

**Report of David Garabrant, MD, MPH
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Re: Perrine et al v DuPont et al

I am a licensed physician specializing in occupational medicine. I am board certified in both occupational medicine and internal medicine. I am qualified as a specialist in the fields of occupational medicine and internal medicine and in the field of epidemiology, especially as it relates to the study of diseases related to exposures to chemical agents. I am Professor of Occupational Medicine in the Department of Environmental Health Sciences and Professor of Epidemiology in the Department of Epidemiology, University of Michigan School of Public Health, and I also hold an appointment as Associate Professor of Emergency Medicine in the Department of Emergency Medicine, University of Michigan School of Medicine. I am engaged full-time in research in environmental and occupational epidemiology; teaching risk assessment, occupational and environmental diseases, and research methods in occupational and environmental epidemiology to graduate students in public health; teaching medical students; and in the clinical practice of occupational medicine.

I received my undergraduate degree with high honors in chemical engineering from Tufts University, Medford, Massachusetts in 1972. I received my M.D. degree from Tufts University School of Medicine, Boston, in 1976. From 1976-77, I served as an intern in internal medicine at Georgetown University Hospital in Washington, D.C. and from 1977-78, I served as a fellow in medical ambulatory care at that same institution. From 1978-80, I served as a resident in occupational medicine at the Harvard School of Public Health, Boston, and received the Master of Public Health degree in 1979 and the Master of Science in Physiology degree in 1980 from that institution. From 1980-81, I served as senior resident in internal medicine at University Hospital, Boston.

Upon completion of my training in 1981, I joined the faculty of the University of Southern California School of Medicine, where I was engaged in research in occupational cancer epidemiology and in the practice of occupational medicine until 1988, when I joined the faculty of the University of Michigan. While I have been on the faculty of the University of Michigan, I have served as Director of Occupational Medicine (1988-94), head of the Occupational Health Program (1992-95), Director of the Center for Occupational Health and Safety Engineering (1990-95), Director of the Occupational & Environmental Epidemiology program (2001-present), and Director of the Center for Risk Science and Communication (2003-present). My research has focused for the past 20 years on the long term health effects of chemicals on humans and I have published over 100 research articles, book chapters, and abstracts related to this area. A copy of my curriculum vitae is attached.

I have served on the Editorial Board of the Journal of Occupational Medicine and currently serve as a reviewer of scientific papers for a number of other journals, including Environmental Health Perspectives, Journal of the National Cancer Institute, Cancer Causes and Control, Cancer Epidemiology Biomarkers and Prevention, American Journal of Epidemiology, and Cancer Research. I was appointed to membership on the Safety and Occupational Health Study Section of the National Institutes of Health (NIH) in 1992 and served on that Study Section until 1996, having been appointed by Secretary of HHS Donna Shalala as Chair for the 1995-96 year. In that position I served as a peer-reviewer of research proposals for the NIH. I am currently a member of a number of professional organizations, including the Society for Epidemiologic Research, the International Epidemiological Association and the American College of Occupational and Environmental Medicine.

Materials reviewed

I have reviewed the following materials that your office forwarded to me regarding this case:

1. Anticipated health effects of the contamination of Spelter, WV and surrounding communities with arsenic, cadmium, and lead, and recommendations for medical monitoring. Charles Wertz, DO. March 30, 2007
2. Proposed Medical Monitoring due to the contamination of Spelter, WV and surrounding communities with arsenic, cadmium, and lead. Including estimations of participation rates and testing outcomes for economic purposes. March 30, 2007. Charles Wertz, DO.
3. Medical Surveillance Guidelines and Recommendations. James Kornberg, MD, ScD. November 11, 2005
4. Deposition and exhibits of Dr. Charles Wertz, April 12, 2007
5. Expert report of Kirk W. Brown, Ph.D., April 2, 2007

In addition, I have reviewed the medical and scientific literature relevant to the issues in this case (1-148)

Dr. Wertz alleges that plaintiffs have been exposed to hazardous levels of arsenic, cadmium, and lead from the former zinc smelter as a result of plant emissions, soil contamination, exposure to fugitive emissions from waste piles, contaminated household dust, and incidental soil and dust ingestion.

It is my understanding that Dr. Wertz relies on the opinions of Dr. Kornberg's initial medical monitoring recommendations from 11/11/2005 and his revised report from 3/3/2006. Dr. Wertz concurs with most of Dr. Kornberg's recommendations, but made numerous significant changes to the monitoring tests. Dr. Wertz also relies on the risk assessment and contour maps created by Dr. Brown to calculate minimum residency requirements to enter the medical monitoring program he has spelled out. Dr. Wertz concluded that "the residents in the area around Spelter have been exposed to arsenic, cadmium, and lead an extended time", and that "the residents in all areas have a significantly increased risk of developing disease based upon their residence and

exposure in the area”. He also claims there have been several studies documenting increased cancer risk around smelter sites where similar exposures have occurred.

Dr. Werntz proposes a medical monitoring program under the circumstances below:

- Only for diseases clearly associated with arsenic, cadmium, and lead.
- Testing is limited to diseases and medical tests clearly supported by the literature or general medical practice.

As the following discussion demonstrates, Dr. Werntz does not adhere to his own criteria for medical monitoring. The diseases that Dr. Werntz considers to be related to exposures from the smelter site are:

- Arsenic:
 - o Skin cancer
 - o Lung cancer
 - o Bladder cancer
 - o Kidney cancer
- Cadmium:
 - o Lung cancer
 - o Kidney cancer
 - o Decreased Renal Function
 - o Renal Failure
 - o Bone Fragility
- Lead:
 - o Plumbism (lead poisoning)
 - o Lung cancer
 - o Stomach cancer
 - o Kidney cancer
 - o Decreased Renal Function
 - o Renal Failure
 - o Bone Fragility
 - o Loss of Teeth
 - o Hypertension
 - o Increased Rates of Criminal Activity

My opinions regarding Dr. Werntz’s proposed cancer screening program

1. Dr. Werntz’s basis for claiming increased cancer risk around smelter sites is not reliable

Dr. Werntz relies on four studies (6, 18, 121, 137) in support of his claim that cancer risks are increased around smelter sites. The Tokudome study (137) has nothing to do with residents around a smelter site. It is a study of copper smelter workers in Japan, not residents in the area of the smelter. Furthermore, the study contradicts Dr. Werntz’s opinions regarding stomach cancer in relation to smelter exposures. The Tokudome study shows that smelter workers are at no increased risk of stomach cancer. The ATSDR Study of National Zinc Company in Bartlesville, Oklahoma (6) examines cancer risks in the vicinity of a zinc smelter and finds that the occurrence of lung and kidney cancer was

as expected from the state rates. There was no evidence that the residents in the area around the zinc smelter were at increased risk of cancer, contrary to Dr. Werntz's opinion. The Pershagen study (121, 122) is a study of arsenic exposure in the area of a copper smelter. There was no clearly increased risk of lung cancer in the community near the smelter among non-smokers and the authors concluded that no firm conclusions could be reached on the cause of the increased lung cancer risks in the exposed area (121). The Brown study (18) found increased risk of lung cancer associated with residence near a zinc smelter, but concluded that the limited size of the study precludes causal interpretation.

Furthermore, Dr. Werntz fails to cite studies that show no apparent excess of cancer risk in communities around smelters or in which soil is contaminated with arsenic, cadmium, or lead. ATSDR conducted a public health assessment of the Murray Smelter in Murray, Utah (5), found no apparent public health hazard, and concluded that health effects from arsenic, cadmium, and lead were unlikely due to limited exposures to these metals. A study of soil contaminated with arsenic (146) showed the skin cancer rates to be within the range for other locations in the US. A study of high environmental arsenic concentrations in soil and water of rural populations (60) in which there were mine tailings with known arsenic concentrations was conducted in Australia. The study found no increased risk of cancer of the lung, bladder, kidney, stomach, colon, or rectum. Frost (50) reported on lung cancer risks in relation to the ASARCO smelter in Ruston, Washington and found no evidence of increased lung cancer risk in the communities exposed to the smelter's arsenic emissions.

Overall, Dr. Werntz's conclusion appears to be based on an incomplete consideration of the scientific literature, selective review of favorable articles, and occasional mischaracterization of their contents.

2. Lead is not known to be carcinogenic and the medical monitoring proposed by Dr. Werntz related to lead exposure has no scientific foundation

Dr. Werntz fails to establish that lead is a carcinogen in humans or that the cancers for which he proposes medical monitoring are causally related to lead. Lead is not known to be a carcinogen in humans. The evidence regarding the carcinogenicity of lead to humans is not adequate to support a conclusion of causation. Dr. Werntz relies on the evaluation of the International Agency for Research on Cancer (IARC) for his opinion regarding the carcinogenicity of lead. IARC indicates "There is limited evidence in humans for the carcinogenicity of inorganic lead compounds" (72). The fact that IARC has classified inorganic lead compounds in category 2A means, by definition, that there is not sufficient evidence of carcinogenicity in humans to classify lead as being a human carcinogen (73). Similarly, the US EPA considers the available human evidence to be "inadequate to refute or demonstrate any potential carcinogenicity for humans from lead exposure." (46) Therefore it is unknown whether people exposed to lead are at increased risk of cancer. Consequently, there is no justification for medical monitoring for lung cancer, stomach cancer, kidney cancer, or any other cancer Dr. Werntz alleges is related to lead.

There is a large body of scientific evidence on the carcinogenicity of lead in human populations (2, 14, 33-39, 52, 80, 87, 93, 94, 98, 104, 112, 123, 125, 127, 128, 134, 138). These studies fail to show consistent excesses of any cancer site, fail to show evidence of dose response, and are potentially confounded by other concurrent exposures. Furthermore, studies of environmental lead exposures have failed to find any excess risk of cancer whatsoever (80).

Dr. Werntz admitted in his deposition at pages 89-92 that the scientific literature linking stomach cancer to lead and arsenic exposure was not strong “I was troubled -- I would agree that the literature for stomach cancer is not especially strong. There is some.” These associations are not accepted within the scientific community as reliable and Dr. Werntz’s inclusion of these cancers in his monitoring program is not based on scientific evidence. He has provided no scientific literature that supports his position. His position appears to be based on speculation that there might be associations.

There are no guidelines for cancer monitoring for any type of cancer among lead exposed populations that have been proposed, accepted, or used by the medical community in any instance in the history of medicine, in spite of the widespread commercial use of this material and the abundant scientific studies on its health effects. Dr. Werntz’s proposal for medical monitoring for cancer related to lead is simply outside of the realm of accepted medical practice and has no scientific basis. Dr. Werntz provides no evidence that medical monitoring for lead induced cancer has ever been accepted or utilized by the medical community in any setting.

3. Dr. Werntz has not properly considered exposure pathways, dose, and latency in designing his medical monitoring program

Exposure pathways are critically important in assessing cancer risks. Dr. Brown does not consider drinking water to be an exposure pathway in the class area. Therefore cancer risks related to drinking water are not at issue. Dr. Brown’s report considers ingestion of soil and inhalation of dust to be the only pathways by which exposure creates risks to the people in the class area. He does not consider drinking water to be a pathway that conveys cancer risk.

Dr. Werntz has proposed screening for cancers (and other diseases) that have been linked to high exposure to arsenic or cadmium only through drinking water exposure, even though there is no drinking water pathway in the class area. Skin cancer has been linked to drinking water containing excessive arsenic, but not to soil ingestion or inhalation of arsenic in communities near industrial sites, smelters, or waste sites. There is no basis for believing that the alleged exposures in the class area resulted in arsenic exposures via pathways that put people at increased risk of skin cancer. Skin cancer has never been linked to exposure to cadmium or lead under any circumstances. In light of these facts, there is no scientific rationale for screening for skin cancer in this community.

Bladder cancer is not associated with exposure to cadmium or lead. Although bladder cancer has been linked to drinking water containing excessive arsenic (22), drinking water is not at issue in the class area. Bladder cancer is not known to be linked to either

inhalation or soil ingestion of arsenic. Insofar as Dr. Werntz has provided no basis for a claim that people in the class area are at increased risk of bladder cancer due to their alleged soil ingestion or inhalation exposures, there is no basis for his proposed medical monitoring for bladder cancer.

Lung cancer has been associated with inhalation of arsenic and cadmium among workers in industry. As discussed above, it has not been shown reliably to be associated with community exposures to arsenic or cadmium. The principal concern with respect to lung cancer is the dose of dust that is inhaled and the latency between exposure and cancer risk. Dr. Brown has calculated estimates of inhalation doses for these materials and has calculated lifetime risks of cancer related to these alleged doses. While I do not agree with his estimates, even if they are correct the cancer risks are too small for medical monitoring to yield any benefit to the class members. I will discuss this in detail below.

Kidney cancer is not associated with exposure to cadmium or lead. Although transitional cell carcinomas of the kidney have been linked to drinking water containing excessive arsenic (22), drinking water is not at issue in the class area. Kidney cancer is not known to be linked to either inhalation or soil ingestion of arsenic. Insofar as Dr. Werntz has provided no basis for a claim that class members are at increased risk of kidney cancer due to their alleged soil ingestion or inhalation exposures, there is no basis for his proposed medical monitoring for kidney cancer.

Stomach cancer is not known to be caused by arsenic, cadmium, or lead by any route of exposure. Although Dr. Werntz believes that lead causes stomach cancer, the scientific literature does not support this belief and Dr. Werntz has offered no scientific publications that support his conclusions. The single study he cites (134) does not conclude that lead causes stomach cancer. Because there is no basis for Dr. Werntz's claim that class members are at increased risk of stomach cancer there is no basis for his proposed medical monitoring for this cancer.

Dr. Werntz has not considered latency in designing his cancer screening program. In the Tokudome study upon which he relies, the latency from first exposure to arsenic to lung cancer was 37.6 years (137). This suggests that screening for lung cancer in relation to inhaled arsenic will not reveal any arsenic-related lung cancers until at least 35-40 years after first exposure. While I do not believe the Tokudome study alone provides a thorough assessment of the latency between arsenic inhalation and onset of lung cancer or any other cancer, it is clear that Dr. Werntz's medical screening program has not adequately considered latency. Much of the cancer screening he proposes will be done during periods when the class members are at no increased risk of cancer from their alleged exposures and therefore his screening program cannot provide any benefit whatsoever during these periods. Similarly, the period of increased cancer risks ends at some point after exposure ceases. Dr. Werntz has not considered this issue in designing his program and has no plan to terminate screening at the end of the susceptible period. Because of these issues, his program cannot be viewed as having a scientific basis and will not reasonably lead to the detection of cancers purportedly related to exposures from the Spelter site.

4. Cancer risks alleged from these exposures are too small to be detectable in relation to background cancer risk

Dr. Werntz fails to properly consider the risk of cancer related to the exposures he alleges. Dr. Werntz has defined three exposure zones and the incremental cancer risks in each of those zones.

- Zone 1 is alleged to have a 5×10^{-4} incremental risk of cancer.
- Zone 2 is alleged to have between a 5×10^{-4} and a 1×10^{-4} incremental risk of cancer.
- Zone 3 is alleged to be outside the 1×10^{-4} cumulative incremental risk contour.

Dr. Werntz then proposes that one year of residence in Zone 1, three years of residence in Zone 2, and five years of residence in Zone 3 are required for entry into the medical monitoring program.

Comparison of these alleged incremental lifetime cancer risks to the lifetime cancer risks in the US general population indicates that these incremental cancer risks are so small that they cannot be regarded as evidence of appreciably increased risk. The lifetime cumulative cancer risks in the US general population are currently 45 percent (or 0.45) among males and 38 percent (or 0.38) among females (70). Adding an incremental risk of 5×10^{-4} (or 0.0005) to this risk would change the lifetime cancer risk of a man allegedly exposed in Zone 1 from 0.45 to 0.4505. In other words, exposure to Zone 1 (adequate to justify inclusion in Dr. Werntz's medical monitoring program) would increase the man's lifetime cancer risk by a factor of 0.0012 or 0.12 percent.

5. Dr. Werntz's proposed medical monitoring is not known to be effective for some cancers under any circumstances

For some of the cancers for which Dr. Werntz proposes medical monitoring there is no medical monitoring that is known to be effective and no medical monitoring tests have been accepted by the medical community as efficacious. There are no medical monitoring tests for lung cancer that have been shown to be effective in reducing the mortality due to this disease. Medical monitoring for lung cancer is not endorsed by any medical organization or specialty. Dr. Werntz's recommendations for monitoring for lung cancer have no basis in science and are outside the realm of accepted medical practice.

There are no medical monitoring tests for kidney cancer that have been shown to be effective in reducing the mortality due to this disease. Medical monitoring for kidney cancer is not endorsed by any medical organization or specialty. Dr. Werntz's recommendations for monitoring for kidney cancer have no basis in science and are outside the realm of accepted medical practice. Medical monitoring for bladder cancer is not endorsed by any medical organization or specialty. Dr. Werntz's recommendations for monitoring for bladder cancer have no basis in science and are outside the realm of accepted medical practice.

My opinions regarding Dr. Werntz's proposed screening for non-cancer effects

6. Exposure assessments must establish that subjects have been adequately exposed to be at increased risk of the disease in order to justify medical monitoring

It is accepted scientific practice in occupational health to quantify exposure levels from measurements of exposure. The causes of medical conditions must be determined through a process that considers whether chemical exposures occurred, what the specific chemical agents were, and whether the circumstances of those exposures are adequate to cause the medical condition. Scientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the individual was exposed to such levels are minimal facts necessary to support a scientific conclusion that exposure caused a person's disease. Dr. Werntz's methods are not consistent with these practices. His plans for screening for decreased renal function, renal failure, bone fragility, loss of teeth, hypertension, and increased rates of criminal activity are not grounded in any consideration of the dose or duration of exposure adequate to cause these conditions. In fact, his decision to screen people for these conditions is based entirely on their putative cancer risk. Because his screening program has no relationship to the dose or duration of exposure that is known to be capable of causing any of these conditions, it is entirely without a scientific basis and cannot be viewed as reasonably likely to reveal any medical condition related to the alleged exposures to arsenic, cadmium, or lead.

7. Dr. Werntz fails to consider dose response relationships and latency

The dose response relationship is a fundamental concept in toxicology (and in all medical sciences). Before one can assert that exposure to a chemical has harmed a person, the dose must be understood so that it can be compared to the doses that have been shown to cause that particular health effect. Dr. Werntz has not done this.

Dr. Werntz does not discuss the levels or duration of exposure (to any of the agents which he believes were present at the zinc smelter site) that are reported in the medical literature as likely to cause the medical conditions from which the plaintiffs suffer. In the absence of knowledge of the levels to which the plaintiffs were exposed and failing to compare those levels to exposure levels that are established as harmful, Dr. Werntz's opinions on causation are entirely without any scientific basis. Dr. Werntz provides no scientific evidence that the lead, cadmium, or arsenic he believes were present in the class area are known to cause any of the health conditions under the circumstances that existed for residents in that community.

Furthermore, Dr. Werntz has failed to address in his opinions the time course of these health effects he believes are consequences of the former zinc smelter exposures or to even comment on whether any of them would ever resolve. He has provided no scientific basis for his assumption that such effects would persist for the remaining lifetimes of all the plaintiffs and thereby necessitate 40 years of medical monitoring, as he proposes. The medical monitoring plan proposed by Dr. Werntz does not address these issues and has no evident scientific rationale. Consequently, there is no reason to believe that any of the tests he recommends will provide any benefit whatsoever to the plaintiffs in this case.

8. Cadmium and renal disease

Dr. Werntz alleges that cadmium has been linked to decreased renal function and renal failure. Although these disorders have been seen in industrial settings and under circumstances of gross overexposure, there is no scientific basis for a conclusion that the exposure levels alleged to have occurred in the class area would have caused these disorders in this community. Dr. Werntz has not provided any scientific literature that indicates that the exposure levels were at levels adequate to have caused any impairment whatsoever in renal function.

Published studies support a threshold for the adverse effects of cadmium on renal function. In a study of subjects who ate rice contaminated with cadmium in Japan (107), subjects who had a blood cadmium level of 0.38-0.41 ug/dL had no greater risk of renal dysfunction than did non-exposed subjects whose blood cadmium levels were 0.21-0.25 ug/dL. Ikeda studied the relationship between urinary cadmium excretion and early markers of renal dysfunction (urinary β 2-microglobulin and others)(74). The authors found that there was a threshold urine cadmium concentration of 4 micrograms cadmium/gram of creatinine excretion that led to an increase in urinary β 2-microglobulin excretion. Cadmium excretion below this level was not associated with any evidence of renal effects. Urinary β 2-microglobulin is regarded as the most sensitive indicator of renal dysfunction related to cadmium exposure. Jin studied the critical concentration of urinary cadmium required for the development of renal dysfunction (81). The authors found that the urinary cadmium concentration associated with a lower confidence limit on the 5% excess risk (the 5% Bench Mark Dose) of renal tubular dysfunction was 3-4 ug cadmium/gram of creatinine. Taken together, these studies and others (61, 79) indicate that there is a threshold of cadmium exposure below which there is no clear risk of adverse renal effects.

Dr. Werntz also alleges that lead has been linked to decreased renal function and renal failure. Although these disorders have been seen in industrial settings and under circumstances of prolonged high exposure, there is no scientific basis whatsoever for a conclusion that the exposure levels alleged to have occurred in the class area would have caused these disorders in this community. Dr. Werntz has not provided any scientific literature that indicates that the exposure levels were adequate to have caused any impairment whatsoever in renal function.

Dr. Werntz apparently has not considered the information discussed above or any other information on dose-response in designing his screening program. In consequence, his screening program is not designed to reasonably anticipate finding evidence of renal dysfunction related to cadmium, lead, or arsenic. The proposed program has no scientific basis and has no likelihood of providing any benefit to the population of the class area.

Dr. Werntz does not propose biomonitoring for arsenic or cadmium in spite of their established efficacy in quantifying exposure to these metals. Dr. Werntz concedes that if arsenic and cadmium testing were conducted it would be unlikely to detect the low levels of exposure that he expects in this community today. Plaintiffs have presented no

evidence of exposure to these materials adequate to necessitate medical monitoring and have done no tests that have documented exposure in any plaintiff. It is therefore surprising that Dr. Werntz declines to perform the biological tests that would document exposure if it existed. In light of the absence of evidence of plaintiffs' exposure and the unwillingness of plaintiffs' expert Dr. Werntz to even look for evidence of exposure, there is no basis for medical monitoring. Plaintiffs must provide evidence of exposure adequate to have put them at increased risk of disease as a foundational requirement for medical monitoring. They simply have not done so. As a consequence, their medical monitoring program has no scientific basis.

9. Bone fragility

Dr. Werntz alleges that lead has been linked to bone fragility. Lead is not known to be associated with this disorder and Dr. Werntz has provided no scientific basis for his claim. Moreover, he proposes no tests to screen for bone fragility, nor have any tests been used for this purpose among lead exposed populations.

Dr. Werntz alleges that cadmium has been linked to bone fragility. Effects on bone mineralization due to cadmium have been reported only in settings of gross overexposure and in the presence of renal damage. There is no scientific basis whatsoever for a conclusion that the exposure levels alleged to have occurred in the class area would have caused this disorder in this community. Dr. Werntz has not provided any scientific literature that indicates that the exposure levels were at levels adequate to have caused any impairment whatsoever in bone mineralization. Moreover, he proposes no tests to screen for bone fragility, nor are there any tests that are known to be efficacious for screening for this disorder in a community setting.

10. Loss of teeth

Dr. Werntz alleges that lead causes loss of teeth. He provides no basis for this claim and cites no scientific literature to support his belief. Lead is not known to cause periodontal disease, dental caries, or any other disorder associated with loss of teeth (64, 97). High lead exposure in the presence of poor dental hygiene can cause a bluish discoloration of gums, known as Burtonian lines, which indicate lead absorption and not lead intoxication (58). Good dental hygiene causes the discoloration to disappear. Moreover, tooth loss is self-apparent and no medical screening is necessary.

11. Hypertension

Dr. Werntz alleges that lead causes hypertension and that blood pressure should be recorded every two years. There is considerable scientific debate as to whether lead exposure and hypertension are causally associated (8, 10, 59, 111, 114). Concerns about this body of scientific literature focus principally on whether covariates (such as age, body mass, race, smoking, alcohol consumption, family history of cardiovascular/renal disease, and dietary factors) account for the association. In addition, studies that have relied on multiple blood pressure measurements or 24-hour ambulatory measurements rather than a single measurement have failed to confirm the associations between lead and blood pressure. Regardless of whether there is or is not a causal association, the magnitude of the relationship between lead and blood pressure is small: it is estimated

that blood pressure increases by approximately 1 mm of mercury per doubling of the blood lead level. Changes of this small magnitude cannot be reliably measured in individuals and cannot be reliably attributed to lead, especially in the presence of other factors related to high blood pressure (age, body mass, race, smoking, alcohol consumption, family history of cardiovascular/renal disease, and dietary factors). Because of these issues, blood pressure screening is not known to be a reliable procedure for detecting disease caused by lead in any individual.

12. Criminal activity

Dr. Werntz alleges that lead is associated with increased rates of criminal activity. This association is not accepted by the scientific community as being proven and there is substantial evidence that does not support it. Moreover, Dr. Werntz agreed in his deposition that he knows of no way to monitor for this. Medical screening for criminal activity is neither justified nor feasible.

My opinions in this report are expressed to a reasonable degree of medical and scientific certainty. I reserve the right to supplement my opinions as additional materials and scientific studies become available.



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