Eye Problems Related to Mercury

Bernard Windham (Ed.)

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Studies document that mercury and similar toxic metals accumulate in endothelial cells such as those in the eye retina, cornea, and macula depleting glutathione and lipoate by binding to thiols; these are needed to counteract free radicals caused by such as toxic metals that damage the endothelial layers of the the retina, cornea and macula, as major factors in such conditions (see Pub Med abstracts of studies). Such free radicals generated have been found to cause cataracts and such conditions that can be prevented, slowed, and even reversed to some degree by detox and antioxidant eye drops (according to studies). Mercurialentis (brown discoloration of anterior capsule of eye lens-caused by mercury, is documented in medical texts as the first sign of mercury toxicity: It is an indicator and early sign of further eye damage. Cataracts, retinitis pigmentosa, iritis, color vision problems, and other eye conditions are documented to commonly be caused by mercury/metals toxicity.

Medical Dictionary, *www.medilexicon.com/medicaldictionary.php*[1]. It is commonly caused by systemic poisoning from absorption of mercury vapor through the respiratory tract or through the gastrointestinal tract: *www.llnl.gov/es_and_h/hsm/supplement_21.11/inorg.html*[2]

From my experience I know of 5 eye problems related to mercury. There are probablymore. One eye problem mercury causes is chronic iritis- I don't know much about that but it's documented in the medical literature and someone else I know had it. Another is color vision; that's also documented in the medical literature and several I know have had color vision improve after amalgam removal, including me.

I have Fuch's disease (clouding of cornea caused by deterioration/ glumping of endothelial cells in the cornea. Aggressive form of cataracts. Animal studies and in vitro studies have shown mercury causes similar damage to endothelial cells in various parts of the body due to deterioration and free radical effects. Since having my amalgams removed over the last 2 years, my opthamologist says that the deterioration of the endothelial layer of my corneas has slowed considerably compared to 2 years ago. My vision has also improved so much that I cannot see at all through my glasses that I got 4 years ago. My optomitrist who did the glasses and reexamined me was really surprised, said my vision had improved almost 50%. I no longer wear glasses.

Another eye problem related to mercury is dry eyes. Several clinics have had success with improvements after amalgam replacement. The other eye problem known to sometimes be related to mercury is macula degeneration. The buildup of mercury in the eye is similar to in Fuch's, etc. and causes clouding and degeneration. Someone I know says a relative got better after amalgam replacement. The following are a few abstracts or references I'm aware of. (B Windham)

Strabismus is where one eye moves freely of the other, either inwardly or outwardly to focus independently. This condition, which was present at an early age in my now 5 year old, was quickly

[1] **Internet:** "http://www.medilexicon.com/medicaldictionary.php".
[2] **Internet:** "http://www.llnl.gov/es_and_h/hsm/supplement_21.11/inorg.html".

corrected by supplementing the RDA of vit A in cis form and cutting out all other vit A sources (Megson protocol). Pupil dilation was always a factor in him too. Interestingly, his pupils reacted normally within the first two doses of DMSA he ever received, but the dilation returned post-round. After 5 months of chelation, I see VERY normal eye function, with normal pupil reactivity for the most part. I believe the dilation is a symptom of mercury toxicity.

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Dilation, poor accomodative function (focussing) and convergence insufficiency - which if severe is strabismus - are characteristic of mercury poisoning. They are all due to a mild, symmetrical impairment of the third cranial nerve. Since this nerve connects to the brain right next to the hypothalamus, where mercury is known to concentrate.

Dr. A. Cutler, (see his web site)

Macular Degeneration - Degeneration of the macula lutea of the eye. Often caused by free radical or oxidation damage.

www.nutritionfocus.com/nutrition_supplementation/glossary/GlossaryM.html[3]

The first symptom I had of mercury toxicity was double vision, then drooping eyelids. I also had floaters for years and bright lights blinded me. When I was 42 my keen eyesight started to go and I had to wear glasses to sew or read. By the time 1998 rolled around I was wearing 250 magnifying glasses. Within a short period of time after amalgam removal I no longer needed reading glasses, and today I do not wear glasses and am able to read any size print.

However, I still have very slight double vision to the extreme right and left, I don't have floaters any longer but still have some sensitivity to bright lights.

Freya Koss, *Frekoss@aol.com*[4]

Mercury has been found to be a factor in retinitis pigmentosa and retina degeneration.

Olynyk F, Sharpe DH; Mercury poisoning in paper pica, New Eng J Med, 1982, Apr 29: 306(17):1056-57; & Uchino M, Tanaka Y, Ando M, et al; Neurologic features of chronic minamata disease (organic mercury poisoning) J Environ Sci Health B 1995, Sep; 30(5): 699-715.

Distributions of elements in the human retnal pigment epitheliaum; Arch Ophthalmol, 1990, Jan; 108(1):113-117; Ulshafer RJ, Allen CB, Rubin ML.

> Transport of thiol-conjugates of inorganic mercury in human retinal pigment epithelial cells, Bridges CC, Battle JR, Zalups RK. Toxicol Appl Pharmacol. 2007 Jun 1; 221(2):251-60. Epub 2007 Mar 23
>
> Division of Basic Medical Sciences, Mercer University School of Medicine, Macon, GA 31207, USA. bridges_cc@mercer.edu
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> Inorganic mercury (Hg (2+)) is a prevalent environmental contaminant to which exposure to can damage rod photoreceptor cells and compromise scotopic vision. The retinal pigment epithelium (RPE) likely plays a role in the ocular toxicity associated with Hg (2+) exposure in that it mediates transport of substances to the photoreceptor cells. In order for Hg (2+) to access photoreceptor cells, it must first be taken up by the RPE, possibly by mechanisms involving transporters of essential nutrients. In other epithelia, Hg

[3] **Internet:** "http://www.nutritionfocus.com/nutrition_supplementation/glossary/GlossaryM.html".
[4] **Internet:** "mailto:Frekoss@aol.com".

(2+), when conjugated to cysteine (Cys) or homocysteine (Hcy), gains access to the intracellular compartment of the target cells via amino acid and organic anion transporters. Accordingly, the purpose of the current study was to test the hypothesis that Cys and Hcy S-conjugates of Hg (2+) utilize amino acid transporters to gain access into RPE cells. Time- and temperature-dependence, saturation kinetics, and substrate-specificity of the transport of Hg (2+), was assessed in ARPE-19 cells exposed to the following S-conjugates of Hg (2+): Cys (Cys-S-Hg-S-Cys), Hcy (Hcy-S-Hg-S-Hcy), N-acetylcysteine (NAC-S-Hg-S-NAC) or glutathione (GSH-S-Hg-S-GSH). We discovered that only Cys-S-Hg-S-Cys and Hcy-S-Hg-S-Hcy were taken up by these cells. This transport was Na (+)-dependent and was inhibited by neutral and cationic amino acids. RT-PCR analyses identified systems B (0, +) and ASC in ARPE-19 cells. Overall, our data suggest that Cys-S-Hg-S-Cys and Hcy-S-Hg-S-Hcy are taken up into ARPE-19 cells by Na-dependent amino acid transporters, possibly systems B (0, +) and ASC. These amino acid transporters may play a role in the retinal toxicity observed following exposure to mercury.

Transport of thiol-conjugates of inorganic mercury in human retinal pigment epithelial cells, Bridges CC, Battle JR, Zalups RK. Toxicol Appl Pharmacol. 2007 Jun 1; 221(2):251-60. Epub 2007 Mar 23

Division of Basic Medical Sciences, Mercer University School of Medicine, Macon, GA 31207, USA. bridges_cc@mercer.edu

Inorganic mercury (Hg (2+)) is a prevalent environmental contaminant to which exposure to can damage rod photoreceptor cells and compromise scotopic vision. The retinal pigment epithelium (RPE) likely plays a role in the ocular toxicity associated with Hg (2+) exposure in that it mediates transport of substances to the photoreceptor cells. In order for Hg (2+) to access photoreceptor cells, it must first be taken up by the RPE, possibly by mechanisms involving transporters of essential nutrients. In other epithelia, Hg (2+), when conjugated to cysteine (Cys) or homocysteine (Hcy), gains access to the intracellular compartment of the target cells via amino acid and organic anion transporters. Accordingly, the purpose of the current study was to test the hypothesis that Cys and Hcy S-conjugates of Hg (2+) utilize amino acid transporters to gain access into RPE cells. Time- and temperature-dependence, saturation kinetics, and substrate-specificity of the transport of Hg (2+), was assessed in ARPE-19 cells exposed to the following S-conjugates of Hg (2+): Cys (Cys-S-Hg-S-Cys), Hcy (Hcy-S-Hg-S-Hcy), N-acetylcysteine (NAC-S-Hg-S-NAC) or glutathione (GSH-S-Hg-S-GSH). We discovered that only Cys-S-Hg-S-Cys and Hcy-S-Hg-S-Hcy were taken up by these cells. This transport was Na (+)-dependent and was inhibited by neutral and cationic amino acids. RT-PCR analyses identified systems B (0, +) and ASC in ARPE-19 cells. Overall, our data suggest that Cys-S-Hg-S-Cys and Hcy-S-Hg-S-Hcy are taken up into ARPE-19 cells by Na-dependent amino acid transporters, possibly systems B (0, +) and ASC. These amino acid transporters may play a role in the retinal toxicity observed following exposure to mercury.

Homocysteine, system b0, + and the renal epithelial transport and toxicity of inorganic mercury, Bridges CC, Zalups RK. Am J Pathol. 2004 Oct; 165(4):1385-94

Mercer University School of Medicine, Division of Basic Medical Sciences, 1550 College St., Macon, GA 31207, USA.

Proximal tubular epithelial cells are major sites of homocysteine (Hcy) metabolism and are the primary sites for the accumulation and intoxication of inorganic mercury (Hg (2+)). Previous in vivo data from our laboratory have demonstrated that mercuric conjugates of Hcy are transported into these cells by unknown mechanisms. Recently, we established that the mercuric conjugate of cysteine [2-amino-3-(2-amino-2-carboxy-ethylsulfanylmercuricsulfanyl) propionic acid; Cys-S-Hg-S-Cys], is transported by the luminal, amino acid transporter, system b (0, +). As Cys-S-Hg-S-Cys and the mercuric

conjugate of Hcy (2-amino-4-(3-amino-3-carboxy-propylsulfanylmercuricsulfanyl) butyric acid; Hcy-S-Hg-S-Hcy) are similar structurally, we hypothesized that Hcy-S-Hg-S-Hcy is a substrate for system b (0, +). To test this hypothesis, we analyzed the saturation kinetics, time dependence, temperature dependence, and substrate specificity of Hcy-S-Hg-S-Hcy transport in Madin-Darby canine kidney (MDCK) cells stably transfected with system b (0, +). MDCK cells are good models in which to study this transport because they do not express system b (0, +). Uptake of Hg (2+) was twofold greater in the transfectants than in wild-type cells. Moreover, the transfectants were more susceptible to the toxic effects of Hcy-S-Hg-S-Hcy than wild-type cells. Accordingly, our data indicate that Hcy-S-Hg-S-Hcy is transported by system b (0, +) and that this transporter likely plays a role in the nephropathy induced after exposure to Hg (2+). These data are the first to implicate a specific, luminal membrane transporter in the uptake and toxicity of mercuric conjugates of Hcy in any epithelial cell.

(mercury accumulates in cornea endothelial cells and causes oxidative damage resulting in cataracts, etc.)

SEAFOOD/CATARACTS Methylmercury in seafood may cause lens clouding, contributing to cataract development. Optometrist Ben Lane noted that his cataract patients liked seafood, while those who didn't like fish were clear-eyed. A study of 17 patients revealed that the cataract patients had eaten salt water fish or shellfish at least once a week on the average, but those cataract-free reported using these foods an average of once every five weeks. The cataract patients showed far higher concentrations of mercury in their hair. Dr. Lane's study showed that the presence of 2.3 ppm or more of mercury in hair samples was related to a 23-fold increase in the risk of cataracts. Dr. Lane encourages his patients to eat such foods as garlic and pectin-rich foods such as apples to help remove the mercury, and to receive adequate, while avoiding excessive, amounts of vitamins A, C, and E.

Dr, Ben Lane, O.D., Methylmercury in seafood contributes to cataract development, Medical World News, December 20, 1982

I would look into the possibility of mercury poisoning which is capable of causing both cataracts and light sensitivity. (So are other things.) I have seen many babies born with mercury poisoning (further exacerbated by the mercury in vaccinations.) from their mother's dental amalgams. I would get a Hair Mineral Analysis right away. Good luck.Steve Rochlitzrochlitz@wellatlast.com

Cataract reversal through mercury detox www.digitalnaturopath.com/treat/T33633.html

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Mobilization AND excretion are required for mercury detoxification. Consuming foods high in sulfur such as garlic, onions, beans, and eggs or supplemental sulfur in the form of MSM can help move mercury around but it is only bound loosely and caution is advised. There have been reported cases of reversible cataract development from individuals mobilizing mercury without excreting it. Consult a qualified doctor for a detoxification protocol appropriate for you. Alan Thal, MD

Antioxidant eye drops (n-acetylcarnosine) have been documented to prevent and sometimes reverse cataracts (such as Can C drops).

Rudolph CJ, Samuels RT, McDanagh EW. Cheraskin E. Visual Field Evidence of Macular Degeneration Reversal Using a Combination of EDTA Chelation and Multiple Vitamin and Trace Mineral Therapy.In: Cranton EM, ed. A Textbook on EDTA Chelation Therapy, Second Edition. Charlottesville, Virginia: Hampton Roads Publishing Company; 2001

Dr. G. E. Poesnecker, Its Only Natural, 2001, http://www.oneflesh.org/only-22.html

Disorders that Chelation Can Help

Following is a list of conditions successfully treated by chelation that has been assembled by physicians who did much of the early research work. Many of these problems are common and are generally considered incurable: scleroderma; digitalis intoxication; heavy-metal poisoning (especially acute plumbism); calcinosis (pipestem calcinosis of the vessels, prostatic calcinosis); vascular atheromatous disorders including atherosclerosis, atheromatous deposits, arteriosclerosis obliterans, peripheral vascular insufficiency with intermittent claudication, and acute brain syndrome secondary to cerebral ischemia secondary to calcific atherosclerosis; myocardial or coronary insufficiency; collagenosis; arteriosclerosis including cerebrovascular arteriosclerosis; arthritis including hypertrophic and rheumatoid; calcific tendinosis; calculi; diabetic retinopathy; multiple sclerosis; macular degeneration of the retina; cataracts; Parkinsonism; emphysema; poisonous snake and insect bites; calcified necrotic ulcers; heart valve calcification; hemochromatosis; calcific bursitis; calcified granulomas; and hypertension.

Mercury accumulates in the uvea and retina of the eye.

Khayat A, Dencker L. Whole body and liver distribution of inhaled mercury vapor in the mouse: influence of ethanol and aminotriazole pretreatment. J Appl Toxicol. 1983 Apr; 3(2):66-74

Inorganic mercury has been found to be associated with cataract formation.Hachet E. Cataracts, Bull Soc Ophtalmol Fr. 1985 Nov; Spec No:87-107.

Dr. Victoria Buntine, Mercury Effects, Healthinasia Incorporated, 2001 www.healthinasia.com/mercury.ht

The problem with mercury is that it affects our nervous system. Mercury accumulates in what we call end organs, like kidneys, brain, thyroid and eyes, and this is why it is detected on hair analysis It may contribute to cataracts, headaches, numbness and tingling, irritability, joint pain and autism in kids, as well as chronic fatigue syndrome and general allergies.

Dr. D.A. Carroll, O.D.& Dr. B.C. Lane, Preventing mercury related cataracts.

www.medicalvisioncenter.com/prevention.html

Vitamin C also helps to pull out the toxic mercury that results from the consumption of large fish, such as tuna, swordfish and shark. Dr. Lane said that his 1982 study found that mercury, which would accumulate in the crystalline lens, resulted in the depression of enzymes such as superoxide dismutase and glutathione peroxidase. The latter is the primary enzyme that helps prevent mercury cataracts from forming. 'Organic mercury is the worst offender because it's able to penetrate membranes and get into organic tissues, ' he said.

Cavalleri A, Belotti L, Gobba FM, Luzzana G, Rosa P & Seghizzi P. Colour vision loss in workers exposed to elemental mercury vapour. Toxicology Letters 77(1-3):351-356 (1995)

ABSTRACT: "We evaluated colour vision in 33 workers exposed to elemental mercury (Hg) vapour and in 33 referents matched for sex age, alcohol consumption and cigarette smoking. The results were expressed as colour confusion index (CCI). In the workers urinary excretion of Hg (HgU) ranged from 28 to 287 μg/g creatinine. Subclinical colour vision loss, mainly in the blue-yellow range, was observed in the workers. This effect was related to exposure, as indicated by the correlation between HgU and CCI (r=0.488, P¡0.001)."

& Urban P, Gobba F, Nerudova J, Lukas E, Cabelkova Z, Cikrt M, .Color Discrimination Impairment in Workers Exposed to Mercury Vapor. Neurotoxicology. 2003 Aug; 24(4-5):711-716;

If you want to know something about retinitis pigmentosa (or retinopathia pigmentosa) and mercury poisoning, you should read the following two articles:

1. Olynyk F, Sharpe DH: Mercury poisoning in paper pica. (retinitis pigmentosa), N Engl J Med 1982 Apr 29; 306(17):1056-1057

2. Uchino M, Tanaka Y, Ando Y, Yonehara T, Hara A, Mishima I, Okajima T, Ando M: Neurologic features of chronic minamata disease (organic mercury poisoning) and incidence of complications with aging. J Environ Sci Health B 1995 Sep; 30(5):699-715

Also: Arch Ophthalmol 1990 Jan; 108(1):113-117

Distributions of elements in the human retinal pigment epithelium.

Ulshafer RJ, Allen CB, Rubin ML

Department of Ophthalmology, College of Medicine, Univ. of Florida, Gainesville 32610.

Distributions of elements above the atomic number of sodium were mapped in the retinal pigment epithelia of eight human eyes. X-ray energy spectra and maps were collected from cryofixed, freeze-dried, and epoxy-embedded tissues using energy-dispersive x-ray microanalysis. All eyes had high concentrations of phosphorus in the nuclei of retinal pigment epithelial cells. Melanosomes were rich in sulfur, zinc, calcium, and iron. Lipofuscin and cytoplasm contained only phosphorus and sulfur in detectable amounts. Drusen, when present, contained phosphorus and calcium. Six eyes had a prominent aluminum peak recorded from melanosomes, nuclei, and Bruch's membrane. In one pair of 90-year-old eyes, small, electron-dense deposits surrounded many melanosomes and contained mercury and selenium. Retinal pigment epithelial melanosomes may bind and accumulate metals and other potentially toxic ions over time, preventing them from reaching the neural retina.

Journal of Environmental Science and Health Part B Pesticides Food Contaminants and Agricultural Wastes 30(5): 699-715. 1995

Abstract: Tessier-Lavigne M, Mobbs P, Attwell D, Invest Ophthalmol Vis Sci 1985 Aug; 26(8):1117-1123 "Lead and mercury toxicity and the rod light response".

Lead and mercury have been reported to alter selectively the rod component of the electroretinogram, and to inhibit the phosphodiesterase in rod outer segments which may be responsible for generating the rods' light response. The authors have investigated the effect of lead and mercury on the voltage response to light of rods, and compared these effects with those of the phosphodiesterase inhibitor papaverine. Lead and mercury, like papaverine, slow the light response. In addition, papaverine increases the light response amplitude while lead decreases it. Mercury initially increases and then decreases the amplitude. The late decrease in amplitude "produced by mercury is associated with rod degeneration": an effect which may mimic degenerative diseases in which the rod phosphodiesterase is insufficiently active. These results demonstrate that the changes of electroretinogram induced by lead and mercury can be accounted for by the changes in receptor potential these heavy metals produce. The changes in receptor potential seen are consistent with mercury inhibiting the rod phosphodiesterase, and with lead having an action in addition to phosphodiesterase inhibition.

PMID: 2991162, UI: 85260515

God Zb Med Fak Skopje 1978; 24:289-291

[Changes in the crystalline lens of the eye in workers occupationally exposed to mercury vapors]. [Article in Serbo-Croatian (Cyrillic)]

Delivanova S, Popovski P, Orusev T PMID: 757176, UI: 80092857

Abstract

"Effect of the ophthalmic preservative thimerosal on human and rabbit corneal endothelium".

Van Horn DL, Edelhauser HF, Prodanovich G, Eiferman R, Pederson HF

Widespread use of the mercurial-containing preservative thimerosal as an antibacterial agent in ophthalmic drugs and solutions warranted an investigation into its possible cytotoxic effects on the functional and ultrastructural integrity of the corneal endothelium. No changes in corneal thickness were observed during 5 hours' perfusion of the endothelium of rabbit and human corneas with 0.0001 and 0.0005 percent thimerosal in glutathione bicarbonate Ringer's solution (GBR). Scanning electron microscopy (SEM) and transmission electron microscopy (TEM) of the endothelium of the 0.0001 percent group revealed normal ultrastructure. SEM and TEM of the endothelium of corneas perfused with 0.0005 percent thimerosal for 5 hours revealed condensed mitochondria, cytoplasmic vacuoles, and cytoplasmic flaps at the apical end of the cellular junctions. Perfusion of higher concentrations (0.001 and 0.005 perecnt) of thimerosal in GBR resulted in increases in corneal thickness after 2 hours and irreversible ultrastructural damage to the endothelial cells by 5 hours. Corneas perfused with 0.01 and 0.1 percent thimerosal in GBR showed a rapid and immediate increase in corneal thickness and endothelial cell death and necrosis within 1 hour. It is postulated that the mercury in thimerosal becomes bound to the cell membrane protein sulfhydryl groups, causing an increase in cellular permeability; These results suggest that the prolonged exposure of the corneal endothelium

to thimerosal in the accepted antimicrobial dosage of 0.005 to 0.001 percent may result in functional and "structural damage to the endothelium".

PMID: 844986, UI: 77140310

Garron LK, Wood IS, Spencer WH, Hayes TL. A clinical pathologic study of mercurlalentis medicamentosus. Trans Am Ophthalmol Soc 1977; 74:295-320

Thirty-one patients who used eye drops containing the preservative, phenylmercuric nitrate for from 3 to 15 years, developed a brownish pigmentation of the anterior capsule of the pupillary area. Light and electron microscopic studies on two lenses demonstrated deposits of dense particulate material resembling melanin pigment on and in the anterior capsule of the lens in the area of the pupil. Special studies, including electron microprobe analysis and neutron activation analysis established the presence of mercury in a lens with mercurialentis. No mercury was found in two lenses used as controls.

PMID: 867632, UI: 77196922

Klein, CL; Kohler, H; Kirkpatrick, CJ.

Increased Adhesion and Activation of Polymorphonuclear Neutrophil Granulocytes to Endothelial Cells under Heavy Metal Exposure in Vitro. Pathobiology. 62(2):90-98, 1994.

ABSTRACT: Heavy metals have been implicated in the mechanisms of endothelial damage. Influences of heavy metal ions on diverse cell types have been studied using a variety of in vitro and in vivo methods. Polymorphonuclear neutrophil granulocytes (PMNs) have physiological and pathological functions, including the modulation of adhesion to and destruction of endothelial cells (ECs).

PMNs were studied during interaction with human umbilical vein ECs under exposure to zinc, nickel and cobalt using an in vitro model. We studied adhesion processes with the help of a computer-controlled image-analyzing system and examined the activation of PMNs by quantification of leukotriene B4 (LTB4) release. The biphasic effects of the valuated heavy metals on PMN-EC adhesion, with stimulation at very high and very low molar concentrations, were observed. The release of LTB4 by PMNs increased during exposure to very low metal concentrations. The initiation of these important pathogenetic mechanisms of inflammation at very low metal ion concentrations, which give no morphologic changes, must be regarded as potentially significant with respect to the toxic effects of heavy metals. BIO-PROBE COMMENT: Damage to the inner lining of blood vessels (endothelium) is widely regarded to be the initial step in the disease process that leads to cardiovascular disease. Although mercury was not included in this study, it is a heavy metal that has previously been shown to cause endothelial damage. The three metals examined in this study nickel, cobalt and zinc) are all used in dental restorative materials. Research, published in peer-reviewed dental journals, has demonstrated the release and bioavailability of nickel (and mercury). Cardiovascular disease has become widespread only since the 1920's, about the time of increased use of heavy metals in dental therapy and long after humans consumed eggs, meat, milk, butter and cheese.(other studies documenting mercury damage to is available on the web: use EXCITE search engine or MEDLINE http:///www.nlm.nih.gov/)

Abstract

Exp Eye Res 1993 Nov; 57(5):549-555

Low levels of inorganic mercury damage the corneal endothelium.

Sillman AJ, Weidner WJ

Section of Animal Physiology, University of California, Davis 95616.

The effect of inorganic mercury on the integrity of the endothelium of isolated bullfrog (Rana catesbeiana) corneas was examined by spectrophotometric analysis of corneal uptake of the vital stain Janus green, and by both transmission (TEM) and scanning (SEM) electron microscopy. The uptake of Janus green by the endothelium is dose related between 1.0 and 30.0 microM HgCl2. The effect of mercury is not altered by changes in external calcium concentration, nor is it influenced by the calcium ionophore A23187, indicating that inorganic mercury damages the corneal endothelium through a mechanism which does not involve competition with external calcium or interaction with calcium channels. TEM and SEM demonstrate significant ultrastructural damage to the endothelium exposed to inorganic mercury, including cellular swelling, increased vacuolization, focal denuding of Descemet's membrane, and diminished integrity at the intercellular junctions.

PMID: 8282041, UI: 94109509

Toimela TA, Tahti H. Effects of mercuric chloride exposure on the glutamate uptake by cultured retinal pigment epithelial cells. Toxicol In Vitro 2001 Feb; 15(1):7-12

Tampere University Medical School, FIN-33014 University of Tampere, Finland.

The cytotoxicity of mercuric chloride and the effects of mercuric chloride on glutamate and calcium uptake and the factors regulating glutamate uptake were studied in retinal pigment epithelium (RPE) cell cultures. RPE cells isolated from pig eyes and human RPE cell line (D407) cells were cultured to confluency and further subcultured according to the test protocol in question. The cytotoxicity caused by 15 min of exposure to mercuric chloride (0.01–1000 microM) was evaluated by WST-1 assay based on the activity of mitochondrial dehydrogenases. [(3)H]Glutamate uptake was measured after the cells were exposed to 0.1–100 microM mercuric chloride and the selected regulators of protein kinase C (PKC) pathway: PKC activator SC10, PKC inhibitor chelerythrine chloride, phospholipase A (2)/C inhibitor manoalide, tyrosine kinase inhibitor lavendustin A, competitive NMDA receptor antagonist AP7 and IP (3) receptor antagonist heparin. Intracellular calcium was monitored with Fluo-3 probe starting immediately after the exposure to 1–1000 microM mercuric chloride. Mercuric chloride showed concentration-dependent effects on cell viability, on glutamate uptake and on intracellular calcium concentration. The results give some support to the concept that glutamate uptake is affected by PKC. The PKC inhibitor chelerythrine chloride decreased glutamate uptake by 25%, but the PKC activator SC10 could partly prevent the inhibitory effect of mercuric chloride. Lavendustin A, manoalide and heparin had smaller, but statistically significant, effects. All these substances act on mediators which can regulate the activity of PKC. However, PKC is not likely to be the only regulator of glutamate uptake. The rise observed in [Ca (2+)](i) may initiate various cellular events during mercury intoxication.

Hum Toxicol 1987 May; 6(3):253-256

Prenatal and early postnatal intoxication by inorganic mercury resulting from the maternal use of mercury containing soap.

Lauwerys R, Bonnier C, Evrard P, Gennart JP, Bernard A.

A case of slight renal tubular dysfunction associated with cataract and anaemia was diagnosed in a 3-month-old black boy in whom high levels of mercury were found in blood and urine. Several arguments suggest that the renal, ocular and haematological defects may have resulted from exposure to mercury during foetal life and the 1-month lactation period due to the extensive use of inorganic mercury containing cosmetics by the mother.

1: Bull Soc Belge Ophtalmol 1978; 181:21-37

[Cataract of toxic origin (mercury)] [Article in French]

Michiels J.

Bull Soc Ophtalmol Fr 1985 Nov; Cataracts:87-107

[Cataracts (mercury)].

[Article in French]

Hachet E.

Oftalmol Zh 1974; 29(7):501-503

[Eye manifestations of chronic mercury poisoning].

[Article in Russian]

Fomicheva IV.

Bull Environ Contam Toxicol 1991 Feb; 46(2):230-236

Inhibition of corneal epithelial cell migration by cadmium and mercury.

Ubels JL, Osgood TB

Mount Desert Island Biological Laboratory, Salsbury Cove, Maine 04672.

PMID: 2018869, UI: 91208463

Klein, CL; Kohler, H; Kirkpatrick, CJ.

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Toxicology 1996 Mar 18; 107(3):189-200

Mercury accumulation in the squirrel monkey eye after mercury vapour exposure.

Warfvinge K, Bruun A

Department of Ophthalmology, University Hospital of Lund, Sweden.

Squirrel monkeys were exposed to mercury vapour at different concentrations and for different numbers of days. The calculated total mercury absorption ranged between 1.4-2.9 mg (range of daily absorption 0.02-0.04 mg). The monkeys were killed at different intervals after the end of exposure (range 1 month - 3 years) and the eyes were enucleated. Eyes from four un-exposed monkeys were used as control material. Mapping of the mercury distribution in the eye revealed that the non-myelin-containing portion of the optic disc was densely loaded with mercury deposits, which are mostly confined to the capillary walls and the glial columns. The white matter of the brain does not accumulate mercury at these exposure levels, which might suggest that the myelinization process inhibits the accumulation of mercury. The pigmented epithelium of the pars plicata of the ciliary body and of the retina contained a considerable amount of mercury. This finding indicates that mercury is trapped within the melanocytes, which keeps potentially dangerous material from reaching the neural retina. In addition, the retinal capillary walls were densely loaded with mercury deposits, even 3 years after exposure. It was also found that the inner layers of the retina accumulated mercury during a 3-year period. It is known that the biological half-time of mercury in the brain may exceed years. This seems also to be the case for the ocular tissue.

PMID: 8604479, UI: 96180636

Toxicology 1988 Sep; 51(1):67-76

Enhanced electroretinogram in cats induced by exposure to mercury acetate.

Gitter S, Pardo A, Kariv N, Yinon U

Institute for Occupational Health, Maurice and Gabriela Goldschleger Eye Research Institute, Chaim Sheba Medical Center, Tel-Hashomer, Israel.

The present study was undertaken in order to verify whether, and how, retinal functions are affected by subacute poisoning with organic mercury. Mercury acetate in various concentrations (0.025-0.25 mg/kg per day) was injected subcutaneously every second day to adult cats (N = 20) throughout a 2.5-4.0-week period. The electroretinogram (ERG) was recorded and the Hg2+ concentrations in the blood were determined. In nearly 90% of the intoxicated cats an enhanced electroretinogram (scotopic b-wave amplitude) was found as compared to its level in the normal control cats (N = 10). The latency of the ERG was found to be appropriately shorter, up to a maximal difference of nearly 20% in comparison to the controls. Hg2+ was present in the blood of the exposed cats during a 2.5-month period following the exposure. It is concluded that exposure to mercury acetate induces a permanent increase in the excitability level of the cat's retina.

Toxicology 1983 Jan; 26(1):1-9

A cell aggregation model for the protective effect of selenium and vitamin E on methylmercury toxicity.

Kleinschuster SJ, Yoneyama M, Sharma RP

Histotypic aggregation of embryonic neural retinal cells was chosen as a test model to evaluate mercury toxicity. After 24 h rotational culture with methylmercury (CH3HgCl) at 4 microM, aggregation was completely inhibited. A dose-response relationship between concentrations of methylmercury and final sizes of aggregates was found. Selenium (Na2SeO3) at concentrations of 1, 3 and 5 microM provided a protective effect for methylmercury (1 microM) toxicity. Vitamin E (DL-alpha-Tocopherol acetate) at concentrations 5, 7 and 10 microM also provided protection against the same concentration of methylmercury; however, it was less effective than selenium. Histotypic embryonal retinal cell aggregation may be a useful assay system for in vitro neurotoxic studies in morphogenesis.

PMID: 6829026, UI: 83147085

Science 1979 Oct 5; 206(4414):78-80

Heavy metals affect rod, but not cone, photoreceptors.

Fox DA, Sillman AJ

Low concentrations of lead, mercury, or cadmium depress the amplitude of the rod receptor potential in the perfused bullfrog retina. Responses from the cones were not affected. The data implicate the rods as a lesion site in animals exhibiting scotopic vision deficits as a result of heavy metal poisoning.

PMID: 314667, UI: 80014468

K.Warfvinge et al, "mercury accumulation in the monkey eye after mercury vapour exposure". Toxicology, 1996, 107: 189-200.

mercury from vapor exposure accumulates over time in the various parts of the eye.

Kishimoto T, Ohno M, Yamabe S, Tada M

Methylmercury Injury of Cultured Human Vascular Endothelial Cells

Journal of Trace Elements in Experimental Medicine. 6(4):155-162, 1993

Abstract The effect of methylmercury chloride (MeHg) on cultured human vascular endothelial (HVE) cells was investigated. Umbilical vein-derived HVE cells were collected by enzymatic digestion with collagenase. At concentrations of 0-50 mu M, MeHg had only barely detectable effects on cell viability. However, the viability of HVE cells decreased dose-dependently at concentrations ¿100 mu M.

Morphologic examination by phase-contrast microscopy revealed a markedly damaging effect of MeHg at concentrations exceeding 500 mu M. The cytotoxic effect of MeHg on DNA synthesis was also concentration-dependent. These results suggest that HVE cells are susceptible to concentration-dependent MeHg cytotoxicity and that MeHg could induce vascular endothelial injury, which may be involved in the pathogenesis of arteriosclerosis. (C) 1993 Wiley-Liss, Inc. [References: 34]

I have seen several abstracts related to eye degeneration and mercury, due to my friends father's eye degeneration, I researched abstracts for my friend to give to his father. I do not have any of them handy yet anymore, but I found 1-2 from Sam Queen's Chronic Mercury Toxicity book (see anotated bibliog.), and I found quite a few more when I researched glutathione and lipoic acid, in addition to mercury, and it has been proven that mercury depletes glutathione and lipoate by binding to the thiols in these 2 as well as inhibiting various important glutathione system enzymes.

I found the abstracts from Medline, additionally, some where in the Life Extension Foundation Magazine abstract sections, and the same can be found also from Medline. I am sorry that these days I do not have enough time to go fetch the abstracts for you. But, what I found from tbe abstracts, lipoate, NAC, GSH, E-vitamin and C-vitamin have the greatest potential to be used as a protective treatment against mercury also in all eye disease that are due to oxidative damage and thiol-binding by mercury.

Please go to Medline at www.medscape.com, join them free, and then seach with "mercury" and "macular" or "lipoate" and "macular" or "glutathione" and "macular" and cross all the years down to 1980 at least, and you will fork many abstracst to read.

Hope this helps, Ray

From: "Amy L. Riskedahl, O.D." ¡ariskedahl@seanet.com¿

Subject: Re: Vision

Cataracts and macular degeneration are the two eye diseases most often attributed to oxidative damage. Cataracts can be taken out surgically but macular degeneration often can't be helped. The progression can be slowed with large amounts of certain anti-oxidants such as lutein.

The macula is the part of the eye that has the highest number of cones and thus the sharpest vision and the best color vision. Mercury and other things that cause oxidative damage can damge the macula over time.

Generally what you do to heal the body, heals the eye. Get rid of the mercury. Vit. C, Vit. E, essential fatty acids, proanthocyanadins and several of the carotenoids are things that have been proven to heal the eye. Keep the cardiovascular system healthy. The retinal artery is a branch off of the carotid artery so plaques thrown off the carotids can cause strokes in the eye just as they do in the brain.

Cavalleri A, Gobba F. Reversible color vision loss in occupational exposure to metallic mercury. Environ Res 1998 May; 77(2):173-7

Sezione di Medicina Preventiva dei Lavoratori, Universita di Pavia, Pavia, Italy.

Color vision was evaluated in twenty-one mercury exposed workers and referents matched for sex, age, tobacco smoking, and alcohol habits. The Lanthony 15 Hue desaturated panel (D-15 d) was applied. In the workers, mean urinary Hg (HgU) was 115+/-61.5 microg/g creatinine; in all but one the values exceeded the biological limit (BEI) proposed by the American Conference of Governmental Industrial Hygienists. A dose-related subclinical color vision impairment was observed in Hg-exposed workers compared to the referents. Just after the survey, working conditions were improved. Twelve months later the workers were reexamined. Mean HgU was 10.0 microg/g creatinine and in no subjects was the BEI exceeded. Color perception was significantly improved compared to the first examination and, furthermore, no differences were observed between exposed workers and referents. The results add evidence that the color vision loss observed during the first part of the study was related to Hg exposure and, moreover, show that this effect is reversible. These data indicate that metallic Hg can induce a reversible impairment in color perception. This suggests that color vision testing should be included in studies on the early effects of Hg. The possibility of applying the D-15 d as an early effect index in the biological monitoring of Hg exposed workers should also be entertained. Copyright 1998

I do not know of any direct science references tying mercury with Macular Degeneration. However, from indirect information there appears to be a connection.

Dr. Johnathon Wright and Alan Gaby have developed a nutritional protocol that is quite effective. It is interesting to note that the nutrients are all those that mercury reduces in the body. ie Taurine, Vit E, Selenium, and Zinc. I am sure most ACAM physicians would be happy to get the formula (if it isn't already included in their data) and administer it. It is given by IV, except the Vit E.

Sam Ziff

¡bpinfo@bioprobe.com¿

¡http://www.bioprobe.com/¿

Myasthenia Gravis

Quite common with mercury poisoned, relates to antibodies against acetylcholine receptors, mercury has major effects of impairing acetylcholine metabolism and neural transmission, and bindind to the receptors. Quite a few abstracts is available of that, so the linking of myastenia gravis to the changes mercury cause is not that great jump to make. I know numerous people that had myastenic symptoms, and continue to have from amalgams, I am one of those, and Freya on the list has similar, and it was quite frequent when I used to go to amalgam poisoned's meetings in the early part of the decade, we had meetings of up to 50 poisoned in Tampere Finland and even more in Helsinki, and discussed diagnosises and symptoms each had from amalgams, and myastenia was one fairly common in addition to MS/ALS, autoimmunities, thyroid, prostata, liver, kidney, fibromyalgia, chronic fatique and various other symptoms/ conditions resulting or contributed by the mercury and copper leaking from the amalgams en masse.

Ray Sarela

Washington University Medical School

Neuropothy http://www.neuro.wustl.edu/neuromuscular/nother/toxic.htm

(another source of eye mercury & eye problems)

Mercury is still used in eye makeup as a preservative. From the FDA site:

http://vm.cfsan.fda.gov/ dms/cos-hdb3.html

Mercury compounds (21 CFR 700.13).

The use of mercury compounds as cosmetic ingredients is limited to eye area cosmetics at concentrations not exceeding 65 parts per million (0.0065%) of mercury calculated as the metal (about 100 ppm or 0.01% phenylmercuric acetate or nitrate) and provided no other effective and safe preservative is available for use.

Mercury compounds are readily absorbed through the skin on topical application and have the tendency to accumulate in the body. They may cause allergic reactions, skin irritation, or neurotoxic manifestations.

List members may be interested to note that information on this subject was published in the American Journal of Clinical Pathology in 1973 (Vol. 59. 515-7) by my colleagues in the Department of Forensic Medicine, University of Glasgow, Scotland following research in Africa.

It is surprising that such a toxic metal should continue to be used in cosmetics.

Ian Dale

Occupational Hygienist

Glasgow Occupational Health

"Conjunctivitis sicca" or "dry eye study"

Conjunctivitis sicca (dry eyes) is a major health problem for about 4 million people in Germany.

The "dry eye study" with 36 patients has shown that people with heavy metals like amalgam or palladium used in their goldcrowns often have fungi in the large intestine and also food allergies. Patients which have been treated had very good results. Other visual problems (spectacles) have shown to be highly correlated with the number of amalgam fillings as well.

Marburg Amalgam Study. (there also is a published version)

From: Dr.B. Weber, Amalgam information Marburg.

http://home,t-online.de/home/Institut_f._Naturheilverfahren/patinf.htm

¡b.weber@FIREMAIL.DE¿

Subject: Information about treatment of 3000 patients with amalgam-problems in german and englisch

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Dr. D.A. Carroll, O.D. & Dr. B.C. Lane, Preventing mercury related cataracts.

www.medicalvisioncenter.com/prevention.html; & Alan Thal, MD, Cataract reversal through mercury detox, www.digitalnaturopath.com/treat/T33633.html; & Dr, Ben Lane, O.D., Methylmercury

in seafood contributes to cataract development, Medical World News, December 20, 1982; & Dr. Victoria Buntine, Mercury Effects, Healthinasia Incorporated, 2001, www.healthinasia.com/mercury.html, & Dr. G. E. Poesnecker, Its Only Natural, 2001, *www.oneflesh.org/only-22.html*[5];

Iritis, Inflammation of the Eye[6], (autoimmune condition, can be caused by mercury)

www.hfhut.com/iritis-inflammation-of-the-eye[7]

Syphilitic iritis after treatment with mercury common; likewise use of thimerosal eyedrops.

Photophobia often accompanies mercury poisoning, *www.causeof.org/sensitivity.htm#TreatIritis*[8]

Astaxanthin, a natural supplement has been documented to relieve eye fatigue and improve visual acuity and accommodative amplitude. Reduces inflammation. Life Extension Foundation Life Extension Jan 2009

Mercury - inorganic

Toxicity elemental metal usually airborne exposure often produces neural disease without systemic disorders ?Salts: GI absorption produce systemic & neural effects ?Organic mercurials: Methyl mercury Little peripheral nerve toxic toxicity Early paresthesias & ataxia related to CNS effects Converted from inorganic mercury by microorganisms then enters food chain Outbreaks of toxicity

Minimata Bay: Spillage of HgCl into sea ?Iraq: Ethyl Hg fungicide in grain used for baking bread ?Subacute: Metallic mercury vapor ?Neuropathy ?Motor ?Axonal ?Myokymia

Encephalopathy òOther: Mouth inflammation; GI; Fetid breath òChronic ò

CNS: Encephalopathy; Psychosis; Extrapyramidal; Ataxia òNeuropathy:

Sensory & Motor; Pain & paresthesias ò

Children: Acrodynia òEncephalopathy òAutonomic: Tachycardia; Hypertension;

Sweating on trunk òInsomnia; Weight loss; Constipation

òDiagnosis: 24 hour urinary excretion òInorganic toxicity only ò

Treatment: ?Chelation; Spironolactone

Optic Nerve & Eye

òAtaxias òFriedreich òMitochondrial - NARP Syndrome: (Neuropathy; Ataxia; Retinitis Pigmentosa) òPosterior column ataxia + Retinitis pigmentosa òHMSN VI òToxic òCarbon disulfide òDisulfiram òMercury (Hg) òNutrition: Cuban neuropathy òVernant's disease

3. Cerebellum

òA-beta-lipoproteinemia òAtaxia telangectasia òFriedreich Ataxia ò

Paraneoplastic: Hu; CV2 òInfantile Onset Spinocerebellar Ataxia (IOSCA) òMetachromatic Leukodystrophy òRefsum òSCA 2, 3, 4

4. Supratentorial òHereditary òCowchock Syndrome òFabry's òHexosaminidase A (Late Onset) òMulti-Infarct Dementia (CADASIL) òPorphyria òPrion protein (PrP27-30)

mutation: Glu200Lys òPolyglucosan body syndromes òFamilial ALS: Higher prevalence of dementia (15%) than sporadic ALS òInfections òHIV òLyme disease òRabies òSyphilis òInflammatory

[5]**Internet:** "http://www.oneflesh.org/only%1e22.html".
[6]**Internet:** "http://www.hfhut.com/iritis-inflammation-of-the-eye".
[7]**Internet:** "http://www.hfhut.com/iritis-inflammation-of-the-eye".
[8]**Internet:** "http://www.causeof.org/sensitivity.htm#TreatIritis".

& Immune òSarcoid òVasculitis òMetabolic òThyroid òVitamin B12 deficiency òHypophosphatemia òMitochondrial: òMELAS (Mitochondrial Encephalomyopathy; Lactic Acidosis; Stroke)

òMERRF (Myoclonic Epilepsy; Ragged Red Fibers

òMNGIE Syndrome (Myopathy and external ophthalmoplegia; Neuropathy; Gastro-Intestinal; Encephalopathy) òMotor neuron disorders with dementia

(Sporadic) òWestern Pacific ALS òFrontal Dementia followed by motor system disease òUpper motor neuron: Especially bulbar òLower motor neuron: Fasciculations; Less prominent weakness ò? Atypical Creutzfeld-Jacob syndromes òNeoplastic: Lymphoma (angiotropic large-cell); Carcinomatous meningitis òParaneoplastic (anti-Hu) òToxic: Alcohol; Anticholinergic; Arsenic; Lead; Mercury; Podophyllin; Thallium; Vacor

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