Nutrition and Mental Illness
Sampling of the Current Scientific Literature – Part 1

by Gary Null, Ph.D. and Martin Feldman, M.D.

For the past 40 years the mainstream medical establishment has denied the connection between nutrition and physical disease states. The mental health establishment has followed suit, with orthodox psychiatry and psychology contending that nutrition has no bearing on mental conditions. Yet the reality is that an objective review of mainstream scientific literature reveals the following: For virtually every major mental illness, quality studies exist showing that there are significant nutritional influences. So there is a kind of schizophrenia on the part of most mental health practitioners in that their reality is at odds with what has actually been going on in the real world of research.

The sad fact is that just because something important has been discovered and the results replicated, a paradigm shift will not necessarily occur. Where long-held beliefs conflict with new findings, it is usually the case that the beliefs continue to hold sway for a considerable time. In fact, it is often the belief in a therapy, rather than its true safety and efficacy, that supports its continued use. As a result, we see the phenomenon of old therapies – even antiquated and iatrogenic ones – dying a protracted death; these outdated treatments are chronically terminal but never seem to completely expire, as they linger on in that limbo of being supported, not by patient cures, but by medical megalomania and conceit.

To counter this state of affairs, and to set the record straight for those who would denigrate nutritional and orthomolecular approaches to mental health, we have compiled a sampling of scientific references that support these newer approaches. All of the following references are from peer-reviewed journals. They are based on sound science, not on tradition. Hopefully, as those in the mental health field become more aware of what has been happening in the research arena, the results herein will be widely acknowledged and applied.

Aggression

• A neuropsychopharmacological profile of "Cinkara," a polyherbal preparation. Sakina MR; Khan EA; Hamdard ME; Dandiya PC. *Indian Journal of Physiology and Pharmacology*, 1989 Jan-Mar, 33(1):43-6.

In rats, the herbal preparation known as Cinkara appears to stimulate the central nervous system, but, unlike other such stimulants, it lowers aggressive behavior.

• Acute and chronic effects of ginseng saponins on maternal aggression in mice. Yoshimura H; Watanabe K; Ogawa N. *European Journal of Pharmacology*, 1988 Jun 10, 150(3):319-24.

Ginseng root contains an ingredient that suppresses maternal aggression in mice, without impairing their movement abilities.

• Anxiolytic activity of Panax ginseng roots: an experimental study. Bhattacharya SK; Mitra SK. *Journal of Ethnopharmacology*, 1991 Aug, 34(1):87-92.

Ginseng root was shown to be effective in reducing anxiety and aggression in rats and mice, when given over a period of 5 days (as opposed to single-dose administration, which had little effect). Ginseng's effectiveness was comparable to that of diazepam (Valium).

Cerebrospinal fluid magnesium and calcium related to amine metabolites, diagnosis, and suicide attempts. Banki CM; Vojnik M; Papp Z; Balla KZ; Arato M. *Biological Psychiatry*, 1985 Feb, 20(2):163-71.

Suicidal female psychiatric patients suffering from depression, schizophrenia, or adjustment disorder had decreased levels of magnesium in their cerebrospinal fluid.

• Lithium in scalp hair of adults, students, and violent criminals. Effects of supplementation and evidence for interactions of lithium with vitamin B12 and with other trace elements. Schrauzer GN; Shrestha KP; Flores-Arce MF. *Biological Trace Element Research*, 1992 Aug, 34(2):161-76.

Lithium levels in human hair are low in certain pathological conditions, such as heart disease, and in learning disabled subjects and violent criminals. Hair levels of lithium rise with extradietary supplementation, and it is suggested that lithium may help distribute vitamin B12 in the body. Lithium also interacts with other trace elements.

• Magnesium alters the potency of cocaine and haloperidol on mouse aggression. Kantak KM. *Psychopharmacology*, 1989, 99(2):181-8.

Magnesium given to mice was shown to increase the potency of a single dose of cocaine, and a magnesium-deficient diet reduced its potency. With chronic cocaine use, however, magnesium countered cocaine's effects.

• Psychotropic effects of ginseng saponins on agonistic behavior between resident and intruder mice. Yoshimura H; Watanabe K; Ogawa N. *European Journal of Pharmacology*, 1988 Feb 9, 146(2-3):291-7.

Crude ginseng saponins and pure ginsenocide given to mice reduce aggressive behavior in certain situations.

• Stimulant-like effects of magnesium on aggression in mice. Izenwasser SE; Garcia-Valdez K; Kantak KM. *Pharmacology, Biochemistry and Behavior*, 1986 Dec, 25(6):1195-9.

Low levels of magnesium in mice are linked to reduced aggression, heightened levels to increased aggression, and extremely high levels to reduced aggression. Since magnesium works with the neurotransmitters dopamine, norepinephrine, and serotonin, which affect aggressive behavior, the effects shown may be related to these systems.

Alcoholism

• A hypothetical mechanism for fetal alcohol syndrome involving ethanol inhibition of retinoic acid synthesis at the alcohol dehydrogenase step. Duester G. *Alcoholism, Clinical and Experimental Research*, 1991 Jun, 15(3):568-72.

A mechanism is offered to explain how ethanol causes the bodily abnormalities of fetal alcohol syndrome. To develop normally, embryonic tissues require certain levels of retinoic acid – the active form of vitamin A – and ethanol inhibits the enzyme needed to create this essential molecule.

• ABC of Nutrition: Nutritional advice for other chronic diseases. Truswell, AS. *Brit Med J.* London: British Medical Association. July 20, 1985, v. 291, 197-200.

Nutritional guidelines are given for preventing various chronic diseases, including cirrhosis of the liver due to alcoholism.

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• Abnormalities of peripheral nerve conduction in relation to thiamine status in alcoholic patients. D'Amour ML; Bruneau J; Butterworth RF. *Canadian Journal of Neurological Sciences*, 1991 May, 18(2):126-8.

Alcoholic patients were shown to be severely thiamine-deficient, a condition that may contribute to the nervous-system abnormalities seen in alcoholics. (Other factors that may be involved in these abnormalities are deficiencies of other vitamins, as well as the direct effects of alcohol itself.)

• Age-related effects of chronic ethanol intake on vitamin A status in Fisher 344 rats. Mobarhan S; Seitz HK; Russell RM; Mehta R; Hupert J; Friedman H; Layden TJ; Meydani M; Langenberg P. *Journal of Nutrition,* 1991 Apr, 121(4):510-7.

In rats, chronic ethanol ingestion alters tissue distribution of vitamin A.

• Alcohol and bone disease. Rico H. *Alcohol and Alcoholism*, 1990, 25(4):345-52.

Excessive alcohol consumption leads to decreased bone formation, defective mineralization, and osteoporosis, the latter due possibly to excessive zinc excretion induced by alcohol.

• Alcohol, liver, and nutrition. Lieber CS. *Journal of the American College of Nutrition*, 1991 Dec, 10(6):602-32.

Liver disease in alcoholics used to be attributed mainly to dietary deficiencies, but now more is understood about how alcohol affects the liver directly. It's been shown, for instance, that animals given ethanol, along with vitamin-A-rich diets, had low levels of the vitamin in their livers, and this was especially so when the ethanol was combined with other drugs, mimicking a common circumstance in humans. When supplementing patients with vitamin A, however, it is essential to understand that too much of the vitamin is toxic to the liver – and that this is particularly so in alcoholics – so that the amount given is crucial. This decreased "therapeutic window" for alcoholics taking vitamin A applies to other nutritional supplements as well.

• Alcohol-induced bone marrow damage: status before and after a 4-week period of abstinence from alcohol with or without disulfiram. A randomized bone marrow study in alcohol-dependent individuals. Casagrande G; Michot F. *Blut*, 1989 Sep, 59(3):231-6.

Alcohol can induce bone marrow damage, which has been shown to be reversed in patients who totally abstain. However, patients who detoxified while taking the drug disulfiram (Antabuse) continued to have bone marrow pathology.

• Alcoholism in the elderly. How to spot and treat a problem the patient wants to hide. Tobias CR: Lippmann S; Pary R; Oropilla T; Embry CK. *Postgraduate Medicine*, 1989 Sep 15, 86(4):67-70, 75-9.

Increased awareness of alcoholism by physicians, with early diagnosis and treatment, can reduce its damaging effects. Especially in the elderly, all medications used should be monitored, and nonessential ones should be discontinued. Also suggested are treating withdrawal symptoms with thiamine, multivitamins, and perhaps sedatives; treating any underlying psychiatric disorder; psychosocial support; and possibly the use of disulfiram (Antabuse).

• Anemia in alcoholics. Savage D; Lindenbaum J. *Medicine*, 1986 Sep, 65(5):322-38.

A deficiency of folic acid in alcoholics is a factor in anemia in these patients. A diagnostic approach to anemia in alcoholics was developed, as were suggestions for therapy.

• Ascorbic acid chronic alcohol consumption in the guinea pig. Susick RL Jr; Abrams GD; Zurawski CA; Zannoni VG. *Toxicology and Applied Pharmacology,* 1986 Jun 30, 84(2):329-35.

Protection against the toxic effects of chronic alcohol consumption was observed in guinea pigs maintained on a high-ascorbic-acid diet, as opposed to those on a low-ascorbic-acid diet.

• Assessment of nutritional status and in vivo immune responses in a disease. Mills PR; Shenkin A; Anthony, RS; McLelland, AS; Alistair NH; MacSween RNM; Russell RI. *Am. J. Clin. Nutr.*, Bethesda, MD.: American Society for Clinical Nutrition 1983. v.38(6)p.849-859.

High alcohol intake resulted in metabolic and cellular changes, including the depletion of potassium, magnesium, and phosphate in the blood.

• Blood thiamine and thiamine phosphate concentrations in excessive drinkers with or without peripheral neuropathy. Poupon RE; Gervaise G; Riant P; Houin G; Tillement JP. *Alcohol and Alcoholism*, 1990, 25(6):605-11.

Thiamine phosphate (but not free thiamine) was found to be at low levels in groups of excessive drinkers with and without peripheral nerve damage.

• Bone and mineral metabolism and chronic alcohol abuse. Lalor BC; France MW; Powell D; Adams PH; Counihan TB. *Quarterly Journal of Medicine*, 1986 May, 59(229):497-511.

Significant changes in bone structure and mass appear to be common among heavy drinkers. In a group of alcoholic patients with varying degrees of liver damage, but with no clinical evidence of metabolic bone disease, osteoporosis and osteomalacia were found, and related to various factors, including magnesium deficiency, low blood levels of calcitriol, the state of liver function, and the type of alcohol consumed.

• Calcium status and calcium-regulating hormones in alcoholics. Bjorneboe GE; Bjorneboe A. Johnsen J; Skylv N; Oftebro H; Gautvik KM; Hoiseth A; Morland J; Drevon CA. *Alcoholism, Clinical and Experimental Research*, 1988 Apr, 12(2):229-32.

Vitamin D3 levels were shown to be lower in alcoholics than in a control group, during the winter season. Dietary intake of the vitamin did not differ significantly between the groups, and so it seems that the activities of enzymes crucial in vitamin D3 metabolism may be altered in alcoholics, resulting in low calcium levels.

• Carotenoids and liposoluble vitamins in liver cirrhosis. Rocchi E; Borghi A; Paolillo F; Pradelli M; Casalgrandi G. *Journal of Laboratory and Clinical Medicine,* 1991 Aug, 118(2):176-85.

The role of carotenoids, retinol, and tocopherol in quenching oxidative cellular damage and combatting tumor growth is well documented; this research looked at their activity in human liver cirrhosis. In patients with this disease, significantly reduced blood levels were found of alpha- and beta-carotene and several other vitamin factors. Improved diet for patients with liver cirrhosis is discussed.

• Changes in the activation of red blood cell transketolase of alcoholic patients during treatment. Jeyasingham MD; Pratt OE; Shaw GK; Thomson AD. *Alcohol and Alcoholism*, 1987, 22(4):359-65.

An enzyme test can monitor the effectiveness of thiamin therapy used in alcohol detoxification.

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• Chronic administration of ethanol with high vitamin A supplementation in a liquid diet to rats does not cause liver fibrosis. 1. Morphological observations. Bosma A; Seifert WF; Wilson JH; Roholl PJ; Brouwer A; Knook DL. *Journal of Hepatology*, 1991 Sep, 13(2):240-8.

Rats fed a high-ethanol diet supplemented with vitamin A did not develop liver fibrosis, suggesting that the main effects of chronic ethanol consumption to the liver may be secondary to interference with host resistance to infections.

• Chronic administration of ethanol with high vitamin A supplementation in a liquid diet to rats does not cause liver fibrosis. 2. Biochemical observations. Seifert WF; Bosma A; Hendriks HF; Blaner WS; van Leeuwen RE; van Thiel-de Ruiter GC; Wilson JH; Knook DL; Brouwer A. *Journal of Hepatology*, 1991 Sep, 13(2):249-55.

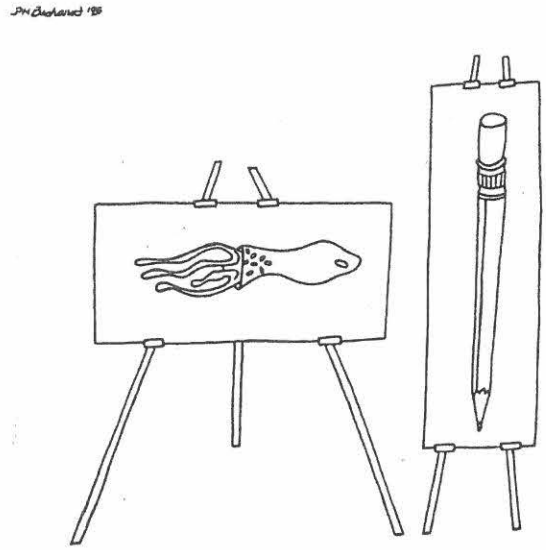
The inability of a high-alcohol, high-vitamin-A diet to induce liver fibrosis in rats (see abstract above) was further evaluated. The hypothesis that interaction between alcohol and retinoids is a major factor in alcoholic liver disease needs to be reconsidered.

• Chronic alcohol treatment results in disturbed vitamin D metabolism and skeletal abnormalities in rats. Turner RT; Aloia RC; Segel LD; Hannon KS; Bell NH. *Alcoholism, Clinical and Experimental Research*, 1988 Feb, 12(1):159-62.

Rats on a high-alcohol diet, when compared to a control group, had low blood levels of magnesium and of substances metabolized from vitamin D.

• Chronic ethanol feeding and acute ethanol exposure in vitro: effect on intestinal transport of biotin. Said HM; Sharifian A; Bagherzadeh A; Mock D. *American Journal of Clinical Nutrition*, 1990 Dec, 52(6):1083-6.

Alcohol-fed rats showed lowered biotin levels in their blood, as well as lowered ability to absorb biotin from the intestine.



OBSERVE THE TWO PICTURES.
WHAT IS THE SAME ABOUT SQUID AND A PENCIL ERASER?
ANSWER: BOTH ARE RUBBERY TO THE TOOTH.

• Concentrations of zinc and copper in pregnant problem drinkers and infants. Halmesmaki E; Ylikorkala, O; Alfthan G. *Brit. Med. J. London* British Medical Association. Nov 23, 1985. v. 291, 1470-1471.

Reduced zinc levels were found in infants of mothers who were problem drinkers.

• Current progress toward the prevention of the Wernicke-Korsakoff syndrome. Bishai DM; Bozzetti LP. *Alcohol and Alcoholism*, 1986, 21(4):315-23.

Wernicke-Korsakoff syndrome, a neurological disorder seen mainly in alcoholics, may be prevented by supplementing alcoholic beverages with thiamin. Also relevant to the disease are folate and magnesium levels.

• Decreased serum selenium in alcoholics as related to liver structure and function. Korpela H; Kumpulainen J; Luoma PV; Arranto AJ. *Am. J. Clin. Nutr.*, Bethesda, MD.: *American Society for Clinical Nutrition* 1985. v. 42(1):147-151.

A group of alcoholic patients showed low blood levels of selenium, with those patients having the most damaged livers showing the lowest levels. Inadequate dietary selenium intake, as well as alcohol-caused changes in liver structure and function, are probable factors.

• Depressed selenium and vitamin E levels in an alcoholic population. Possible relationship to hepatic injury through increased lipid peroxidation. Tanner AR; Bantock I; Hinks L; Lloyd B; Turner NR; Wright R. *Digestive Diseases and Sciences*, 1986 Dec, 31(12):1307-12.

Blood levels of both selenium and vitamin E were shown to be significantly depressed in alcoholics, with selenium more markedly depressed in those with established liver disease. Depressed selenium correlated closely with poor nutritional status, and liver disease activity was more markedly abnormal in subjects with combined vitamin E and selenium deficiency.

• Diminished serum concentration of vitamin E in alcoholics. Bjorneboe GE; Johnsen J; Bjorneboe A; Bache-Wiig JE; Morland J; Drevon CA. *Annals of Nutrition and Metabolism,* 1988, 32(2):56-61.

A group of alcoholic subjects showed low blood levels of vitamin E when compared with a control group, and it was reported as well that their estimated dietary intake of this vitamin was significantly lower than that of the controls. Selenium was also lower in the alcoholics, and the reduced levels of these substances may affect cell structure and function, and contribute to development of diseases frequently observed in alcoholics.

• Discovery and importance of zinc in human nutrition. Prasad AS. *Fed. Proc. Fed. Am. Soc. Exp. Biol.*, Bethesda, MD.: The Federation. Oct 1984. (13):2829-2834.

Zinc appears to be involved in many biological functions; its roles in enzymatic functions, cell membranes, and immunity have been well established. Cases of deficiency of this trace element can be traced to several causes, and alcoholism is a predisposing factor.

• Disorders of divalent ions and vitamin D metabolism in chronic alcoholism. Pitts TO; Van Thiel DH. *Recent Developments in Alcoholism,* 1986, 4:357-77.

Deficient vitamin D metabolism in alcoholics can result from liver problems, lack of sun exposure, poor diet, and malabsorption. Low vitamin D may contribute to calcium and phosphate deficiencies, and to osteoporosis. Alcoholics should be screened for vitamin D deficiency and given supplements if needed.

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• Effect of abstinence from alcohol on the depression of glutathione peroxidase activity and selenium and vitamin E levels in chronic alcoholic patients. Girre C; Hispard E; Therond P; Guedj S.; Bourdon R; Dally S. *Alcoholism, Clinical and Experimental Research*, 1990 Dec, 14(6):909-12.

Chronic alcoholics without severe liver disease were shown to have deficiencies in their antioxidant defense systems. Blood factors indicating this were seen to normalize during 14 days of alcohol abstinence.

• Effect of alcohol consumption on serum concentration of 25-hydroxyvitamin D3, retinol, and retinol-binding protein. Bjorneboe GE; Johnsen J; Bjorneboe A; Rousseau B; Pederson JI; Norum KR; Morland J; Drevon CA. *American Journal of Clinical Nutrition*, 1986 Nov, 44(5):678-82.

Chronic alcohol consumers had significantly lower levels of vitamin D in their blood than did a control group, even though the two groups seemed to have similar dietary intake of the nutrient. The alcoholics also had lower calcium levels.

• Effect of chronic consumption of ethanol and vitamin E on fatty acid composition and lipid peroxidation in rat heart tissue. Pirozhkov SV; Eskelson CD; Watson RR; Hunter GC; Piotrowski JJ; Bernhard V. *Alcohol*, 1992 Jul-Aug, 9(4):329-34.

Rats were given large amounts of ethanol and vitamin E, and the latter was shown to have a stabilizing effect on phospholipids in the heart, by preventing their deterioration.

• Effect of chronic ethanol administration on thiamine transport in microvillous vesicles of rat small intestine. Gastaldi G; Casirola D; Ferrari G; Rindi G. *Alcohol and Alcoholism*, 1989, 24(2):83-9.

Intestinal absorption of thiamine was markedly lower in rats that had been administered ethanol over a period of time than in nonalcoholic rats.

• Effect of free radical scavengers on superoxide dismutase (SOD) enzyme in patients with alcoholic cirrhosis. Feher J; Lang I; Nekam K; Muzes G; Deak G. *Acta Medica Hungarica*, 1988, 45(3-4):265-76.

Silymarin and other antioxidants have an effect protective of the liver in alcoholics.

• Effect of heavy alcohol consumption on serum concentrations of fat-soluble vitamins and selenium. Bjorneboe GA; Johnsen J; Bjorneboe A; Morland J; Drevon CA. *Alcohol and Alcoholism*, 1987, Suppl 1:533-7.

A group of alcoholics showed blood levels of vitamin E and selenium that were significantly lower than those of a control group, and it is noted that these antioxidants protect against cell damage. Also lower in the alcoholics was vitamin D; this may be a factor – through disturbance of calcium and phosphate metabolism – in the high frequency of bone fractures and osteomalacia in alcoholics.

• Effect of silibinin on the activity and expression of superoxide dismutase in lymphocytes from patients with chronic alcoholic liver disease. Feher J; Lang I; Nekam K; Csomos G; Muzes G; Deak G. *Free Radical Res.Communications*, 1987, 3(6):373-7.

Silibinin acts to protect the liver, possibly through antioxidant activity.

• Effects of acute ethanol on urinary excretion of 5-methyltetrahydrofolic acid and folate derivatives in the rat. Eisenga BH; Collins TD; McMartin KE. *Journal of Nutrition*, 1989 Oct, 119(10):1498-505.

Ethanol-treated rats were shown to excrete more folic acid in their urine than did a control group. This effect has been implicated in the deficiency of this vitamin often seen in alcoholics.

• Ethanol and fetal nutrition: effect of chronic ethanol exposure on rat placental growth and membrane-associated folic acid receptor binding activity. Fisher SE; Inselman LS; Duffy L; Atkinson M; Spencer H; Chang B. *Journal of Pediatric Gastroenterology and Nutrition*, 1985 Aug, 4(4):645-9.

Rat fetuses whose mothers were fed alcohol were smaller than those of control-group mothers, and their placentas were less able to process folic acid.

• Folate absorption in alcoholic pigs: in vitro hydrolysis and transport at the intestinal brush border membrane. Naughton CA; Chandler CJ; Duplantier RB; Halsted CH. *American Journal of Clinical Nutrition*, 1989 Dec, 50(6):1436-41.

An enzymatic process required for intestinal absorption of folic acid was seen, in the miniature pig, to be impeded by chronic consumption of alcohol.

• Food and nutrient intake of alcoholic laborers. Chhabra KB; Ramesh P; Mehta U. *Ecol. Food Nutr.* London: Gordon & Breach Science Publishers. 1991. v.2, 51-57.

Fifty subjects – 30 alcoholics and 20 nonalcoholics – were selected from an industrial area of Ludhiana City, Punjab, India, and their dietary intake was assessed. Although both groups consumed about the same number of calories, the nutrient intake of the alcoholics was lower, resulting in deficiencies.

• Hypothesis: prenatal ethanol-induced birth defects and retinoic acid. Pullarkat RK. *Alcoholism, Clinical and Experimental Research*, 1991 Jun, 15(3):565-7.

Prenatal exposure to alcohol causes birth defects in humans and animals, specifically, central nervous system and limb abnormalities. It is hypothesized that this comes about as a result of ethanol's inhibitory effect of the formation of retinoic acid from retinol. Retinoic acid is important in the development of the central nervous system, and of limbs.

• Inhibitory effect of maternal alcohol ingestion on rat pup hepatic 25-hydroxyvitamin D production. Milne M; Baran DT. *Pediatric Research*, 1985 Jan, 19(1):102-4.

Eighteen days of alcohol consumption had no effect on liver synthesis of vitamin D in pregnant rats, but did inhibit fetal production of the vitamin.

• Interaction of alcohol with other drugs and nutrients. Implication for the therapy of alcoholic liver disease. Lieber CS. *Drugs*, 1990, 40 Suppl 3:23-44.

New understanding of how alcohol damages the liver has led to more successful therapy with drugs and nutritional factors, such as vitamin A. Vitamin A is depleted in the alcoholic, but excess vitamin A is extra-toxic in the alcoholic.

• Interaction of niacin and zinc metabolism in patients with alcoholic pellagra. Vannucchi H; Moreno FS. *American Journal of Clinical Nutrition*, 1989 Aug, 50(2):364-9.

In patients with alcoholic pellagra, zinc interacts with niacin metabolism, through a probable mediation by vitamin B-6.

• Intestinal absorption, liver uptake, and excretion of 3H-folic acid in folic acid-deficient, alcohol-consuming nonhuman primates. Blocker DE; Thenen SW. *American Journal of Clinical Nutrition*, 1987 Sep, 46(3):503-10.

Chronic alcohol ingestion in nonhuman primates impaired folic acid utilization.

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• Iron uptake from transferrin and asialotransferrin by hepatocytes from chronically alcohol-fed rats. Potter BJ; McHugh TA; Beloqui O. *Alcoholism, Clinical and Experimental Research*, 1992 Aug, 16(4):810-5.

Alcohol-fed rats showed impaired ability to use iron.

• Lipoprotein cholesterol, vitamin A, and vitamin E in an alcoholic population. D'Antonio JA; LaPorte RE; Dai WS; Hom DL; Wozniczak M; Kuller LH. *Cancer*, 1986 May 1, 57(9):1798-802.

Elevated alcohol consumption is associated with increased cancer risk, due possibly to altered vitamin A, vitamin E, and cholesterol metabolism in alcoholics.

• Liver cell protection in toxic liver lesion. Feher J; Cornides A; Pal J; Lang I; Csomos G. *Acta Physiologica Hungarica* 1989, 73(2-3):285-91.

In animal experiments, silymarin, silibinin, and Aica-P were shown to have liver-protecting effects related to their actions as free-radical scavengers.

• Metabolism of vitamin D in patients with primary biliary cirrhosis and alcoholic liver disease. Mawer EB; Klass HJ; Warnes TW; Berry JL. *Clinical Science*, 1985 Nov, 69(5):561-70.

Alcoholism may lead to impairment of the liver's function in processing vitamin D.

• Nutrition and alcoholic encephalopathies. Thomson AD; Jeyasingham MD; Pratt OE; Shaw GK. *Acta Medica Scandinavica. Supplementum*, 1987, 717:55-65.

Chronic alcoholism may cause vitamin B deficiencies due to impaired uptake of thiamin as well as disruption of thiamin metabolism. This may subsequently cause brain damage.

• Plasma amino acid patterns in alcoholic pellagra patients. Vannucchi H; Moreno FS; Amarante AR; de Oliveira JE; Marchini JS. *Alcohol and Alcoholism*, 1991, 26(4):431-6.

"Well, what did you expect to find?"

Alcoholics with pellagra (a disease resulting from lack of B complex vitamins) showed lowered levels for 11 amino acids in the blood.

• Plasma osteocalcin levels in liver cirrhosis. Capra F; Casaril M; Gabrielli GB; Stanzial A; Ferrari S; Gandini G; Falezza G; Corrocher R. *Italian Journal of Gastroenterology*, 1991 Mar-Apr, 23(3):124-7.

Cirrhosis of the liver results in lowered levels of osteocalcin, and therefore a lowered ability to replace bone. The low osteocalcin levels may be due to low vitamin D and blood calcium levels.

• Prenatal ethanol exposure decreases hippocampal mossy fiber zinc in 45-day-old rats. Savage DD; Montano CY; Paxton LL; Kasarskis EJ. *Alcoholism, Clinical and Experimental Research*, 1989 Aug, 13(4):588-93.

In rats, a brain region important in the process of memory consolidation is affected by prenatal exposure to alcohol. Pregnant rats on an alcohol diet had offspring with lower than normal zinc levels in the hippocampal formation.

• Randomized controlled trial of silymarin treatment in patients with cirrhosis of the liver. Ferenci P; Dragosics B; Dittrich H; Frank H; Benda L; Lochs H; Meryn S; Base W; Schneider B. *Journal of Hepatology*, 1989 Jul, 9(1):105-13.

Silymarin, the active principle of the milk thistle, Silybum marianum, protects experimental animals against various substances toxic to the liver. In a double-blind study of human patients with cirrhosis, silymarin was shown to have an effect protective of the liver.

• Reduced concentration of hepatic alpha-tocopherol in patients with alcoholic liver cirrhosis. Bell H; Bjorneboe A; Eidsvoll B; Norum KR; Raknerud N; Try K; Thomassen Y; Drevon CA. *Alcohol and Alcoholism*, 1992 Jan, 27(1):39-46.

The vitamin E content in the liver was significantly lower in patients with alcoholic cirrhosis compared with patients with normal livers.

• Role of acetyl-L-carnitine in the treatment of cognitive deficit in chronic alcoholism. Tempesta E; Troncon R; Janiri L; Colusso L; Riscica P; Saraceni G; Gesmundo E; Calvani M; Benedetti N; Pola P. *International Journal of Clinical Pharmacology Research*, 1990, 10(1-2):101-7.

Acetyl-L-carnitine can be a useful and safe therapeutic agent in ameliorating the cognitive disturbances of chronic alcoholics. Fifty-five one-month-abstinent alcoholics were put in a double-blind placebo-controlled study to assess the effects of the substance, which did help the group that took it perform better or regain performance abilities faster than those who did not. Memory, logic, and constructional abilities were among those improved.

• Selenium status in patients with liver cirrhosis and alcoholism. Johansson U; Johnsson F; Joelsson B; Berglund M; Akesson B. *British Journal of Nutrition*, 1986 Mar, 55(2):227-33.

Blood levels of selenium and vitamins A and E were shown to be reduced in patients with alcoholic cirrhosis.

• Some aspects of antioxidant status in blood from alcoholics. Bjorneboe GE; Johnsen J; Bjorneboe A; Marklund SL; Skylv N; Hoiseth A; Bache-Wiig JE; Morland J; Drevon CA. *Alcoholism, Clinical and Experimental Research*, 1988 Dec, 12(6):806-10.

Blood levels of vitamin E were 30% lower in a group of alcoholics compared to a control group of nonalcoholics. After this measurement was taken, half of the alcoholics in the study received vitamin E supplementation, as did half of the nonalcoholics; the other halves of each group were supplemented

with placebo capsules. Of the four groups, only the alcoholics receiving the vitamin E supplements showed increased blood levels of the vitamin, showing that reduced levels of vitamin E can be normalized by supplementation.

• The Wernicke-Korsakoff syndrome in Queensland, Australia: antecedents and prevention. Price J. *Alcohol and Alcoholism*, 1985, 20(2):233-42.

Wernicke-Korsakoff syndrome may be the end result of thiamine deficiency in alcoholics. To prevent the syndrome, fortification of alcoholic beverages with thiamine has been proposed in Queensland, Australia, and the publicity this suggestion has generated has alerted some heavy drinkers to the need for supplementary B vitamins.

• The antioxidant status of patients with either alcohol-induced liver damage or myopathy. Ward RJ; Peters TJ. *Alcohol and Alcoholism*, 1992 Jul, 27(4):359-65.

Alcoholics showed low blood levels of beta-carotene, zinc, and selenium, and in patients with alcoholic cirrhosis, alpha-tocopherol levels were also low.

• The clinical spectrum of alcoholic pellagra encephalopathy. A retrospective analysis of 22 cases studied pathologically. Serdaru M; Hausser-Hauw C; Laplane D; Buge A; Castaigne P; Goulon M; Lhermitte F; Hauw JJ. *Brain*, 1988 Aug, 111 (Pt 4):829-42.

Alcoholic pellagra has often gone unrecognized, and therefore untreated with niacin. Multiple vitamin therapy should be given in the treatment of undiagnosed brain abnormalities in alcoholic patients

• The concentration of thiamin and thiamin phosphate esters in patients with alcoholic liver cirrhosis. Tallaksen CM; Bell H; Bohmer T. *Alcohol and Alcoholism*, 1992 Sep, 27(5):523-30.

Current alcohol misuse was shown to be associated with low thiamin concentrations in the blood.

• The effect of vitamin E (alpha-tocopherol) supplementation on hepatic levels of vitamin A and E in ethanol and cod liver oil fed rats. Odeleye OE; Eskelson CD; Alak JI; Watson RR; Chvapil M; Mufti SI; Earnest D. *International Journal for Vitamin and Nutrition Research*, 1991, 61(2):143-8.

Ethanol consumption in rats resulted in decreased levels of vitamins A and E in their livers, but supplementation with vitamin E restored levels of this vitamin to normal, and restored levels of vitamin A somewhat. Rats consuming cod liver oil along with ethanol also had lowered vitamin A and E levels, although the levels were higher than those of the rats not receiving cod liver oil.

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• Thiamin deficiency and prevention of the Wernicke-Korsakoff syndrome. A major public health problem. Yellowlees PM. *Medical Journal of Australia*, 1986 Sep 1, 145(5):216-9.

In order to prevent Wernicke-Korsakoff syndrome in Australia, it is recommended that flour and bread, as well as alcoholic beverages, be fortified with thiamin.

• Thiamin status and biochemical indices of malnutrition and alcoholism in settled communities of !Kung San. van der Westhuyzen J; Davis RE; Icke GC; Jenkins T. *Journal of Tropical Medicine and Hygiene*, 1987 Dec, 90(6):283-9. ➤

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Settled groups of !Kung San in the northern Kalahari Desert of Namibia show a high prevalence of thiamin deficiency, and alcohol abuse seems to be the main factor.

• Tissue thiamin levels of hospitalized alcoholics before and after oral or parenteral vitamins. Baines M; Bligh JG; Madden JS. *Alcohol and Alcoholism,* 1988, 23(1):49-52.

Oral supplementation of thiamin is effective for most alcoholics.

• Trace element and vitamin deficiency in alcoholic and control subjects. Cook CC; Walden RJ; Graham BR; Gillham C, Davies S; Prichard BN. *Alcohol and Alcoholism*, 1991, 26(5-6):541-8.

A wide range of trace elements and vitamins was studied in alcoholic patients admitted for detoxification and in healthy controls. The alcoholics were found to be deficient relative to the controls in magnesium and vitamin E, but there was also a surprising range of deficiencies in the control group, which points to the prevalence of undetected nutritional deficiency in the general population.

• Vitamin A status of alcoholics upon admission and after two weeks of hospitalization. Chapman KM; Prabhudesai M; Erdman JW Jr. *Journal of the American College of Nutrition*, 1993 Feb, 12(1):77-83.

Elevated bilirubin levels seen in alcoholics may indicate low vitamin A levels. Caution in levels of vitamin A therapy in these cases is advised, and consideration should instead be given to beta-carotene supplementation.

• Vitamin B-12 and folate function in chronic alcoholic men with peripheral neuropathy and encephalopathy. Gimsing P; Melgaard B; Andersen K; Vilstrup H; Hippe E. *Journal of Nutrition*, 1989 Mar, 119(3):416-24.

Folate deficiency may contribute to the development of nerve problems in alcoholics.

• Vitamin B6 status in cirrhotic patients in relation to apoenzyme of serum alanine aminotransferase. Ohgi N; Hirayama C. *Clinical Biochemistry,* 1988 Dec, 21(6):367-70.

Alcoholic cirrhotic patients have vitamin B6 deficiency.

• Vitamin K deficiency in chronic alcoholic males. Iber FL; Shamszad M; Miller PA; Jacob R. *Alcoholism, Clinical and Experimental Research*, 1986 Dec, 10(6):679-81.

Blood clotting defects are frequently present in alcoholics, suggesting vitamin K deficiency. Alcoholics given vitamin K did show more normal clotting protein in their blood than those not given the vitamin.

• Zinc and vitamin A status of alcoholics in a medical unit in Sri Lanka. Atukorala TM; Herath CA; Ramachandran S. *Alcohol and Alcoholism*, 1986, 21(3):269-75.

Alcoholics had lower blood levels of zinc and vitamin A than did controls, with female alcoholics having levels lower than those of males, although they drank less.

• Zinc nutrition in fetal alcohol syndrome. Keppen LD; Moore DJ; Cannon DJ. *Neurotoxicology*, 1990 Summer, 11(2):375-80.

Experiments with mice suggest that zinc intake should be optimized during pregnancy; the Recommended Daily Allowance should not be exceeded.

Alzheimer's Disease

• A histochemical study of iron, transferrin, and ferritin in Alzheimer's diseased brains. Connor JR; Menzies SL; St. Martin SM; Mufson EJ. *Journal of Neuroscience Research*, 1992 Jan, 31(1):75-83.

Iron, and iron-regulating proteins, are abnormally distributed in the brains of Alzheimer's disease patients.

• A natural and broad spectrum nootropic substance for treatment of SDAT – the Ginkgo biloba extract. Funfgeld EW. *Progress in Clinical and Biological Research*, 1989, 317:1247-60.

Ginkgo biloba extract was found to be therapeutic, and without side effects, in Parkinson's patients with additional signs of Alzheimer's-type dementia.

• A search for longitudinal variations in trace element levels in nails of Alzheimer's disease patients. Vance DE; Ehmann WD; Markesbery WR. *Biological Trace Element Research*, 1990 Jul-Dec, 26-27:461-70.

Progressive changes in trace-element levels occur in the nails of Alzheimer's disease patients, and imbalances are detected even in the earliest stages of the disease. Mercury levels were seen to decrease progressively with the level of the disease and with age, and potassium and zinc to increase with these same factors.

• Acetyl-L-carnitine: a drug able to slow the progress of Alzheimer's disease? Carta A; Calvani M. *Annals of the New York Academy of Sciences*, 1991, 640:228-32.

Clinical studies suggest that acetyl-L-carnitine, which has protective effects against aging processes and nerve degeneration, may slow the natural course of Alzheimer's disease.

• Changes in calcium homeostasis during aging and Alzheimer's disease. Peterson C; Ratan R; Shelanski M; Goldman J. *Annals of the New York Academy of Sciences*, 1989, 568:262-70.

Alzheimer's disease patients and normal aged patients had altered calcium regulation compared to that of young patients.

• Cultured cells as a screen for novel treatments of Alzheimer's disease. Malow BA; Baker AC; Blass JP. *Archives of Neurology*, 1989 Nov. 46(11):1201-3.

L-carnitine normalized two properties normally measured as abnormal in Alzheimer's diseased cells.

• Double-blind parallel design pilot study of acetyl levocarnitine in patients with Alzheimer's disease. Sano M; Bell K; Cote L; Dooneief G; Lawton A; Legler L; Marder K; Naini A; Stern Y; Mayeux R. *Archives of Neurology,* 1992 Nov, 49(11):1137-41.

Acetyl levocarnitine shows the ability to retard the deterioration in some cognitive areas in those suffering from Alzheimer's disease.

• Effects of free Ca2+ on the [Ca2++ Mg2+]-dependent adenosinetriphosphatase (ATPase) of Alzheimer and normal fibroblasts. Rizopoulos E; Chambers JP; Wayner MJ; Martinez AO; Armstrong LS.*Neurobiology of Aging*, 1989 Nov-Dec, 10(6):717-20.

Calcium regulation is different in Alzheimer's disease cells than in normal cells.

• Essential fatty acids in Alzheimer's disease. Corrigan FM; Van Rhijn A; Horrobin DF. *Annals of the New York Academy of Sciences*, 1991, 640:250-2.

Essential fatty acids are abnormal in Alzheimer's disease patients. Twenty-week treatment with essential fatty acids improved the levels.

• Folate, vitamin B12 and cognitive impairment in patients with Alzheimer's disease. Levitt AJ; Karlinsky H. *Acta Psychiatrica Scandinavica*, 1992 Oct, 86(4):301-5.

An inverse relationship was found between vitamin B12 levels and the severity of cognitive impairment in Alzheimer's disease patients.

• Hair aluminum in normal aged and senile dementia of Alzheimer type. Kobayashi S; Fujiwara S; Arimoto S; Koide H; Fukuda J; Shimode K; Yamaguchi S; Okada K; Tsunematsu T. *Progress in Clinical and Biological Research*, 1989, 317:1095-109.

In Alzheimer's disease, decreased calcium and magnesium levels enhance accumulation of aluminum in the brain. In normal aged individuals, cerebral blood flow levels decrease as hair aluminum levels increase, suggesting that aluminum may contribute to aging of the brain.

• Hypothesis regarding amyloid and zinc in the pathogenesis of Alzheimer disease: potential for preventive intervention. Constantinidis, J. *Alzheimer Disease and Associated Dis.*, 1991 Spring, 5(1):31-5.

It is suggested that amyloid production in the cerebral cortex causes a zinc deficiency in the brain; toxic metals (such as iron, aluminum, and mercury) then displace the zinc in some enzymes. Application of a zinc complex that crosses the blood-brain barrier may mitigate these effects.

• Lipid peroxidation and free radical scavengers in Alzheimer's disease. Jeandel C; Nicolas MB; Dubois F; Nabet-Belleville F; Penin F; Cuny G. *Gerontology*, 1989, 35(5-6):275-82.

The blood of a group of Alzheimer's patients, when compared with that of a group of healthy age-matched controls, showed lower levels of glutathione peroxidase activity in red blood cells, as well as lower levels of vitamins E, C, and A, and zinc.

• Long-term acetyl-L-carnitine treatment in Alzheimer's disease. Spagnoli A; Lucca U; Menasce G; Bandera L; Cizza G; Forloni G; Tettamanti M; Frattura L; Tiraboschi P; Comelli M. *Neurology*, 1991 Nov, 41(11):1726-32.

The effects of acetyl-L-carnitine on Alzheimer's patients were assessed in a double-blind, placebo-controlled study over one year. After this period, both the treated and placebo groups worsened, but the treated group showed a slower rate of deterioration in 13 of the 14 outcome measures, with statistically significant results in 5 of them. No significant side effects were seen.

• Low B12 levels related to high activity of platelet MAO in patients with dementia disorders. A retrospective study. Regland B; Gottfries CG; Oreland L; Svennerholm L. *Acta Psychiatrica Scandinavica*, 1988 Oct, 78(4):451-7.

Vitamin B12 levels were shown to be reduced in the blood of Alzheimer's patients and patients with confusional states.

• Magnesium depletion and pathogenesis of Alzheimer's disease. Durlach J. *Magnesium Research*, 1990 Sep, 3(3):217-8.

Magnesium depletion in a particular region of the brain, along with aluminum incorporation into the brain, is associated with Alzheimer's disease. Further research should seek to control the alterations of albumin, which may induce the magnesium depletion.

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• Nutrient intakes and energy expenditures of residents with senile Alzheimer's type. Litchford MD; Wakefield LM. *J. Am. Diet Assoc.*, Chicago, Ill.: The Association, Feb 1987. v. 87(2).

In a study conducted over three days, Alzheimer's patients were seen to exhibit lower nutrient intake than did a control group. Significant intake differences were noted for vitamin A, thiamin, niacin, riboflavin, and calcium, as well as for total calories and other factors.

• Oxidative damage in Alzheimer's dementia, and the potential etiopathogenic role of aluminosilicates, microglia and micronutrient interactions. Evans PH; Yano E; Klinowski J; Peterhans E. *Exs*, 1992, 62:178-89.

In laboratory experiments, aluminosilicate particles have stimulated the generation of tissue-damaging free radicals in nervous-system cells. Similar aluminosilicate deposits have been found in the brains of Alzheimer's patients, and it is suggested that antioxidant micronutrients and pharmacological agents would be useful in preventing and treating Alzheimer's disease.

• Plasma concentrations of vitamins A and E and carotenoids in Alzheimer's disease. Zaman Z; Roche S; Fielden P; Frost PG; Niriella DC; Cayley AC. *Age and Ageing*, 1992 Mar, 21(2):91-4.

Compared to controls, Alzheimer's patients had lower levels of vitamins E and A, and of beta-carotene, in their blood.

• Possible participation of calcium-regulating factors in senile dementia in elderly female subjects. Ogihara T; Miya K; Morimoto S. *Gerontology*, 1990, 36 Suppl 1:25-30.

Calcium and calcium-regulating hormones may play several roles in senile dementia.

• Regional distribution of iron and iron-regulatory proteins in the brain in aging and Alzheimer's disease. Connor JR; Snyder BS; Beard JL; Fine RE; Mufson EJ. *Journal of Neuroscience Research*, 1992 Feb, 31(2):327-35.

Levels of blood proteins that regulate the body's use of iron are altered in the aging brain, particularly in Alzheimer's disease. The decreased availability of iron that results could be important in explaining the degenerative changes that occur in the disease.

• Specific reduction of calcium-binding protein (28-kilodalton calbindin-D) gene expression in aging and neurodegenerative diseases. Iacopino AM; Christakos S. *Proceedings of the National Academy of Sciences of the United States of America*, 1990 Jun, 87(11):4078-82.

In Alzheimer's disease, and in aging in general, decreased levels of calcium-binding protein have been observed in humans, and in rats. Disturbances in calcium balance within nerves may be responsible for some of the degeneration seen in these conditions.

• The hypothesis of zinc deficiency in the pathogenesis of neurofibrillary tangles. Constantinidis J. *Medical Hypotheses*, 1991 Aug, 35(4):319-23.

Functional zinc decreases leading to abnormal metals reaching the brain may be responsible for a number of conditions, including Alzheimer's disease. A nontoxic zinc compound crossing the blood-brain barrier may be useful in treating Alzheimer's, which is associated with decreased levels of zinc and increased brain levels of aluminum and iron.

• Thiamine and Alzheimer's disease. A pilot study. Blass JP; Gleason P; Brush D; DiPonte P; Thaler H. *Archives of Neurology*, 1988 Aug, 45(8):833-5.

In a double-blind, placebo-controlled study, Alzheimer's patients showing no signs of thiamine deficiency, but treated with thiamine over three months, showed cognitive improvements.

• Vitamin B12 levels in serum and cerebrospinal fluid of people with Alzheimer's disease. Ikeda T; Furukawa Y; Mashimoto S; Takahaski K; Yamada M. *Acta Psychiatrica Scandinavica*, 1990 Oct, 82(4):327-9.

Low levels of vitamin B12 in the cerebrospinal fluid of Alzheimer's patients are characteristic of the disease.

• Vitamin B12-induced reduction of platelet monoamine oxidase activity in patients with dementia and pernicious anaemia. Regland B; Gottfries CG; Oreland L. *European Archives of Psychiatry and Clinical Neuroscience*, 1991, 240(4-5):288-91.

There is a significant connection between vitamin B12 deficiency and Alzheimer's disease. When Alzheimer's patients were treated with B12, their increased platelet monoamine oxidase activity (a characteristic of the disease), was significantly reduced.

• Vitamin E and Alzheimer's disease in subjects with Down's syndrome. Jackson CV; Holland AJ; Williams CA; Dickerson JW. *Journal of Mental Deficiency Research*, 1988 Dec, 32 (Pt 6):479-84.

People with Down's syndrome are at high risk of developing Alzheimer's disease; they seem, because of their genetic makeup, to be more susceptible to oxidative damage. Blood levels of vitamin E in 12 Down's syndrome subjects with Alzheimer's disease were lower than those in 12 Down's subjects without the disease, suggesting an interaction between risk of Alzheimer's and the protective action of vitamin E against oxidative damage.

Next month: Anorexia

Direct Correspondence to the *Townsend Letter*

Gary Null, Ph.D. has 50 published books on health and nutritopics as well as numerous articles published in leading magazines. He is the publisher of the *Natural Living Newsletter*, a monthly publication on health and nutrition. Null is host and health and science reporter on the nationally syndicated Gary Null Show on WBAI. Dr. Null holds a B.S. in human nutrition from Edison State College and Ph.D. in human nutrition and public health science from the Union Graduate School. Dr. Null's investigative reporting has won prestigious awards. To listen to Gary Null's weekly Saturday/Sunday program in your area, call Virtual Network at 800-USA-1718.

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