

# White Flour Report

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Why white flour is an abomination - and is killing you.  
"Dangers of White Flour" - White Flour is Killing Us!!!

[Aspartame NM] Shaw: white flour bleach, MSO (excitotoxin), ALS Dec 1998: hypothalamic lesions, obesity 1.18.00

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Jan 14 2000 - Did consumption of flour bleached by the agene process contribute to the incidence of neurological disease?

Shaw CA, Bains JS Christopher A. Shaw Department of Ophthalmology, University of British Columbia, Vancouver, Canada.

The present report proposes the hypothesis that increased levels of neurodegenerative disorders in humans may have arisen due to inclusion in the diet of methionine sulfoximine (MSO), a byproduct of the bleaching of flour by nitrogen trichloride. This method of bleaching, the 'agene process' was in use from early in the century and continued until at least 1949/1950.

Estimates indicate that, at least in the UK, as much as 80% of all flour during this period was produced by this process.

MSO acts directly to inhibit the production of two crucial molecules, glutathione (GSH) and glutamine. Decreases in GSH, a key antioxidant and free radical scavenger, diminish the body's antioxidant defenses and may lead to increased oxidative stress.

Decreases in glutamine synthesis may act to increase free glutamate and give rise to increased levels of ammonia. Cells in the nervous system are particularly sensitive to a decline in either GSH or glutamine. The combined effects of decreases in these molecules, particularly with long-term exposure to MSO in bleached flour, may have had quite drastic effects on neuronal health and survival.

The present hypothesis may provide clues to the etiology of neurological disorders such as Alzheimer's disease (AD), Parkinson's disease (PD) and amyotrophic lateral sclerosis (ALS), suggesting that such disorders may arise in part due to toxic actions of some compounds in processed human foods. PMID: 10052866, UI: 99160231 Brain Res Brain Res Rev 1997 Dec.25 (3):335-58 Neurodegenerative disorders in humans: the role of glutathione in oxidative stress-mediated neuronal death. Bains JS, Shaw CA Department of Ophthalmology, The University of British Columbia, Vancouver, Canada. [jbains@unixg.ubc.ca](mailto:jbains@unixg.ubc.ca) Oxidative stress has been implicated in both normal aging and in various neurodegenerative disorders and may be a common mechanism underlying various forms of cell death including necrosis, apoptosis, and excitotoxicity.

In this review, we develop the hypothesis that oxidative stress-mediated neuronal loss may be initiated by a decline in the antioxidant molecule glutathione (GSH). GSH plays multiple roles in the nervous system including free radical scavenger, redox modulator of ionotropic receptor activity, and possible

neurotransmitter. GSH depletion can enhance oxidative stress and may also increase the levels of excitotoxic molecules; both types of action can initiate cell death in distinct neuronal populations.

Evidence for a role of oxidative stress and diminished GSH status is presented for Lou Gehrig's disease (ALS), Parkinson's disease, and Alzheimer's disease. Potential links to the Guamanian variant of these diseases (ALS-PD complex) are discussed. In context to the above, we provide a GSH-depletion model of neurodegenerative disorders, suggest experimental verifications of this model, and propose potential therapeutic approaches for preventing or halting these diseases. PMID: 9495562, UI: 98154950 Can J Physiol Pharmacol 1999 Nov;77(11):871-7 Methionine sulfoximine shows excitotoxic actions in rat cortical slices.

Shaw CA, Bains JS, Pasqualotto BA, Curry K Department of Ophthalmology, The University of British Columbia, Vancouver, Canada. cshaw@interchange.ubc.ca

Methionine sulfoximine (MSO) is a rare amino acid. It occurs in nature or as a by-product of some forms of food processing. A notable example of the latter was a former method for bleaching wheat flour, using nitrogen trichloride, the "agene process," in use for most of the first 50 years of this century.

"Agenized" flour was found to be responsible for various neurological disorders in animals, and MSO was identified as the toxic factor. The agene process was subsequently discontinued in the United States and the United Kingdom circa 1950.

MSO inhibits the synthesis of both glutathione and glutamine, and it is possible that its actions on the nervous system arise from alterations in the amount or distribution of these molecules. Structurally, MSO resembles glutamate, an observation that has also raised the possibility that it might have more direct glutamate-like actions on neurons.

In the present investigation, we report excitatory and toxic actions of MSO in an in vitro preparation of adult rat cortex.

Field potential recordings in this preparation show that MSO application evokes a sustained depolarization, which can be blocked by the N-methyl-D-aspartate (NMDA) antagonist L-(+)- 2-amino-5-phosphonovalerate (AP5). However, competition assays using MSO on [3H]CGP-39653 (DL-(E)-2- amino-4-propyl-1-phosphono-3-pentenoate) binding in rat cortical homogenates show only 20% displacement of total binding, suggesting that MSO is acting indirectly, perhaps by releasing glutamate.

To investigate this possibility, we measured glutamate release during MSO application.

Time course and dose-response experiments with MSO showed significant [3H] glutamate release, which was partially attenuated by AP5. To assess cellular toxicity, we measured lactate dehydrogenase (LDH) release from cortical sections exposed to MSO. MSO treatment led to a rapid increase in LDH activity, which could be blocked by AP5. These data suggest that MSO acts by increasing glutamate release, which then activates NMDA receptors, leading to excitotoxic cell death. These data suggest the possibility that MSO in processed flour had excitotoxic actions that may have been contributing factors to some human neuronal disorders.

Are Loaves and Lou Gehrig's Disease Linked? Researcher blames onetime additive to bleach flour By Nicolle Charbonneau Health SCOUT Reporter THURSDAY, Jan. 13 (Health SCOUT) -- White bread may be

synonymous with blandness. But if a Canadian researcher's belief that a flour bleaching process may be linked to amyotrophic lateral sclerosis has any merit, the implications would be anything but boring.

Amyotrophic lateral sclerosis, or ALS, attacks the motor nerve cells in the spinal cord. It causes progressive muscle weakness leading to total paralysis. However, the brain is virtually unaffected. It's also known as Lou Gehrig's disease, after the baseball player who died of the illness in 1941.

Roughly 30,000 Americans have the disease, which usually appears between ages 40 and 70. Death from respiratory paralysis normally occurs within five years, and there is no known cure.

As yet, there is no known cause, although there are many theories. The latest came this week, when researchers announced that they had isolated a virus in the motor nerve cells of ALS victims.

But Christopher Shaw, a neuroscientist at the University of British Columbia in Vancouver, suspects that the disease may have another cause, one as prosaic as white bread.

Or, more specifically, white flour.

Bleached flour became popular in Western countries in the early 1900s because it rose more and with greater consistency. The most popular method of bleaching the flour was a process called the agene method, which used nitrogen trichloride gas to whiten the flour. In Britain and North America, this method eventually was used to treat roughly 80 percent of all flour. But by the 1940s, scientists had realized that the agene method produced a byproduct called methionine sulfoximine (MSO), a toxic neurochemical that could induce epilepsy in dogs. MSO stayed in the flour during baking, and was eventually eaten. By 1950, Britain and the United States had banned the agene method.

Two years ago, Shaw was researching a compound called buthionine sulfoximine (BSO) which is sometimes given in combination with chemotherapy drugs. BSO reduces levels of glutathione, which shields cancer cells from chemotherapy but at the same time protects the body from damaging free radicals. Chemical damages nerves In the course of his research on BSO; Shaw discovered 50-year-old reports on MSO, the parent chemical of BSO. In lab studies, Shaw has shown that along with reducing glutathione levels (and leaving nerve cells vulnerable to free radical damage), it prevents the synthesis of glutamine, an amino acid. Without this synthesis, toxic levels of ammonia can build up around neurons. Finally, MSO locks open the calcium channels on nerve cells, until the cells overload and die. Shaw was stunned to realize that millions of people had essentially been taking a powerful drug for years.

Shaw contacted Christopher Martin, a British neuroepidemiologist who feels that neurodegenerative diseases have become two to four times more common in the last century. According to his research, the incidence of diseases like ALS has actually peaked, and should continue to decline as the generations exposed to MSO age and die.

But neither Shaw nor Martin expects that ALS will disappear once MSO disappears. Other chemical toxins may have the same neural effect, or other viral or genetic factors may be at work.

Still, Shaw's research raises the question of why more people haven't developed ALS. Shaw suggests that it's similar to how different people are more or less vulnerable to certain diseases or toxins. "Some

people may be able to detoxify molecules in a way that's a lot more successful than others," says Shaw. Or they may be able to carry more of the toxin in their system before an effect is felt.

According to Dr. Peter St. George-Hyslop, director of the Centre for Neurodegenerative Diseases at the University of Toronto, such a threshold may also explain why exposure earlier in life doesn't have an effect until decades later. "If you are exposed to some toxin that kills, say, 30 percent of the cells, it lowers your reserve," he says.

"It means that you will reach a threshold, or a point where you no longer have enough cells left" to "function normally," he continues. Then, during "normal aging, you lose cells and you reach the minimum threshold earlier." What To Do"

This Health SCOUT story describes a new study that links a virus to ALS. This story looks at a protein that may help fight the symptoms of ALS. For more information, contact the ALS Association, the Muscular Dystrophy Association or the National Institute of Neurological Disorders and Stroke.

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Shaw: excitotoxins cause hypothalamic lesions, thus obesity 1.18.00 Jan 18 2000

The basic idea is based on the work of John Olney which you are familiar with. Excitotoxic compounds like MSG, aspartate, cysteine seem to create hypothalamic lesions, particularly in young animals. The reason for the latter is likely the fact that the blood brain barrier closes most slowly (if ever completely) around structures like hypothalamus.

The outcome for such animals (rats) was obesity, severe behavioral changes, etc. Needless to say, any compromise to the bbb can let such compounds gain access in older animals. Regarding MSO, some studies by one of our colleagues (Dr. Mike Wilkinson, Dalhousie Univ, Halifax) showed that MSO was extremely toxic with hypothalamic damage occurring in new born rat pups. The latter is still an ongoing project.

Regarding the various disorders on the increase: obesity and mood disorders clearly fit with hypothalamic abnormality; as for asthma you are completely right that it is definitely on the increase. My fast impression would be an immune disorder mediated via abnormal HPA axis, although whether this is due to food borne toxin or some airborne junk like gasoline additives MMT or MTBE, or is due to a combination of air and food toxins, may be hard to sort out. Epidemiology is not good at multiple variable/long term toxin effects, especially where the toxic action is chronic rather than acute.

Reference the experiment you propose: I sincerely hope someone tries this ASAP. I have relatively little doubt what the outcome would be.

Also, MRI of hypothalamus of long-term diet soft drink drinkers' vs. 'control' (which may be hard to find given the ubiquity of aspartame and other such toxins) would likely be highly informative and likely quite distressing.

Med Hypotheses 1998 Dec;51(6):477-81 Did consumption of flour bleached by the agene process contribute to the incidence of neurological disease? Shaw CA, Bains JS Christopher A. Shaw Department of Ophthalmology, University of British Columbia, Vancouver, Canada.

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This method of bleaching, the 'agene process' was in use from early in the century and continued until at least 1949/1950....etiology of neurological disorders such as Alzheimer's disease (AD), Parkinson's disease (PD) and amyotrophic lateral sclerosis (ALS)..." Brain Res Dev Brain Res 1997 Aug 18;102(1):97-104 Recovery of hypothalamic NMDA-induced c-fos expression following neonatal glutamate (MSG) lesions. Natarajan M, Wilkinson M Department of Obstetrics and Gynecology, IWK-Grace Health Centre, Halifax, NS, Canada.

The neonatal brain is susceptible to neurotoxic insult. In a previous report we showed that a single neonatal injection of MSG, known to cause damage in the arcuate nucleus (ARC), induces a precocious yet otherwise normal puberty in female rats. We have examined this ability of the medial basal hypothalamus (MBH) to recover from an excitotoxic insult..."

Best of Health,

Margie "The Arthritis Lady"

This article is only for information and does not take the place of medical advice. It mainly gives you a starting place to explore what is best for you. I agree with everything in these articles, but it is up to you to make your own decisions.