EXPERT REPORT

PREPARED BY

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Expert Witness appointed by the Commission of Inquiry
into Excess Lead Found in Drinking Water

1 December 2015

Commission of Inquiry into Excess Lead Found in Drinking Water

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Specialist Field	:	See **Appendix I**
Appointed on behalf of	:	The Commission of Inquiry into Excess Lead Found in Drinking Water (the "**Commission**")
Prepared for	:	The Commission
On instructions of	:	Messrs. Lo & Lo, Solicitors for the Commission ("**Lo & Lo**")
Subject matter / Scope of engagement:	:	To assist the Commission in discharging its duties under the Terms of Reference and by acting as an expert witness in the inquiry hearings
Documents reviewed	:	See **Appendix II**

The Terms of Reference of the Commission are as follows:

(a) to ascertain the causes of excess lead found in drinking water in public rental housing developments;

(b) to review and evaluate the adequacy of the present regulatory and monitoring system in respect of drinking water in Hong Kong;

(c) make recommendations with regard to the safety of drinking water in Hong Kong

Instructions

I have been instructed to give my opinion on the matters under the Terms of Reference.

In providing my opinion, I have also been instructed to consider the following areas and undertake the following tasks:

(1) to explain the short, medium and/or long term health effect(s) (if any) of elevated blood lead level on human beings in general, and in particular on (a) infants; (b) children under six years of age; (c) children/teenagers between six and eighteen years of age; (d) pregnant women; (e) lactating mothers; (f) elderly persons; (g) immunocompromised patients and (h) long term patients with chronic illnesses;

(2) to explain the internationally accepted or recognised guidelines and/or parameters (and their rationales), particularly those adopted by the World Health Organization ("**WHO**"), on the content of lead in (a) tap water and (b) blood in human beings;

(3) if the guidelines and parameters considered in (2) above have changed/ evolved over time, to explain the reasons for such changes;

(4) to opine on the adequacy and suitability of the reference values for blood lead level and the care plan published or followed by the Hong Kong Special Administrative Region Government;

(5) to opine on the adequacy and suitability of the acceptance criteria laid down by the Water Supplies Department ("**WSD**") for heavy metals and make recommendations, if necessary.

Introduction

I, Professor David C. Bellinger of Boston, U.S.A., have been appointed as the Commission's expert to assist the Commission in determining the matters under the Terms of Reference. The opinion and conclusions which are set out in this Report were formed on the basis of the evidence and the selected documents collected by the Commission from the Involved Parties since 20 August 2015 that I have seen. I appear as an independent expert for the Commission unrelated to any other work.

My Opinion

(1) **The short, medium, and/or long term health effect(s) (if any) of elevated blood lead level on human beings in general, and in particular on (a) infants; (b) children under six years of age; (c) children/teenagers between six and eighteen years of age; (d) pregnant women; (e) lactating mothers; (f) elderly persons; (g) immuno-compromised patients and (h) long term patients with chronic illnesses**

Introductory Comments

More is known about the adverse effects of lead on human health than about any other environmental chemical. When the National Toxicology Program (NTP) of the U.S. National Institute of Environmental Health Sciences evaluated the scientific literature on lead's health effects, its search identified more than 28,900 peer-reviewed publications (as of April, 2012). Therefore, the evidence base permits robust inferences about the range of effects of exposure to lead, as well as the characteristics of the dose-response and dose-effect relationships that describe the levels of exposure associated with increased risk. In my responses to the queries, I focus on the so-called "subclinical" health effects of chronic exposure to lead, that is, at levels of exposure that do not cause clinical signs and symptoms, as the likelihood that clinical lead poisoning would occur from consuming water with the lead concentrations measured in the Hong Kong estates is very low. To establish the context within which to consider such exposures, however, I briefly discuss the blood lead levels at which such clinical signs and symptoms occur. A very high level of lead exposure can be fatal, although it rarely occurs unless a child's blood lead level exceeds 150 µg/dL, and some children have survived a blood lead level of several hundred µg/dL. Overt signs of acute intoxication occur at blood lead levels of 100-120 µg/dL in adults and at 80-100 µg/dL in children. These signs include restlessness, irritability, poor attention span, headaches, muscle tremor, abdominal

cramps, kidney damage, hallucinations, loss of memory, cerebral oedema or haemorrhage, confusion, coma, and seizures. Chronic lead exposure (e.g., blood lead levels of 50–80 µg/dL in adults and 25-50 µg/dL in children) can also produce sleep problems, fatigue, irritability, constipation, poor appetite, anaemia, headaches, and joint pain. As such non-specific signs and symptoms can occur as the result of other medical conditions, it can be difficult to diagnose lead exposure as the cause.

Lead is often characterized as a "multi-media" pollutant because of the diverse ways in which human exposure can occur. The major classes of sources/pathways of exposure to inorganic lead (the form of lead in solder) include food, air, soil, paint, and water, although exposure can also occur as a result of many other activities (e.g., use of contaminated folk or herbal medicines). Once lead enters the body, its toxicity is the same regardless of the source/pathway through which exposure occurred. An individual's blood lead level reflects exposures from all sources/pathways, so to evaluate the magnitude of the contribution of a specific source/pathway, it is necessary to consider information about other important sources/pathways. The lead in a person's body resides in three major "pools," and can move among them. Among adults approximately 90% of the total body burden is in mineralized tissues, such as bone. The lead accumulated in hard (cortical) bone might remain there for several decades, whereas the lead accumulated in more porous (trabecular) bone, which is in greater contact with the circulatory system, might remain there for much less time. In children, lead in bone accounts for approximately 70% of the total body burden, and appears to move in and out of bone much more rapidly than it does in adults due to the rapid changes in bone turnover that occur during childhood. Most of the rest of an individual's body burden of lead is in soft tissues such as the brain, liver, and kidneys. Only a small percentage, about 5%, is in blood. Lead can be mobilized from mineralized tissues and re-introduced into blood by a variety of physiologic and pathophysiologic states that increase bone turnover, such as pregnancy, lactation, menopause, infection, and osteoporosis. Therefore, the blood lead level measured for an individual at any given time reflects the equilibrium between an individual's current exposure to "new" lead and the "legacy" lead from past exposures. The half-life of lead in blood is approximately 30 days, meaning that if two atoms of lead enter the blood, in a month's time only one will remain there, and the other one either excreted from the body or moved to storage in hard or soft tissue. Because of the re-equilibration processes, the half-life of 30 days does not mean that an individual's blood lead level will fall by half in a month's time if major exposure sources/pathways are removed. The greater an individual's past exposures to lead, the harder it will be to reduce blood lead by an intervention (e.g., chelation, removal of

exposure source/pathway). Indeed, in one study of children who had a blood lead level of 25-29 µg/dL and were placed in a case management system, it took an average of two years for the blood lead to drop below 10 µg/dL. On the other hand, if an individual's exposure is acute and prior exposure to lead has been low, the total body lead burden would be modest, and the individual's blood lead level would be expected to decline relatively rapidly following cessation of the current exposure.

1a and 1b. Infants and children under six years of age

Humans of any age can be harmed by exposure to lead, but young children are considered to the most vulnerable subgroup of the population, and the developing central nervous system is considered to be the most vulnerable organ. Children with blood lead levels below 25 µg/dL generally do not show any signs or symptoms that bring them to medical attention. What a plethora of studies show, however, is that children with such levels are at increased risk of a variety of cognitive and behavioural adversities that are persistent and affect many aspects of an individual's health and well-being. In its recent evaluation, the U.S. NTP characterized the weight of evidence on an association between blood lead level and a health outcome as sufficient, limited, or inadequate. Evidence for an association was characterized as "sufficient" if methodological factors such as chance, bias, and confounding could be ruled out with reasonable confidence, as "limited" if the association had been observed but that the methodological factors could not be ruled out with reasonable confidence, and as "inadequate" if, "The available studies are insufficient in quality, consistency, or statistical power to permit a conclusion regarding the presence or absence of an association...or no data in humans are available." The NTP concluded that, in children, the evidence is *sufficient* to conclude that blood lead levels <5 µg/dL are associated with adverse neurological effects, including reduced intelligence, neuropsychological function, and academic achievement and increased incidence of attention-related and other problem behaviours. The most complete and compelling evidence available pertains to children's intelligence. A set of analyses in which the data from 7 prospective studies were pooled (a sample size of 1,333 children) found that the inverse association between children's IQ scores and their blood lead concentrations had a supra-linear form, such that the slope of the association (the rate of decline in IQ per µg/dL increase in blood lead concentration), was steeper over the range below 10 µg/dL than it was over the range between 10 and 30 µg/dL. Although the biological mechanism of this is not known, the finding has now been replicated in several independent studies. The details of this dose-effect relationship suggests a child with a blood lead level of 0 will, all other things being equal, have an IQ score about 5 points

(1/3 standard deviation) higher than a child with a blood lead level of 10 µg/dL. A large number of studies support the hypothesis that this effect of lead on children's IQ scores is only the "tip of the iceberg." Even among children with blood lead levels less than 10 µg/dL, those with higher levels perform significantly worse on tests of academic achievement and more often require special educational supports in school.

A series of neuroimaging studies of young adults (mean age 20 years) in whom detailed histories of lead exposure prior to the age of 6 years were available provides evidence that early-life exposure produces persistent changes in brain structure and function. Higher blood lead levels in childhood, which in these children were generally than 10 µg/dL, were associated with inverse linear decreases in grey matter volume, most strikingly in the frontal regions of the brain (anterior cingulate cortex, ventro-lateral prefrontal cortex). Diffusion-tensor imaging studies revealed lead-related changes in myelination and axonal integrity throughout the white matter of the brain (i.e., reduced fractional anisotropy). Proton magnetic resonance spectroscopy revealed that blood lead levels in childhood predicted reduced level of metabolites in several regions of grey matter and white matter, suggesting altered patterns of brain function. Functional magnetic resonance studies showed that activation patterns in the left frontal cortex and left middle temporal gyrus while performing a language task differed among individuals with different levels of childhood lead exposure. It is noteworthy that these differences in brain structure and function were related to blood lead levels measured nearly two decades earlier.

Much less information is available about the associations in children between lead exposure and other organ systems. The NTP considered the evidence to be *limited* for the association between blood lead levels <10 µg/dL and aspects of immune function (increased hypersensitivity/allergy by skin prick test of allergens and increased IgE). The evidence was considered to be *inadequate* for associations between blood lead levels <10 µg/dL and other aspects of immune function (asthma, eczema, non-allergy immune function) as well as with cardiovascular function and decreased kidney function in children <12 years of age.

1c. Children and Teenagers between 6 and 18 years

Several of the major prospective studies of lead and neurodevelopment have included follow-up intervals that extend as far as the fourth decade of life (Boston, Port Pirie, Cincinnati, Kosovo). These studies provide evidence that the inverse associations between early-life lead exposure and neurodevelopment persist, though perhaps in somewhat weakened form, over this interval. In one prospective study, the lead

concentration in umbilical cord blood, reflecting prenatal exposure, was still inversely related to IQ scores at more than 30 years of age.

The US NTP evaluation concluded that the evidence is *sufficient* for the association between blood lead level <10 µg/dL and both decreased hearing, delayed puberty, and reduced postnatal growth. In cross-sectional data on pubertal development in girls aged 8-18 years from the U.S. NHANES III (National Health and Nutrition Examination Survey), blood lead levels above 3 µg/dL were associated with delays of 2 to 6 months in breast and pubic hair development (i.e., progression to the next Tanner stage), and the onset of menarche, though these associations were stronger in African-American and Mexican-American girls than in Non-Hispanic White girls. In another study of Native American girls 10-17 years of age, those with a blood lead concentration greater than the median of 1.2 µg/dL reached menarche 10.5 months later than girls with a concentration below the median. In a study in 8 to 9 year old boys, a blood lead concentration ≥5 µg/dL was associated with slower progression through stages of genital development and, at a follow-up examination several years later, boys with higher blood lead levels were less likely to have begun puberty, with the differences in testicular volume and staging of genitalia and pubic hair corresponding to delays of 6 to 8 months.

A considerable body of evidence now exists in support of the hypothesis that greater lead exposure places a child at increased risk of meeting diagnostic criteria for Attention Deficit Hyperactivity Disorder. In one study of 6 to 16 year olds (NHANES 1999-2002), children in the fifth quintile of current blood lead (>2 µg/dL) were 4.1 times more likely than children in the first quintile (<0.8 µg/dL) to have parent-reported ADHD. In a subsequent study, when outcome classification was based on a diagnostic interview rather than parent-report (NHANES 2001-2004), 8-15 year olds with a blood lead concentration in the upper tertile of the distribution (>1.3 µg/dL) were 2.3 times more likely than children in the lowest tertile to meet diagnostic criteria. In a study conducted in South Korea, children with a blood lead level >3.5 µg/dL were 1.96 times more likely than children with a blood lead level <1 µg/dL to have ADHD. In a case-control study conducted in China, children with a blood lead level of 5 to 10 µg/dL were 5.2 times more likely to have ADHD than children with a blood lead level <5 µg/dL, and children with a blood lead level ≥10 µg/dL were 7.2 times as likely.

Several studies, varying in design from case series, ecologic, case-control, cross-sectional, and prospective cohort, have suggested that greater childhood lead exposure is associated with an increased propensity for violence and aggression, as reflected by homicide rates, the diagnosis of Conduct Disorder, being an adjudicated delinquent, ratings by parents or teachers of rule-breaking or antisocial behaviour, self-reported

offenses and convictions, and arrests. Such studies are difficult to conduct and subject to a variety of biases, but, in aggregate, the evidence for this association is strongest for blood lead levels >10 µg/dL.

The NTP considered the evidence to be *limited* for the associations between blood lead levels <5 µg/dL and decreased kidney function in children ≥12 years of age (reduced glomerular filtration rate). Among 12-20 year olds who participated in the NHANES III survey, adolescents with a blood lead level in the highest quartile (≥3 µg/dL) had a significantly lower estimated glomerular filtration rate (based on serum cystatin-C or, to a lesser extent, serum creatinine) than adolescents with blood lead levels in the first quartile (≤1 µg/dL).

1d. Pregnant Women

Lead crosses the placenta by the process of passive diffusion. As a result, the concentration of lead in the umbilical cord blood of a neonate will be similar to the concentration of lead in maternal blood at delivery. In other words, the lead exposure of a foetus is essentially the same as that of the pregnant woman. Certain physiologic changes associated with pregnancy, and with the progression of pregnancy, alter lead kinetics in complex ways. These include increasing blood volume, decreasing haematocrit, saturation of the lead-binding capacity of red blood cells, increased bone re-sorption (and thus mobilization of long-term lead stores), and possibly increased intestinal absorption of lead. Studies using lead isotopic ratios have shown that a substantial fraction of the lead in the blood of a pregnant woman cannot be attributed to her current external exposure but reflects lead from past exposure that has been mobilized by the rapid turnover of bone that occurs during the second and third trimesters of pregnancy. Animal studies suggest that up to 40% of the lead in the foetal skeleton came from maternal bone.

Considerable research has been conducted on the potential effects of lead exposure on the health of the pregnant woman herself, in particular her reproductive health, the course of pregnancy, and the health of her foetus at birth and in the postnatal period. In 2010, the U.S. Centers for Disease Control issued guidelines for the identification and management of lead exposure in pregnant and lactating women. The literature review conducted evaluated the evidence regarding the associations between lead exposure and a variety of health endpoints.

Fertility. Greater lead exposure in a woman is associated with a longer time to conceive a pregnancy, but only at blood lead levels exceeding 10 µg/dL.

Gestational hypertension. Cohort and case-control studies have shown that a greater concentration of lead in maternal blood during pregnancy or in cord blood is significantly associated with pregnancy hypertension and elevated blood pressure during pregnancy or at delivery. Although these associations are statistically significant, they are very modest in magnitude (e.g., correlations <0.10, suggesting little shared variance). In many such studies, the mean blood lead levels of the participants were less than 10 µg/dL and, in some, less than 5 µg/dL. The dose-effect relationship has not been well-characterized, however, and it is uncertain whether the elevated risk is associated with a woman's acute exposure to lead during pregnancy, to chronic exposure, or both. Pre-existing hypertension could affect a woman's renal function during pregnancy, altering lead kinetics, causing increased lead retention (i.e., reverse causation). Evidence linking lead exposure to the risk of pre-eclampsia (elevated blood pressure accompanied by proteinuria) is weak and based on only a few studies.

Spontaneous abortion. Most studies evaluating the risk of spontaneous abortion in relation to maternal lead exposure have not identified a significant relationship at blood lead levels below 30 µg/dL. One high-quality prospective study conducted in Mexico City did, however, report that the risk began to increase, and increased consistently thereafter, when maternal blood lead level exceeded 5 µg/dL (Borja-Aburto et al., 1999).

Fetal growth. Many studies have evaluated lead exposure as a risk factor for outcomes such as length of gestation, birth weight, low birth weight (<2500 grams), infant birth length, infant head circumference, and congenital anomalies. Although significant associations have been reported, the evidence is somewhat inconsistent, and the maternal blood lead level at which risk begins to increase is uncertain.

In considering the overall evidence, the U.S. CDC recommended that a pregnant woman with a blood lead level ≥5 µg/dL receive follow-up testing, education, and environmental, nutritional, and behavioural interventions to reduce, if possible, her exposure and that of her foetus and newborn child. It does not recommend universal screening of pregnant women.

The NTP concluded that the evidence was is *limited* for the association between blood lead levels <5 µg/dL and "decrease in measures of cognitive function," as well as for the association between blood lead levels <10 µg/dL and "decreased IQ, increased incidence of attention-related and problem behaviours, and decreased hearing."

1e. Lactating Mothers

Bone turnover is greater during lactation than pregnancy and continues as long as an infant is nursing. As much as 5% of a woman's bone mass is mobilized during this period. Isotopic studies of the lead in breast milk indicates that at least some of the lead comes from maternal bone, and the concentration of lead in maternal bone, measured using X-ray fluorescence, is positively related to the concentration of lead in breast milk. However, the concentration of lead in breast milk is low, comparable to that in the plasma fraction of blood (which accounts for only about 1% of the lead in whole blood). Thus, this pathway of exposure likely contributes relatively little to an infant's lead exposure.

Water can be a very important pathway of lead exposure for infants who consume formula made up with water that contains lead. Balancing the known benefits of breastfeeding and the slight risks of substantial lead exposure from breastfeeding , the U.S. CDC encourages mothers with a blood lead level ≤40 µg/dL to breastfeed.

1f. Elderly Persons

Relatively few studies have investigated the association between lead exposure and health outcomes in the elderly, and most of these have relied on measurements of bone lead as the exposure index rather than blood lead. This is because, as noted, lead accumulates in bone over time so that its concentration in this tissue is thought to provide a more accurate measure of cumulative exposure than does an individual's current blood lead level, which tends to reflect largely recent exposure. Another factor that might render an elderly person's current blood lead level less informative about his or her current exposure is the fact that physiologic and pathophysiologic processes that involve increased bone turnover, such as osteoporosis, result in some of the lead stored in bone being mobilized into soft tissues and to blood. As a result, some of an elderly person's blood lead likely reflects not only lead to which he or she was recently exposed but also so-called "legacy" lead, that is lead to which exposure occurred in the past. In a study conducted in Boston, the concentration of lead in patella (knee-cap, a trabecular bone) was significantly higher in older men who, 14-19 years previously, had lived in homes with a first-flush water lead concentration greater than 50 µg/L.

In aggregate, the available studies, which have been conducted mostly in elderly men, provide reasonably consistent evidence that numerous aspects of health, including cognitive function, mental health, renal function, hearing, and cardiovascular function, are inversely related to bone lead concentration. Because lead is an accumulative toxicant, however, it is difficult to determine the contribution, if any, of current (or recent) lead exposure to these associations.

One recent development in lead toxicology that is generating considerable interest is the evidence, in animal models, that early-life lead exposure is a risk factor for accelerated neurodegeneration in later life. In rodents and non-human primates, early-life lead exposure, but not current lead exposure, is associated with later overexpression, in adulthood, of genes involved in the production of a protein, beta-amyloid, that is a constituent of the amyloid plaques that are found in the brains of patients with Alzheimer's Disease. The extent to which this might be true in humans is unknown.

1g. Immuno-compromised Patients

There is some limited evidence that greater exposure to lead can produce changes in immune function. To my knowledge, however, immuno-compromise has not been investigated as an effect modifier of lead toxicity.

1h. Long-Term Patients with Chronic Illnesses

Relatively limited information is available about the effects of lead exposure on individuals with comorbid conditions that might make them more vulnerable to lead. In general, such information has come from subgroup analyses of study cohorts. These analyses have sometimes, but not always, suggested that lead exposure of a given intensity is more harmful to individuals with some pre-existing medical conditions.

Kidney Function. In analyses of adults (≥20 years) in the NHANES III survey, significant associations between concurrent blood lead level and kidney function (e.g., elevated serum creatinine, reduced glomerular filtration rate) were found only in individuals with hypertension. Compared to individuals in the lowest quartile of blood lead level (0.7 to 2.4 µg/dL), individuals in the second (2.5 to 3.8) and third quartiles (3.9 to 5.9 µg/dL) were more likely to have chronic kidney disease. In a prospective study, a lead-related decline in renal function (specifically the rise in serum creatinine concentration) over a 6-year follow-up period was greater in individuals who, at baseline, had diabetes. Lead burden was measured in bone (tibia), however, reflecting chronic exposure, so it is difficult to determine the blood lead level at which this increased vulnerability is expressed. A study using data from NHANES 1999-2006 reported that the risk of lead-associated reduction in kidney function (albuminuria and reduced glomerular filtration rate) was increased two-fold among individuals who had greater exposure to cadmium, another metal that is well-known to impair kidney function.

Cardiovascular Function. Hypertension has long been known to be one result of greater lead exposure in adults. In one study of adult men, greater bone lead

concentrations (patella but not tibia) were associated with changes in heart rate variability (higher low frequency power, ratio of low to high frequency power) but only among men with metabolic syndrome.

Cognitive function. Little evidence is available regarding co-morbidities that affect the likelihood or severity of lead-associated impact on cognitive function. Several genetic polymorphisms are hypothesized to modify lead neurotoxicity, but the findings are inconsistent across studies. One study of individuals exposed to lead occupationally suggested that the inverse associations between an index of lifetime lead exposure and scores on neuropsychological tests were greater in individuals who had "low cognitive reserve" operationalized as poorer reading achievement, an outcome that presumably predated the onset of occupational exposure to lead. The idea is that such individuals are less able to weather an insult to the brain. A similar hypothesis has been advanced with regard to children. Several studies have suggested that the inverse association between lead and neurodevelopmental test scores is greater among children who, for reasons other than lead, have scores that place them in the lower reaches of the distribution. In other words, children whose neurodevelopment is imperiled by factors such as low socioeconomic status, stress, nutritional deficiencies, and other co-morbidities, suffer more from a given exposure to lead than do children without these characteristics. In one study, the impact of blood lead level on children's neurodevelopment was greater among children with nutritional deficiencies (e.g., folate, iron).

(2) The internationally accepted or recognised guidelines and/or parameters (and their rationales), particularly those adopted by the World Health Organization, on the content of lead in (a) tap water and (b) blood in human beings;

(3) If the guidelines and parameters considered in (2) have changed/evolved over time, to explain the reasons for such changes

(a) Lead in tap water

World Health Organization. In 1958, the WHO recommended a maximum allowable concentration of 0.1 mg/L (100 µg/L) in water. This recommendation was health-based. In 1963, it was reduced to 0.05 mg/l" (50 µg/L), however, a value of 0.1 mg/kg was re-established, "...because this level was accepted in many countries and the water consumed for many years without apparent ill effects, and it was difficult to reach a lower level in countries where lead pipes were used." In the 1st edition of the WHO Guidelines for Drinking water Quality (1984), a health-based guideline of 0.05 was again

recommended. Based on the JECFA Provisional Tolerable Weekly Intake (PTWI) of 3.5 µg/kg of body weight per day (1986) for infants and children, the 1993 edition of the drinking water guidelines recommended a value of 0.01 mg/l, based on the assumption of a 50% allocation of the PTWI to drinking-water for a 5 kg bottle-fed infant consuming 0.75 litre of drinking-water per day. As infants were considered to be the most sensitive subgroup of the population, this guideline value was thought to also be protective for other age groups. In light of JECFA's recent withdrawal of the PTWI (25 µgPb/kg body weight/week), the WHO guideline for drinking water lead was re-evaluated in 2011. The value of 10 µg/L was retained. The reasons were that lead exposure arises from a range of sources, of which water is frequently a minor one, and it is extremely difficult to achieve a concentration lower than 10 µg/l by central conditioning, such as phosphate dosing. However, this is designated as "provisional" on the basis of treatment performance and analytical achievability. The WHO further stated that, "... lead is exceptional, in that most lead in drinking-water arises from plumbing in buildings, and the remedy consists principally of removing plumbing and fittings containing lead, which requires much time and money. It is therefore emphasized that all other practical measures to reduce total exposure to lead, including corrosion control, should be implemented." (Lead in Drinking Water: http://www.who.int/water_sanitation_health/dwq/chemicals/lead.pdf?ua=1).

U.S. Environmental Protection Agency. In the U.S., the allowable concentration of lead in water was established under the Safe Drinking Water Act of 1974. Until 1991, the limit for lead in drinking water was 0.05 mg/L (50 µg/L). With passage of the Lead and Copper Rule in 1991, this concentration was reduced to 0.015 mg/L (15 µg/L). For most contaminants, the EPA establishes a Maximum Contaminant Level (MCL). Lead is handled differently because it does not enter water at the source but as a result of corrosion of plumbing materials in the distribution system, and typically those materials close to the point of consumption. Therefore, rather than establishing an MCL for lead, the EPA established a treatment technique that water authorities must use to reduce the corrosivity of water. If more than 10% of the tap water samples collected exceed the action level of 0.015 mg/L (15 µg/L), a water system is required to take steps such as corrosion control treatments, lead service line replacement, source water monitoring and treatment, and public education about reducing exposure.

(b) Lead in the blood of human beings

At the present time, the World Health Organization identifies a blood lead level of

10 µg/dL as the upper limit of the acceptable range. A WHO committee is currently reviewing the guidelines for the diagnosis and treatment of lead poisoning, however. As noted, in its most recent evaluation of lead, the FAO/WHO JECFA withdrew the PTWI, of 25 µgPb/kg body weight/week, which had been established in 1993. The rationale was that the absence of a threshold for lead toxicity means that no level of exposure is safe (thus "tolerable"). Moreover, it was not possible to establish a new PTWI that would be considered to be health protective."

In the U.S., the reference value (explained below) is currently 5 µg/dL for children. Among adults, 5 µg/dL is the upper value for pregnant and lactating women, 10 µg/dL for others. These values are of relatively recent origin. The toxicity of lead exposure at high dose has been recognized for two millennia, but it is only in the past 40 years that we have learned that exposures that do not produce clinical toxicity can, nevertheless, reduce an individual's health and quality of life. Over this recent period, the steady accumulation of evidence has motivated a series of reductions in the blood lead level identified as "acceptable" in young children. In paediatric textbooks of the 1960's, a blood lead level of 60 µg/dL was considered to be the upper limit of a "normal" value. In 1971, the U.S. Surgeon General reduced this cut-off to a blood lead level of 40 µg/dL. The U.S. CDC subsequently identified an "action level" of 30 µg/dL in 1975, 25 µg/dL in 1985, and 10 µg/dL in 1991. Each reduction in the concentration considered to be acceptable stimulated a new round of research to determine whether the new action level did, indeed, provide children with an adequate margin of safety. Each time, these studies clearly indicated that the answer was "no" because adverse health effects were consistently found at blood lead levels below the action level. The current consensus is that there is no "safe" blood lead concentration below which adverse effects do not occur. As a result, in 2012, the U.S. CDC abandoned the concept of an action level, substituting for it a "reference level". This level is defined solely on a statistical basis, as the 97.5th percentile of the blood lead distribution of young children in the U.S. Therefore, this is not a health-based value, and its purpose is simply to identify children who are the most highly exposed. This concentration is currently 5 µg/dL, but it will be updated, as necessary, every four years using the blood lead distribution measured in the two most recent NHANES surveys. If these surveys indicate that the blood lead level corresponding to the 97.5th percentile has changed, the reference value will be changed accordingly. As stated by the U.S. CDC, because of the absence of an identified threshold for adverse effects, it cannot specify "an allowable exposure level, level of concern, or any other bright line intended to connote a safe or unsafe level of exposure" (2010, p.12).

(4) The adequacy and suitability of the reference values for blood lead level and the care plan published or followed by the Hong Kong Special Administrative Region Government

The reference values selected by the Hong Kong Special Administrative Region Government for prioritizing individuals for follow-up based on blood lead level are appropriate and consistent with those identified by authoritative international bodies. The following tables present the recommendations of the U.S. CDC for responding to the identification of a child or adult with a blood lead level greater than the reference value (5 µg/dL for children, 10 µg/dL for adults).

Recommendations for Follow-up Actions, Children (U.S. CDC, 2012)

Blood Lead Concentration < 5 µg/dL	Blood Lead Concentration 5-45 µg/dL
Lead education (dietary, environmental)	Lead education (dietary, environmental)
Environmental assessment for pre-1978 housing	Follow-up blood lead monitoring (see following table for schedule)
Follow-up blood lead monitoring (see following table for schedule)	Complete history and physical examination
	Lab studies (iron status—consider haemoglobin or haematocrit)
	Environmental investigation
	Lead hazard reduction
	Neurodevelopmental monitoring
	Abdominal X-ray (if particulate lead ingestion suspected; bowel decontamination if indicated)

Schedule for Follow-up Blood Lead Testing in Children (U.S. CDC)

Venous Blood Lead	Early Follow-up Testing (2-4 Months after Identification	Later Follow-up Testing After Blood Lead is Declining
≥reference value but <10 µg/dL	3 months	6-9 months
10-19 µg/dL	1-3 months	3-6 months
20-24 µg/dL	1-3 months	1-3 months

Source: http://www.cdc.gov/nceh/lead/acclpp/final_document_030712.pdf.

Recommendations for Follow-up Actions, Adults (Association of Occupational and Environmental Clinics, U.S., 2007)

Blood lead concentration 5-9 µg/dL	Blood lead concentration 10-29 µg/dL
Lead education (occupational, environmental, reproduction)	Consider clinical assessment
Follow-up blood lead monitoring	History (occupational, environmental, medical)
	Examination, lab work
	Identify risk factors
	Blood lead levels of family members
	Exposure assessment (air testing, Workplace)
	Consider consultations (occupational medicine, industrial hygiene, public health department

	Lead hazard reduction
	Follow-up blood lead monitoring

Source: http://www.aoec.org/documents/positions/MMG_FINAL.pdf.

In developing my opinion about the adequacy of the care plan to address the issue of lead exposure in the 11 affected public rental housing estates, I considered the information provided on the second blood tests conducted on individuals who first blood lead results exceeded 5 µg/dL (line-listing of 163 persons, with results as of 22 October 2015) [Bundle E1/537-546]. The results of repeat blood lead tests are reported for 28 individuals. For 24 individuals, the interval between the initial and repeat test was approximately 3 months. For 4 of the remaining individuals (all pregnant women), the interval ranged from 1 week to 1 month. These groups are considered separately. For the 24 non-pregnant individuals, the average decline in blood lead level was 30.8% (2.7-55.5). There is some evidence of regression to the mean (i.e., if a variable is extreme on its first measurement, it will tend to be closer to the average on its second measurement). The 10 individuals who showed the largest decreases between measurements had an initial blood lead level that averaged 8.46 µg/dL (range 5.58-14.18), while the other 14 individuals had an initial level that averaged 6.30 µg/dL (range 5.28-8.62). It would have been helpful to conduct repeat blood lead tests on a sample of individuals whose initial result was below the reference value. This would have permitted a more certain interpretation of how much of the decline in blood lead observed in all of the non-pregnant individuals can be attributed to reduction in lead exposure and how much to regression to the mean.

The 4 pregnant women showed changes in blood lead level that were smaller than those observed in the non-pregnant individuals (-0.81, -1.55, +0.09, and -0.91). The average change was a decrease of 13.7%. Several factors could explain this. First, the interval between tests was much shorter, providing less time for change. Second, as noted previously, the kinetics of lead change during pregnancy. Several studies show that blood lead level tends to decrease over the early stages of pregnancy. This is likely due to haemodilution, i.e., the approximately 50% increase in plasma blood volume that occurs during this period. On average, however, a woman's blood lead level begins to increase from the middle of pregnancy on. This is likely due to the mobilization of maternal bone to meet the increasing calcium required to meet the needs of the fetal skeleton as it develops. As noted earlier, this process mobilizes lead, as well as calcium, into the blood. These considerations complicate the interpretation of short-term blood

lead changes in pregnant women. No information is provided in the line-listing about the stage of pregnancy of the 4 individuals so it is difficult to draw conclusions.

In my opinion, the general components of the care plan proposed for residents are appropriate, although some are not described in detail, making the adequacy of the plan somewhat difficult to evaluate. Continued follow-up blood lead testing for individuals whose blood lead concentration remains above the age-appropriate reference value is very important. Because of the multiplicity of sources and pathways of lead exposure, it might be that sources/pathways other than water contribute to an individual's continued blood lead elevation. Therefore, while the plan to conduct an "exposure assessment" is sound, no information is provided about what this assessment will include and what methods will be used. In general, such an assessment involves consideration of lead hazards in an individual's home environment (paint, food, hobbies, use of folk medicines, children's toys, etc.), outside activities (soil, proximity to lead-emitting point sources), and any other environments in which an individual spends substantial time (e.g., school, day care centre, workplace). The care plan also stipulates a "health evaluation" (children <18 years, pregnant women) for individuals with a blood lead level of 5-44 µg/dL and a "medical assessment" for individuals with a blood lead level >44. What will be included in these activities, and whether the clinicians performing them will be experienced in assessing lead-exposed individuals are not described. Whether the "developmental assessment" will involve use of screening tools, parent-questionnaires, or in-person clinical evaluations is not described.

(5) The adequacy and suitability of the acceptance criteria laid down by the Water Supplies Department for heavy metals and, if necessary, to make recommendations

In my opinion, the acceptance criteria specified by the Water Supplies Department for four metals, lead (≤10 µg/L), cadmium (≤ 3 µg/L), chromium (≤ 50 µg/L), and nickel (≤ 70 µg/L), are all based on sound reasoning. They are either more protective or equally as protective of human health than are guidelines for these metals in drinking water established by authoritative bodies such as the World Health Organization and the U.S. Environmental Protection Agency. In establishing the guidelines, these bodies have relied on thorough consideration of the routes of human exposure, kinetics of the metals in the human body, and the critical health effects. The guidelines have undergone extensive peer review and reflect the medical and scientific consensuses at the times that the guidelines were established.

Lead. As discussed earlier, the WHO guideline for lead in drinking water is 10 µg/L, the same as the acceptance criteria established by the Hong Jong WSD. This guideline is somewhat stricter than that used by the U.S. Environmental Protection Agency. If more than 10% of the tap water samples collected exceed 0.015 mg/L (15 µg/L), a water system is required to take steps such as corrosion control treatments, lead service line replacement, source water monitoring and treatment, and public education about reducing exposure.

Cadmium. It is known that cadmium in the zinc of galvanized pipes or cadmium-containing solders in fittings, water heaters, water coolers and taps can contaminate drinking water. The concentration is increased in the presence of low pH, as this would tend to make water more corrosive. WHO has established a guideline of 0.003 mg/L for cadmium in drinking water. In re-evaluating the PTWI for cadmium in 2011, the FAO/WHO JECFA substituted a Provisional Tolerable Monthly Intake of 25 µg/kg of body weight for the PTWI of 7 µg/kg of body weight due to the very long half-life of cadmium in the kidney. This change did not affect the water cadmium guideline, however. The change from a PTWI to a PTMI had no effect on the guideline value however, which remained 0.003 mg/L (3 µg/L).

The US EPA maximum contaminant level for cadmium in water is 0.005 mg/L (5 µg/L), a level that is considered to be protective of public health.

Chromium. Chromium differs from lead and cadmium in that it is an essential nutrient, with the daily requirement for adults estimated to be 0.5–2 µg of absorbable trivalent chromium (chromium-III). Hexavalent chromium (chromium-VI), however, is extremely toxic. If a fractional absorption value of 25% for "biologically incorporated" chromium-III is assumed, the requirement would be met by a daily dietary intake of 2–8 µg of chromium-III, equivalent to 0.03–0.13 µg of chromium-III per kg of body weight per day for a 60-kg adult. Ideally, different guideline values should be set for chromium-III and chromium-VI, but for a variety of reasons the chromium guideline value refers to total chromium in water. This is because these forms of chromium can convert back and forth in water and in the human body depending on environmental conditions. The WHO has established a guideline of 0.05 mg/litre (50 µg/L) for total chromium. This is considered to be unlikely to give rise to significant risks to health.

In the U.S. the drinking water standard for total chromium is 0.1 mg/L (100 µg/L). In order to ensure that the greatest potential risk is addressed, this regulation assumes that the more toxic form, chromium-VI accounts for all of the total chromium value. This is considered to be protective because the actual fraction of chromium-VI varies depending on the water type (ground water versus surface water, raw water versus treated drinking water, etc.), geographical location, and the oxidation-reduction potential of the water.

Nickel. Like chromium, nickel is an essential trace mineral. Drinking water is thought to contribute only a minor proportion of daily intake, although this depends on the concentration of nickel in groundwater. Based on a "lowest observed adverse effect level" of 12 µg/kg body weight per day, the WHO estimated that a guideline value of 0.07 mg/L (70 µg/L) would adequately protect human health (assuming a 60 kg adult drinking 2 litres of water per day, and assuming that this intake accounts for 20% of the daily intake of nickel).

In the U.S., the EPA recommends that drinking water levels for nickel should not be more than 0.1 mg/L (100 µg/L).

Summary of Conclusions

Lead serves no biological purpose in the body. There is no "safe" level of lead, as adverse effects in different organ systems, particularly the central nervous system, have been observed at blood lead levels less than 5 µg/dL. Therefore, the ideal blood lead concentration for a human is 0 µg/dL. Because of the ubiquity of lead in the contemporary environment, this will not be achievable in the near term. All lead exposure is preventable, however. The goal, therefore, must be to reduce exposure as much as is feasible. The many sources and pathways of lead exposure complicate the path to achieving this goal. Removing one pathway/source might produce only a modest reduction in blood lead level. That lead is an accumulative toxicant stored in multiple pools in the body besides blood introduces an additional complication. The partial data available demonstrating an average reduction of approximately 30% in the blood lead levels of residents of the affected public housing estates following interruption of the water pathway suggests to me that lead in the drinking water was, indeed, contributing to the exposure of the residents. I would anticipate that, over time, the residents' blood lead levels will re-equilibrate and reach a new steady state that reflects their lead exposure from other (non-water) sources and their endogenous lead sources reflecting past exposures. The blood lead concentrations achieved will therefore depend on what other sources/pathways contribute to an individual's lead exposure, as well as the magnitude of the individual's historical lead exposure.

Expert's Declaration

I, PROFESSOR DAVID C. BELLINGER DECLARE THAT:

1. I declare and confirm that I have read the Code of Conduct for Expert Witnesses as set out in Appendix D to the Rules of High Court, Cap. 4A and agree to be bound by it. I understand that my duty in providing this written report and giving evidence is to assist the Commission. I confirm that I have complied and will continue to comply with my duty.
2. I know of no conflict of interests of any kind, other than any which I have disclosed in my report.
3. I do not consider that any interest which I have disclosed affects my suitability as an expert witness on any issues on which I have given evidence.
4. I will advise the Commission if, between the date of my report and the hearing of the Commission, there is any change in circumstances which affect my opinion above.
5. I have been shown the sources of all information I have used in Appendix II.
6. I have exercised reasonable care and skill in order to be accurate and complete in preparing this report.
7. I have endeavoured to include in my report those matters, of which I have knowledge or of which I have been made aware, that might adversely affect the validity of my opinion. I have clearly stated any qualifications to my opinion.
8. I have not, without forming an independent view, included or excluded anything which has been suggested to me by others, including my instructing solicitors.
9. I will notify those instructing me immediately and confirm in writing if, for any reason, my existing report requires any correction or qualification.
10. I understand that:
    (a) my report will form the evidence to be given under oath or affirmation;
    (b) questions may be put to me in writing for the purposes of clarifying my report and that my answers shall be treated as part of my report and covered by my statement of truth;
    (c) the Commission may at any stage direct a discussion to take place between the experts for the purpose of identifying and discussing the issues to be investigated under the Terms of Reference, where possible reaching an agreed opinion on those issues and identifying what action, if any, may be taken to resolve any of the outstanding issues between the parties;

(d) the Commission may direct that following a discussion between the experts that a statement should be prepared showing those issues which are agreed, and those issues which are not agreed, together with a summary of the reasons for disagreeing;

(e) I may be required to attend the hearing of the Commission to be cross-examined on my report by Counsel of other party/parties;

(f) I am likely to be the subject of public adverse criticism by the Chairman and Commissioners of the Commission if the Commission concludes that I have not taken reasonable care in trying to meet the standards set out above.

Commission of Inquiry into Excess Lead
Found in Drinking Water

Statement of Truth

I confirm that I have made clear which facts and matters referred to in this report are within my own knowledge and which are not. Those that are within my own knowledge I confirm to be true. I believe that the opinions expressed in this report are honestly held.

Professor David C. Bellinger
1 December 2015

APPENDIX I

CURRICULUM VITAE OF PROFESSOR DAVID C. BELLINGER

DAVID C. BELLINGER

Professor of Neurology and Professor of Psychology at Harvard Medical School
Professor in Department of Environmental Health at Harvard School of Public Health
Developmental Psychologist (with additional training in epidemiology)

PERSONAL DETAILS

Year of birth:	1950	
Nationality:	United States of America	
Qualifications:	1971 B.A.	Williams College
	1977 Ph.D.	Psychology, Cornell University
	1987 M.Sc.	Epidemiology, Harvard School of Public Health
Awards:	1971	B.A. magna cum laude with Highest Honors, Williams College
	1970	Phi Beta Kappa, Williams College
	1977	Phi Kappa Phi, Cornell University
	1985-1990	National Institute of Environmental Health Sciences Research Career Development Award
	1996	MillerComm Lecturer, University of Illinois at Urbana-Champaign
	2008	Elsevier Distinguished Lecturer, Neurobehavioral Teratology Society
	2011	M.I.N.D. Institute Distinguished Lecturer, University of California-Davis
	2011	Jakob Hooisma Plenary Lecture, International Neurotoxicology Association
	2011	Environmental Health Perspectives, Reviewer of the Year
	2013	Newburger-Bellinger Cardiac Neurodevelopmental Award (inaugural recipient, with Jane W. Newburger)
	2013	Bernstein Lecturer, Department of Psychiatry, Boston Children's Hospital
Professional interests:	Environmental Epidemiology, Toxicology, Child Development, Public Health, Neuropsychology	

Other (voluntary) positions:

International Society for Children's Health and the Environment, President 2010-present
Editor-in-Chief, *Toxics* (2012-present)
Associate Editor (children's environmental health), *Environmental Health*, 2013-present

1993	World Health Organization/International Programme on Chemical Safety Task Group on Environmental Criteria for Lead, Brisbane, Australia
1997	World Health Organization/International Programme on Chemical Safety, Consultation on Methods Used to Study Neurobehavioral Development of Children Exposed In Utero to Methylmercury, Montreal, Canada
1998-1999	WHO Temporary Advisor, Joint FAO/WHO Expert Committee on Food Additives and Contaminants, Work Group on Methyl Mercury, Rome, Italy
1999-2000	WHO Temporary Advisor, Joint FAO/WHO Expert Committee on Food Additives and Contaminants, Work Group on Cadmium, Geneva, Switzerland
2002	World Health Organization/International Programme on Chemical Safety, Project to Update the Principles and Methods for the Risk Assessment of Chemicals in Food, London, England
2002-2003	WHO Temporary Advisor, Joint FAO/WHO Expert Committee on Food Additives and Contaminants, Work Group on Methylmercury, Work Group on Cadmium, Rome, Italy
2004-2005	Member, Joint FAO/WHO Expert Committee on Food Additives and Contaminants, Rome, Italy
2007-	Member, World Health Organization, Foodborne Disease Burden Epidemiology Reference Group (core group, Chemical Task Force, Source Attribution Task Force, Country Burden Task Force)
2009	Consultant, European Food Safety Authority, Use of the Benchmark Dose

	Approach in Risk Assessment
2009-2010	WHO Temporary Advisor, 72nd Joint FAO/WHO Expert Committee on Food Additives and Contaminants, Rome, Italy
2009-2010	WHO Temporary Advisor, 73rd Joint FAO/WHO Expert Committee on Food Additives and Contaminants, Geneva, Switzerland
2011-	WHO Guidelines on the Prevention and Management of Lead Poisoning, World Health Organization (Chairperson)
2011-	Member, WHO Expert Advisory Panel on Food Safety, World Health Organization
2014-	Member, Biology and Medicine Panel, Research Grants Council, University Grants Committee, Hong Kong

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Co-Edited

Needleman H, **Bellinger D**, editors. Prenatal Exposure to Environmental Toxicants: Developmental Consequences. Baltimore: The Johns Hopkins Press; 1994.

EMPLOYMENT EXPERIENCE

1977-1978	Public Health Service Postdoctoral Fellow, Department of Psychiatry, University of Rochester School of Medicine
1978-1979	Public Health Service Postdoctoral Fellow, Department of Psychology, Boston University
1983-1987	Instructor in Neurology (Psychology), Harvard Medical School
1987-1994	Assistant Professor of Neurology (Psychology), Harvard Medical School
1994-2003	Associate Professor of Neurology (Psychology), Harvard Medical School
2003-	Professor of Neurology, Harvard Medical School
2004-	Professor in the Department of Environmental Health, Harvard School of Public Health

2013- Professor of Psychology in the Department of Psychiatry, Harvard Medical School

1 December 2015

APPENDIX II

Commission of Inquiry into Excess Lead
Found in Drinking Water

DOCUMENTS PROVIDED TO PROFESSOR DAVID C. BELLINGER (for the purpose of this report)

1. Selected documents collected by the Commission from the Involved Parties since 20 August 2015.