

## Ophthalmic Findings of Acute Mercury Poisoning in Primary School Students

Lokman Aslan<sup>1\*</sup>, Murat Aslankurt<sup>1</sup>, Cengiz Dilber<sup>2</sup>, Murat Özdemir<sup>1</sup>, Adnan Aksoy<sup>1</sup> and Tahir Dalkıran<sup>2</sup>

<sup>1</sup>Department of Ophthalmology, KSU Faculty of Medicine, Kahramanmaraş, Turkey

<sup>2</sup>Department of Pediatric Neurology, KSU Faculty of Medicine, Kahramanmaraş, Turkey

### Abstract

**Purpose:** To report ophthalmic findings in acute mercury poisoning in the primary school students.

**Methods:** Seventy two children exposed to mercury vapor and 42 healthy controls were enrolled in the study. Full ophthalmologic examination including best corrected visual acuity, external eye examination, a slit-lamp examination, funduscopy, intraocular pressure measurements, Visual Field (VF), Visual Evoked Potential (VEP) and Color Vision (CV) tests were performed at the presentation and after six months. The parametric values of VF, Mean Deviation (MD) and Pattern Standard Deviation (PSD) were compared between groups.

**Results:** The visual acuity less than two lines in ETDRS chart in 7(9.7%) patients, color vision impairment in 6(8.3%) patients were determined with ophthalmic examination. There were a significant difference in the color confusion index of patients, ( $p<0.05$ ). The mean parametric VF values of MD and PSD were found statistically, significant difference ( $p<0.001$ ,  $p<0,001$  respectively), The latency values of VEP were 7% of 20 patients over 100 ms. There were no correlation between ophthalmic findings and mercury levels in urine and blood.

**Conclusion:** While visual acuity minimally affected, advanced visual functions were significantly impaired in a way independent of mercury level. The goal of this paper is to draw attention to the importance of public education on potentially hazardous effects of mercury in terms of preventive community health. In particular, both primary school teachers and students should be trained concerning poisonous gases such as mercury.

**Keywords:** Mercury poisoning; Ophthalmic finding; Children

### Introduction

Pediatric population is at the highest risk for the hazardous effects of elemental mercury poisoning [1]. They attain much higher body concentration of mercury than adults in the same exposure. Mercury vapor tends to settle near the floor for heavier than air and children have higher minute volume respiration per unit. Therefore, they inhale higher quantity of vapor and more air [1-4]. Also the blood-brain barrier of children is less able to keep mercury out of the brain, and their nervous system is developing. Metallic mercury is rapidly absorbed via inhalation or through the skin, but poorly absorbed after oral ingestion. The respiratory system is the main absorption of the mercury vapor [5,6]. Inhaled vapor is absorbed up to 80% by the lung into the bloodstream, and it needs to be demethylated to elemental form to pass into brain, retina and vitreous [1-3].

The visual system has been shown that is susceptible to the toxic effect of mercury ions [6,7]. Its damage to the eye results from direct accidental, or occupational exposure and systemic uptake of mercury and their action on the retina and optic nerve [7,8]. In vivo animal studies have demonstrated the presence of mercury deposits in the retina and vitreous [9-14]. Retinal Pigment Epithelium (RPE) likely plays a role in the ocular toxicity associated with mercury exposure in that it mediates transport of substances to the photoreceptor cells [11,12]. The mercury deposits are prevalent in the photoreceptor layer, in the inner and outer nuclear layers, vessel walls, a lesser extent in plexiform layers and the ganglion cell layer [9,12]. Visual symptoms of mercury exposure are decreased visual acuity, constriction of visual field and color vision impairment [2,6,15]. One of the earliest signs of mercury poisoning is a disturbance of night (scotopic) vision [4,9]. Although, the chronic effects of mercury exposure on the visual system are well known in the literature, there is not enough information of acute mercury poisoning regarding human visual system. We aimed

to report the ocular manifestations of acute mercury poisoning in a pediatric population in the present study.

### Methods

#### Patients

The study was approved by local ethics committee and conducted in accordance with the ethical principles described by Declaration of Helsinki. Informed consent was obtained from the parents of participants.

Primary school students had taken liquid mercury, which is held for experimental purpose in the school laboratory from there without any permission, and then taken it to their home where mercury had vaporized on the stove as a fun. The spilled mercury vapor in their living area had caused to acute mercury poisoning. Thus, 48 adults of family members also affected. Seventy two children exposed to mercury vapor were examined ophthalmologically. The acute mercury poisoning was diagnosed at the Emergency Department (ED) and Pediatric Neurology department (PN) with clinical examination and laboratory analysis. The children who contain mercury level more than 10  $\mu\text{g/L}$  (normal

**\*Corresponding author:** Lokman Aslan, Department of Ophthalmology, KSU Faculty of Medicine, Kahramanmaraş, Turkey, 46050 Kahramanmaraş, Turkey, Tel: +905326069808; E-mail: [lokasian46@yahoo.com](mailto:lokasian46@yahoo.com)

**Received** October 06, 2012; **Accepted** November 19, 2012; **Published** November 21, 2012

**Citation:** Aslan L, Aslankurt M, Dilber C, Özdemir M, Aksoy A, et al. (2012) Ophthalmic Findings of Acute Mercury Poisoning in Primary School Students. J Clin Toxicol S1:010. doi:10.4172/2161-0495.S1-010

**Copyright:** © 2012 Aslan L, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

range: 0-10 µg/dL) in their blood and/or 15 µg/L (normal range 0-15) in the urine were hospitalized and referred to ophthalmology clinic for ophthalmic examination. The mean serum creatinine level of children was found 0.45 mg/dL at first examination and 0.46 mg/dL at final visit. The kidney and liver function tests of all children was normal during six months follow up period. From now on, we call them as patient.

The ophthalmologic examination of patients was carried out on the day after accepted PN exposure and after six months later. Full ophthalmologic examination including Best Corrected Visual Acuity (BCVA), external eye examination, pupillary light response evaluation, a slit-lamp examination, intraocular pressure measurement, funduscopy were performed on the each patient. The auto refractor measurements were done in all of them, and refractive errors were corrected according to auto refractometer measurement results. BCVA was measured with the standard ETDRS chart. Intraocular Pressure (IOP) was measured with hand held device (tonopen) under topical anesthesia in all participants. Note that, those who had previous ophthalmic diseases leading to visual impairment were excluded from the study.

Visual field test was performed in 70 patients, and 42 age, sex matched control group who did not have any ophthalmic pathology leading to visual impairment at ophthalmologic examination. Visual Field (VF) assessments were made using Humphrey visual field analyzer (Carl Zeiss, Meditec Dublin CA). Central 24-2 SITA fast strategy protocol was used for all participants and right eyes data were used for statistical analysis. The tests with low (<20) false positive, false negative and fixation loss parameters were recorded for interpretation. The right eyes of 57(81%) patients and 35(83%) controls were found to be appropriate for evaluation. The mean parametric values of Mean Deviation (MD) and Pattern Standard Deviation (PSD) were compared between groups.

Color discrimination was evaluated with Anthony 15-D test. Color vision measurement was performed monocularly, closing each eye alternately. The participants with glasses kept them during the examination. The color confusion index value of Anthony 15-D test was used for statistical analysis. Five out of subjects and two in controls who had a family history or were aware of color-blindness was excluded.

Visual Evoked Potential (VEP) test was done in randomly selected

20 of patients. All measurements were performed by an experienced electrophysiology technician under the same conditions. Pattern VEP was conducted by applying the visual stimulus alternately to both eyes. Test measurements were repeated at the least three times until we received the best results. The parametric values of amplitude (P1-N2) and latency (P100) of right eyes were evaluated.

### Statistical analysis

The data were presented as the mean ± standard deviation for continuous variables and frequencies (in percents) for the categorical variables. The differences between groups were compared with Independent Student's t Test for continuous variables. Chi-square Test was used for the categorical variables. Correlation analyses were performed with Pearson correlation test. A probability of p<0.05 was considered statistically significant. The statistical analysis was performed using SPSS package program for Windows (version 16.0, Chicago, IL, USA).

### Results

The study group consisted of 72 children of whom 35 boys and 37 girls and the average age was 10.45 ± 2.9 years. Control group consisted of 35 (17 boys, 18 girls) healthy school children, the mean age was 10.82 ± 2.36 years.

The average mercury level in subjects was found to be 8.3 ± 7.3 (0.6-37.9) µg/L in blood and 318.9 ± 855 (2.4-4508) µg/L in urine at the time of the first ophthalmological examination. The blood and urine mercury levels of all subjects were found to be below the toxic level in the latest ophthalmologic examination. The mercury levels in controls were found in the normal range, for blood (0-10 mg/dL) and urine (0-15 mg/dL).

Clinical evidence was detected in 13 patients and it was summarized in table 1. The decreased visual acuity was detected in 13(18%) of patients. Six out of 13 patients had prior ophthalmic pathology leading decreased visual acuity and were excluded from the study. The rest of seven (9.7%) patients with decreased visual acuity had no history of previously existing ophthalmic disease. Three of them had some complains such as blurred vision, irritation, but others did not have. The visual acuity was found to be decreased two lines

Age	Sex	Laboratory 1*		Laboratory 2**		Laboratory 3***		Ophthalmologic Findings****					Clinical Signs
		Blood	Urine	Blood	Urine	Blood	Urine	CV	VA	IOP	MD	PSD	
7	B	15,6	27,3	9,2	87,4	0,9	1,7	N	0.8	17	-1.74	1.37	Cough, arthralgia
9	G	28,1	36,4	7,2	145,4	1,4	5,4	N	1.0	14	NA	NA	Nausea, arthralgia
11	B	34,9	44,3	30,4	3873	3,4	9,5	N	1.0	13	-3.75	1.95	Rash diarrhea
11	G	163,5	43,5	7,7	174	1,1	1,9	N	1.0	11	-2.26	1.65	Nausea
14	B	6,7	37,9	3,6	10,7	1,3	6,5	N	1.0	14	-2.01	1.55	Rash, headache
9	G	119	280	37,9	4508	8,9	333,5	P	0.8	13	-5.02	3.7	Sore throat, fever, cough
12	G	107	215	15,5	1919	9,5	184,6	N	1.0	12	-4.57	3.57	Rash, sore throat
11	G	11,6	49	1,9	21,5	1,6	5,9	N	1.0	16	-3.43	2.17	Rash
14	B	28,9	7,4	2,8	106,4	2,3	6,2	N	1.0	13	-1.36	2.35	Rash
15	G	10,4	12,3	6,1	42,1	2,3	7,3	N	0.8	19	-2.66	1.65	Rash, fever
9	B	24,2	13,4	16	2366	4,4	33,6	N	1.0	16	-2.87	2.29	Rash
7	B	3,6	15,3	1,7	28,8	5,7	38,2	P	0.8	17	-2.5	1.38	Rash
10	G	12,8	25,2	7,2	94,5	1,5	10,4	N	1.0	15	-4.11	2.01	Rash

\* At the presentation \*\* One week later \*\*\* One month later, \*\*\*\* Right eye

Abbreviations: B: Boy; G: Girl; CV: Color vision; VA: Visual acuity; IOP: Intraocular pressure; MD: Mean deviation of visual field test; PSD: Pattern standard deviation of visual field test

Table 1: Laboratory and ophthalmic examination findings in patients with clinical evidence.

with ETDRS chart in these patients, and mean visual acuity was not changed along six months period. There was no correlation between the BCVA and the level of blood/urine mercury ( $r=0.143$ ,  $p=456/r=0.128$ ,  $p=486$  respectively). Ocular findings of right eyes in patients at the presentation and six months period were summarized in table 2. At the first examination, mid-dilated pupil was seen and reduction of the light reaction in 5(6.9%) patients, but this finding was disappeared at the later visits.

The mean IOP of patients was  $15.21 \pm 2.46$  mmHg in the right eyes and  $16.09 \pm 2.20$  mmHg in the left eyes. That of the control group was found to be  $14.49 \pm 2.34$  mmHg in the right eye and  $15.03 \pm 2.38$  mmHg in the left eye. There were not statistically different between groups ( $p=0.745$ ). There were no pathologically high IOP levels at any time. Fundus examination revealed normal optic nerve, macula and retinal vasculature. Weakness of the light reaction was found in 5(6.9%) patients at the first examination which disappeared in the later controls.

The mean values of MD were  $-3.66 \pm 1.8$  in the study group and  $-2.21 \pm 1.18$  in controls and were found to be statistically significant, ( $P<0.001$ ). The mean values of PSD were  $2.60 \pm 1.39$  in the study group and  $1.62 \pm 0.42$  in controls and found to be statistically significant ( $p<0.001$ ). The parametric values of visual field test were summarized in table 3. There was no correlation between the mean MD value and the level of blood/urine mercury ( $r=0.052$ ,  $p=0.852/r = -0.136$ ,  $p=0.556$  respectively), and the mean PSD value and the level of blood/urine mercury ( $r = -0.117$ ,  $p=0.614 / r = -0.11$ ,  $p=0.961$  respectively).

Color vision was assessed with Anthony 15-D test. Color vision impairment in 6(8.3%) patients was determined with this test. Response to light was normal in case of all six patients. VA was 0.8 in one patient. VF was abnormal in three of them. The color confusion indexes of patients compared to controls, there was statistically significant difference ( $p<0.05$ ). They had failed to distinguish between blue-yellow tints. There was no correlation between color impairment and blood-urine mercury levels (respectively  $r=0.257$ ,  $p=486/r=0.328$ ,  $p=532$ ).

The VEP test was evaluated as parametric values of amplitude (P1-N2) and latency (P100). VEP latency was found over 100 ms in 7% of 20 patients who were tested. There was no correlation between the VEP amplitude and the level of blood/urine mercury and ( $r=427$ ,  $p=0.053/r=0.266$ ,  $p=0.244$  respectively), and the latency and the level of blood/urine mercury ( $r=0.525$ ,  $p=0.052$  and  $r=0.334$ ,  $p=0.138$  respectively).

	At the presentation	Six months later
<b>Reduction of Visual Activity</b>	7(9.7%)	7(9.7%)
<b>Intraocular Pressure</b>	$15.21 \pm 2.46$	$14.13 \pm 2.26$
<b>Color Confusion Index</b>	$1.35 \pm 1.03$	$1.41 \pm 0.86$
<b>Mean Deviation</b>	$-3.66 \pm 1.8$	$-3.75 \pm 1.67$
<b>Pattern Standard Deviation</b>	$2.60 \pm 1.39$	$2.65 \pm 1.46$
<b>Reduction of light response</b>	5(6.9%)	0(0%)

Table 2: Ocular findings of right eyes in patients at six months period.

	Minimum	Maximum	Mean	P
<b>MD*</b>				
Study group (n=57)	-5.40	-0.79	$-3.66 \pm 1.8$	
Control group (n=35)	-3.12	-0.13	$-2.12 \pm 1.18$	$P<0.001$
<b>PSD**</b>				
Study group (n=57)	1.34	4.66	$2.60 \pm 1.39$	
Control group (n=35)	1.20	2.75	$1.62 \pm 0.42$	$P<0.001$

\* Mean deviation, \*\* Pattern standard deviation.

Table 3: Parametric values of visual field analysis in the groups.

## Discussion

Acute mercury vapor poisoning is a prominent topic for public health. It is rare but may result in a devastating damage in the human body [1]. The children are very susceptible to mercury intoxication during the developmental period. Mercury is a metal odorless, colorless and attractive, so children like to play with it and they are often fascinated by the sight of spilled elemental mercury [2-4]. Mercury and its compounds are widespread in nature and can be obtained easily by children [16]. Zeitz et al. [17] reported that children playing with mercury caused 46% of reported evaporated mercury spills in elementary and secondary schools. Seventy two children exposed to acute mercury vapor became subject of this study. The following reasons such as to use stove, to live in closed environment, to be unaware of mercury's hazardous effect on the human body and to live in crowded family has played a pivotal role in mercury poisoning. Especially some families had significant symptom and higher mercury level in laboratory analysis. Five of them were accepted to pediatric neurology department because of some complains such as headache, ataxia, slurred speech, arthralgia, abdominal pain, skin rash and movement restriction. Initially mercury poisoning was diagnosed with detailed history and clinical findings, and diagnosis was supported with laboratory analysis. Then laboratory analysis was performed for all students and their family members, suspected mercury poisoning.

Ideally neither children nor adults should have any mercury in their bodies since, it does not provide physiological benefit [2,3]. Mercury entering the body is carried to target tissues such as kidney, lung, heart, brain and eye through the bloodstream [3,5]. The mercury concentration in the tissues and protein-bound ions is crucial for the formation of toxic effects [1,18,19]. Mercury has an extremely short half life in the blood, but a relatively long half time of approximately two months in the body. The laboratory analysis of blood and urine sample can be used to assess an acute poisoning [2]. Blood samples within first three days after the acute exposure are useful primarily in short-term, higher-level exposures. If exposure is prevented, the toxic level in the blood can return to normal within a few days. However, because the mercury released from tissues is eliminated through urine excretion, toxic level in the urine can resume for several weeks [3,4]. In this study, the blood level of mercury fell under toxic level within a few days, but the urine level initially increased within a few days, then decreased after several weeks. The blood and urine mercury levels were found normal range at the end of the six-month period.

Despite high laboratory levels, the majority of patients were subclinical or without significant clinical evidence. Possible extremely toxic effect of mercury was prevented due to early detection of poisoning and those who exposed were removed from contaminated area. In a study from Iraq in 1972 regarding acute exposure was reported that mercury poisoning resulted in serious toxic effects such as complete visual loss in some of the patients [20]. The family members living at the same environment and those exposed to the same dose has determined different clinical variation in the mentioned study. Even though one in the family members was blind, sister or brother had no detectable any visual disturbance. Moreover, there was no correlation between clinical findings and laboratory level of mercury in chronic exposure [21-23]. Patients mainly remain subclinical in the event of such exposure, but the hazardous of mercury can be emerged in the health screenings. In a case report, mercury has been defined during etiological investigation, through consumed food [24]. In the present study despite the fact that family members had some clinical symptoms such as skin rash, ataxia, restriction of movement and high laboratory

results, their ophthalmic findings showed no correlation with laboratory analysis. As well as exposure time and the amount of mercury taken from the body, individual susceptibility has also a significant influence on the visual system in the mercury toxicity.

In this study, although seven patients who had no any previous ophthalmic disease lead to visual impairment, they had decreased visual acuity mean two lines in ETDRS chart. As visual acuity is previously unknown, the relationship between minimal decreased visual acuity and mercury poisoning is not quite clear. There was not found a correlation between the central visual impairment and laboratory analysis.

The color confusion index of Anthony 15-D test was found different as being compared to the control. Congenital color blindness usually affects men and the red-green color band [25-27]. The previous studies regarding color vision has been associated with predominantly male sex. The advantage of the present study had a gender distribution close to each other. However, we did not found any difference between the sexes. Moreover, there was no difference between right and left eyes and color impairment and blood and urine levels of mercury. Acquired dyschromatopsia has been reported mostly in the chronically exposed subjects to mercury. Although, a number of studies about mercury poisoning have been found in the loss of blue yellow color discrimination, some studies have been described altered red-green discrimination. Both chromatic systems, blue-yellow and red-green can be affected and color vision impairment remains irreversible in the long term period follow up [25-27]. Jedrejko et al. [27] showed qualitative changes, which are borderline corresponding to the early stage of developing dyschromatopsia type III in the men employed in a chloralkali plant. They reported that the right eye was more affected, level of urinary mercury and duration of exposure were not found to have any correlation between color confusion indexes.

Pattern VEP test was performed on 20 patients and latency was found over 100 ms in 7% of them. Although, latency and amplitude values showed no correlation with laboratory blood and urine levels of mercury, there was not any compliance with clinical findings. To make VEP test in children is more difficult than in adult and should be repeatedly tested for the reliability of the results. As abnormal test results can be associated with retina and the optic disc defect, it may also be related to visual cortex abnormalities [28].

Visual field test shows the damage of retinal ganglion cells and of visual pathways, which is from the optic disc to the occipital cortex. Even if the central vision is not affected it can give information about the peripheral vision. While visual field test is an indispensable diagnostic tool for glaucoma and optic nerve disease, it is also used in the diagnosis of drug or chemical intoxications [28,29]. However, the test is subjective and may be affected by patient compliance. Moreover, test outcomes are based on the probability as related to the presence of damage. The children with mercury poisoning in each parameter of visual field test were found statistically significant differences, compared with healthy controls. In addition to that the visual field loss not only associated with the retina and optic nerve damage, but also it may be seen in the nervous system defects [28-30]. Korogi et al. [31] reported that the range of visual field might correlate with the degree and extent of atrophy in striate cortex on MR images.

As a result, ophthalmic manifestations including central vision, color vision, visual field and VEP tests were found slightly affected in the present cases. These findings were not correlated to urine and blood mercury levels. The ophthalmic findings based on subjective data are a disadvantage in this study, but we found statistically significant differences than controls. The liquid mercury is readily available in the

natural environment. Its potential toxic effects on visual systems are not known by the public, particularly children. The first step in case of acute mercury poisoning, those exposed to mercury should be removed from contaminated environment. The second step is an education regarding hazardous effects of mercury on the human body. Informing of the children about this issue should be addressed as a significant public health task.

## References

1. Ozuah PO (2000) Mercury poisoning. *Curr Probl Pediatr* 30: 91-99.
2. Bose-O'Reilly S, McCarty KM, Steckling N, Lettmeier B (2010) Mercury exposure and children's health. *Curr Probl Pediatr Adolesc Health Care* 40: 186-215.
3. Bernhoft RA (2012) Mercury toxicity and treatment: a review of the literature. *J Environ Public Health* 2012: 460508.
4. Devlin EW (2006) Acute toxicity, uptake and histopathology of aqueous methyl mercury to fathead minnow embryos. *Ecotoxicology* 15: 97-110.
5. Mahajan VK, Sharma NL (2011) Metallic mercury vapour poisoning revisited. *Australas J Dermatol* 52: e5-e7.
6. Lansdown AB (2011) Metal ions affecting the skin and eyes. *Met Ions Life Sci* 8: 187-246.
7. Gabal MS, Raslan OA (1995) Ocular disorders among workers exposed to mercury. *J Egypt Public Health Assoc* 70: 1-14.
8. Dasari S, Yuan Y (2009) Low level postnatal methylmercury exposure in vivo alters developmental forms of short-term synaptic plasticity in the visual cortex of rat. *Toxicol Appl Pharmacol* 240: 412-422.
9. Mela M, Cambier S, Mesmer-Dudons N, Legeay A, Grötzner SR, et al. (2010) Methylmercury localization in Danio rerio retina after trophic and subchronic exposure: a basis for neurotoxicology. *Neurotoxicology* 31: 448-453.
10. Warfvinge K, Bruun A (2000) Mercury distribution in the squirrel monkey retina after in Utero exposure to mercury vapor. *Environ Res* 83: 102-109.
11. Bridges CC, Battle JR, Zalups RK (2007) Transport of thiol-conjugates of inorganic mercury in human retinal pigment epithelial cells. *Toxicol Appl Pharmacol* 221: 251-260.
12. Toimela TA, Tähti H (2001) Effects of mercuric chloride exposure on the glutamate uptake by cultured retinal pigment epithelial cells. *Toxicol In Vitro* 15: 7-12.
13. Tanan CL, Ventura DF, de Souza JM, Grotzner SR, Mela M, et al. (2006) Effects of mercury intoxication on the response of horizontal cells of the retina of thraira fish (*Hoplias malabaricus*). *Braz J Med Biol Res* 39: 987-995.
14. Korbas M, Krone PH, Pickering IJ, George GN (2010) Dynamic accumulation and redistribution of methylmercury in the lens of developing zebrafish embryos and larvae. *J Biol Inorg Chem* 15: 1137-1145.
15. Weber DN, Connaughton VP, Dellinger JA, Klemer D, Udvadia A, et al. (2008) Selenomethionine reduces visual deficits due to developmental methylmercury exposures. *Physiol Behav* 93: 250-260.
16. Baughman TA (2006) Elemental Mercury Spills. *Environ Health Perspect* 114: 147-152.
17. Zeitz P, Orr MF, Kaye WE (2002) Public health consequences of mercury spills: Hazardous Substances Emergency Events Surveillance system, 1993-1998. *Environ Health Perspect* 110: 129-132.
18. Sarikaya S, Karcioğlu O, Ay D, Cetin A, Aktas C, et al. (2010) Acute mercury poisoning: a case report. *BMC Emerg Med* 10: 7.
19. Alhamad T, Rooney J, Nwosu A, Maccombs J, Kim YS, et al. (2012) Lessons learned from a fatal case of mercury intoxication. *Int Urol Nephrol* 44: 647-651.
20. Sabelaish S, Hilmi G (1976) Ocular manifestations of mercury poisoning. *Bull World Health Organ* 53: 83-86.
21. Satoh H (2000) Occupational and environmental toxicology of mercury and its compounds. *Ind Health* 38: 153-164.
22. Grandjean P, Herz KT (2011) Methylmercury and brain development: imprecision and underestimation of developmental neurotoxicity in humans. *Mt Sinai J Med* 78: 107-118.

23. Beuter A, Edwards R (2004) Effect of chronic exposure to methylmercury on eye movements in Cree subjects. *Int Arch Occup Environ Health* 77: 97-107.
24. Remø SC, Olsvik PA, Torstensen BE, Amlund H, Breck O, et al. (2011) Susceptibility of Atlantic salmon lenses to hydrogen peroxide oxidation ex vivo after being fed diets with vegetable oil and methylmercury. *Exp Eye Res*. 92: 414-24.
25. Feitosa-Santana C, Barboni MT, Oiwa NN, Paramei GV, Simões AL, et al. (2008) Irreversible color vision losses in patients with chronic mercury vapor intoxication. *Vis Neurosci* 25: 487-491.
26. Feitosa-Santana C, Costa MF, Lago M, Ventura DF (2007) Long-term loss of color vision after exposure to mercury vapor. *Braz J Med Biol Res* 40: 409-414.
27. Jedrejko M, Skoczynska A (2011) Color vision impairment in workers exposed to mercury vapor. *Med Pr* 62: 227-235.
28. da Costa GM, dos Anjos LM, Souza GS, Gomes BD, Saito CA, et al. (2008) Mercury toxicity in Amazon gold miners: visual dysfunction assessed by retinal and cortical electrophysiology. *Environ Res* 107: 98-107.
29. Herba E, Pojda-Wilczek D, Plech AR, Pojda SM, Szkilnik R (2004) Influence of dopamine on flash visual evoked potentials (FVEP) in prenatally mercury intoxicated rats. *Pol J Pharmacol* 56: 415-419.
30. Roels H, Lauwerys R, Buchet JP, Bernard A, Barthels A, et al. (1982) Comparison of renal function and psychomotor performance in workers exposed to elemental mercury. *Int Arch Occup Environ Health* 50: 77-93.
31. Korogi Y, Takahashi M, Hirai T, Ikushima I, Kitajima M, et al. (1997) Representation of the visual field in the striate cortex: comparison of MR findings with visual field deficits in organic mercury poisoning (Minamata disease). *AJNR Am J Neuroradiol* 18: 1127-1130.

This article was originally published in a special issue, **Epidemiology of Poisoning** handled by Editor(s). Dr. John F Gamble, Consultant, Somerset, New Jersey, USA; Dr. Monath Sanjaya Kuruppu, Monash University, Australia