Why Copper in Your Multi Does Not Cause Alzheimer's or Senile Dementia

By David Seaman, DC, MS, DABCN

There is typically 1-2 mg of copper in a multivitamin/mineral. It cannot cause Alzheimer's or dementia.

Facts

- The upper tolerable limit for copper ingestion is set at 10 mg per day.
- "Copper toxicity is rather rare in humans and animals, because mammals have evolved precise homeostatic control of copper due to the high reactivity of the free metal."
- Free copper in cells and in circulation always exists in low concentrations and is primarily bound to proteins.

Collins JF, Klevay LM. Copper. Adv Nutr. 2011;2:520-22.

Point 1:

Transition metals, such as iron and copper, when circulating in excess can generate free radicals.
This does not mean that supplemental iron and copper cause disease.

Point 2:

If 1-2 milligrams of copper in a multi-vitamin actually caused Alzheimer's or dementia in normal individuals, would not 8 mg of supplemental copper in patients in the processes of developing Alzheimer's likely lead to A GREATER progression toward Alzheimer's?

In fact, patients in the early stages of Alzheimer's were supplemented with placebo or 8 mg of copper daily for 12 months. The outcome was that the rate of progression was identical between groups and there were no additional toxic side effects identified in the 8 mg per day copper-supplemented group.

Kessler H, Bayer TA, Bach D et al. Intake of copper has no effect on cognition in patients with mild Alzheimer's disease: a pilot phase 2 clinical trial. J Neural Transm. 2008;115:1181-87.

Point 3:

Without relation to supplements or diet, why may circulating copper be elevated in Alzheimer's disease, type 2 diabetes, lymphocytic leukemia, inflammation, atherosclerosis, hypertension in the absence of diabetes, vascular disease, malignancy and inflammation? (1-3).
The answer is because copper, copper-containing ceruloplasmin, ferritin, C-reactive protein, and serum amyloid A all function as acute phase reactants and are elevated during acute and chronic inflammatory states. Normally they quickly elevate during an acute inflammatory state and reduce just as quickly as the situation resolves. The problem with the conditions mentioned above is that they are conditions wherein the acute phase response has become chronic.
In other words, if copper is found to be elevated in Alzheimer's or type 2 diabetes, it is because copper is part of the chronic inflammatory state of these conditions and has nothing to do with diet or supplementation.

1. Brewer GJ. Risks of copper and iron toxicity during aging in humans. Chem Res Toxicol. 2010;23:319-26.

2. Walter RM, Uriu-Hare JY, Olin KL et al. Copper, zinc, manganese, and magnesium status and complications of diabetes mellitus. Diabetes Care. 1991;14:1050-56.
3. Murphy P, Wadiwala I, Sharland DE, Rai GS. Copper and zinc levels in "healthy" and "sick" elderly. J Am Geriatr Soc. 1985;33(12):847-9.

Point 4:

Correlations have been identified between hyperglycemia and acute phase reactants, including copper. This further demonstrates that "copper" intake is not the problem. The issue that augments endogenous copper levels is a chronic pro-inflammatory state.

- Hyperglycemia, as measured by hemoglobin A1c (HbA1c), is correlated to hs-CRP levels (1).
- Emergent type 2 diabetes correlates with rising levels of hs-CRP (2).
- As glycemic control worsens, serum copper levels may rise accordingly (3).

1. Meshram A, Agrawal U, Dhok A, Adole P, Meshram K, Khare R. HbA1c, hs-CRP and anthropometric parameters evaluation in the patients of Diabetes Mellitus of Central Rural India. Int J Med Sci Public Health. 2013; 2(2): 293-296.
2. Tabak AG, Kivimaki M, Brunner EJ. Changes in C-reactive protein levels before type 2 diabetes and cardiovascular death: the Whitehall II study. Eur J Endocrinol. 2010;163:89-95.
3. Viktorinova A, Toserova E, Krizko M, Durackova Z. Altered metabolism of copper, zinc, and magnesium is associated with increased levels of glycated hemoglobin in patients with diabetes mellitus. Metab Clin Exp. 2009;58:1477-82.

Point 5:

The fat content of the modern diet is characterized by elevated levels of trans fats, omega-6 fatty acids and saturated fatty acids, and a reduced level of omega-3 fatty acids; a pro-inflammatory pattern. A study in 2006 demonstrated that high dietary intake of copper in conjunction with a diet high in saturated and trans fats may be associated with accelerated cognitive decline. In contrast, intake of copper was not associated with cognitive change among persons whose diets were not high in these fats.

This study further demonstrates copper is not the problem. The pro-inflammatory diet is the problem.

Morris MC, Evans DA, Tangney CC et al. Dietary copper and high saturated and trans fat intakes associated with cognitive decline. Arch Neurol. 2006;63:1085-88.

Point 6:

The goal of patient care from the perspective of nutrition is to avoid chronic inflammation and therefore suppress the inappropriate elevation of acute phase reactants, such as copper.

Several markers of inflammation can be monitored in clinical practice (1-3). Table 1 outlines the markers of the metabolic syndrome. Three of the five criteria must be present in order for a patient to be characterized as having the metabolic syndrome.

Table 1. Markers of the metobolic syndrome

Markers		Dates	Dates	Dates	Dates
Metobolic Syndroms	Abnormal value				
1. Fasting blood glucose	≥ 100 mg/dL				
2. Triglycerides	≥ 150 mg/dL				
3. HDL cholesterol	< 50 for women; < men				
4. Blood Pressure	≥ 130/85				
5. Waist circumfrance	> 35" women > 40" men				

While the metabolic syndrome is considered to be a pro-inflammatory state, there are additional direct and surrogate markers of chronic inflammation that are readily available for use in clinical practice. Making these measurements a routine part of patient care allows the practitioner to identify which patients need lifestyle intervention.

Table 2. Laboratory and anthropometric markers for chronic inflammation

Pro-Inflammatory Markers		Dates	Dates	Dates	Dates
Pro-inflammatory markers	Parameters				
2-hour postprandial glucose	<140 mg/dl = normal 140-199 = prediabetes 200+ = diabetes				
Fasting triglycerides	<1.0= normal 1.0-3.0= moderate >3.0 = high				
hsCRP in mg/L (marker of chronic inflammation)	<1.0= normal 1.0-3.0= moderate >3.0 = high				
25(OH)D (vitamin D)	32-100 ng/ml (goal >40 ng)				

Table 2. Continued

Pro-Inflammatory Markers		Dates	Dates	Dates	Dates
Pro-inflammatory markers	Parameters				
Body mass index (BMI)	18.5-24.9 = normal 25-29.9 = overweight ≥30 = obese				
Waist/hip ratio women (risk factor for diabetes)	<0.80 = low risk 0.81-.85 = moderate risk >0.85 = high risk				
Waist/hip ratio men (risk factor for diabetes)	<0.95 = low risk 0.96-1.0= moderate risk >1.0 = high risk				

Table 3 contains lifestyle and psychological markers for inflammation, which are typically not viewed as markers of systemic inflammation. To reduce the markers in Tables 1-3, diet, exercise, and other lifestyle factors are required. Several supplements are also recommended.

Table 3. Lifestyle and psychological markers for inflammation

Pro-Inflammatory Markers		Dates	Dates	Dates	Dates
Pro-inflammatory markers	Parameters				
Lack of sleep	Less than 6 hrs				
Sedentary living	Associated with systemic inflammation				
Stress	Associated with systemic inflammation				
Depression	Associated with systemic inflammation				
Poor self-rated health (use HSQ-12)	Associated with systemic inflammation				
If NSAIDs relieve pain	Suggests need for dietary balance of omega-6:omega-3 fatty acids)				

1. Seaman DR. Body mass index and musculoskeletal pain: is there a connection? Chiropractic Man Ther. 2013;21:15. http://www.chiromt.com/content/21/1/15
2. Seaman DR. Anti-inflammatory diet for pain patients. Pract Pain Management. 2012;12(10)36-46. http://www.practicalpainmanagement.com/treatments/complementary/anti-inflammatory-diet-pain-patients
3. Seaman DR. When chronic inflammation prevents success with manual care. Dynamic Chiro. August 1, 2013. Vol 31, Issue 15. http://www.dynamicchiropractic.com/mpacms/dc/article.php?id=56628

Point 7:

Several of the markers in Table 1 and 2, as well as non-alcoholic fatty liver, have been normalized within 12 weeks by adhering to what the researchers called the ketogenic Spanish Mediterranean diet. The diet included the following calorie sources (1-3):

- olive oil
- moderate red wine
- green vegetables and salads
- fish as the primary protein
- lean meat
- fowl
- eggs
- shellfish
- cheese

Experimental evidence indicates that a ketogenic diet can also reduce pain and inflammation (4,5).

The Nutritional Foundation booklet from Anabolic Laboratories is consistent with the ketogenic Spanish Mediterranean diet and can be used to educate patients to avoid the chronic inflammatory state. Dietary and supplement recommendations are clearly outlined and consistent with the available evidence (6). The key foods to avoid are refined sugar, flour, refined omega-6 oils, and trans fats.

Supplementation should be viewed from two perspectives. Those include a general health supplementation approach and additional supplements for more specific goals.

General health program

- Multivitamin/mineral
- Magnesium
- Vitamin D
- Omega-3 fatty acids from fish oil
- Probiotics

Pain and inflammation

- Ginger/turmeric etc.
- Proteolytic enzymes
- White willow

Cellular energy

- Coenzyme Q10
- Lipoic acid
- Acetyl-L-carnitine

Glycemic support

- Lipoic acid
- Chromium

Digestive support

- Digestive enzymes
- Probiotics
- Fiber
- Glutamine

Joint and bone support

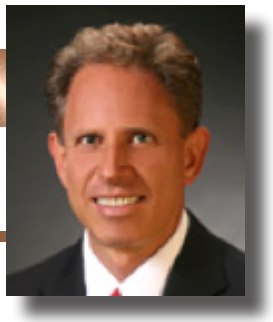
- Glucosamine/chondroitin
- Calcium
- Hydroxyapatite

Specific botanicals

- Ginseng
- Saw palmetto
- Milk thistle
- Valerian root

1. Pérez-Guisado J et al. Spanish Ketogenic Mediterranean Diet: a healthy cardiovascular diet for weight loss. Nutr J. 2008;7:30.
2. Pérez-Guisado J. A pilot study of the Spanish Ketogenic Mediterranean Diet: an effective therapy for the metabolic syndrome. J Med Food. 2011;14(7-8):681-7.
3. Pérez-Guisado J, Muñoz-Serrano A. The effect of the Spanish Ketogenic Mediterranean Diet on nonalcoholic fatty liver disease: a pilot study. J Med Food. 2011;14(7-8):677-80.
4. Ruskin DN, Kawamura M, Masino SA. Reduced pain and inflammation in juvenile and adult rats fed a ketogenic diet. PLoS One. 2009;4(12):e8349.

5. Ruskin DN, Masino SA. The nervous system and metabolic dysregulation: emerging evidence converges on ketogenic diet therapy. Front Neurosci. 2012;6:33. \
6. Seaman DR. Anti-inflammatory diet for pain patients. Pract Pain Management. 2012;12(10)36-46. http://www.practicalpainmanagement.com/treatments/complementary/anti-inflammatory-diet-pain-patients



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Prior to his graduation in 1986 from New York Chiropractic College, Dr. David Seaman received his B.S. in biology from Rutgers University. He earned his M.S. in nutrition from the University of Bridgeport in 1991 and completed his postdoctoral studies in neurology at Logan College of Chiropractic the next year. He also a diplomate of the American Chiropractic Academy of Neurology and the American Clinical Board of Nutrition.

A popular and prolific author of numerous nutrition and neurology articles and of the text Clinical Nutrition for Pain, Inflammation, and Tissue Healing, Dr. Seaman is a Professor of Clinical Sciences at National University of Health Sciences in St. Petersburg, Fla., and is on the postgraduate faculty of several chiropractic colleges.

A master's degree in nutrition and neurology, combined with his chiropractic background, gives Dr. Seaman a diverse perspective from which to observe the affects of diet and nutrition on neurological processes with regards to acute-to-chronic pain.

Dr. Seaman has also provided a wealth of articles for chiropractic and sports-related publications.