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Sulfur Amino Acid

Sulfur amino acids can prevent browning in various food products like fruit juices, being N-acetyl-l-cysteine and others – SH-containing compounds – as effective as sodium bisulfite in decreasing the rate of browning.

From: Encyclopedia of Food and Health, 2016

Related terms:

Amino Acid, Cysteine, Methionine, Taurine, Homocysteine, Metabolism, Enzyme, Protein, Protein Intake, Sulfur

Sulfur Amino Acid-Restricted Diets: Mechanisms and Health Benefits

Gene P. Ables, in Reference Module in Life Sciences, 2020

Rodents

When sulfur amino acids were restricted by 80% (normal = 0.86% (CF); experimental = 0.17% (SAAR)) in young rats, the main significant effect observed was lifespan extension (Orentreich *et al.*, 1993; Richie *et al.*, 1994). The SAAR diet was based on the American Institute of Nutrition formulation 76 (AIN-76) wherein dietary protein is replaced by an amino acid mixture in which methionine is reduced, cysteine is removed, and glutamic acid is increased to balance nitrogen (Table 1). In those studies, Fischer 344 rats (F344) on the SAAR diet starting at 4–6 weeks of age showed greater median lifespan (SAAR = 1059 days vs CF (control fed) = 818 days), while maximum lifespan was extended (SAAR = 1252 days vs CF = 1116 days) despite the SAAR group consuming similar amounts of food to the CF group (Orentreich *et al.*, 1993). The lifespan extension benefit of SAAR has been replicated in other commonly used strains of rats, namely Sprague–Dawley, Wistar–Hannover, and Brown Norway which were fed CF or SAAR diets beginning at 6 weeks of age (Zimmerman *et al.*, 2003).

Table 1. Composition of SAAR diet in rats. Control diet ingredients are in parentheses

Ingredients	g/100 g
L-arginine	1.12
L-histidine	0.33
L-isoleucine	0.82

Ingredients	g/100 g
L-leucine	1.11
L-lysine	14.4
DL-methionine	0.17 (0.86)
L-phenylalanine	1.16
L-threonine	0.82
L-tryptophan	0.18
L-valine	0.82
L-glutamic acid	3.39 (2.7)
Glycine	2.33
Corn Starch	43.61
Sucrose	20.0
Solka-Floc	5.00
Corn oil	8.00
Minerals	3.50
Vitamins	1.00
Choline bitartrate	0.20

In mice, lifespan extension by SAAR has been demonstrated in a non-inbred but genetically homogeneous strain of CB6F1 mice that was generated from a cross of BALB/cJ and C57BL/6J strains (Miller *et al*., 2005; Sun *et al*., 2009). Controls were fed a diet containing 0.43% methionine (w/w) while the SAAR groups were treated with incremental reduction of methionine down to 0.15% methionine (w/w) as the mice aged (Miller *et al*., 2005; Sun *et al*., 2009). CF mice lived for 1144 ± 26 days, while SAAR mice survived until 1261 ± 32 days (Miller *et al*., 2005). When male CB6F1 mice were started on the diet beginning at 12 months of age, median lifespan was 1011 days in SAAR mice compared to 948 days in CF mice (Sun *et al*., 2009). Compared to controls, females on SAAR experienced low levels of IGF-1, insulin, glucose and thyroid hormone; delayed lens turbidity development; and, improved response to hepatic oxidative stress (Miller *et al*., 2005). It is possible that SAAR achieves these effects in part by inducing upregulation of Jnk2 and downregulation of Akt protein levels (Sun *et al*., 2009). SAAR also extends lifespan in 2 mouse models of Hutchinson-Gilford progeria by downregulating transcriptome pathways related to DNA damage (p53 pathway, DNA repair, and apoptosis) and inflammation (TNFA signaling via NF- kB and IL-6, JAK, STAT3 signaling) (Barcena *et al*., 2018). Because SAAR improves the lipid profile and bile acid levels in wild-type and progeroid mice, it is possible that SAAR-induced alterations in different metabolic pathways may be beneficial in treating progeria (Barcena *et al*., 2018).

Overall, studies of SAAR in rodents have identified putative molecular mechanisms and revealed their potential for delaying age-related conditions and for extending lifespan.

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Disorders of Amino Acid Metabolism

Marc Yudkoff, Marc Yudkoff, in Basic Neurochemistry (Eighth Edition), 2012

Disorders of Sulfur Amino Acid Metabolism: Homocystinuria 745

- The transsulfuration pathway is the major route for the metabolism of the sulfur-containing amino acids 745
- Homocystinuria is the result of the congenital absence of cystathionine synthase, a key enzyme of the transsulfuration pathway 747
- Homocystinuria can be treated in some cases by the administration of pyridoxine (Vitamin B_6), which is a cofactor for the cystathionine synthase reaction 747
- Patients with homocystinuria are at risk for cerebrovascular and cardiovascular disease and thromboses 747
- Prognosis is more favorable in the pyridoxine-responsive patients 747
- Homocystinuria can occur when homocysteine is not remethylated back to form methionine 748
- One form of remethylation deficit involves defective metabolism of folic acid, a key cofactor in the conversion of homocysteine to methionine 748
- Methionine synthase deficiency (cobalamin-E disease) produces homocystinuria without methylmalonic aciduria 748
- Cobalamin-c disease: remethylation of homocysteine to methionine also requires an 'activated' form of vitamin B_{12} 748
- Hereditary folate malabsorption presents with megaloblastic anemia, seizures and neurological deterioration 749

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Homocysteine

J.W. Miller, in Encyclopedia of Human Nutrition (Third Edition), 2013

Homocysteine is a sulfur amino acid and a product of methionine metabolism. The metabolism of homocysteine requires B-vitamins, including folate, vitamin B_{12}, vitamin B_6, and riboflavin. Disruption of homocysteine metabolism due to B-vitamin deficiencies, genetic defects, or other pathophysiological conditions leads to elevation of homocysteine in the blood (hyperhomocysteinemia). Hyperhomocysteinemia is a risk factor for vascular disease, neurodegenerative disease, and other clinical conditions. B-vitamin supplements are effective in lowering blood homocysteine concentrations, but it may have little or no effect in reducing the risk of vascular events. Homocysteine may therefore be a risk marker but not a cause of vascular disease.

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Minerals and Trace Elements

Martin Kohlmeier, in Nutrient Metabolism (Second Edition), 2015

Nutritional Summary

Function: The sulfur amino acids methionine and cysteine are necessary for the synthesis of proteins and serve as precursors of important cofactors and metabolites. Development and maintenance of brain and nerves, spermatogenesis, joint repair, hormone action, and many other body functions are critically dependent on undisturbed sulfate metabolism. Sulfate is an essential constituent of many proteins, glycans, and glycolipids, and plays an important role in the activation and elimination of hormones, metabolites, phytochemicals, and xenobiotics. Various other sulfurous compounds, such as taurine, thiamin, biotin, and thiosulfate, have specific functions in the body.

Food sources: Most utilizable sulfur comes with sulfur amino acids in proteins of animal and plant origin. Much smaller amounts are consumed as sulfate. Some sulfur compounds, such as the potentially toxic hydrogen sulfide, arise when bacterial in the distal intestine act on nonabsorbed amino acids.

Requirements: Daily sulfur needs are met when adequate amounts (13 mg/kg) of methionine and cysteine are consumed (Young & Borgonha, 2000).

Deficiency: Since symptoms of methionine and cysteine deficiency will occur with inadequate intake, the consequences of isolated sulfur deficiency cannot be determined. Experience with the rare patients with defective sulfate transporters indicates a particular vulnerability of the brain and connective tissue.

Excessive intake: Excessive sulfate intake may accelerate bone mineral loss and increase the risk of osteoporosis. Exposure of sensitive individuals to large doses of sulfite (>100 mg) can trigger asthma attacks, urticaria, and related symptoms.

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Diet Modulation of Chronic Inflammation in Individuals with Acquired Immune Deficiency Syndrome

Lauren E. Lawson, Ronald Ross Watson, in Health of HIV Infected People, 2015

6.5.1 Sulfur Amino Acids

The levels of sulfur amino acids and amino acids metabolically related to them are higher during inflammation [2]. Patients in the late stages of HIV infection have an increased concentration and production of free radicals accompanied by activation of lymphocytes and phagocytic cells and chronic inflammation [9]. Additionally, patients with HIV exhibit abnormally low cysteine and glutamine levels in the subsequent stages [2,4,9]. These same levels have been observed in both humans and simian immunodeficiency virus(SIV)–infected rhesus macaques [2,4,9]. Although it is understood that a diet lacking in protein reduces the glutathione content in the lungs and the liver, it is not known whether the low levels are the

result of protein deficiency as a whole or deficiency of cysteine, glycine, or glutamate alone [2,10,11].

However, the influence of methionine and cysteine supplementation was observed in protein-depleted rats receiving cytokine injections, which served to stimulate inflammation in the rats [2]. Addition of cysteine and methionine to the low-protein diet permitted and increased the glutathione content of both the liver and the lungs in response to the cytokine injection, suggesting that these amino acids are important in maintaining and enhancing glutathione levels in tissue [2]. Cysteine, a semi-essential amino acid, meaning it can be biosynthesized in humans, amplifies several lymphocyte functions [2]. Additionally, by increasing the intake of sulfhydryl compounds such as cysteine and glutathione, free-radical stimulation of cytokine production can be inhibited [4,10].

Subsequently, no direct data were found with regard to the impact of increased intake of sulfur amino acids on human immune function, whereas glutathione, homocysteine, and taurine were found to influence inflammation [12]. However, limited direct experimentation has been performed on humans, and thus, the full significance of the effect of amino acids on immune function is still being defined despite the theoretical importance of sulfur amino acids in immune function [12].

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HOMOCYSTEINE

J.W. Miller, in Encyclopedia of Human Nutrition (Second Edition), 2005

Introduction

Homocysteine is a sulfur amino acid and an intermediate in the biochemical conversion of methionine to cysteine, a process called trans-sulfuration. Vincent Du Vigneaud and others elucidated the biochemistry of homocysteine over the period from the 1930s to the 1950s. In the early 1960s, the description and characterization of the inborn error of metabolism, homocystinuria, initiated a 40-year (and continuing) period of investigation that has revealed homocysteine as an independent risk factor for vascular disease. The association between elevated blood levels of homocysteine (hyperhomocysteinemia) and vascular disease may be similar in magnitude to the association between cholesterol and vascular disease, thus implicating hyperhomocysteinemia as a significant public health concern. Currently, large-scale intervention trials are being conducted to determine if supplements of the B vitamins folate, vitamin B_{12}, and vitamin B_6, each of which plays an integral role in homocysteine metabolism, reduce the incidence of vascular disease. If successful, B vitamin supplements may prove to be an inexpensive and safe prophylactic to reduce the risk of heart attacks and strokes.

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Taurine Deficiency and the Eye

Nicolas Froger, ... Serge Picaud, in Handbook of Nutrition, Diet and the Eye, 2014

Abstract

Taurine is a small sulfur amino acid found in high amounts in mammalian eyes and is the most abundant amino acid in the retina. Although taurine can be synthesized endogenously, the main source is the diet. This exogenous taurine intake is regulated by active taurine uptake into tissues by a specific taurine transporter. Taurine depletion, by administering a taurine-free diet or selective blocker of the taurine transporter, generates severe retinal damage in the photoreceptor layer. Taurine depletion has been implicated in the retinal toxicity of the antiepileptic drug vigabatrin, characterized by cone damage and loss of retinal ganglion cells (RGCs). The taurine dependency for RGC survival was further demonstrated on purified RGC cultures and animal models of RGC degeneration. Reduced retinal blood perfusion may lead to decreased retinal taurine uptake from the blood and to RGC loss. High levels of taurine are present in the anterior part of the eye. Taurine may prevent the development of cataract and dry eye through its osmoregulatory action. Taurine dietary intake is therefore a major factor in eye health.

Sulfur Amino Acids and Skeletal Muscle

Isabelle Papet, ... Isabelle Savary-Auzeloux, in Nutrition and Skeletal Muscle, 2019

Abstract

Methionine and cysteine are the two sulfur amino acids amongst the 20 standard ones incorporated into proteins. In addition to the dietary supply, cysteine can be synthetized from methionine and serine mainly in the liver but not in skeletal muscle. A large part of the whole body flux of methionine transmethylation contributes to the synthesis of creatine and carnitine. Cysteine is the rate-limiting substrate of the synthesis of glutathione and the precursor of taurine, coenzyme A, reduced and oxidized inorganic sulfurs. Only the coenzyme A synthesis pathway is complete in skeletal muscle. Skeletal muscle sulfur amino acid metabolism depends on the arterial availability of methionine and cysteine providers (cysteine, cystine, glutathione, other peptides). Creatine, cartinine, and coenzyme A are essential for ATP production and regeneration, and taurine plays also a role in muscle physiology. Conceptually, supplementations with sulfur amino acids or precursors might palliate some muscle impairments.

Thiols in Cancer

JASON M. HANSEN, DEAN P. JONES, in Nutritional Oncology (Second Edition), 2006

OPTIMAL SULFUR AMINO ACID INTAKE IN HUMANS REMAINS UNCERTAIN

The average American diet contains ~2.4 g of sulfur amino acids per day (i.e., about 2.4 times the recommended daily allowance [RDA]) (Flagg et al., 1994). Intake ranges from <0.5 g to >5 g and is, in general, highly related to animal protein consumption. Sulfur amino acid content in legumes and some nuts is ~20–25% of that in animal protein; other vegetarian food sources often contain only ~10% of that in animal protein. Because protein content is low in foods derived from plants, vegetarians are at risk of being sulfur amino acid deficient. However, sulfur amino acid balance studies are necessary because this effect may be counteracted by a higher content of phytochemicals that induce GSH synthesis and GSH-dependent detoxification systems. Epidemiological studies often show protective effects associated with intake of fruits and vegetables. Thus, if a carcinogenic risk occurs in some individuals due to deficient sulfur amino acid intake, this effect must be restricted to a subgroup rather than to the general population or to specific periods of risk. This can occur, for instance, following illness, surgery, or antitumor therapy that results in prolonged nutritional insufficiency. Increased sulfur amino acid requirements occur during healing, with evidence that individuals with extensive burns have a threefold increase in requirement for sulfur amino acids. To optimize individual health, some individuals may benefit from supplemental sources of sulfur amino acid precursors to protect against mutagenic electrophiles. At present, no clinical tests are available to assess optimal intake of sulfur amino acids.

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Drug–Nutrient Interactions in Renal Failure

Raimund Hirschberg, in Nutritional Management of Renal Disease, 2013

Nutrients and Cytochrome P-450-Dependent Drug Metabolism

Increased dietary protein augments hepatic microsomal cytochrome P-450 content probably through tryptophan and sulfur amino acids [13]. In contrast, the activity of this enzyme system is reduced by low protein, high carbohydrate or fat diets, flavonoids (i.e., contained in citrus fruits and some vegetables), large doses of riboflavin and possibly during total parenteral nutrition [12]. The cytochrome P-450IIE1 (CYP2E1) isoenzyme is induced by dietary lipids [14]. This isoenzyme participates in the metabolism of acetaminophen, enflurane and halothane [11]. Moreover, the CYP2E1 isoenzyme is also activated by fasting, as well as by thiamine deficiency [14]. Several other alterations in the micronutrient status can affect the oxidative metabolism of drugs (Table 44.4).

TABLE 44.4. Effects of Vitamin and Trace Element Alterations on Oxidative Drug Metabolism[1]

Vitamin A deficiency	↓ P-450
	↓ Metabolism of aminopyrine, Coumadin
Vitamin A, high dose	↑ Metabolism of Coumadin
Niacin deficiency	↓ Metabolism of anesthetics

Riboflavin deficiency	↓ NADPH:P-450 reductase
	↑ Aminopyrine metabolism
Vitamin C deficiency	↓ P-450
	↓ NADPH:P-450 reductase
	↓ Monooxigenase activities
Folic acid deficiency	↓ Induction of P-450IIB1 by barbiturates
Aluminum, high dose	↓ Hepatic P-450
Selenium deficiency	↓ Induction of P-450 by phenobarbital
Zinc deficiency	↓ Phenobarbital and aminopyrine metabolism

1
Adapted from [11].

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