

windows, construction, steel welding, casting bullets or fishing sinkers, glazed pottery or certain types of artistic paint, firing range instructors or cleaners and cable men.²⁹ Other concerns related to lead exposure are from foreign folk remedies, foods, supplements, cosmetics, jewelry or toys.²⁹ According to the American Association for Clinical Chemistry, blood lead levels should be under 10 mcg/dL in children and under 25 mcg/dL in adults.²⁹ Lead intoxication primarily affects the renal, central nervous, cardiovascular and hematopoietic systems.⁵

As previously mentioned, low levels of lead exposure can induce endothelial cell dysfunction and raise blood pressure. One observational study by Chaikittiporn et al measured blood lead levels in 444 male bus drivers in Bangkok. The subjects were categorized into three groups. The first group was categorized as the low exposure group with blood lead levels less than 4.1 mcg/dL. The second group had blood lead levels between 4.1-8.5 mcg/dL and the third group was considered the high exposure group with blood lead levels of 8.5 mcg/dL or more. The majority of the population being studied was in the medium exposure group. The mean lead exposure and blood pressure of the entire population was 6.3 mcg/dL and 130.9/81 mmHg respectively.²⁶

The low and high lead exposure groups were compared. The blood pressure of the low and high exposure groups were 127.5/78.1 mmHg and 136.3/84.9 mmHg respectively.²⁶ The average age, body mass index, smoking history and working years were taken into prospective. After analysis of these factors, the authors concluded that low levels of lead exposure were an independent risk factor for CVD.²⁶ However, the contributions of poor diet, sedentary lifestyle, smoking, alcohol consumption and age cannot be discounted as risk factors for CVD.

It should be noted that all of the individuals involved in this observational study had lead exposure levels well below the safety standards established within the United States for both children and adults. However, the results show that a majority of the subjects with lead exposure experienced blood pressures that was elevated above normal levels. Even the low exposure group, which had less than 4.1 mcg/dL of lead in their blood, still had an average blood pressure above normal. Therefore, prolonged exposure to low levels of lead appear to damage the vasculature and surrounding tissue increasing peripheral resistance and elevating blood pressure.

A study performed by Fiorim et al evaluated the effects of exposure to low levels of lead on blood pressure and endothelial cell function in rats. The rats were injected with 4 mcg/100 g and an additional 0.5 mcg/100 g of body weight daily for seven days and then sacrificed. After seven days of lead exposure, the rats had blood lead levels of 9.98 mcg/dL and a systolic blood pressure of 137 mmHg compared to the systolic blood pressure of the control group, which was 121 mmHg.³⁰ The investigators also found an increase in NO bioavailability and a higher level of angiotensin converting enzyme (ACE) activity in rats injected with lead.³⁰

A cascade of different hormones within the body regulate blood pressure. One hormone that is produced and stored in granular juxtaglomerular cells of the kidney is renin. Renin is secreted in response to a reduction in arterial blood pressure, a reduction in the amount of sodium chloride within the macula densa, stimulation of the sympathetic nervous system and elevated concentrations of prostaglandins during periods of tissue injury.³¹ Renin converts angiotensinogen, which is a hormone produced in the liver and stored in the proximal tubule of the cortex of the kidney, into angiotensin I by cleaving the leucine-valine bond.³¹

The next step of the process requires ACE. Vascular endothelial cells produce ACE for the regulation of blood pressure. ACE regulates blood pressure through two mechanisms. The first function is the conversion of angiotensin I into angiotensin II, which occurs adjacent to the vascular smooth muscle, by cleaving the histidine-leucine dipeptide bond.³¹ Angiotensin II acts as a potent vasoconstrictor. The second function is the degradation and subsequent inactivation of bradykinin, which is a vasodilating agent.

Although blood lead levels were under 10 mcg/dL, which is considered to be below the level of toxicity, an abrupt elevation in blood pressure was observed. Based on this information it seems possible that low levels of lead exposure accumulating over a short period of time to near toxic quantities alters blood pressure by stimulating the renin-angiotensin system due to damage to the vascular tissues resulting in the release of prostaglandins. The renin-angiotensin system promotes ACE activity increasing the concentration of angiotensin II. Higher concentrations of angiotensin II activate the sympathetic nervous system inducing vasoconstriction causing an increase in blood pressure.

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