Editorial

iMedPub Journals
www.imedpub.com

Journal of Headache & Pain Management
ISSN 2472-1913

2017
Vol. 2 No. 3:13

DOI: 10.4172/2472-1913.100043

Lead: An Environmental Neurotoxic Agent

JH Lange* and AV Condello III

Envirosafe Training and Consultants, Pittsburgh, USA

Received: November 07, 2017; **Accepted:** November 10, 2017; **Published:** November 13, 2017

***Corresponding author:**
JH Lange

jhlange1@hotmail.com

Envirosafe Training and Consultants, 2366 Golden Mile Highway, Pittsburgh, USA

Tel: 412 908-9274

Citation: Lang JH, Condello III AV (2017) Lead: An Environmental Neurotoxic Agent. J Headache Pain Manag Vol. 2 No.3:13

The metal lead (Pb) has been well established as a toxic agent to man, other organisms and the environment [1,2]. There is no essential biochemical or physiological function for this metal. Hazards of lead have been recognized for thousands of years with physiological ailments being reported since antiquity. Most risks have been associated with high levels of exposure and the industrial environment. This concept of hazards from lead has, in part, continued today where the current Occupational Safety and Health Administration permissible exposure level has remained the same for decades; although, current research has shown dramatic health effects from low exposure levels [1]. From a public health prospective, lead is a legacy pollutant that arose from leaded gasoline, lead-based paint, lead pipes/solder and industrial activities. Lead can impact a large number of organ systems including the renal, blood, nervous, reproductive, and has been identified as an ototoxic substance [3,4]. Occupational exposure levels have dramatically declined over the last 20 years [5], which has in some circles reduced concern for this metal. Current events, such as observed in Flint, Michigan and other locations, have resulted in the re-emergence of environmental concerns for lead [6]. Lead can substitute for other metals that are considered to be essential including bivalent cations (Zn^{2+}, Ca^{2+}, Mg^{2+}, Fe^{2+}) and monovalent cations (Na^{+}) [7,8]. It has been suggested that the site of action for lead appears to be associated with locations of zinc and calcium on proteins [9]. Thus, cation interaction is an important mechanism for many of the molecular pathways involving this metal [10]. Most discuss calcium in association with lead, which appears to not be predominant as a physiological mechanism (e.g. bone) related to toxicity at low levels.

Neurotoxicity as a result of lead has been recognized for decades, but a multitude of recent investigations are reporting health effects at very low levels [5,7,11,12]. These investigations have begun focusing on impacts not easily evaluated and occurring at low levels with most associated with neurobehavioral issues [5]. Historically, lead has been reported to impact the central nervous system (CNS) in two ways-damage to the blood-brain barrier (BBB) and plasticity of the brain through blocking the N-methyl-D-aspartate receptor [5]. Classical toxicity from lead focused on impacts to organ systems with these occurrences observed at high blood concentrations (e.g. 20 to 30 ug/dl). Today, studies [13] have shown toxicity at levels less than 10 ug/dl (blood) which is the currently accepted level of safety (level of concern) according to the Center for Disease Control and Prevention (CDC). These detrimental effects from lead may even exist at 3 ug/dl, when evaluated against various neurobehavioral functions such as visual-motor disturbance criteria [14]. Some of these impacts may be a result of a combination of damage to the BBB along with inhibition of biochemical pathways. These combinations may be resulting in observed changes that contribute in changed social behavior and crime rates [9]. Investigations have suggested the CNS is more sensitive in children and fetuses while the peripheral nervous system (PNS) is more predominantly impacted in adults [7]. It has been suggested that the most critical time period of lead toxicity exists before the age of 2 years; although, toxic insult clear occur at other times [15].

Mechanisms of lead toxicity appear to be widely varied but have been suggested to include induction of oxidative stress, occurrence of apoptosis and interference with synaptic transmission [9]. Oxidative stress induced by lead is thought to result in formation of free radicals due to lipid peroxidation and can include generation of reactive oxidative species (ROS) [10]. This results in the depletion of antioxidants (superoxide dismutase, glutathione peroxidase and glutathione reductase) in the brain (e.g. hippocampus, cortex) [9]. Damage or dysfunction in these areas of the brain (e.g. hippocampus) could explain observed behavioral issues associated with lead (e.g. memory problems, conflict issues). This can also explain lead-related effects on IQ and neuropsychological performance outcomes. Reduced antioxidant activity can also explain observed damage in experimental animals extending the plausibility of molecular mechanisms. Oxidative stress is associated within mitochondria and damage to these cellular structures can result in the generation of secondary reactive products [11].

Physiologically, lead at low levels has been shown to impact numerous neurological functions, including the CNS and PNS [12]. The problem with physiological based evaluations is these indicators are not sensitive to neurobehavioral and cogitative endpoints. Many studies have shown various measurable impacts at levels below 10 ug/dl, even in the range of less than 5 ug/dl (2.9 ug/dl and 4.3 ug/dl) (e.g. abstract reasoning and attention defects) [8]. Exposure in the past may also carry long-term effects, especially involving high exposure, decades after the event [8]. Such exposures may be of even greater importance since lead has been observed to function as an endocrine disruptor [16,17]. These impacts have been shown to effect cortisol levels in fish. Although this association has not been directly associated with lead poisoning, there is at least one report of metals having a psychological effect on man, which based on ecological studies could be extended to lead [18,19]. Reports of lead in the ecological environment causing disruption of endocrine systems may be of major importance related to the recent lead exposure from water sources. It is likely these factors in impacting endocrine activities are in combination with oxidative stress mechanisms (e.g. ROS).

Seasonally has been also shown to impact lead levels with the highest value observed in the summer and early fall time period [20]. It has been postulated that soil suspension in air is the contributing factor [20]. Modeling studies have suggested the major contributor of blood lead in children is soil. When examining recent issues with lead in water, most regulatory factors do not include other contributors which are the major causes of increased blood lead. Thus, mitigation of water sources, such as seen in Flint, Michigan and other locations [21], will likely have limited practical impact on reducing blood lead levels over a protracted period of time. The real question will be what are the most economic remediation solutions and effective long-term measures? This must also coincide with the realities of economic costs of remediation.

References

1 Hernberg S (2000) Lead poisoning in a historical perspective. Am J Ind Med 38: 244-254.

2 Taylor MP, Forbes MK, Opeskin B, Nick Parr, Bruce PL (2016) The relationship between atmospheric lead emissions and aggressive crime: an ecological study. Environ Health 15: 23.

3 Lange JH, Condello AV (2017) Ototoxicity in the Occupational Environment: A Neurological Problem. J Headache Pain Manag 2: 9

4 Brochin R, Leone S, Phillips D, Shepard N, Zisa D, et al. (2008) The cellular effect of lead poisoning and its clinical picture. The Georgetown Undergraduate J Health Sci 5: 2

5 Feng C, Gangemi S, Alibrandi A, Costa C, Micali E (2016) Relationship between lead exposure and mild cognitive impairment. J Prev Med Hyg 57: E205-E210.

6 Laidlaw MA, Filippelli GM, Sadler RC, Gonzales CR, Ball AS, et al. (2016) Children's Blood Lead Seasonality in Flint, Michigan (USA), and Soil-Sourced Lead Hazard Risks. Int J Environ Res Public Health 13: 358.

7 Farhat AS, Khadem G (2015) Blood lead level and seizure: a narrative review. Reviews in Clinical Medicine 2: 84-87.

8 Lidsky TI, Schneider JS (2003) Lead neurotoxicity in children: basic mechanisms and clinical correlates. Brain 126: 5-19.

9 Hsiang J, Diaz E (2001) Lead and developmental neurotoxicity of the central nervous system. Current Neurotoxicity 2: 35-42.

10 Flora G, Gupta D, Tiwari A (2012) Toxicity of lead: a review with recent updates. Interdiscip Toxicol 5: 47-58.

11 Sanders T, Liu Y, Buchner V, Paul BT (2009) Neurotoxic effects and biomarkers of lead exposure: a review. Rev Environ Hlth 24: 15-45.

12 Mason LH, Harp JP, Han DY (2014) Pd neurotoxicity: neuropsychological effects of lead toxicity. BioMed Research International 14: 840547.

13 Olympio KPK, Goncalves C (2009) Neurotoxicity and aggressiveness triggered by low-level lead in children: a review. Pan Am Public Health 26: 266-275.

14 Chiodo LM, Jacobson SW, Jacobson JL (2004) Neurodevelopmental effects of postnatal lead exposure at very low levels. Neurotoxicol Teratol 26: 359-371.

15 Murta K, Iwata T, Dakeishi M, Karita K (2009) Lead toxicity: does the critical level of lead resulting in adverse effects differ between adults and children? J Occup Health 51: 1-12.

16 Łuszczek-Trojnar E, Drąg-Kozak E, Szczerbik P, Socha M, Popek W (2014) Effect of long-term dietary lead exposure on some maturation and reproductive parameters of a female Prussian carp (Carassius gibelio B). Environ Sci Pollut Res Int 21: 2465-2478.

17 Thang NQ, Huy BT, Van Tan L, Phuong NTK (2017) Lead and Arsenic Accumulation and Its Effects on Plasma Cortisol Levels in Oreochromis sp. Bull Environ Contam Toxicol 99: 187-193.

18 Butts CD, Bloom MS, Frye CA, Walf AA, Parsons PJ, et al. (2014) Urine cortisol concentration as a biomarker of stress is unrelated to IVF outcomes in women and men. J Assist Reprod Genet 31: 1647-1653.

19 Polat F, Akın Ş, Yıldırım A, Dal T (2016) The effects of point pollutants-originated heavy metals (lead, copper, iron, and cadmium) on fish living in Yeşilırmak River, Turkey. Toxicol Ind Health 32: 1438-1449.

20 Laidlaw MA, Filippelli GM, Sadler RC, Gonzales CR, Ball AS, et al. (2016). Children's Blood Lead Seasonality in Flint, Michigan (USA), and Soil-Sourced Lead Hazard Risks. Int J Environ Res Public Health 13: 358.

21 Bryant SD (2004) Lead-contaminated drinking waters in the public schools of Philadelphia. J Toxicol Clin Toxicol 42: 287-294.