.g., leucine, tryptophan) reduce brain tyrosine and its rate of conversion to dopa. The rate of catecholamine synthesis, however, appears to be influenced less by precursor levels than is serotonin formation: tyrosine hydroxylase, which catalyzes the rate-limiting step in catecholamine synthesis, responds strongly to end-product inhibition and to other controls that reflect variations in neuronal activity. The synthesis of acetylcholine in brain responds to substrate (choline) availability much like serotonin synthesis. Short-term alterations in brain choline levels are mirrored by similar changes in brain acetylcholine concentration. Variations in the daily dietary intake of choline also modify brain choline and acetylcholine. The relationship between choline
availability and acetylcholine synthesis has already found a clinical application: choline has been used successfully in the treatment of tardive dyskinesia, a disorder of the central nervous system thought to reflect a deficiency in cholinergic transmission. These relationships between precursor availability from the periphery and brain neurotransmitter synthesis may ultimately provide the brain with information about peripheral metabolic state.

PMID: 13261
“Plasma free choline, betaine and cognitive performance: the Hordaland Health Study.”

“Plasmafreies Cholin, Betain und kognitive Leistung: die Hordaland Health Study.”

Ελεύθερη χολίνη στο πλάσμα, βεταϊνη και νοητικές επιδόσεις: η μελέτη Hordaland Health Study.

“Choline exempte de plasma, bétaïne et performance cognitive : L’étude sanitaire Hordaland.”

“Volný cholin v plazmě, betain a kognitivní výkonnost: Hordaland Health Study (Studie zdraví Hordalandu).”
Plasma free choline, betaine and cognitive performance: the Hordaland Health Study.

Nurk E, Refsum H, Bjelland I, Drevon CA, Tell GS, Ueland PM, Vollset SE, Engedal K, Nygaard HA, David Smith A.

Source Department of Nutrition, Faculty of Medicine, Institute of Basic Medical Sciences, University of Oslo, Oslo, Norway.

Abstract

Choline and betaine are nutrients involved in one-carbon metabolism. Choline is essential for neurodevelopment and brain function. We studied the associations between cognitive function and plasma concentrations of free choline and betaine.

In a cross-sectional study, 2195 subjects (55% women), aged 70-74 years, underwent extensive cognitive testing including the Kendrick Object Learning Test (KOLT), Trail Making Test (part A, TMT-A), modified versions of the Digit Symbol Test (m-DST), Block Design (m-BD), Mini-Mental State Examination (m-MMSE) and Controlled Oral Word Association Test (COWAT). Compared with low concentrations, high choline (>8-4 µmol/l) was associated with better test scores in the TMT-A (56-0 v. 61-5, P = 0-004), m-DST (10-5 v. 9-8, P = 0-005) and m-MMSE (11-5 v. 11-4, P = 0-01). A generalised additive regression model showed a positive dose-response relationship between the m-MMSE and choline (P = 0-012 from a corresponding linear regression model). Betaine was associated with the KOLT, TMT-A and COWAT, but after adjustments for potential confounders, the associations lost significance. Risk ratios (RR) for poor test performance roughly tripled when low choline was combined with either low plasma vitamin B12 (≤ 257 pmol/l) concentrations (RRKOLT = 2-6, 95% CI 1-1, 6-1; RRm-MMSE = 2-7, 95% CI 1-1, 6-6; RRm-BD = 2-8, 95% CI 1-3, 6-1). Low betaine (≤ 31-1 µmol/l) combined with high MMA was associated with elevated RR on KOLT (RRKOLT = 2-5, 95% CI 1-0, 6-2). Low plasma free choline concentrations are associated with poor cognitive performance. There were significant interactions between low choline or betaine and low vitamin B12 or high MMA on cognitive performance.

PMID: 22717142
"Oral choline increases choline metabolites in human brain."

Orales Cholin erhöht die Cholinmetaboliten im menschlichen Gehirn.

Η χορήγηση χολίνης από στόματος αυξάνει τους μεταβολίτες της χολίνης στον ανθρώπινο εγκέφαλο.

La choline orale accroît les métabolites de la choline dans le cerveau humain.

Perorální cholin zvyšuje množství metabolitů cholinu v lidském mozku.
Oral choline increases choline metabolites in human brain.


Source Department of Psychiatry, Harvard Medical School, McLean Hospital, 115 Mill Street, Belmont, MA 02478, USA.

Abstract

Choline, a precursor of acetylcholine and phosphatidylcholine, is largely obtained from the diet. Animal studies demonstrate increased choline metabolites in brain following oral administration. Several proton magnetic resonance spectroscopy ((1)H-MRS) reports differ as to whether similar increases are observable in human subjects. This study was designed to minimize intra-subject variance and thereby maximize the ability to determine if a significant increase in brain choline can be detected after choline ingestion. (1)H-MRS was performed continuously for 2.5 h on 11 healthy young males following choline ingestion. Nine of the original subjects returned for identical scans without choline ingestion. Following oral choline, there was a statistically significant increase in the choline signal (Cho) measured from the left putamen, representing choline-containing compounds, as measured against creatine (Cr) or N-acetylaspartate (NAA). The mean increase in Curve maxima (C(max)) is 6.2% for Cho/Cr and 3.0% for Cho/NAA. The Mean Time to C(max) (T(max)) was approximately 2 h after ingestion. A 3-6% increase in Cho by MRS likely corresponds to a 10-22% increase in phosphocholine, similar to findings in animal studies. In conclusion, a significant increase in choline-containing compounds in human brain can be detected by (1)H-MRS after choline ingestion in young subjects.

PMID: 14972364
"Nutritional modifiers of aging brain function: use of uridine and other phosphatide precursors to increase formation of brain synapses."

Modifikation der alternden Hirnfunktion durch Ernahrung: Anwendung von Uridin und anderen Phosphatidvorläufern zur erhöhten Bildung von Hirnsynapsen.

Διατροφικοί τροποποιητές της εγκεφαλικής γήρανσης: χρήση ουριδίνης και άλλων πρόδρομων ουσιών των φωσφατιδίων για αύξηση του σχηματισμού εγκεφαλικών συνάψεων.

Modificateurs nutritionnels du fonctionnement du cerveau vieillissant : utilisation d’uridine et d’autres précurseurs phosphatidiques pour accroître la formation de synapses cérébraux.

Funkce nutričních modifikátorů stárnoucího mozků: příjem uridinu a ostatních fosfatidových prekurzorů ke zvýšení formace synapsí v mozků.
Nutritional modifiers of aging brain function: use of uridine and other phosphatide precursors to increase formation of brain synapses.

Wurtman RJ, Cansev M, Sakamoto T, Ulus I.

Source

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Abstract

Brain phosphatide synthesis requires three circulating compounds: docosahexaenoic acid (DHA), uridine, and choline. Oral administration of these phosphatide precursors to experimental animals increases the levels of phosphatides and synaptic proteins in the brain and per brain cell as well as the numbers of dendritic spines on hippocampal neurons. Arachidonic acid fails to reproduce these effects of DHA. If similar increases occur in human brain, administration of these compounds to patients with diseases that cause loss of brain synapses, such as Alzheimer's disease, could be beneficial.

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PMID: 21091953

Free PMC Article
„Dietary phosphatidylcholine improves maze-learning performance in adult mice."
Dietary phosphatidylcholine improves maze-learning performance in adult mice.

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Abstract

The effect of phosphatidylcholine (PC) on maze-learning performance was investigated in adult mice. Three-month-old mice were given a semipurified diet of 1%, 2.5%, or 5% PC over a period of 4 months, with their maze-learning ability assessed beginning at 3 months after the start of the experiment and again 4 and 8 days later. This entailed the measurement of the time required by the mice to reach the maze exit and counting the number of times that mice strayed into blind alleys in the maze. During trial 1, mice in the 5% PC diet group required significantly less time to reach the maze exit compared with the control group (P < .05), while mice in the 1% and 2.5% PC dietary groups tended to require a shorter time to find the exit, but the differences were not significant. The number of times that mice strayed into blind alleys in the maze was significantly fewer in the 2.5% and 5% PC diet groups than in the control group during trial 1 (P < .05). The PC diets increased the percentages of docosahexaenoic and arachidonic acids in serum but had a lesser effect on brain fatty acid composition. These results suggest that the intake of 5% PC diet improves learning ability in adult mice and that the improved brain function may be related to the provision of choline.

PMID: 18361742

CholinScientific Study Collection (9/11)


Source Institute for CNS Disorders, Basic and Clinical Neurosciences Research Center, La Coruña, Spain.

Abstract

CDP-choline was given to patients with Alzheimer's disease (AD) at a daily dose of 1000 mg/day p.o. for one month. This compound slightly improved mental performance, tended to reduce theta activity in fronto-temporal regions, increasing alpha power in occipital areas, and enhanced cerebrovascular perfusion by increasing blood flow velocity and reducing pulsatility and resistance indexes. In addition, CDP-choline diminished histamine and interleukin-1 levels in blood and serum, respectively, and increased plasma TNF.

PMID: 8624120
Gene response elements, genetic polymorphisms and epigenetics influence the human dietary requirement for choline.

Gen Response Elemente, genetische Polymorphismen und Epigenetik beeinflussen den diätetischen Bedarf von Cholin beim Menschen.

Στοιχεία απόκρισης των γονιδίων, γενετικοί πολυμορφισμοί η επιγενετική επηρεάζουν τις ανθρώπινες διατροφικές ανάγκες χολίνης.

Les éléments de réponse génique, les polymorphismes génétiques et l'épigénétique influencent les exigences nutritionnelles humaines relatives à la choline.

Responsivní elementy genu, genetický polymorfismus a epigenetika ovlivňují požadavky na cholin v potravě člověka.
Gene response elements, genetic polymorphisms and epigenetics influence the human dietary requirement for choline.

Zeisel SH.

Source Nutrition Research Institute, Department of Nutrition, School of Public Health and School of Medicine, University of North Carolina at Chapel Hill, North Carolina 27599, USA.
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Abstract

Recent progress in the understanding of the human dietary requirement for choline highlights the importance of genetic variation and epigenetics in human nutrient requirements. Choline is a major dietary source of methyl-groups (one of choline's metabolites, betaine, participates in the methylation of homocysteine to form methionine); also choline is needed for the biosynthesis of cell membranes, bioactive phospholipids and the neurotransmitter acetylcholine. A recommended dietary intake for choline in humans was set in 1998, and a portion of the choline requirement can be met via endogenous de novo synthesis of phosphatidylcholine catalyzed by phosphatidylethanolamine N-methyltransferase (PEMT) in the liver. Though many foods contain choline, many humans do not get enough in their diets. When deprived of dietary choline, most adult men and postmenopausal women developed signs of organ dysfunction (fatty liver, liver or muscle cell damage, and reduces the capacity to handle a methionine load, resulting in elevated homocysteine). However, only a portion of premenopausal women developed such problems. The difference in requirement occurs because estrogen induces expression of the PEMT gene and allows premenopausal women to make more of their needed choline endogenously. In addition, there is significant variation in the dietary requirement for choline that can be explained by common polymorphisms in genes of choline and folate metabolism. Choline is critical during fetal development, when it alters DNA methylation and thereby influences neural precursor cell proliferation and apoptosis. This results in long term alterations in brain structure and function, specifically memory function.

PMID: 17613168

Free PMC Article
Lecithin and choline in human health and disease.

Lecithin and choline in human health and disease.

Canty DJ, Zeisel SH.

Source Department of Nutrition, Food, and Hotel Management at New York University, NY.

Abstract

Choline is involved in methyl group metabolism and lipid transport and is a component of a number of important biological compounds including the membrane phospholipids lecithin, sphingomyelin, and plasmalogen; the neurotransmitter acetylcholine; and platelet activating factor. Although a required nutrient for several animal species, choline is not currently designated as essential for humans. However, recent clinical studies show it to be essential for normal liver function. Additionally, a large body of evidence from the fields of molecular and cell biology shows that certain phospholipids play a critical role in generating second messengers for cell membrane signal transduction. This process involves a cascade of reactions that translate an external cell stimulus such as a hormone or growth factor into a change in cell transport, metabolism, growth, function, or gene expression. Disruptions in phospholipid metabolism can interfere with this process and may underlie certain disease states such as cancer and Alzheimer's disease. These recent findings may be appropriate in the consideration of choline as an essential nutrient for humans.

PMID: 7816350