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上午 10 時 05 分恢復聆訊

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許偉強先生：係，主席，喺 Professor Bellinger 畀證供之前，因為我哋之前就住有一位證人陳小華先生，咁佢就係何標記嘅前職員嚟。咁我哋就出過信畀佢，就係要求佢就做一張證人供詞，咁當時佢就冇回覆。然後我哋大概係喺呢個月 7 號、8 號左右，就出咗一個證人傳票畀佢，咁陳先生今日亦都嚟咗我哋聆訊嘅。

主席：好呀，唔該。陳先生。

陳小華：係。

主席：我係今次呢個專職委員會嘅主席陳慶偉法官。咁我哋較早之前就出咗一張證人傳票畀你，因為我哋就想就住今次嘅研訊有一啲問題係想

問你嘅。

陳小華：好。

主席：咁你冇律師代表你，係咪？

陳小華：冇。

主席：得，唔緊要，你自己代表自己都唔緊要嘅。

陳小華：唔。

主席：咁樣樣，我哋基本上就係想問你一啲問題關於你喺何標記工作嘅時候嘅情況，尤其是係買一啲焊料嘅時候嘅情況，明白吖嘛？

陳小華：明白。

主席：咁嗰個證人傳票就要求你今日就出庭，不過就--就出庭，但係就冇講到就話你係幾時會畀口供。咁樣樣我哋就首先就會我哋嘅律師團隊就會係同你約一個時間，可能係今日，可能係稍後嘅時間，咁樣樣就係方便你，如果你有時間嘅話，咁佢哋就想見你，同你攞一份嘅證人供詞，你明白吖嘛？

陳小華：明白。

主席：總之係一個大家都方便嘅時間。咁畀咗呢份證人供詞之後，你做完呢份證人供詞之後，咁喺稍後嘅時間，你就會係被傳召係出嚟做證人。至於實際上係邊一個月，邊一日，我哋暫時都未知，不過我哋又唔想要你係日日都嚟呢度等，所以我哋遲啲如果知道係幾時會傳召你，咁我哋早幾日就會通知你，就話畀你聽係幾時係會嚟，有冇問題？

陳小華：冇問題。

主席：冇問題，得，唔該。基本上就而家你就可以離開，跟住離開之後，我哋嘅律師團隊喺出面就會同你約一個時間，睇下係幾時同你落一份口供，明白吖嘛？

陳小華：明白。

主席：好，唔該。

陳小華：好。

許偉強先生：咁我而家要請 Professor Bellinger。

主席：好呀。

PROF DAVID CHUDLEIGH BELLINGER (sworn)

(All questions and answers were in English)

CHAIRMAN: Thank you. Take a seat please, Professor.

Examination-in-chief by MR KHAW

MR KHAW: Prof Bellinger, first of all, thank you very much for coming all the way to Hong Kong to provide assistance, as an expert appointed by this Commission for the purpose of this Inquiry.

We understand that you have prepared a report, dated 1 December this year. This report can actually be found in our e-bundle at V1. It is now shown on the screen. Your report has also been downloaded to the website of this Commission, so that in fact the public could have access to the same.

I don't intend to read out the contents of your report verbatim, as I don't think people here can bear my voice for so long. Instead, I will try to go through and summarise most of the issues that you have addressed in your report.

Unlike the chairman, my science knowledge is rather limited, so perhaps, in the course of going through your report, I will ask you to further explain some of the matters which might call for further discussion.

If I may now first turn to internal page 3 of your report, page 47 of the bundle, where you have set out the areas on which you have been instructed to provide your opinion for the purpose of this Inquiry. Perhaps I will just go through those items:

"(1) to explain the short, medium and/or long-term health effect(s) ... 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 ... ('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 ... for heavy metals and make recommendations, if necessary."

If we first look at the introductory part of your report, at internal page 4, page 48 of this bundle -- I think here, before you deal with the first question that we have just seen, you have provided some introductory comments on the availability of information on the adverse effects of lead on human health. It seems that you have come to a conclusion, at I think around the fifth line under "Introductory comments", that:

"... 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."

May I just ask you to briefly explain what you mean by "dose-response and dose-effect relationships"?

A. Yes. That's a distinction made in toxicology, referring

to how an adverse effect is related to exposure. A dose-response relationship, if one exists, means that the higher the dose an individual is exposed to, the greater the risk of some adverse effect such as cancer. And a dose-effect relationship pertains to an outcome that is not dichotomous, such as do you have cancer or don't you have cancer, but something like an intelligence quotient score. So a dose-effect relationship would mean that the higher the dose of some chemical an individual is exposed to, the IQ goes down, as dose increases.

Q. Thank you. Then you go on to say:

"In my response to the queries" -- ie the questions set out above -- "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."

If I may just ask you to explain this observation.

A. My understanding is that the highest water lead concentration that was measured in the Hong Kong estates was 83 micrograms per litre, but most of the levels were in the range of 10 to 20 micrograms per litre.

According to our current knowledge, regularly consuming water with water lead in the concentration of 10 to 20 would be unlikely to give a large enough dose that someone would show clinical signs of lead toxicity, which tend to occur at much higher blood lead levels, in the range of -- it varies from individual to individual, but it's rare to see clinical symptoms in someone with a blood lead below 40, 45 micrograms per decilitre, and higher than the blood lead levels observed in the residents of those estates.

Q. Under the same paragraph, you then continue to talk about the overt signs of acute intoxication.

CHAIRMAN: Before you ask that question, can I ask you, Prof Bellinger, how long can one be regarded as chronically exposed?

A. That's a very difficult question to answer. It typically refers to a period of months to years, would be chronic exposure. Anything else shorter would be considered more of an acute exposure.

CHAIRMAN: Thank you.

MR KHAW: Under the same paragraph, you then continue to talk about the acute -- the overt signs of acute intoxication, acute lead intoxication, and also chronic lead exposure.

Then, if we turn to internal page 5, the second

paragraph, about the second sentence, you mention that:

"The major classes and 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 ([including the] use of contaminated folk or herbal medicines)."

Then further down, in about the middle, you talk about the places in a person's body where lead resides. I think, in short, just to summarise what you have set out here, in adults lead in bone accounts for about 90 per cent of the total body burden, whereas in children, lead in bone accounts for about 70 per cent of the total body burden, because in the case of children lead will move in and out of bone more rapidly, due to the changes in bone structure.

Then there is a discussion regarding re-equilibration process. If I may just quote what you have stated here, about the last ten lines on this page, starting from the word "Therefore" -- can you see that?

"Therefore, the blood lead level measure for an individual at any time reflects the equilibrium between an individual's current exposure to 'new' lead and the 'legacy' lead" -- ie the old lead which has accumulated in our body for a relatively long period of

time -- 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 ..."

May I just ask you to elaborate a bit more on the re-equilibration process that you have just mentioned here.

A. Certainly. Over time, the lead that is distributed into the three major compartments, the mineralised tissue, the soft tissue -- by which I mean organs such as the brain, kidneys and the liver -- and the blood is the third compartment, will come to a balance that reflects the amount of lead someone is taking in and the amount that they are excreting.

Now, if there's some intervention that changes an individual's exposure at a given time, such as removing exposure to water lead or administering -- in

cases of much more severe exposure, giving drugs that bind to the lead and promote its excretion from the body -- then the amount of lead that's distributed in those three major compartments will find a new balance. And what happens is if blood lead is reduced because of cutting off an exposure pathway, blood lead will decline reflecting that, but lead from the other two compartments, the hard and the soft tissue, will come into the blood compartment and a new balance will be achieved between those three compartments.

How long that takes will reflect how much the exposure is decreased and how much past lead an individual has been exposed to and is storing in those other compartments.

Q. Thank you. Then you continue to talk about a study, one study of children -- at internal page 6, page 50 of the bundle -- who had a blood lead level of 25 to 29 micrograms per decilitre and were placed in a case management system; it took an average of two years for the blood lead to drop below 10.

Can you provide any reasons for this slow drop in that study?

A. Yes. That's because children who had a blood lead of 25 to 29, in this particular study were living in homes that had deteriorated lead paint, and they had been

living there for some time, so they had been continuously exposed and had built up a considerable amount of lead in their bones and in their soft tissues, so that when the children were removed from those homes and placed into the case management system, which means that their current exposure was drastically reduced, because they were removed from the hazardous environment they had been in, then their blood lead fell quite slowly, because of this equilibration process, because they had been exposed to so much lead in the past that during the re-equilibration it was being drawn out of the hard tissues and the soft tissues into the blood, so that the blood lead fell very, very slowly, certainly longer you would expect from the simple fact of a half-life of lead being 30 days. That's why you can't expect blood lead to drop by a half in 30 days. It depends on the entire exposure history of an individual.

Q. Thank you.

I think most people would be interested in the next topic, because that goes to the IQ score of children. Most parents want their children to obtain a high IQ score and they would like to see whether the lead content in water --

CHAIRMAN: You have to speak up, Mr Khaw.

MR KHAW: Sorry. I think most parents want their children

to achieve a high IQ score, and they also want to know the effect of lead content in water on this particular aspect.

You have started your discussion by saying that young children are considered to be most vulnerable subgroup of the population. Then you went on to say that children with blood lead levels below 25 micrograms per decilitre generally do not show any signs or symptoms that bring them to medical attention. But studies also show 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 wellbeing.

Then you talk about what you call the most complete and compelling evidence in respect of children. That is I think the last ten lines on this page:

"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 [microgram per decilitre] increase in blood lead concentration), was steeper over the range below 10 ...

than it was over the range between 10 and 30 ... Although the biological mechanism of this is not known, the finding has now been replicated in several independent studies."

Professor, just one question here. We all know that children's IQ score may be affected by various factors -- environmental factors, behavioural factors, et cetera -- so to what extent can these studies on lead content demonstrate the extent of the impact of lead on children's IQ scores?

A. That's a very important question, and it's always -- whether or not investigators have addressed and measured and taken adequately into account the other factors that influence a child's IQ is always the major point of contention in interpreting a study such as lead, because we can't do an experimental study with children like we can with animals. We have to observe samples of children who are exposed to different amounts of lead and see if we can see an association with their cognitive outcomes.

So in each study -- and as I say, it's very important to measure the parents' socioeconomic status, their education, if possible the parents' IQ, because that's very strongly predictive of a child's IQ. It's important to take account of a child's nutrition, how

stimulating the home environment is, whether they are exposed to other things that can pose a threat to their cognition.

So the statistical models that have to be implemented in these studies can be quite sophisticated and very detailed. And what we find is if we take account of all of these other factors to the best that we can, is there still a relationship between lead and children's IQ, and what these studies indicate is yes, there is.

We can also get a sense of how important lead is in the context of all these other factors by asking what per cent of the variability in children's IQ scores can be explained by their lead exposure. Typically, what we find is that it's a relatively small amount. It's statistically significant and consistent across studies, but it explains usually less than 5 per cent of the variation in children's IQ, whereas something like socioeconomic status captures about 30 per cent of the variation in children's IQ.

So lead is important but it's not the most important predictor of children's outcomes, and this is something that I counsel parents about when they call me, when they are concerned that their child has a blood lead higher than we would like to see. I tell them, yes,

that's very important and we certainly want to prevent that lead exposure, but it's important to put it in the perspective of what else may be influencing how their child does, and I try to help them relax a little bit in this regard; that yes, we should prevent lead exposure, but it's also important to do these other things that parents do that maximise their child's endpoints.

CHAIRMAN: Can I ask you a more basic question first: why is lead so toxic?

A. That's a good question. It works at a very general biological level, and so it impairs processes that are fundamental to many aspects of the way our bodies work. Chemically, it's what's called a divalent cation; it has a 2-plus charge, and many important things in the body like calcium have a 2-plus charge. So lead interferes with a lot of the biological processes that depend upon calcium, and many of our body processes do depend on calcium.

So how the nerve cells in our brain communicate with one another is dependent on calcium, so lead interferes with the communication between cells in the brain. It also interferes with processes that are important in long-term memory and storage of information. In fact, lead interferes with just about everything that anybody has looked at in the brain. So that explains his

pervasive and widespread toxicity.

CHAIRMAN: Thank you. So that's why you say the central nervous system is considered to be the most vulnerable organ among all the organs within our bodies?

A. That's right, in particular the developing central nervous system. As a colleague of mine says, a child gets only one chance to develop a brain, and so we want to prevent lead from interfering with the complex spatial and temporal choreography that's involved in putting a brain together, that involves billions of nerve cells that have to be in the right place at the right time in order for a brain to be normal.

CHAIRMAN: So when you say the central nervous systems, you are not confining yourself to simply the brain; you are also talking about the nerve cells, the nerves that can be found in our bodies, the whole body as well?

A. Well, the central nervous system typically is used to refer only to the brain and the spinal cord, and the connections of the central nervous system to, like, the hands and sensory organs in other parts of our bodies is the peripheral nervous system. That is also sensitive to lead but less so. Often the effects in the peripheral nervous system are reversible, once exposure stops. But we are concerned that the effects are more persistent in the central nervous system.

CHAIRMAN: Is that because the lead displaces the calcium in the process of transmission of nerve messages; that actually adversely affects the development of children?

A. Yes, that's part of it. These chemicals that lead interferes with are involved not just in the function, the transmission of information, but also they are involved in the formation, the laying down of the structure, the fine structure of the nervous system. So lead is both a developmental neurotoxicant and a functional neurotoxicant. That's why the developing nervous system is of particular concern, because lead can interfere with actual structural formation of the brain.

CHAIRMAN: Thank you.

MR KHAW: Still at internal page 6, the last sentence is perhaps quite important:

"The details of this dose-effect relationship suggest 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 [micrograms per decilitre]."

The reason why I ask you in relation to this particular sentence is since your report has been released, there has been some media coverage which has taken this sentence to mean that the IQ of a child with

a blood lead level of 10 micrograms per decilitre would be one-third lower than that of a child with zero blood lead content, but in fact you are talking about one-third standard deviation here. Can you explain a bit more on the standard deviation point?

A. Yes, that would be an incorrect interpretation.

Standard deviation is a property of a bell curve distribution. It refers to how much variability there is in a particular characteristic. So, for instance, with regard to IQ, we expect that the average person will have an IQ of 100, and the standard deviation of the IQ distribution is 15 points.

So this is just a way of saying how many people we expect to have IQ scores within certain ranges, so between the mean of 100 and plus 1 standard deviation, that is a score of 115, we would expect 34 per cent of the population to be between 100 and 115. Similarly, because the distribution is symmetrical, we would expect 34 per cent to have a score between 85 and 100.

So the one-third standard deviation just simply means that the effect that has been observed in studies that's attributable to lead, the effect on IQ, amounts to five points, for an increase in blood lead from 0 to 10, which is one-third of that standard deviation. It doesn't mean that the child with a blood lead of 10

would have an IQ that's only one-third as large as the child with a score or a blood lead of 0.

Q. Thank you. In the next paragraph on the same page, you have referred to a series of neuroimaging studies of young adults, and those studies actually show some structural or ischemic changes of various parts of the brain, including the grey matter volume and also the white matter volume.

Can you just briefly explain the importance of these parts of our brain?

A. The grey matter is basically what we think with, so it's the cerebral cortex; we have a frontal lobe, occipital lobe, parietal lobe on both sides of our brain, and that's where most of our thinking goes on; and there are also some subcortical structures that are part of the grey matter that are also important, the basal ganglia and so on.

The white matter are basically the information highways in our brain. The white matter tracts are quite long and they connect different parts of our brain, so they are the way that our different parts communicate with one another.

So they are all important in terms of having a healthy brain. We want both grey and white matter to be functioning as they were intended.

Q. In these studies which actually show persistent changes in brain structure and function, are we talking about blood lead content which is quite high?

A. We are. The neuroimaging studies come from a particular study that was conducted in the United States in Cincinnatti, among mostly minority African-American children who were living in fairly dire situations of poverty; inner city, in poor housing, exposed to lots of lead. They had blood leads that were -- most of the children in the study, there were about 250 children, had at some point in their life a blood lead above 25, and some of them had blood lead so high that they were actually hospitalised and given chelation therapy to reduce their lead burden.

So these were children with exposures that were not that uncommon at that time -- this study was begun in around 1980, in the US -- but fortunately they're fairly rare these days, and it's uncertain the degree to which these studies apply to children with lower blood lead levels. They are simply a lesson that lead is actually doing observable damage to the brain.

Q. Then the last paragraph on the same page, where you are addressing the issue regarding children and teenagers between 6 and 18 years. I think you have also referred to various studies which provide evidence regarding the

inverse association between early-life lead exposure and neurodevelopment.

Then at page 8 you talk about the US National Toxicology Program evaluation, which concluded that the evidence is sufficient for the association between blood lead level under 10 micrograms per decilitre and both decreased hearing, delayed puberty, and reduced postnatal growth.

Again, I just want to ask whether these studies showed relatively high blood lead content?

A. Well, this conclusion pertains to a blood lead range of less than 10 micrograms per decilitre. So, again, these are findings that are relatively consistent across studies, sufficiently consistent that the NTP was willing to consider that in aggregate the evidence was sufficient to draw this conclusion.

But again I would say that finding a statically significant association is one thing, and finding an association of a large magnitude in the effect is another thing.

Q. Yes.

A. So, for instance, this reduced postnatal growth association, it may be relatively modest and something that you would not notice in an individual child, but if you are looking at large groups of children then you

might find that those children with higher blood lead within this range were a centimetre smaller than children with blood lead levels lower than this range. Statistically significant but not really very meaningful for an individual child.

Q. Then in the next paragraph you talk about the association between lead exposure and ADHD, attention deficit hyperactivity disorder. Then you have referred to various studies in this respect.

May I just ask you how these studies were conducted, regarding the association between lead exposure and ADHD?

A. A variety of study designs were used. In some of the studies, which are called cross-sectional studies, a sample of children were recruited for participating in some large survey, and as part of the survey a blood sample was taken, and that was measured for blood lead content.

Then the parents were interviewed and asked, "Has a medical professional ever told you that your child has ADHD?" In other studies, the parents were given a structured interview that was linked to the criteria for diagnosing ADHD, and in yet other kinds of studies, called case control studies, two groups of children were studied. One group had been identified and diagnosed

with ADHD, and the other had not, and then blood samples were collected to see whether or not the group carrying the diagnosis of ADHD had a significantly higher blood lead level than the group that did not.

Q. In the last paragraph on this page, you talk about various studies which show the association between childhood lead exposure and propensity for violence and aggression. Then you went on to say that in fact these studies are difficult to conduct and they are subject to a variety of biases. Why do you say that?

A. Well, in order to try to see if there's an association between early childhood lead exposure, which is the primary concern, and the propensity to engage in these kinds of behaviours 20 years later, it's very difficult to identify a situation in which you have those data.

If you do the study by identifying individuals who already have committed offences, it's difficult to look back and get reliable information about their early childhood lead exposure. Typically, you don't have access to those data. So one of the biases would be -- it's called a retrospective bias; whereas people or parents of individuals who are offenders, as adults may have different levels of recall. They may be more likely to say, "Oh, yes, he was lead poisoned as a child" and you don't really know whether that's true

or not or if they are just looking for an explanation for their child's behaviour.

So it's difficult to find exactly the right situation in which you can address this question in a very compelling way.

The study that I mentioned where the neuroimaging was conducted, a study in Cincinnatti, has provided the best evidence, in my mind, of this association, because they enrolled these children during pregnancy. As the children grew older, they collected periodically blood lead histories, and then they followed the children until they reached their 20s, and they went to administrative county records where these individuals now resided and obtained information from those objective records as to the number of times these participants had been arrested for some offence or another.

So that study, which is very difficult to carry out over that period of time, is less subject to these biases because the blood lead information was collected prospectively, long before the offending occurred, and so there's no concern about bias there. Also because this study had collected a lot of information about factors that I mentioned earlier that are important to take account of -- family socioeconomic status,

nutrition, stimulating-ness of the home environment, and so on -- the associations that they saw between early childhood lead exposure and risk of offending and being arrested later in life I think are much more solid; they provide a much more compelling base of evidence for drawing a conclusion that lead is linked to a propensity for aggression and violence.

But I would add, again, that participants in the study had lead exposures that were quite high by contemporary standards and many of these children actually got chelated because of their lead poisoning.

Q. Then in relation to pregnant women, page 9, in the third line you mention that:

"... the lead exposure of a foetus is essentially the same as that of the pregnant woman."

Then in the next paragraph you refer to research on the potential effects of lead exposure on the health of pregnant women, in particular reproductive health, the course of pregnancy, the health of the foetus at birth, et cetera. Then you have also referred to the US Centers for Disease Control, which "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."

Then you go on to discuss various aspects regarding pregnant women, including fertility, hypertension, et cetera.

In relation to gestational hypertension, at page 10, there's just one question I have. Here, you say:

"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 ..."

Just one question: would such limited magnitude affect the validity of the findings in those studies?

A. No, it doesn't affect the validity of the findings, because the findings have been reported in multiple studies. So I think that they are real. But the modest magnitude does affect the interpretation of the clinical significance.

Again, as I indicate, the correlation, which is a measure of the degree of relatedness of blood pressure to blood lead, is quite small, meaning that most of the variation among pregnant women in their blood pressure or risk of hypertension is not due to lead, it's due to

other factors. Again, we know how to prevent lead exposure and so it's worthwhile to do everything we can, but it won't solve the problem of pregnancy hypertension entirely.

Q. Then, in relation to spontaneous abortion, you first mention that:

"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 [micrograms per decilitre]."

Then you refer to one high-quality prospective study conducted in Mexico City which actually reported that "the risk began to increase, and increased consistently thereafter, when maternal blood lead level exceeded 5 [micrograms per decilitre]".

Are there any special features in this particular study conducted in Mexico City which show this result?

A. Well, I say it's high quality because of the care that the investigators took to try to identify potential confounding factors that may also be related to risk of spontaneous abortion. As I say, this study did suggest that the risk of spontaneous abortion begins to rise once maternal blood lead exceeds 5 micrograms per decilitre. I have no reason to think that the study is wrong, but in this kind of work one never draws a strong

conclusion based on one epidemiological study, because it's always possible there's something unique about Mexico City and the women and their practices that may have contributed to this result. So it's always helpful to be able to have evidence that's been collected in multiple studies, that all point towards the same direction.

So I think this study is important in telling us that there may be something, an important signal here, but until we have additional evidence I would be reticent to draw a very strong conclusion.

Q. Then in relation to foetal growth, that is the next passage, you have mentioned that the evidence regarding the association between lead exposure and foetal growth is perhaps not that consistent.

A. Right.

Q. Can you explain a bit more on this?

A. Well, I just mean that some studies find that increased blood lead during pregnancy is associated with a reduced length of gestation. Other studies don't find that. The same with the other endpoints, the birth weight, head circumference, birth length. So I find it very difficult to know what the truth is. Until we get a heavier weight of evidence pointing in one direction, it's difficult to draw a strong conclusion, in my mind.

CHAIRMAN: But logically, it should have a greater adverse effect on the baby's growth, because the baby's brain is developing at the time?

A. I'm sorry?

CHAIRMAN: Because the baby's brain is developing at the time, so logically it should have a greater adverse effect on foetal growth?

A. Yes, you would think so. Interestingly, the evidence has not borne that out.

CHAIRMAN: I see.

A. And I don't have an explanation for it. It appears to be that the exposure of a postnatal child is a stronger predictor of their IQ and educational achievement than is their prenatal exposure. It may be that there are some protective features in the foetal unit that protect them, protect the foetal brain, from the lead.

It's an important question, and it is a little counter-intuitive, but so far the evidence just doesn't bear it out.

CHAIRMAN: Thank you.

MR KHAW: Perhaps there is just one minor typo, in the last paragraph on this page:

"The NTP concluded that the evidence was ..."

I think the word "is" is perhaps redundant.

A. Yes. I apologise for that.

Q. If we turn to the next page, in relation to lactating women, in the first paragraph, about the sixth line, you have mentioned:

"... the concentration of lead in breastmilk is low, comparable to that in the plasma fraction of blood (which accounts for only about 1 per cent of the lead in whole blood). Thus, this pathway of exposure likely contributes relatively little to an infant's lead exposure."

Then you went on to say:

"Water can be a very important pathway of lead exposure for infants who consume formula" -- milk powder -- "made up with water that contains lead. Balancing the known benefits of breastfeeding and the slight risks of substantial lead exposure from breastfeeding, the US CDC encourages mothers with a blood lead level [not exceeding 40 micrograms per decilitre] to breastfeed."

So I take it that so long as the blood lead level of the mother is not higher than 40 micrograms per decilitre, it is still safe for her to breastfeed. Is that the case?

A. That's correct.

Q. Regarding elderly people, we have the same discussion in relation to legacy lead, the old lead that accumulates

in the body.

If we turn to page 12, internal page 12, you have mentioned one recent development in lead toxicology which is generating considerable interest. That is the study regarding the association between overexpression of genes involved in the production of a protein, which is a constituent of the plaques that are found in the brains of patients with Alzheimer's Disease with lead exposure.

Can you explain a bit more on this study, on whether it is applicable to human beings?

A. Well, the simple answer is we don't know. The studies have only been done in rodents and monkeys at this point. This is another case where it's very difficult to do this study in humans because the finding in the animals is that it's the exposure to lead in very early life that predicts this late overexpression of these genes involved in neurodegeneration.

To do this study in humans, we would have to have a prospective cohort that we had early-life lead exposure measures on, and follow them up to age 60 and beyond, and unfortunately that's very difficult to do and usual people have to take advantage of other data that had been collected, in which this question can be asked. But there are relatively smaller opportunities

to do that.

There is one study in humans that I was involved in where, at age 30, we looked to see whether, on children for whom we did have good lead exposure histories, we looked to see whether or not there was a relationship to the level of expression of genes that are involved in this amyloid protein synthesis pathway.

Now, what we are really concerned about is whether or not the genes are being over-expressed in the brain, but we obviously can't do that, so we were limited to seeing whether they were over-expressed in the blood, and we did see some suggestive relationships between gene expression of individuals around 30 years of age in relation to actually their cord blood lead levels. That's the only study that we have in humans at the moment.

So this hypothesis remains highly speculative, but it is of concern because the current elderly population grew up in a time when we were exposed to much more lead than our current cohorts of children, and so we do know that the incidence of Alzheimer's Disease is rapidly increasing as our population ages, so it's reasonably -- and it's biologically plausible to think that lead might be involved in this process, but again it's too soon to draw very strong conclusions.

Q. Thank you.

Further down this page, at 1h, you talk about long-term patients with chronic illnesses. Then you have referred to kidney function, cardiovascular function and also cognitive function.

There's just one aspect regarding cognitive function that I would like you to elaborate. That is, in the middle of this particular passage regarding cognitive function, internal page 13, you have mentioned at line 4:

"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' operationalised as poorer reading achievement, an outcome that presumably pre-dated the onset of occupational exposure to lead."

Can I just ask you to explain a bit about the low cognitive reserve point that you have mentioned here?

A. Yes. That's a concept that pertains to how readily an individual can be protected from an adverse effect of an exposure such as lead. It's thought that if an individual who is cognitively functioning at a higher level, as reflected in a higher reading achievement or other scholastic achievement, may be less affected by

a given exposure, such as lead, and so show fewer decrements in performance as a result. So, basically, they are buffered, and it's people whose cognitive performance is more fragile who may suffer a greater impact.

Q. Then, regarding the WHO standards that we have talked about very often at this Inquiry, you first discussed the lead content in tap water and then you have gone through the changes in relation to the WHO Guidelines. May I just first ask you this: can you briefly let us know your personal participation in the formulation of the WHO Guidelines over the years?

A. I have not been involved in the guidelines pertaining to water lead. I currently chair a committee at the WHO that's developing guidelines for the diagnosis and treatment and prevention of lead poisoning in children and adults. I've been involved in the lead evaluations for the WHO/FAO joint expert committee on food additives and contaminants that has set provisional tolerable weekly intake for lead, that is the basis for the water lead guidelines. Then I have also been involved in the Foodborne Disease Epidemiology Reference Group, which is developing estimates of the global burden of foodborne disease, and I have been involved in the chemical aspect of that process and lead is one of the chemicals that we

have developed estimates for.

Q. Thank you. We all know that the current reference value regarding lead in water is 10 micrograms per litre. At internal page 14, the first paragraph, perhaps the last six or seven lines of this particular paragraph, you have mentioned that:

"... this is designated as 'provisional' on the basis of treatment performance and analytical achievability."

Then you went on to say:

"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 emphasised that all other practical measures to reduce total exposure to lead, including corrosion control, should be implemented."

Do you have anything further to add in this respect?

A. Not really. Unlike many chemicals which are present in the source water, where you can take measures at that point in the system, lead is different, in that it is introduced into the system at places closer to the point of consumption. So I think the WHO is recognising that to remove plumbing and fittings is costly, and so they

recommend that as a first preliminary step, to take measures to reduce corrosivity of the water it, to reduce leaching of lead, and presumably they would endorse, if that doesn't work, then to take the more costly step of replacing the fittings.

CHAIRMAN: So does that mean that that was known to the WHO back even in 1958, when they first developed the allowable limit?

A. I don't know the answer, what they knew back in 1958. Certainly lead in water and the contribution of lead in plumbing and actually the use of lead to make pipes was known back then. But I don't have specific knowledge of that.

CHAIRMAN: But certainly it was known back in 1974, when the United States passed this Safe Drinking Water Act (1974)?

A. Again, that was before my time. I don't know what the --

CHAIRMAN: Okay. Thank you.

MR KHAW: In relation to blood lead content, we know that the current reference values are 5 micrograms per decilitre for children and 10 for adults. You have also mentioned at internal page 15 --

CHAIRMAN: Before you move to page 15, I note on page 14 the United States adopts sort of a higher value,

15 micrograms. Is there a rationale behind that?

A. Well, that's not the only respect in which the EPA's guideline is different than the WHO's, because the EPA does allow up to 10 per cent of violation, of 15, before some action is mandated.

CHAIRMAN: I see.

A. They will allow up to 10 per cent to be above --

CHAIRMAN: So 16.5?

A. Yes. So all of these guidelines really try to balance health-based considerations with practicality, and I think, you know, that's why they are all provisional, because the health information and the technologies improve, and so over time permit the implementation of more stringent guidelines. But this is where they are at the moment for the EPA.

CHAIRMAN: I understand the ideal value of course is zero. Does that mean that 16.5 is still acceptable?

A. According to the EPA --

CHAIRMAN: But what about according to you?

A. You know, it's hard to take one number out of context.

CHAIRMAN: Right.

A. It would depend upon -- because, as I've said, lead is a multimedia pollutant, so we have to take a look at the entire profile of exposure sources -- the paint, the air, the soil, the diet, hobbies, occupations. For some

people, having a 16.5 might be relatively trivial because they are not exposed to lead in any other aspects of their life, but for other people, who have a higher baseline exposure, that may be enough to put them into a range where we need to be concerned.

So I think it really is hard to give one answer.

CHAIRMAN: I see, that's what you mean. So it's the blood lead level that matters, rather than the lead in water?

A. In my view, that's what we use to make decisions, management decisions, as to whether or not we need to help an individual reduce their exposure. It's what's in their body, that to me is the index of likelihood of an adverse effect.

CHAIRMAN: Thank you.

MR KHAW: In relation to the blood lead level, you have come to a conclusion at internal page 16 that the reference values selected by the Hong Kong government for prioritising individuals for follow-up based on blood lead level are appropriate and consistent with those identified by the international bodies.

A. That's right.

Q. You can confirm that?

A. Yes, I do.

Q. If we then take a look at the recommendations for follow-up actions in respect of both children and

adults, as suggested by the CDC of the United States, at internal page 17. If we talk about children first, the first table, we can see in the left-hand column, blood lead concentration less than 5 micrograms per decilitre, and then we can see that the recommendations for follow-up actions are more limited. They consist of lead education, environmental assessment, and then follow-up blood lead monitoring.

In the next column, if we are talking about blood lead concentration which is in the range of 5 to 45 micrograms per decilitre, then there have been more recommendations for follow-up actions. For example, we have things like environmental investigation, lead hazard reduction, neurodevelopmental monitoring, abdominal X-ray.

First of all, in relation to environmental investigation, and neurodevelopmental monitoring, can you tell us what steps are needed for these follow-up actions?

A. Yes. Usually, the environmental investigation would involve querying a parent about possible lead sources in the environment, finding out where a child spends time, in addition to the home. That of course would be age-dependent. Then, depending on the blood lead level, actually going into the home to collect samples of

different media that the child might come in contact with, such as collecting air samples or dust samples, samples of the soil outside the home, the paint, and noting whether or not the paint is chipping, and asking the parent whether or not they have seen the child put things in their mouth, the child put things in their mouth that are not food. In the extreme it's called pica; asking about occupations of the parents, whether they might be involved in an occupation that involves exposure to lead and so may bring lead home on work clothes; asking whether the parents engage in hobbies that might involve the use of lead and so could contaminate the home environment; asking about foods that the family eats, the medicines they take, whether they take herbal medicines, which unfortunately have been reported, in some instances, to have fairly high lead content; looking at the materials that the family use to prepare food and to serve food -- sometimes, at least in the United States, they are glazed with lead glaze which can leach lead and so get into the food and be consumed -- looking at the toys the child plays with and the furniture, whether it's painted with potentially lead paint.

Really, it's taking a comprehensive look at the environment that the child lives in, to identify

potential sources, because it can be very hard to tell.

Q. What about neurodevelopmental monitoring?

A. This could vary in intensity as well, depending upon a child's blood lead concentration. It could be as simple as asking the parent about a child's developmental milestones, when they first sat unaided, when they first took their first step, said their first word, said their first sentence; or it could involve an in-person assessment using a screening tool by a medical professional; or, if there are red flags that are raised by any of these screening methods, actual administration of more comprehensive neurodevelopmental assessments for diagnostic purposes.

So it really is based on the level of lead exposure and a clinical indication.

Q. Thank you.

In relation to adults, that is the last table on the same page, we can see that for blood lead concentration which is in the range of 10 to 29 micrograms per decilitre, then various follow-up actions are recommended.

Then we can see things like exposure assessment. I take it that that is similar to the environmental investigation that you have just mentioned?

A. That's correct, yes.

Q. Thank you.

Then, talking about the test results that we have got here in Hong Kong, the next part of your report actually addresses the adequacy of the care plan as devised by the Department of Health. Before you talk about the adequacy of the care plan, you have provided a summary of the results, and I understand that at the fifth line you have mentioned that the results of repeat blood lead tests are reported for 28 individuals.

I understand that at the time when you were compiling the report, the information set out here was based on the first list of test results that you gathered at the time?

A. That's right, the 22 October 2015 line listing.

Q. Because in fact subsequently there have been two updated lists, one dated 19 November 2015, and also the latest one is as of 3 December 2015.

I think now you have had a chance to look at those two updated lists, and just for reference sake, the second list, as of 19 November this year, can be found at E2/770 to 773, and the latest one is at E2/850 to 853.

I understand, Professor, that you have had a chance to now look at these updated lists of blood test results. Do you have anything to add to your original

summary of the test results?

A. Well, I didn't have a chance to carry out the same calculations that I carried out on the 22 October list.

I do note that there is one additional follow-up test on a pregnant woman.

Q. Yes.

A. I reported on the four pregnant women, so there are now five. I had calculated an average change, a decrease in blood lead level, over the one month follow-up interval, of 13.7 per cent for those four women for whom I had the data at the time.

The fifth woman had a decline of 14.3 per cent between her initial and follow-up blood test. So her data are very consistent with those that I saw for the other four.

Looking at non-pregnant individuals, I think what I had seen in the initial group of 24 non-pregnant individuals was a decline between the baseline or first blood test and the follow-up blood test of about 30 per cent. Without having carried out the calculations, by just estimation, it looks like the additional data that are now represented in the line listing are quite consistent with that.

There is one individual that was of some concern to me: a two-year-old female whose first blood test was

6.48 micrograms per decilitre, and her follow-up three months later was 27.32 micrograms per decilitre, and then a blood lead conducted one month after that, it was 21.37, and three weeks after that it was 15.3.

So I'm very happy to see that this child was followed up so assiduously and it's good that her blood lead was coming down. It does suggest, however, that there was something in her environment, certainly not the water because that had been stopped, but she was exposed to something between the first and the follow-up -- first follow-up blood lead -- that led to quite a spike in her blood lead level.

But apart from her, everyone else seems to be a nice decline in their blood lead; as I say, averaging about 30 per cent.

Q. Thank you.

Just for record purposes, the two-year-old girl that you have just mentioned, I think the result of her blood test can be shown at page 851 of E2. I think her result is the one just above the middle of this page. The first follow-up blood test result, it's about 27.32.

I will now move on to discuss your recommendations in relation to the care plan devised by the Department of Health.

I am just wondering if this is an appropriate time

for a break?

CHAIRMAN: Yes. Let's first take a 20-minute coffee break and then resume later. Thank you.

(11.28 am)

(A short adjournment)

(11.49 am)

MR KHAW: Regarding the care plan which is discussed at internal page 19 of your report, you have provided your opinion that "the general components of the care plan proposed for the residents are appropriate, although some are not described in detail".

Then you go on to say:

"... 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 ... outside activities [et cetera] ..."

Then you have also mentioned that:

"The care plan also stipulates a 'health evaluation' ... for individuals with a blood lead level of 5 [to] 44 ... and a 'medical assessment' for individuals with a blood lead level [exceeding] 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."

So there you talk about what is actually lacking or what was then lacking in the care plan that you saw at that time.

Now, I understand that the lawyers representing the Department of Health issued a letter to us yesterday, providing more information regarding the care plan. Perhaps we can take a look at such information and then I'll probably ask for your views on whether you find those plans, those existing plans, adequate or not.

If we can first take a look at E2, tab 71, at page 846. Page 846 talks about the updated tables and also the updated blood test results. I think we have just clarified that point earlier.

Then if we can focus on the next page, that is page 847. Again, we have dealt with the issues arising from the first paragraph, regarding the difference between 28 individuals and 29 individuals, because according to the first list there were 28 individuals, as mentioned in your report, and if we look at the updated list, there are actually 29 individuals.

If we can then move on to look at the second

paragraph, from the second paragraph onwards it says:

"As for the 'exposure assessment' mentioned in page 19 of [your report], a lead exposure assessment ... is conducted by a nurse of the Department of Health ... for every individual with elevated blood lead level. For selected circumstances, [lead exposure assessment] will be supplemented by a home visit. Relevant documents including the questionnaire and reference materials used for conducting lead exposure assessments, protocol and flowchart for home visits of cases with elevated blood lead level are included in the above-mentioned updated 'Master List of documents published'."

If we can now perhaps take a quick look at the issue of the home visit, at page 870. In fact, this is a document entitled, "Investigation protocol for elevated blood lead level", published by the Department of Health. If we take a look at "Home visit", page 870, paragraph 13, it says:

"Home visit provides a good opportunity for health education to the case and household members. Also, any probable source(s) of lead exposure of the case may be identified through home visit so as to guide risk reduction measures. The visiting team should take photo(s) of any highly suspicious item(s) in the

household and discuss with the Government Laboratory for the standard and the methodology of analysis prior to collection of any specimen(s).

14. Home visit should be conducted under the following circumstances:

-- Single venous [blood lead level exceeding or equal to 20 micrograms per decilitre] ...; or

-- Any suspicious source(s) of lead exposure is identified ...

-- Follow-up BLL(s) is persistently elevated ..."

So these are the circumstances which call for home visits, according to the measures now taken by the Department of Health.

Do you find this suggestion for home visit sufficient for this specified category of residents?

A. I do. I am very impressed, actually. I particularly like the second bullet point, "Any suspicious source(s) of lead exposure is identified", as cause for a home visit.

Now, I assume it's identified by means of an initial interview and survey of the informant about what potential sources in the home might be. The reason I like this very much is because it really focuses on prevention. It doesn't require that someone be exposed before the home visit is done to reduce exposure and it

holds the potential of actually preventing the exposure.

So I think this is very well thought-out, yes.

Q. If we go back to the letter from the Department of Health, page 847, there are a few more suggestions here. If we take a look at the third paragraph, starting from the words:

"Regarding the 'developmental assessment' mentioned in page 19 of the expert report, all children with elevated blood lead level will receive preliminary developmental assessment at [Department of Health's] Child Assessment Centres ... or Student Health Service Special Assessment Centres ... by a developmental surveillance team composed of [doctors] and nurses. The developmental assessment covers major developmental areas including gross and fine motor, language and communication, cognition, learning, behaviour and emotion, self-care, vision and hearing. This is conducted through history taking (including birth, developmental and medical history, daily and school functioning and parents concern on development and behaviour), reviewing school reports and school work, physical examination (including soft neurological signs), clinical observation and use of questionnaires and assessment tools."

Then finally it says:

"Children identified with developmental or behavioural problems will have follow-up evaluation at Child Assessment Service according to individual needs. Children with largely normal development would receive continuous monitoring through enhanced developmental surveillance at Maternal and Child Health Centres ... during pre-school years and annual health visits at student health centres during school years. Parents are provided with anticipatory developmental guidance and information on children's development in the form of pamphlets."

If we can then take a look at the table which I think basically describes the details of this particular plan, at page 871, we can see various -- the table under paragraph 17, we can see various care plans devised for different groups of people with different ranges of blood lead results.

Basically, it summarises what has been set out in the letter that we have seen.

Also, I take it that you have had a chance to look at the checklist, at page 873, which contains all the items that need to be examined during the home visit.

Now you have had a chance to see the details of such care plans, are you happy with these suggestions now, or do you have any further comments on the adequacy of the

care plan?

A. Again, I am very impressed with the level of detail and the thought that's been invested in the different contingencies.

What I particularly like about the clinical management or developmental assessment plan is that children with blood lead levels of 5 to 20 will receive a preliminary developmental assessment by the Child Assessment Centre or the Maternal and Child Health Centre or the Student Health Services. I like that because it actually is more -- it's providing more services than the US CDC developmental management guidelines provide, which suggests that only children with levels above 20 be surveilled in this way. But I think this is going to provide an opportunity to identify problems that children may be experiencing that have nothing to do with lead, but a child would benefit from receiving some kind of supportive services.

So I think this is generally even beyond the scope of responding to the water lead issue, this is going to benefit children and parents.

I also had a chance to look at the lead exposure assessment questionnaire that will be administered. That's page 877 and beyond. I find that it covers all of the topics that I listed in my report that would be

important to include in an exposure assessment.

So, again, I am very pleased to see this level of detail. I think it's going to serve the purpose for which it was designed.

Q. Thank you. I think finally, we have come to the last question that you have answered in your report, ie the efficacy and suitability of the acceptance criteria laid down by the Water Supplies Department for heavy metals, and if necessary to make recommendations. That's internal page 19, paragraph (5). I think here, apart from lead, you have also discussed the acceptance criteria in relation to three other heavy metals, ie cadmium, chromium and nickel. I think you have come to the conclusion that the acceptance criteria specified by the Water Supplies Department are based on sound reasoning. Can I just for the purposes of general understanding of these heavy metals -- I am sure that you have a lot of experience in dealing with heavy metal poisoning. If we are talking about heavy metals such as cadmium, chromium and nickel, what are the general harm that they can do to human beings?

A. Well, it differs from metal to metal. Cadmium is primarily a renal toxicant. It damages renal function, it reduces the ability to kidneys to filter out poisons in the body. So it can lead to chronic renal failure

and require transplant, at very high doses, mainly occupationalists, people who encounter cadmium working in their jobs.

Chromium is a little more complicated, because there are different forms of chromium. Trivalent chromium or chromium (III) is actually an essential nutrient, so we need a little bit of that. Chromium (VI), hexavalent chromium, is one of the most toxic chemicals known, and it's well known, it's recognised by the International Agency for Research on Cancer as a human carcinogen.

Nickel, there's actually not that much evidence about nickel. Nickel is, like chromium (III), essential, but too much is thought to be an irritant. So that's the reason for establishing exposure limits for nickel.

Q. In general, how can these heavy metals -- we're talking about cadmium, chromium and nickel -- how can they actually enter water?

A. Sometimes they are just in the groundwater. They are natural constituents of the earth's crust. So, depending upon where an aquifer from which drinking water is taken, they may be naturally present, or they could be present as a contaminant. Cadmium is used industrially a lot. Hexavalent chromium is also an industrial pollutant. And so, depending on the care

taken in its disposal, it could contaminate water supplies.

Q. Thank you very much. That leads us to your conclusion at page 22. If I may just quote what you have said here, "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 micrograms per decilitre. Therefore, the ideal blood lead concentration for a human is 0 ... 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 per cent 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."

Just as a sort of general comment, now you've had a chance to look at all the blood test results of the residents involved and the updated test results -- just as from a broad comparative angle, in view of the other studies and cases that you have seen in other countries, what is your observation about the magnitude of this particular incident, in view of the test results that we have seen?

A. As I say, certainly the fact that once the residents were not drinking the lead-contaminated water their blood lead levels came down -- they were being exposed, there's no question about that, and as I say, it is prudent to interrupt any pathways of exposure that one feasibly can. I do look at incidents such as this in

terms of the context of other scenarios that I have been involved in. When I first got involved in lead work, this was back in the late 1970s, at that time the mean blood lead level of US pre-school children was 15 micrograms per decilitre, and 90 per cent, it was 88 per cent, of children had a blood lead level above 10 micrograms per decilitre.

I was thrilled at that time when my two sons had blood lead levels of 6 micrograms per decilitre. In the context looking back now, even that's too high, but at the time it was pretty good.

Looking at the 3 December line listing, I see there were only two individuals who had a blood lead level above 15. One was a 89-year-old who had a 16.7 -- it perhaps may have been an occupational exposure at some point in the past -- and the other was a 15.8 in a one-year-old.

So from that perspective, these levels don't seem very high. They are certainly higher than we would like to see, and it's good that they are coming down.

Another episode that I was just dealing with at WHO was an episode of lead poisoning in Zamfara, Nigeria, where more than 400 children died from lead poisoning and one child had a blood lead level of 700 micrograms per decilitre.

So there are certainly still cases where children are very, very seriously poisoned, and I don't think that's the case here. I don't think any children are going to be showing signs or symptoms of lead toxicity. On a population basis, I think we can conclude that there probably will be some shifts, minor shifts, in mean level of performance, shifts that you would never probably notice at the level of the individual child.

So my overall position is that this is a very unfortunate situation; I don't think the health consequences are going to be terribly serious, and I think that the authorities are taking totally appropriate steps to support the affected people and taking steps to make sure that exposure from water is not a major contributor to the overall exposure.

Q. Thank you. Finally, may I just confirm a few things with you, just as a matter of formality. May I just ask you to confirm that all the facts and matters that you have set out in your report and you have discussed today, which are within your personal knowledge, are true and correct? Can you confirm that?

A. Yes, I do.

Q. You also confirm that the opinions expressed in your report and also expressed today at the hearing are honestly held?

A. Yes, they are.

Q. Can you please also confirm that the contents of your declaration as an expert, as attached to your report, and also your CV attached to your report, are true and correct?

A. Correct, yes.

Q. You are willing to adopt what you have set out in your report and what you have discussed orally at this hearing today as your expert evidence?

A. Yes.

MR KHAW: Thank you very much.

CHAIRMAN: Thank you. Anyone wants to ask any questions? Yes, Mr Pennicott.

MR PENNICOTT: I think there are others as well, but I am happy to go first if nobody else wants to go first.

Cross-examination by MR PENNICOTT

Q. Prof Bellinger, I represent one of the contractors who was responsible for building two of the eleven affected estates. One of those affected estates is an estate called Kai Ching, and as it happens it is Kai Ching in respect of which we have most information in relation to, amongst other things, blood lead levels, and I expect you have picked that up during the course of your reading of the materials for the purposes of your report.

You will know, if you have read that material, that Kai Ching Estate comprises of six separate residential blocks. Is that something you are aware of?

A. I wasn't aware of that level of detail.

Q. It is six residential blocks. There are 5,204 individual units within those six blocks, and what I want to do, I hope fairly briefly, is ask you some questions about the various matters you have been discussing with Mr Khaw, but specifically in relation to the Kai Ching Estate, if I may.

Before I do that, I am going to hand you some documents in a moment that are going help me and hopefully help you as well.

Can I ask you this: so far as the blood lead level tests are concerned, have you established how those tests were actually carried out on the individuals who went for those tests?

A. Only indirectly. I notice that some of the values are reported as less than 3.3 micrograms per decilitre, which leads me to think that a portable LeadCare analyser instrument was used.

Q. Right, and do you know how the blood was taken? Have you made an assumption as to whether it was a venous approach or just a prick on the end of a thumb or a finger?

A. You can use either type of sample with the LeadCare analyser, and I don't know whether venous or capillary blood was used. If the sampling protocol is adequate, it shouldn't matter very much.

Q. That leads me to ask you this: if you are going blood lead level tests in a laboratory, is there any margin for error that you would impose, or are they sort of absolute figures?

A. Oh, absolutely, it does -- the different methods that are used do have different levels of precision.

Q. Can you explain what level of precision, for example, there is on a venous approach?

A. Well, the precision would be more specific to the method, whether it's isotope dilution mass spectrometry or atomic absorption spectrometry or anodic stripping voltammetry. It's not so much whether it's capillary or venous blood that's collected. The same level of precision would apply depending upon which analytical method is used, not the sample collection method.

Q. Can you give an approximation as to the margin of error?

A. Well, for the ICP-MS method, it can be plus or minus only a half of a microgram per decilitre or even a tenth. Yet for the atomic absorption, it may be plus or minus 1 or 2 micrograms per decilitre. Again, it depends on whose hands are applying the method.

Q. Right, the skill of the operator?

A. Right. The US CDC was resisting lowering the action level from 10 to 5 because they were claiming that many labs didn't have the ability to get closer than several micrograms per decilitre in their measurement.

Q. Prof Bellinger, one point that you discussed briefly with Mr Khaw earlier arises on page 58, internal page 14, of your report. It's the last sentence of the paragraph, headed "USA Environmental Protection Agency", and it's this point about "If more than 10 per cent of the tap water samples collected exceed the action level of 0.015 [micrograms per litre] (15 [micrograms per litre]), a water system is required to take steps such as corrosion control treatments", and so forth.

If the percentage is less than 10 per cent -- let's say, for example, 7.5 per cent, just by way of example -- is there anything to be done in those circumstances, or does this only kick in when you get to 10 per cent?

A. Yes, my understanding is it only kicks in when you get to 10 per cent. But there are some -- I'm no water expert, I have to say that right upfront. I believe that if a water system is supplying more than some number of individuals or units, they are required to take appropriate steps to reduce corrosion. And this

10 per cent applies to I think smaller systems, that are providing service to a smaller number of units.

Q. Have you, for the purposes of your report, studied in any detail the water tests that have been done in relation to the various affected estates?

A. No. I have focused on the health side of things.

Q. So you haven't looked at them at all, the results, the test results?

A. Not in detail, no.

Q. Okay. We will come to it a little bit in a moment.

I wonder if I could now hand out some documents that are going to help me to ask a few questions. I hope it won't take too long. (Handed).

If everybody has those, Prof Bellinger, for the first document, it's titled "Table A". You will recall, in your discussion with Mr Khaw earlier, you had identified I think 24 results as at 22 October 2015.

A. Right.

Q. Where there were in fact 28 results, you have taken off four pregnant women and then got an average of 30.8 per cent, I think.

A. (Nodded head).

Q. What we see on table A is in fact 25 results, so similar to you, what we have done is we have taken out the four pregnant women but we have added back the one result

that you didn't have, so we've now got 25 rather than 24. As you can see, we have calculated the average and it's changed very marginally to 30.27.

Can I ask you this first: this table includes two women, lactating women. Why do you think you would include those rather than exclude them for this purpose? You have taken out the pregnant women, I understand, and we see the reasons for that, but why do you include lactating women?

A. Well, to be honest, I didn't notice that. It probably would have been better to calculate the average change including and excluding them. Again, because during lactation, bone is still being mobilised to provide calcium, it does complicate the kinetics a bit.

Q. I just wonder whether there was a particular reason for it. I am not pressing you, I just wanted to know whether there was a particular reason for it.

A. Which two were lactating?

Q. If I am using the references on the left-hand column, about halfway down there's a reference "KC00190"; do you see that?

A. Yes.

Q. The 32-year-and-10-month-old lady -- and the cross-reference for anybody who is interested is E1/542, where we will pick up that there's a Y in the lactating

column on that page -- the other one is KC00257, a little bit further down the page. And similarly, it's the same page where we pick up the reference.

I am not pressing. I just want to know that there was a reason that there is one calculation with and one calculation without?

A. Yes. I see they both declined about 18 per cent, and I would have predicted that they would decline a little bit less than non-lactating individuals. So that would probably boost up the 30 per cent drop a bit.

Q. It would clearly push it up?

A. It would.

Q. The other thing on this page, table A, Prof Bellinger, that I just want to ask you a couple of questions about -- we have highlighted the two extremes in the right-hand column which I think you draw attention to in your report. That is the most dramatic percentage change is in the fourth reference down, of minus 55.54 per cent.

A. Mm-hmm.

Q. And the smallest change appears to be in the fifth reference up, of minus 2.74 per cent.

A. (Nodded head).

Q. Presumably you would agree with me, that's a rather large range for this type of exercise?

A. It's hard to say. I mean, it is a large range, but whether it's larger than one would see elsewhere, I can't say.

Q. What I am quite interested in, Prof Bellinger, is really whether taking an average of all these percentages actually tells us anything? Is it very meaningful, given the wide range that we have here, just to say 30 per cent?

A. Well, it is one way of summarising the evidence. As I mentioned in my report, there is a statistical phenomenon known as regression to the mean, which means that if you take repeat measurements of some biological function, or any function, usually those who are most extreme on the first test will regress towards the mean. If they are high on the first test, they will be closer to the mean on the second test because they will decrease. If they are very low on the first test, on the second test they will probably increase towards the mean.

I did show that the mean -- the percentage decline between the first and the repeat blood test was larger among people whose first blood test result was higher. Now, that does suggest to me that there was some regression to the mean going on. But we can't know how much regression there was unless we also tested people

whose -- did repeat testing on people whose first blood test did not exceed the reference value. That would give us a sense.

Q. Yes.

A. But there are biological reasons why we would expect there to be variability among people in the difference between their first and second blood test. Their age would be one thing.

Q. Yes.

A. Because the older people would have more lead in their bones.

It also depends upon what the other exposure, lead exposure sources are for an individual. An individual who's exposed to -- whose exposure is primarily from non-water sources is not going to show much of a decline once the water pathway is eliminated.

So, you know, for all of those reasons, it's impossible in my mind to draw any conclusion from the variability in the per cent change, without having lots more information.

Q. Yes. That's a very fair answer. But just to point out that -- to really underline the point that you have just made -- the two extremes, the 55 per cent and the 2.74 per cent -- the first one appears to be a female child of 10 months old, that's the 55.54 per cent, and

the other one -- and I should emphasise, for a reason we will come to in a moment, resides in Yuet Ching House -- we will come to the reason or the relevance for that in a moment. At the other end of the scale, as it were, the 2.74 per cent, is another child, a female of one year and four months old. So you've got two children of not great difference in age showing a huge difference in the readings between the first and second blood test. Does that tell you anything?

A. It tells me that water lead was probably a much more significant portion of the first child's -- the ten month old child's overall lead exposure than it was the other child, and that's why you would see a larger drop in the blood lead after the water pathway has been remediated.

Q. Just looking at these percentages and the drops, if you like, in levels, again the passage that Mr Khaw read out to you -- I don't want to go back to it again; I certainly don't want to read it out again -- it's in the passage where you refer to the half-life and you had lengthy exposure and acute exposure, and you explain what the differences are and the implications of that.

Can you apply those principles to these results?

A. Not really, and for the reasons that I suggested: because the half-life has a very narrow applicability,

and how it will play out for an individual will depend upon the other sources of exposure and how much past exposure an individual has had. The half-life of 30 days was actually identified by studies where adults had radioactively labelled lead injected into their blood. A known amount was injected and then measurements were made of the amount of radioactively labelled lead that came out in the urine and the faeces, through the bile. That's how it was arrived at, 30 days, and it's tied to the life cycle of the erythrocyte, the red blood cell, to which 95 per cent of the lead is bound.

Q. Yes.

A. So while that is true, 30 days is the half-life of a labelled atom of lead, for the same reason that the study I mentioned in my report about children who had a blood lead between 25 and 29, it took years for their blood lead to fall by half because of these other toxicokinetic factors that go well beyond the half-life of lead.

Q. With regard to the 10-month-old child referable to 55 per cent reduction in percentage terms -- so we know the child lived for ten months when she had the first blood test, no doubt a few more months older when she had the second blood test -- from your experience, that exposure to lead at the levels we have seen, is that

likely to have any long-term effect, do you think, on the child?

A. Well, you know, again, it's hard to say without knowing more information. If this 10-month-old child was nursed for eight months and then had two months of exposure to formula made up with water that contained lead, then that child probably didn't have much lead in the body, and so that's probably -- that would explain a big drop two months later, once the water lead was remediated. So, you know, under that scenario, I wouldn't anticipate very serious impact on the child's life.

Q. Again, I make the point, Prof Bellinger, just simply because it seems to us that this is very much a sort of micro-analysis that one needs to do on a very much individual-by-individual basis, and therefore taking these broad percentages doesn't really assist the analysis a great deal. Do you agree?

A. Well, I agree that things are always complicated. This is one way of organising the data. Certainly a mean can be misleading. I didn't calculate a median, you know, where it's a 50 per cent value. I suspect that it would not be that much different than 30 per cent.

But yes, these are -- if you are talking about individual management of a child, then an average is meaningless.

Q. Yes.

A. But if you are trying to capture an overall impact, I think it's reasonable, with appropriate caveats.

Q. Okay. Can I then ask you please to go to what I hope is called table B, the next chart. Whereas the previous table just listed out all of those people who had been tested and re-tested, what we have done here, Prof Bellinger, is we have just focused on what I would call abnormal, still abnormal, tests. So you will see, in the box on the left-hand side, we have simply again just replicated the abnormal results first test and those that were still abnormal after the second test, so you can see what we have done here.

Just to explain the blank at number 19 on the top graph and indeed the bottom graph: that's the two-year-old child that you referred to, when you were discussing the matter with Mr Khaw earlier, the one who started at 6.83, went up to 21.32 and then dropped back a little bit on the third test, and then we have seen more recently dropped back a little bit further to 15 on the fourth test. We thought it appropriate just to take that particular person, the child, out of the equation, as it were.

A. I agree.

Q. You agree?

A. Yes.

Q. Okay. We see, if we're just comparing abnormal to abnormal, as it were, the average we get is just under 20 per cent. Do you see that?

A. Yes.

Q. What we see in the bottom graph is in fact, at items 12 and 16, some increases in blood lead levels. Had you spotted those?

A. Yes.

Q. Any comment about those particular ones, the increases?

A. The only conclusion I would draw was that water was not a particularly important pathway for those individuals and that there were other sources in the environment that would have remained the same after the water pathway was interrupted. So they continued to be exposed to those sources and had slight increases in blood lead because of it.

Q. Okay. Then if you go to the next table, table C -- again, this is just referable to Kai Ching. And what we now know, leaving aside the results that I think were only received, certainly by us, yesterday or the last couple of days, that on Kai Ching there have been 59 individuals who have been tested and re-tested. And what we have done here is, if you look at the box in yellow on the left-hand side, those above the line, that

is between references 388 and 324, are the ones we saw on the previous table, the normal to abnormal, save that this time we have included, as it were, the rogue item.

A. 321 per cent.

Q. Yes. Those below the line are, as it were, abnormal but now normal. So this depicts the whole range so far as Kai Ching is concerned, and we see that the average is 22 per cent.

So, again, not miles off the 30 per cent exercise that we saw earlier.

But then over the page to a table I hope called D, this is exactly the same as the previous table, but this time we have taken out the two-year-old child that we were discussing earlier.

A. Yes.

Q. One sees what a dramatic effect that can have on the average percentage.

A. Yes.

Q. Lastly, if I could ask you to go to table E. I hope you can read it, because it's a bit small at the top, but again this is a breakdown of a number of statistics, a number of details, that we have in relation to Kai Ching Estate. You see listed across the top the names of the six blocks and then some totals at the far end. Okay?

A. (Nodded head).

Q. So taking the first one as an example, Hong Ching House, there were 165 blood lead level tests done, 149 of which were normal and 16 which were abnormal. Do you see that?

A. Yes.

Q. So that's now 9.7 per cent, for what that's worth.

If we then go down to the next items in the table, we can see how the normal and the abnormal break down. So there were 12 children -- of the 16 abnormal, 12 were children under 6, one between 6 and 8, none between 8 and 18, and then three people between 18 and 65. Do you see that?

A. Yes.

Q. Can I then just ask you to look at Yuet Ching House, which is the one on the far right-hand side before you get to the "Totals" column, and there, there were 147 -- sorry, there were 150 total tests; do you see that?

A. Yes.

Q. Of which three were abnormal; do you see that?

A. Yes.

Q. Underneath all the details regarding the blood lead level tests, we have put some details of the water sample tests. Do you see that?

A. I'm sorry, where are those?

Q. Under this first table, there's just a little table with two lines.

A. Yes, I'm sorry.

Q. It says on the left-hand side, "Water samples taken"; do you see that?

A. Yes.

Q. We can see on the far right-hand side of that little table that there were 93 water samples taken from the 5,204 units that exist on Kai Ching, of which seven showed excessive lead in water, out of 93.

However, if you look at Yuet Ching House, there were no samples that exceeded the criteria, the 10 micrograms per litre criteria that was being used. Do you see that?

A. Yes.

Q. So, on one view, Yuet Ching, so far as water samples was concerned, was clear, or in the clear, and that was the same for another block, Yan Ching House as well?

A. Yes.

Q. Can I ask you this, Prof Bellinger, by way of introduction: if one assumes, for the sake of argument, that the level or the degree of testing that was done on Yuet Ching House was valid and reliable, doesn't that raise a serious question as to whether or not any high lead levels in blood from the tests is related to the

water?

A. No.

Q. Why do you say that?

A. Because there's nothing magic about 10 micrograms per litre, except that it's a standard. If it were 9 or 8 or 7 or 6, there's still lead in the water and it still could be getting into people and be responsible for some fraction of the lead that's measured in the blood. Just because the water lead level doesn't exceed 10 doesn't mean that the water lead isn't contributing at all to an individual's exposure. It just means it doesn't exceed the regulatory standard.

Q. But the problem -- one issue about that, and this is a point that you fairly mention in your conclusion, is that it's a contributing factor or may be a contributing factor, but you simply can't apportion the contribution, can you?

A. Well, you could if you actually did the study where you went in and measured all of the other potential contributing factors; it would be possible to come up with a per cent of exposure that you attribute to water. That's been done in many, many studies. That wasn't done in this case.

But the fact that we see the declines in blood lead following the interruption of the water pathway, what

other explanation is there, other than that water was no longer contributing to blood lead and the blood lead is coming down? Unless you assume that because of the publicity, people were going out and learning about all the pathways of exposure to lead and taking steps to reduce their overall exposure.

Q. Understood.

Just finally then, can I ask this. I know, and you have already said once, you are not a water expert, you are a medical expert, and of course I accept that. Would you expect there to be a correlation, however, between the water sampling on the one hand and the blood lead levels on the other?

A. In general, yes, other things being equal. But if everybody had the same non-water contributions to their blood lead, but water lead concentration varied among people, yes, you would expect a very high correlation and water lead would explain all of the variability in people's blood lead.

But of course all other things are not equal, and so water will contribute different percentages across individuals of their total exposure to lead, which is why I would expect to see variability in the per cent decline once water lead is eliminated.

MR PENNICOTT: All right. Thank you very much, Professor.

CHAIRMAN: Mr McCoy?

Cross-examination by DR McCOY

DR McCOY: Prof Bellinger, is this your first trip to Hong Kong?

A. No. I am fortunate enough to be able to come once a year, to help with grant reviews.

Q. Have you been here previously on formal scientific reasons?

A. The once-a-year trip is to review grant proposals submitted by Hong Kong investigators for funding.

Q. In biology?

A. Biology and medicine, yes.

Q. I noticed in your report that you refer that there are, as far back as April 2012, some 28,900 peer review publications in relation to excessive lead.

A. That's right.

Q. It's obviously gone up since then, and not just because of this incident. But what proportion of those publications do you think pertain to Hong Kong?

A. Probably very few.

Q. Have you ever used Google Scholar?

A. Yes.

Q. If you put in "lead" and "Hong Kong", can you estimate how many articles might come up, academic scientific papers written about lead poisoning in Hong Kong?

A. I could hazard a guess but it's just a guess.

Q. If I tell you that there have actually been published theses in Hong Kong written on lead poisoning, that would not surprise you?

A. No.

Q. I am going to ask you in due course to have a look at one of those published theses, and it examines the many causes and effects of lead poisoning in Hong Kong.

In fact, I could ask even now that it be put up on the screen, with permission. It's G3, item 88.

This is a thesis in the faculty of science at Hong Kong University. It's slightly dated -- it's April 1987, and I noted that it doesn't actually refer to any of your works, but other Hong Kong publications which I will take you to do -- and it's written in the department of chemistry at Hong Kong University.

You can see the abstract. On (i) -- if you roll over one or two pages, please, past the acknowledgments -- keep going, please.

A. Yes.

Q. The next one. Exactly.

The abstract, of course, is the summary of the academic conclusions. I won't read them all out, but the author shows that lead is detrimental, especially to children, and he identifies some reasons why that is

particularly so, not different from what you have said to the Commission of Inquiry.

He points out that petroleum -- so this was 1987 -- gasoline perhaps in your language, was a major contributor, and also that there was lead in consumer products available in Hong Kong.

Consumer products include, plainly, food and items like paint, for example.

A. (Nodded head).

Q. The methodology of the thesis was to actually examine the lead content inside 6,065 Hong Kong schoolchildren. You will see that in the second paragraph, commencing, "In this survey, the massive screening programmes ..."

It was a study of, as I said, of over 6,000 children in Hong Kong, aged from 6 to 17.

If we come down, please, to the next paragraph:

"In the scheme III screening, 700 primary schoolchildren aged from 6 to 16 years old were studied. The mean blood lead level ... of them was found to be 14 [plus or minus 5.1 micrograms]. The spread of [blood lead level] was from 3.7 to 45 [micrograms]. The blood lead levels of Hong Kong children are similar to or lower than those in large cities. They [are] found to conform to the EEC guidelines on the criteria of undue lead absorption."

Now, the EEC guidelines that the author is referral to, are you able to comment as to whether that conclusion is sound; that, for example, levels like 45 micrograms conform to the EEC guidelines in 1987?

A. Well, I'm not sure actually what the author means by saying they conform, whether that is intended to mean that all levels were below whatever criteria the EEC was using at that time. I personally don't recall what the EEC guidelines were in 1987.

Q. Sorry, I wasn't trying to make it a test of absolute memory. But we will read on and perhaps it will become clear.

The conclusions are:

"[There is an] association of some personal, family, behavioural and environmental factors with blood lead levels of children [when] analysed. It was found that sex, mouthing habit, parents' occupation, floor level of residence as well as the time spent on street(s) do have positive association with [lead] levels in children."

Would you agree with that as a generality, Prof Bellinger?

A. Certainly there are studies that indicate that all of those, under certain circumstances, can contribute to children's lead exposure, yes.

Q. Yes. Then we read on:

"With the use of controlled Pearson Correlation Analysis, it was discovered that the more direct personal factors, namely the occupation of parents and mouthing habits, are more important than the environmental factors such as floor level of residence and the time spent on the street."

Then there was a special reference in the thesis to the risk of fishermen's children, a study at Aberdeen, one of the ports on Hong Kong Island, where the children were often literally handling lead weights and there was an ingestion through the body.

Would that surprise you that that generated an obvious risk of potential excess lead?

A. No. That's been reported numerous times.

Q. I am obviously not going to go through all of this thesis, but there are some matters that I would like to raise with you for your comment which may be of assistance to the Commission of Inquiry.

If we come firstly to page 105, please. This is really near the end of the thesis. You find a conclusion -- it's chapter 4, page 105 -- about 30 per cent of the way down the page, you see the words, "Australia et cetera", full stop. Then this:

"Also the increase in use of copper pipings in domestic water supply system should be carefully

monitored because the leaching of lead from the solders has been reported to be the source of undue lead absorption."

That's a well documented empirical fact; I assume you would agree with that?

A. Yes, if the pipes have been connected by solder that contains lead.

Q. Yes, exactly.

The author in particular identifies a large number of environmental factors that can contribute to lead.

Now, in America, what is the current greatest contributor of lead in the environment?

A. It's the lead in dust, house dust.

Q. The lead in house dust?

A. That's usually the strongest predictor of a child's blood lead level.

Q. And it seems naive but how does the house dust get to contain lead?

A. Well, in numerous ways, and that's why it's a significant predictor. It could be lead-based paint that is chipping and crumbling into fine particles that contribute to the dust. It could be people tracking in soil from outside that may contain lead, because of atmospheric deposition or the deterioration of exterior lead-based paint that gets into the soil. It could be

lead blowing in through the windows; back in the days when lead was added to gasoline, that was a major, major problem. It also could be in the air because of the child's house being proximate to a point source such as an industry that uses lead. It could be because people are carrying out activities in the home that involve lead, and little particles get into the dust.

So it integrates lead from many different sources, and we think that's why it's usually the best predictor.

DR McCOY: Yes.

Is that a convenient time?

CHAIRMAN: Let's take the lunch break first. We will resume at 2.30. Thank you.

(1.00 pm)

(The luncheon adjournment)

(2.35 pm)

CHAIRMAN: Yes, Mr McCoy.

DR McCOY: Good afternoon, Prof Bellinger.

A. Good afternoon.

Q. Just before we broke, I had asked you to confirm what is the number one source in America of lead in the environment, in the free environment, and you indicated that it was basically household dust, or dust?

A. Correct.

Q. That is simply a product of other processes, as opposed

to it being naturally occurring, I assume?

A. Yes. It's not a source in itself. It's a pathway.

Q. That's right.

I see from your very lengthy bibliography that you have written widely, and you have studied, for example, the problem of excess lead with children in India --

A. Yes.

Q. -- Bangladesh, Mexico, Palestine, to name just a few of the places.

Have you ever yourself studied the incidence of excessive lead in any part of Asia?

A. Well, Bangladesh and India.

Q. They may be for FIFA.

A. What?

Q. They may be in terms of football classification, but say east of India, have you ever studied any of those Asian regions, including Hong Kong, China, Japan, Malaysia?

A. No, I haven't.

Q. You haven't? The reason I ask that question is whether there are any well-understood regional differences throughout the world in terms of ordinary background lead standards in daily life.

A. By "lead standards" do you mean regulatory exposure standards?

Q. Yes, first of all regulatory, and then second, as

a matter of fact, the incidence of lead.

A. Well, I'm not familiar with the lead standards used in each country. I know many do refer to the WHO standard of 5.

In terms of background incidence of lead exposure, yes, there are very large differences, mostly between developed and developing countries. The WHO, in an analysis they published in 2003, estimated that about 10 per cent of the world's children have a blood lead level greater than 20 micrograms per decilitre, and 99 per cent of them reside in developing countries. So the success we have seen in reducing population lead exposures in developed countries over the last 30 years have not seen the same benefit in developing countries. So they do have more -- continue to have more of a problem with lead.

Q. Lead does not degrade as such, being an element, does it?

A. That's correct.

Q. And you can't transmute it into anything else, unless you believe in alchemy, I suppose?

A. That's correct.

Q. So once the lead has entered the total environment, it remains, unless dispersed by air or water?

A. That's correct.

Q. So it would be important in understanding the background levels of lead, in any urban environment, to understand the history of that land before its current usage?

A. That can be important, yes.

Q. You see, I will show you in due course that there are well-documented peer-reviewed academic studies in Hong Kong that because certain parts of Hong Kong had been heavily industrialised in the past, the lead effectively has sunk into the land and remains. Does that seem a logical proposition, if the assumption is correct about the prior industrialisation?

A. Yes. There have been similar studies in Massachusetts, where I live, actually, where people have looked at historical land use and identified higher blood lead levels among children living in regions which formerly had industries that used lead.

So, yes, that's a well-described phenomenon.

Q. So if you are living in an area that has previously been relatively heavily industrialised, is there a likelihood that the dust in that area will be significantly higher in lead content than otherwise?

A. I would say -- I would presume yes. It would remain to be confirmed, but it's a very reasonable hypothesis. It would add to the background risk and perhaps raise the background blood lead level against which other sources

would add.

Q. And the human ingests lead by, what, touch, inhalation, consumption; every method possible?

A. Except for skin absorption, yes. Inorganic lead is not absorbed on the skin. But ingestion and inhalation are the main routes of exposure.

Q. Can I ask you to come now to page 1 of the thesis that I have partly shown you this morning. Page 1. I see you have a hard copy now.

75 per cent of the way down the page, it says:

"There are many routes by which lead can enter the air, water and soil as a result of the wide use of lead and its compounds."

I won't read all of this out, but in particular the combustion of coal is identified as a source of lead entering the environment.

Are you familiar with that as a contributing factor to environmental lead?

A. No, actually. That's a new one. Mercury I know is a problem with coal combustion, but it's new to me that lead may also be released.

Q. Again if we can just move down to the last two lines on that page:

"The lead level in the food chain (plant, fish, vegetable, poultry et cetera) is also increased by

several orders of magnitude."

Then this:

"The daily intake of lead of the average person in [the United Kingdom]" -- and (2) is the scientific reference for it -- "and USA (3)" -- ditto -- "is estimated to be 18 [micrograms per day and 175 micrograms per day] respectively."

Are you familiar with those figures?

A. Yes. These are quite old numbers, and the estimates for today are much, much less, in the order of 20 or 25 micrograms per day, because of all the reductions in the last 20 to 30 years and dispersion of lead into the environment.

Q. Then if you come down, please, on page 4 to the first full paragraph, and the last two sentences:

"Lead has a very slow turnover rate in the body."

What dictates that turnover rate?

A. Well, I assume that means the excretion of lead from the body. I had earlier talked about the different pools in the body in which lead resides. Each pool has a different turnover rate, so you can't assign one single number. So lead that's in the type of bone that's hard and compact, like the tibia, has a half-life of maybe decades, three decades, whereas the lead in soft, spongy bone, the trabecular bone that is in

contact with circulation has a half-life of years to a decade, and in soft tissues will be somewhat less, and as I mentioned, in blood it's 30 days.

Certainly in bone there's a very slow turnover rate. It's not so slow in the blood.

Q. Halfway down that page, there's a historical reference to lead poisoning from Roman times. It's probably plumbum, being the chemical name for lead, and plumber going together.

A. Exactly.

Q. Plumbers carry lead.

A. Right.

Q. If you turn the page, please, to page 6, you see the first reported environmental exposure from lead was in Queensland in Australia in 1892. That was paint.

Then an issue the chairman raised with you this morning, the issue about young children. The author here states:

"Young children are widely held to be more susceptible to lead poisoning in the environment than adults for the following reasons.

1. Young children have a higher metabolic rate than adults, so that their intake of food and drink, and hence lead, is greater relative to their body weight than that of an adult."

Do you agree with that?

A. Yes.

Q. "2. Young children may absorb and retain more lead from the gut than adults."

A. Yes.

Q. Thank you.

"3. Young children may have a greater biologically active lead burden than adults."

Do you agree with that?

A. I am not exactly sure what the author means. If he means that a greater or a lesser fraction of lead is in the bone in children than in adults, so it's more available to interact with cellular targets, then yes, I would say I agree with that interpretation of the phrase.

Q. "4. The opportunity for oral intake of significant amounts of lead from non-food sources is higher in young children than in adults due to mouthing habits."

I think that's pica, the obscure word P-I-C-A, the non-nutritive intake by children, eating things that they shouldn't be eating.

A. Right, although even children in whom the behaviour is not as extreme to warrant the application of the term pica still -- typically, developing children explore their environment with their hands and their mouth,

so ...

Q. And the fifth one:

"Deficiencies in nutritional status may also be of greater consequence in the enhancement of absorption of lead in young children."

Do you agree with that?

A. Yes. Children are more likely than adults to be deficient in iron and calcium, and those promote absorption of lead in the gut.

Q. So the conclusion is children are more susceptible to lead poisoning. But can I now ask you: are they more likely, because of the nature and size of their bones, to excrete or eliminate that ingested lead than a comparator adult?

A. I am not familiar with evidence that that's the case, that the excretion rate is not different from adults.

Q. There's no difference at all?

A. Not that I am aware of.

Q. Then perhaps if I could ask you to come across to page 8, and I am certainly not going to go through all of this, but there are just a few highlights. If you come to page 8, please, 10 per cent down the page:

"However the public awareness of this problem" -- that's the problem of excessive lead -- "has increased since early 1980s."

And several factors are set out. I won't read all those factors out, but we have at number 4 a reference to the Hong Kong Consumer Council, a prestigious body that by its very name looks after consumer interests. It was reported by the Hong Kong Consumer Council that the lead in petroleum in 1980 was at 0.84 grams per litre, six times more than in the United States. This was staged down to 0.15 grams in January 1987.

Now, the lead from the petroleum, it enters the environment. It stays, I think we agree, unless it is literally blown away out of Hong Kong.

A. Correct.

Q. So, in a small, dense, compact society like Hong Kong, when lead in petroleum was available, the probability is it remains adjacent to where the vehicle burnt the lead, the petrol?

A. Yes, it is emitted in the area proximal to the roadways. So the farther you go from the roadway, the concentration of lead declines.

Q. If you come over to page 10, please, at number 8:

"High level of lead at kerbside soil was reported. Mean concentration of lead was 2,974 [plus or minus] 408 [micrograms] and the range was from 271 to 19,073 [micrograms]."

Extreme readings.

A. It's quite high.

Q. Then at 9:

"Approximately 7.5 per cent of 1,135 adult blood lead levels were found to exceed generally accepted reference value of 35 [micrograms] in a survey carried out in Hong Kong."

And the reference is to 60 in the bibliography.

"The findings suggested that the group studied did not conform to the EEC guidelines."

Now, with the revised limits, they would be well over, would they not?

A. Correct.

Q. Then 10 -- and I would like you just to go to page 113 briefly. At item 61, because D Barltrop and I Thornton in Hong Kong in 1982 wrote a book, "Lead Pollution in Hong Kong -- Report to the Special Committee on Air Pollution", so that's the source material that the author is referring to.

If you come back then to page 10, at paragraph or item 10:

"A report on 'Lead pollution in Hong Kong' prepared by two experts from UK was published. It stated that a comprehensive evaluation of lead in the environment and its impact on the population were required."

Was there comparable efforts, say, in the

United States at that time?

A. Comparable efforts to ...?

Q. To publish a comprehensive evaluation of lead in the environment.

A. Yes.

Q. And in the United Kingdom?

A. I am not as familiar with ...

Q. Have you seen what these two experts in 1982 recommended to the government should happen? Have you seen the outcome?

A. No, I haven't.

Q. Nor have I.

Now, this thesis involves, as I said, the screening of over 6,000 Hong Kong children. If you turn over, at page 12 -- I won't spend time on this for the non-chemists -- but at page 12 you find the interference with the biosynthesis of the haem, the haemoglobin, and that's set out in diagrammatic form.

The author, at page 11, 80 per cent down, is identifying zinc protoporphyrin. You would be familiar with that as a metabolite.

A. It's not really a metabolite but I am familiar with it.

Q. So the author of this thesis, he examined ZPP, as I will more conveniently call it. You see the chemistry at 13. We can go past that.

Then at page 16 you see what was achieved. He went to government or subsidised Hong Kong schools. The children were all of Chinese race and aged from 6 to 17, and the distribution of schools covered the three major areas of Hong Kong: the Island, which we are now on; Kowloon Peninsula; and New Territories together with the Outlying Islands. Are you generally familiar with the geography?

A. Generally, yes.

Q. We can avoid now much of this, but we come to page 20. The blood sample from each of these children was taken from the finger, from a capillary. Now, this morning, Mr Pennicott, senior counsel, did ask you a question directed at whether there was a margin of error between capillary blood and venous blood for the purposes of determination of excessive lead, and you, would I be right, were hesitant in accepting that there was a difference, or is that unfair?

A. No, no. There should not be a difference if adequate cleaning protocol is done. The problem with capillary samples is that there's sometimes lead dust on the finger, and when you lance and get a drop of blood, it takes up some of that lead on the finger, if you haven't cleaned it properly, and so it can give you a false high reading.

But if you follow accepted practices for the cleaning of the finger before you stick, the capillary blood lead level should be very close to the venous blood lead level.

Q. If I suggest to you that a recent peer-reviewed study actually shows that even with proper cleaning, there is a 10 per cent false positive risk with capillary taking of blood for the testing of lead, would that surprise you?

A. Well, what exactly is -- is it 10 per cent --

Q. I can produce the paper.

A. Is it a 10 per cent difference in the blood lead level or a 10 per cent difference in --

Q. In false positives.

A. That doesn't mean anything to me.

Q. All right.

A. I would have to see the paper.

Q. I am happy to do that and I will do it soon.

A. But I have seen other papers where the difference between the venous and the capillary levels were exactly the same. So I don't know how this study would differ from those.

Q. Now, could we then please move way through much of this and come to page 54. The author notes, 30 per cent down the page, that ZPP, the metabolite, as I'm calling it,

"is firmly bound with haem and persists there for the life span of the red blood cell (about 120 days)."

You agree with that?

A. Yes.

Q. So that's relevant to a 30-day cycle in your 25 per cent equation, the cycle of re-testing the blood every 30 days?

A. Yes.

Q. If we come much further across towards the end -- just give me a moment, please -- the author of this thesis concluded that there is a statistical correlation between the lead levels in children and the occupation of their parents. Is that something you have seen in any other study?

A. Yes. It's quite well described that parents involved in occupations involving lead can bring it home and those children will have higher blood lead levels than children of parents who don't work with lead.

Q. May I ask you to please leave that document, and I would like to look at a study that refers to one of your own works, with appropriate approval, I should add.

Could you come please to item G3/90, please. That's right, "Home sweet home? A case study of household dust contamination in Hong Kong", from the University of Cincinnatti and the Chinese University of Hong Kong in

March 2000.

If we just look at the abstract:

"It is well recognised that many heavy metals have chronic effects ... particularly to young children ..."

I will leave words out in the interests of time.

"... house dust ... This research aims at quantifying the concentrations of heavy metals within the home environment in Hong Kong and their relationships with environmental factors. The results of this study seem to suggest that traffic and the age of the building and neighbourhood are more important factors than the types of industry and socioeconomic status in affecting household dust contamination. The metal burdens in Kwun Tong ..."

Now, that's a particular district in Kowloon.

"... an old area with heavy traffic, are significantly higher than other districts."

If we just move past that and come to a second document, and this is the next one, G3/92, called, "The study of metal contamination in urban soils of Hong Kong". This is written between Imperial College London, the Department of Civil and Structural Engineering at Hong Kong Polytechnic University, and the Department of Land Surveying and Geo-informatics at the Hong Kong Polytechnic University, in September 2003.

If we again look at the abstract, it refers to the highly urbanised Kowloon area:

"A significant spatial relationship was found for [nickel, copper, lead and zinc] in the soils using a GIS-based analysis, suggesting that these metal contaminants in the soils of Kowloon ... had common sources. Several hot-spot areas of metal contamination were identified from the composite metal geochemical map, mainly in the old industrial and residential areas ... The [lead] isotope composition of the contaminated soils showed clear anthropogenic origins."

I will try to deconstruct some of that with you very briefly, and I won't spend long on this, I promise you.

If you come to the second page of this article, at page 114, five or six lines down -- first of all, you can see your own name there, Prof Bellinger, and immediately after that:

"The heavy metal concentrations of soils have been widely studied in Hong Kong ..."

And here a list of Hong Kong academics, from 1978, 1982, 1987, 1996, 1997 and 2001, who have all studied them.

According to a survey conducted in 1981 by Lau and Wong ... in which the heavy metals in soils of different sectors (recreational, commercial, industrial and minor

agricultural) were studied, the highest [cadmium] concentration in Hong Kong was found in a recreational area (Chung Pui), where 54 [micrograms per kilo of cadmium] was found in roadside soils. The highest copper concentration ... was found in an industrial area (Aberdeen). The highest [lead] and [zinc] concentrations in Hong Kong ... were found in an agricultural area (Man Uk Pin)."

That's up near the border, Sha Tau Kok area, for those more familiar.

Then please coming over to the conclusion, you'll see at page 116 the results and discussion, and at page 116, the right-hand column, about three lines down into the last paragraph:

"It has been shown that the concentration of [lead] in Hong Kong is related to Hong Kong's high traffic volumes ... Although [lead] has been banned in petrol for a number of years, the concentration of [lead] in urban soils still reflects the significant degree of historical [lead] contamination nation and the long half-life of [lead] in soils."

That would be consistent with your own experience?

A. Yes.

Q. If we turn over, at 119, the left column, halfway down:

"Several hot-spots [of very high levels] are

identified from the composite geochemical map, including" -- and these will make more sense to others in the room like than yourself -- "Lai Keng, Cheung Sha Wan, Shek Kip Mei, Kowloon City, Ngau Chi Wan, To Kwa Wan, Ho Man Tin. These are mainly old industrial and residential areas in Kowloon. Therefore, the history of an urban site can contribute to heavy metals in soils."

Page 119, in the right-hand column, it is pointed out that "soils in the hot-spot areas were generally about 2.5 times more contaminated than the rest of the urban area". There's even a reference to the fact that "the hot-spot areas were generally found at the northeast or east side of a major road", because of the diffusion of pollutants by the prevailing wind in Hong Kong, which is from the southwest.

One last point. At page 120, the left column, five lines from the bottom:

"3.3.3. The effects of buildings and landscapes on heavy metal dispersion.

... It has been shown that high-rise buildings can obstruct air movements, and prevent the particulates in air from dissipating."

Have you seen this conclusion reached before, Prof Bellinger?

A. No, I haven't.

Q. That's a study by Taiwanese academics, under their own regime.

If we turn over, the effect of it is that the shape, height, density and configuration of buildings disallows the lead particulates to ever leave the area where they originally had been, so they simply stay and remain.

Does that appear at first blush to be a logical conclusion? In a sense, they are trapped where they got to?

A. Oh, it does -- yes. If something prevents the particulates from becoming airborne and carried by the prevailing winds, then it's likely to continue to reside in the soils.

Q. So if we have a combination of what had been in the past a heavily or medium heavy industrialised areas, and they have now been built upon in a dense way, high buildings, the logical inference is that what was originally in the soil and has now become dust will therefore remain in that area; it can't escape?

A. That's correct.

Q. There's more -- I won't go through them all, but there are more to the effect -- other studies. In fact, there are a large number of Hong Kong studies on these issues.

Now, Prof Bellinger, perhaps I could bring these

points, as it were, to a head. At page 49 of your expert report, the bottom right-hand corner of page 5, also marked with a large 49. The first full paragraph:

"Lead is often characterised as a 'multimedia' 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 ..."

And you make a reference to herbal medicine; here, of course, it would be Chinese medicine.

Now, lead in food, and I see you have studied, for example, the effect of lead in turmeric, I think was it in Bangladesh or India. It's a root vegetable. Do root vegetables take in the lead?

A. To a certain extent, yes, and leafy greens will accumulate lead that's in the air or in soil and then becomes entrained as air as soil is worked. Then also in the past there have been some lead-based pesticides used in agriculture -- lead arsenic, a great combination of lead and arsenic.

Then lead is also introduced along the way as food is processed and packaged. Lead-soldered cans used to be a big problem.

So it's possible at a number of points in the production system for lead to get into food.

Q. What about seafood?

A. To my knowledge, lead was not a major problem in seafood, although I can imagine, if there are local hot-spots near discharge points from industry, seafood, especially shell fish, that are feeding in that area on the bottom where lead may settle into the sediments, that may be a problem locally. I am not familiar with it being a problem globally.

Q. Now, the next one you have is air. Hong Kong is positioned adjacent or part of the Pearl River Delta, which is on any view reasonably heavily industrialised. What is the effect of air pollution coming from those sources? It transports lead or it may do so?

A. Well, it depends on what those transports burn, if they are still using leaded petrol, which I don't think they are. Then, if lead is used in any of the industrial processes that go on in those locales, and it's not within my area of expertise, I don't know -- I don't know if there have been recent measurements of air-lead concentrations in Hong Kong.

Q. So you've identified for the Commission there are a number of sources of lead which can have contributed to the aggregate total of lead inside an individual's

body?

A. That's correct.

Q. And on the face of it, the lead in any solder that has been used is a potential source of some of that lead?

A. Presumably, yes.

Q. The outcomes that Mr Pennicott showed you this morning and the variations and the differences, is that likely to be attributable to any particular factor in any particular individual? Is there any predetermined allocation that you can say is due to lead solder?

A. No. I presume you are referring to the per cent decline in blood lead observed?

Q. Yes.

A. As I said, every individual differs in terms of what they are exposed to from all these different potential sources, in terms of how frequently they come in contact with it, what foods they eat, whether they play outside in contaminated soil. So my first hypothesis would be that it's the mix of -- the contributions of these different pathways and sources differs from one individual to the next, and as a result of that you would expect to see some variation among individuals in the per cent decline when one of these pathways, specifically water, is interrupted and no longer contributing.

Q. Yes, because even the difference in an individual's diet could well be material?

A. Absolutely.

Q. I indicated I would show you the materials relating to the protocol for taking of blood. Could I have this given to you, please. (Handed). This is a document called "Pediatric lead toxicity work-up", and it is published on 6 September 2015.

I also have, incidentally, a document from the Oregon Department of Human Services, the Oregon Lead Poisoning Prevention Program, which I would like you to look at, but please look at the first document.

One can see, under the cross-heading "Whole blood lead level":

"Whole blood lead level ... is the criterion standard for confirming the diagnosis of lead poisoning. For convenience, a finger-stick capillary lead level has been used for screening. Properly collected capillary samples have a 10 per cent false-positive rate. Once an elevated lead level is detected, a venous lead level is assessed for confirmation."

A. Yes, that's absolutely right. In fact last week, at the meeting of the guideline work group, producing guidelines for the diagnosis and treatment of lead poisoning in Geneva, this is an important part of our

document, that capillary samples are fine for screening but you don't make treatment decisions based on them; you get a confirmatory venous blood lead level. But that's when you are contemplating clinical management.

Q. Do you know, for the Hong Kong results, whether they were obtained by capillary or venous method?

A. I don't know, no.

Q. So it is important because if it was capillary, but had not been audited by a venous test, then there is significant intrinsic doubt as to the reliability of the original capillary reading?

A. But the pattern of findings over time, in the individuals that were identified as having a result above the reference level, are so coherent in terms of the distribution of declines, I think that's very, very unlikely, that there was contamination of the original blood lead levels.

Usually, when there's a contamination from a capillary sample, the value is greatly elevated, because a microgram of lead is microscopic, and much more than that is on the finger from contamination. So, when that gets in the blood, you are going to see a blood lead level that is much higher than 6 or 7.

Q. Can I ask you a likely different matter for a moment. Do you agree that lead absorption in children is

increased when there is insufficient calcium in the child's diet?

A. There are epidemiological studies showing that children whose diets are insufficient in calcium do have higher blood lead levels, and it's based on the underlying biology. The inference is that the absorption of lead is higher in those children. I don't know of any experimental evidence to back that up, but it makes sense in terms of the known biology.

Q. If I can just ask you the same question but changing the variable. Instead of an insufficiency of calcium, if there was an insufficiency of iron in the diet, is that also likely to lead to increased lead absorption in children?

A. Yes.

Q. Why is that?

A. Well, lead and iron also look very similar to -- they look similar to one another chemically, just as lead looks similar to calcium, so they compete for binding sites in the gut. So if iron is not present, then lead will preferentially be attached to those binding sites and be absorbed into the gut and then into the blood.

DR McCOY: I have no further questions. Thank you, sir. Thank you, Prof Bellinger.

CHAIRMAN: Anybody else? Mr Ho.

Cross-examination by MR HO

MR HO: I just have one or two very minor questions.

Prof Bellinger, I am representing the Housing Authority. I have just got one or two minor questions for you.

When you were answering my learned friend Mr McCoy's question, he showed you a paper which I think was in 1987, and there were some figures about food intake, and you said those are outdated because now, normally, the intake of lead from food is much lower. Do you remember that?

A. Yes. Was that -- I thought that was total lead intake, not intake just from food.

Q. Right.

May I just ask you to look at a background paper to the WHO Guidelines for Drinking-water, which is a paper that's been referred to in this Inquiry, and that is at A1. Page 403 is the cover page. Perhaps you can have a look at that first.

You see the title. This is a background document. If you look at the next page, you will see the date of this paper is 2011; do you see that?

A. Yes.

Q. Are you familiar with this paper?

A. Yes.

Q. You are?

A. Yes.

Q. Did you have a hand in the preparation of this paper?

A. No. I had a hand in the process that this was based on --

Q. I see. Thank you very much.

A. And the JECFA re-evaluation of lead earlier in 2011.

Q. If I may ask you to go to page 2, which is at page 410, please.

Paragraph 2 -- of course you see, I believe, these are the sort of pathways that you identified and explained to us earlier: air; 2.2 is water; 2.3, we see food; 2.4, which is over the page, we see "Other routes of exposure", and that refers to household dust as being one of the significant sources.

If I may ask you to come back to food, at 2.3. At the bottom of that page, do you see it gives examples of the daily dietary lead intake from various countries, for example, like Sweden, and over the page, do you see 66 micrograms for Finland; 23 micrograms for the USA; and further down the page we see a figure for England; we also see Canada, which is 53.8 micrograms per day; Belgium, 90 micrograms per day; Sweden has a lower figure, 24; Mexico has, amongst these countries, the highest, 177. Do you see that?

A. Yes.

Q. So there is a great variety of the daily dietary intake of lead between the countries?

A. Right. One caution I would interject here is that it is important to look at the dates that these figures were taken from, because, looking very quickly at the references, I see that the Finnish number comes from a 1980 study, the USA from 1982 to 1984, and Sweden from 1985. So these are quite outdated numbers.

Q. Right.

A. They are much lower now.

Q. Right. But even amongst what we call the developed countries, for example some of the European countries and even Canada, the daily intake from diet could be much higher than what you said, 20 to 25?

A. They could be. You do have to be careful about dietary estimates, because it depends a lot about how you survey the diet.

Q. Certainly.

A. There's a lot of variability of individuals within the same country and what their diet is. So you just have to be careful to not put too much stock in any single number unless you know exactly how the data were --

Q. Exactly. So there can be different variants which may contribute to a higher figure or a lower figure, even

amongst developed countries on the one side and developing countries on the other side?

A. It's very hard to be certain here.

Q. You can't just generalise, as it were?

A. You can, but at your own peril.

Q. All right. Interestingly, the last sentence of that paragraph talks about even you get lead from drinking wine.

I know quite a lot of my colleagues here drink wine, so do be careful.

Moving down the page to 2.5, you see there is a sentence that starts:

"More than 80 per cent of the daily intake of lead is derived from the ingestion of food, dirt and dust."

From water, that is a relatively small proportion of total intake. That's still true, isn't it?

A. I think on average that's a fair statement, but it will vary from setting to setting.

Q. In fact, the same point is made, if we go to the end of this document at page 423. The last paragraph on that page, if we start with the second sentence of the last paragraph:

"Nevertheless, because lead exposure arises from a range of sources, of which water is frequently a minor one, as it is extremely difficult to achieve

a concentration lower than 10 ..."

Again, I believe that's in fact a sentence that is lifted and becomes part of your report, so that must be correct?

A. Yes, as far as I know.

Q. Thank you very much.

I see that apart from what is stated here -- air, water, food, household dust, and so on -- in your own report at page 63 of the bundle, you also identify children's toys as a potential source of lead intake.

A. (Nodded head).

Q. Is that, in your experience, a general problem with lead in children's toys?

A. In the US, it is a problem with toys imported from certain areas. Mainland China is one of the areas of great concern.

Q. Sorry to say.

A. Sorry to say, yes.

Q. Yes, in mainland China there is a danger or a risk, at least, of producing toys that may contain lead content.

A. Yes, but it's true for toys imported from other areas as well. The surveillance is not really what we would like to see, and if a child is mouthing a toy that's painted with lead-based paint, that can lead to very high exposures.

Q. Of course let's put your report into perspective. You are telling us generally about the effect of lead on various groups of people, but of course, in any individual case, if we want to see the effect of lead on a particular individual, we have to do a lot more study, examination and so on in order to find out the cause; is that correct?

A. What do you mean by particular effects on an individual?

Q. For example, if we isolate one particular person in question, then what you are doing in your report wouldn't give the answer?

A. What I said in my report applies to groups of individuals, and the associations that have been observed are population statistics.

Q. Yes.

A. It can be difficult to identify effects in any individual child. All we can say is that, on average, children with higher blood lead levels or adults with higher blood lead levels have this health adversity.

Q. Yes. Thank you. This is a sort of statistical general observation?

A. Right. We can't say with certainty. We might be able to say more likely than not a child's problems are due to lead.

Q. Thank you. But in order to find out as referring to

a particular individual, we have to study a lot more, we have to have a lot more information about his background, his living environment and so on?

A. I see what you mean. Yes, in order to identify the potential contributors to blood lead level.

MR HO: Yes. Thank you, Professor.

Cross-examination by MS WONG

MS WONG: Prof Bellinger, if I may ask a few questions.

This morning, Mr Chairman has raised the issue of the rationale as to why the US was adopting a higher value of 15 micrograms. I wonder if I may assist by producing or showing you a document. (Handed).

Prof Bellinger, I think this is a meeting note dated 11 January 2006, by National Drinking Water Advisory Committee.

Is it correct that this is a federal advisory committee that supports the EPA in implementing its duties and responsibilities?

A. That's my understanding, yes.

Q. We can look at page 2. We see from that table, there are three different contaminants commonly found in drinking water; correct?

A. Yes.

Q. They include the distribution before water enters the distribution system; the second one is at the end of the

filtration process, before distribution system; and lastly, at the consumer's tap.

I can see there are three different regulatory approaches; correct? Different EPA regulatory standards, in that table.

A. Three approaches, one for each of the contaminants?

Q. Yes, for each contaminant.

Can you explain why we have three different standards for the three different sources?

A. A different standard for arsenic and cryptosporidium and lead?

Q. Yes.

A. Because there's a conclusion that we need to worry about different levels of contamination for each of the three, that they can't all be the same -- the same standard would not be appropriate for all three. They need to have their specific standard, based on the hazard identification process and the whole risk assessment that underlies the setting of standards.

Q. And the purpose is of course to eliminate the lead in each of these possible sources, so that you could achieve the so-called lead offset zero in drinking water?

A. Well, I think the EPA says that it's not really feasible to achieve a level of zero.

Q. Yes.

A. So they set a standard that they hope will be protective of the most vulnerable subgroup of the population.

Q. Yes, and if we turn to page 3, which appears to explain why this 15 micrograms was arrived at -- if we look at paragraph 2, it states:

"To establish the action level, EPA reviewed information from representative water systems, efficacy of different treatment technologies, and cost-effectiveness of these technologies."

And it referred to footnote 1, which states:

"EPA gathered data from 39 medium-sized water systems. Approximately 96 per cent of these systems were able to keep in the 90th percentile in the range of 10-20 [micrograms per litre]. Thus, EPA concluded that 15 [micrograms per litre] represented the feasible level for public water systems."

It appears why they have chosen this particular figure. Do you accept that, Professor?

A. Yes, it's not strictly health-based but it takes into account the feasibility as well. It's a risk management standard, not necessarily a health-based standard.

Q. Yes. Thank you. It refers to an action level. This is my last question. So is it correct that this action level is simply a screening tool for determining whether

certain treatment technique actions are required?

A. That's my understanding, yes.

Q. Thank you.

MS WONG: Thank you, Professor.

Cross-examination by MR LEE

MR LEE: Yes, Professor. You have said that a child gets one chance to develop his brain, something like that. If he misses that chance, presumably, when the child grows older, his IQ level would not compare as well as his peers; is that right?

A. Could you say the last part, may not compare?

Q. May not compare as well as his peers.

A. Not as well as it would have been had the child not been exposed to lead. It may still be higher than his peers.

Q. But he has one chance to develop the brain, and if he misses that chance, can he ever catch up?

A. Unfortunately, with brain development, it may not be possible to catch up. As I mentioned, a variety of processes need to happen at the right time, and putting cells in the right place, and if that chance is lost then there may be permanent effects that cannot be overcome. The effects may be reduced if the child has certain advantages after that point, but the brain cannot go back and rewire itself.

Q. But if the lead content, say from whatever source, gets

into the blood, then presumably it could reach the brain?

A. Unfortunately, yes. There is -- as adults, we have a nice system called the blood-brain barrier that has very tight junctions between the endothelial cells and it limits the size of molecules that can pass into the brain.

In children, those adjunctions are not so tight, they are kind of leaky, for the first couple of years. So things like lead can get into the brain more easily in a young child than in an adult. That's why the central nervous system is more vulnerable in a child than the peripheral nervous system in an adult.

Q. When the lead gets into the brain, does it settle there?

A. Yes. Unfortunately, it has an easier time getting in than getting out.

Q. Ah. So what effect would it be on that child?

A. Well, it depends on the dose, how much lead. What we now think is that there are certain areas of the brain that are more vulnerable to the effects of lead than other areas. One of the primary areas is the frontal lobe, the prefrontal cortex, which is where what are called executive functions tend to lie, and those are things like the ability to do long-term planning and organisation, to develop strategies and to adapt the

strategies in the face of new information, the ability to delay gratification. This is why we think that children with too much lead are more prone to ADHD and behavioural problems, because executive functions are the major underlying deficit of children with ADHD.

We also know that lead is particularly dangerous to a small area of the brain called the hippocampus which is where learning and memory take place, a process called long-term potentiation, which is dependent upon the glutamate system of the brain, which is a neurotransmitter, and lead interferes with the function of that.

So this is a child who may have trouble keeping up in school, may get into behavioural difficulties, and those I think also underlie the propensity I mentioned to aggression and getting into trouble and violence, because they can't delay gratification, don't have as much success in school and so drop out and start making poor choices because of executive function problems.

So I think these issues compound as a child get older and play out in ways that are very disadvantageous.

Q. So once the lead gets into the brain, and supposing the cause of the lead in the brain is actually from lead in the water -- let's assume that -- will the child improve

after he moves away from that source?

A. Well, it's always good if exposure is reduced. So if the child is no longer taking in lead from that source, that's certainly of benefit.

In the literature on lead, you do see that the early deficits associated with childhood exposure are permanent. Personally, I don't believe that. I think it's too pessimistic. I think there are studies -- the prospective studies that have followed children do show that the deficits persist, but usually children remain in the same environment, and so they are continually exposed.

My feeling, and there is actually some animal evidence to support this, is if you raise an animal in an enriched environment, which for a rat means a bigger cage, a cage that includes toys to jump around on and other rats to interact with, that actually can prevent some lead-associated cognitive problems.

Q. Provided there is no lead in the toys?

A. No lead in the toys, correct. But we don't know -- we don't have good studies to indicate whether that's true for children, and I believe that it probably is, but we just haven't done those studies.

Q. But would you agree with me that once lead gets into the brain, it might or it might not be permanent? Would you

accept that?

A. It might not. There are animal studies that show that -- that have sacrificed the animals and measured the actual concentration of lead in different areas of the brain, and those studies have shown that if you put -- if you expose an animal, their blood lead goes up, eventually their brain lead goes up. You take the animal out of that, you stop the exposure, their blood lead comes down, but for the foreseeable future the brain lead remains elevated. But the animals have been sacrificed. If they let the animals live forever, perhaps brain lead would come down, but so far, it doesn't look like that.

Q. Let me come back to lead in the brain of a child. Can you get rid of the lead once it gets into the brain?

A. As far as we know, the chelating drugs that are used when a blood lead level in a child gets above 45 primarily takes the lead out of the blood and to a lesser extent some of the soft tissues. But the brain doesn't seem to be one of those soft tissues, and I think it's because the chelating agent is too large a molecule and even it can't cross the blood-brain barrier. So the answer is, as far as we know, no.

Q. So the chelation treatment, is it an intrusive form of treatment?

A. Certainly it has to be considered --

Q. How would it affect the child, say?

A. Well, there are different options among chelation. Until about a decade and a half ago, the chelation required hospitalisation and giving the child very painful intramuscular injections or IV injections of chelating agents.

Q. How frequent would the injections with?

A. Well, for the most common course of chelation, it's a 19-day course of treatment, and then you stop and let the re-equilibration process that I talked about go on. Usually what happens is the child's blood lead drops precipitously when you start chelation, but then a week or so after you compete it, it comes back up because of the re-equilibration, and so it's necessary to give another course of chelation.

Q. So how many courses of 19 days each?

A. As many as needed. In the Zamfara episode, where children had blood lead levels of several hundred -- I talked to the paediatrician who did the chelation and one child required 68 courses of chelation, and the blood lead still was elevated.

There are now oral chelators, dimercaptosuccinic acid, that can be given on an out-patient basis, which is much less intrusive and more acceptable to parents

and children.

Q. Is it also more effective?

A. It is not effective in reducing the cognitive deficits. It does reduce the blood lead and the soft tissue lead, but there was a randomised trial where children of blood lead levels of 20 to 44 were randomised to receive chelation with oral succimer or placebo, no treatment, and the children were followed up after treatment was completed and there was absolutely no difference in the cognitive outcomes of the children.

So waiting for the child to become poisoned, it's too late, apparently, to prevent the adverse effects of lead on the brain.

Q. Professor, you mentioned chelation. Are there other methods of treatment?

A. That's the first choice. But as I say, chelation is only indicated when a child's blood lead is above 45.

Q. That's very high.

A. Yes. For blood lead levels 44 and below, the interventions are the environmental investigation, to identify an ongoing source, the nutritional counselling, to make sure that the diet is adequate. But unfortunately there's really not much else to offer. The chelation is counter-productive in a child with a blood lead below 44 because it chelates not only lead

but chemicals that look like lead, and the calcium and the zinc that we talked about earlier, and there is some renal toxicity. So it's not performed below 45.

Q. I move on to another topic. You have been asked many questions by other counsel about other sources of lead getting into the body, apart from lead in the water; soil, air, dust and all this. Let me pose a number of questions to you.

I will use two examples, scenario A and scenario B. Before I go to those examples, is there any relationship between the level of lead in water and the level of lead in the blood?

A. Yes.

Q. Scenario A: if the lead content in water is low, like 2 micrograms per litre, but the lead content in the blood is relatively high, say 20 micrograms per decilitre, would you say in this example that the excess of lead in the water is an important contributing factor to the rather high presence of lead in the blood?

A. I would say it's unlikely. It's more likely that there's some other source that's of primary importance for that one child.

When I said there is a relationship between water lead and blood lead, that again is looking at a large population, where those sorts of unusual circumstances

would even out.

Q. So I come to scenario B. Now, this time there's also high lead content in the water, say 50 micrograms per litre in the water, and on the blood side it's also high, say also 50 micrograms per decilitre. In scenario B, would you say the lead content, high lead content in the water, is an important contributing factor to the lead in the blood?

A. Probably, but again I would want to know what the other potential sources are, and I would want to know if the child was actually drinking the water.

Q. Of course, or the baby drinking milk powder where leaded water was used.

A. Yes.

Q. But of course I forgot to mention that if you assume the other factors were equal in scenario A and scenario B -- do you follow me?

A. (Nodded head).

Q. Otherwise, if the other factors are totally different, then you cannot make a comparison. So you have to make a comparison on the basis that the other factors were equal in scenario A and scenario B?

A. I agree.

Q. So, if that is the case, then if the presence of lead in the water is high and the presence of lead in the blood

is also high, then you would say the water is an important contributory factor?

A. More likely than not, and you might want to interrupt the water pathway to see what happens to the blood lead and see if it drops.

Q. Yes. Thank you.

Now, you have in your report made some importance on the age of a child: 0 to 1 and then 1 to 6, and then 6 to 18. I think this is more or less what you are directed to do by the Commission.

A. Yes.

Q. But six years is a rational age; is that right?

A. A threshold?

Q. The threshold of six.

A. What do you mean by "threshold"?

Q. Because I see in your own report, at page 7, internal pagination, that is page 51, the first complete paragraph:

"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."

There, you use six years; is that right?

A. Well, in this study, they measured blood lead quarterly

in these children for the first few years of life, and then up to six years of age, but levels beyond that were not available until they became adults.

Q. I see. So you say this: if you call that a threshold at all of six years, it's really optional? You could put it up to eight years or seven years?

A. I was just describing the data that were presented in this paper. They didn't have blood leads at eight.

Q. But the Commission then designated six years as the threshold?

A. Yes, and I think that's because, for instance, the US CDC says that children up to six are the most vulnerable group. There is no bright line.

Q. But there is logic in it?

A. There's what?

Q. There's logic in six years, is there; you agree?

A. Well, I agree that the risk is greater among young children, but I don't think there's a bright line. For pragmatic reasons, we have to choose a cut-off, and six is the one that people generally use. But, you know, lead isn't good for anybody.

Q. And it should be zero?

A. That would be great. I would love to see that.

Q. Yes.

Now, looking at your report, at page 17, the

internal pagination -- it's the care plan, is that right, that has been used in the US?

A. Correct.

Q. For children, what is the age of those children? What is the threshold there?

A. This is children under six.

Q. Also six?

A. Yes.

Q. Very well. You see on the left-hand side, it's "Blood lead concentration [less than 5 micrograms per decilitre]". Then you have a number of things: lead education, dietary and environmental, and then environmental assessment for pre-1978 housing.

That's old housing; right?

A. Yes. That's when leaded paint for interior use was banned in the United States. So it's a useful screening criterion.

Q. Then follow-up blood lead monitoring. If you look at that and compare it to Hong Kong -- it's bundle E2, page 871. I think you just got these documents yesterday or today. It should be there.

We can see the chart. The first one, 1, then it's less than 5 micrograms per decilitre; "Reassurance and no further follow-up".

So, in other words, as far as the Hong Kong

government is concerned, when the lead content in the blood is less than 5 micrograms, then they don't do anything about the child, it would appear? Do you understand that?

A. Yes.

Q. But in the United States, there were these four things that I have mentioned to you -- three things; right?

A. I'm sorry, could you repeat that?

Q. I referred to your own chart just now. So, even less than 5 micrograms, there are three things done in the United States?

A. Well, I think I actually made an error here, because if you look down the table, for the schedule for follow-up testing, the first --

Q. Your table or the Hong Kong government's table? You say there's a mistake. Mistake in --

A. In my table.

Q. Okay.

A. Yes. There isn't a schedule for following up a blood lead level of a child whose concentration is less than 5 micrograms per decilitre. You can see it's only when it's between 5 and 10, the recommendation is to have a follow-up between two and four months after the initial value, and then, if the blood lead is declining, only after six to nine months.

So, in the United States, there is not any universal screening of children for blood lead. There's targeted screening if the child has some risk factors, if someone suspects that they may be exposed to too much lead.

In my own State, there is mandated screening to age 4, but then thereafter only if there are circumstances that make the paediatrician suspicious.

Q. When there are certain symptoms; is that right?

A. Well, not symptoms of lead toxicity, but if the child has risk factors, so that if the child is living in a pre-1978 home, or if a sibling has been lead poisoned, or if it's known the parent is occupationally exposed to lead; factors such as those.

Q. Now, the Hong Kong government set the threshold at eight years old instead of six.

A. Six? That's prudent. It's conservative.

Q. That's more prudent, because instead of six it's now eight?

A. Right.

Q. But these are only children in the affected public estate, as far as the Hong Kong government is concerned, right, not generally?

A. That's right. Actually, they include children under 12, so that's even more prudent.

Q. But the trouble is, if a child lives in a certain public

housing estate which is affected by lead in the water, and he has been living there for a number of years, but there was no discovery of excess of lead in water yet, and then he moves out, and then say he becomes nine or ten years of age, then he goes above the threshold of eight years, and so he will miss out from the government's plan. Do you follow me? Because the government only looks at children under eight years old.

A. I don't know that that's the case.

Q. But if that is the case, what do you think about it?

A. Well, they are doing screening of all individuals who live in the estates.

Q. You understand that the Hong Kong government does screening for all children in these estates which are affected? Is that your understanding?

A. That was my understanding, yes. And I would hope that there is a system in place to continue tracking these individuals over time. So, in the scenario you mentioned of a child moving out of the estates, if that child still has an elevated blood lead, no matter where that child is, they should be in the case management system until the blood lead falls below the reference value.

Q. But the problem is, if a child is already nine or ten when the whole of Hong Kong knew about this excess of

lead in water, then he is already too old for the threshold of eight.

A. Well, looking at the line listing that I have been provided, there are individuals above the age of eight who are being followed and, if they have a blood lead level above the reference, they are being re-tested.

So your scenario doesn't accord with my understanding of the data that I have been given.

Q. Perhaps I should show you a document which we got from the internet, a government document. It is from the Centre for Health Protection, and the title is "Incident of lead in drinking water". (Handed).

These are what's called the frequently asked questions, and then the government providing the answers. The question is, "When can we arrange for a blood test?"

"For residents of the affected public estates who are children under eight years old, pregnant women and lactating women, they can make arrangements for blood testing via the hotline ... The hotline will operate [between certain hours]. It will be diverted to the Government hotline ... after operating hours."

So it only caters to children under eight years old and pregnant or lactating women. So if the children are over eight years old, then they have to consult their

own laboratory or whatever.

That is why, if a child had lived in one of these estates affected by excess lead in the water but then moved out, and is now nine or ten, then there's no such facility provided to this child.

A. I see what you are saying, yes. It appears so.

Q. Is that satisfactory?

A. Well, in the ideal world, no. This document does say, in this particular situation, we need to channel limited resources to children under eight, pregnant women and lactating women. If I were forced to make a choice about which groups to focus on, I would make the same choice.

But I agree, in an ideal world, the coverage would include other subgroups as well.

Q. But would you not mention somewhere, maybe in a footnote or whatever, that if the children have been living in these estates for a number of years but now they are over the age of eight, then also ring the hotline or at least get their parents to ring the hotline?

A. That's a very point.

Q. Thank you. Now, if the government sets the threshold at eight years, which you say is prudent, more prudent than six, but they should treat it as a reference point only, or should they treat it as at a reference level or

an action level? Do you see the difference between reference level and action level?

A. I don't. In the care plan, they do recommend or mandate certain follow-up activities, if a child's level exceeds the reference value. So it looks to me as if action and reference levels are being interpreted as one and the same.

Q. Now, I want you to look at the letter from the government which you have been looking at. This is page 846 of E2. You have been looking at this letter already, right, earlier?

A. Yes.

Q. You are familiar with this letter.

Go to the next page, towards the bottom of the page, 847. The last paragraph says:

"Children identified with developmental or behavioural problems ..."

Perhaps I should go -- sorry, I want to read the paragraph above that, beginning with "Regarding":

"Regarding the 'developmental assessment' mentioned in page 19 of the expert report ..."

That is your report; right?

A. Yes.

Q. "... all children with elevated blood lead level will receive preliminary developmental assessment at DH's

Child Assessment Centres" -- that's Department of Health -- "(for pre-school children below 6 years) or Student Health Service Special Assessment Centres (for schoolchildren 6 to 12 years) by a developmental surveillance team composed of paediatricians and nurses."

Then it talks about what the development assessment covers. Then the last paragraph:

"Children identified with developmental or behavioural problems will have follow-up evaluation at Child Assessment Service according to individual needs."

So once a child is identified to have these problems, developmental or behavioural, then there will be follow-up. Then:

"Children with largely normal development would receive continuous monitoring through enhanced developmental surveillance at Maternal and Child Health Centres ... during pre-school years and annual health visits at Student Health Centres during school years."

So two types, those who are identified with behavioural or developmental problems, there will be follow-up; those with largely normal development would still have enhanced developmental surveillance. Then:

"Parents are provided with anticipatory developmental guidance and information on children's

development in the form of pamphlets. These pamphlets are available online at [so and so]. Due to the large volume of these documents, hard copies will be provided upon [the Commission of Inquiry's] request."

Do you see that? Have you actually gone online to take a look at these pamphlets?

A. No, I didn't.

MR LEE: Can I have some indulgence? I want to discuss with my junior, because it does say if you want hard copies, it's only upon your request. We couldn't get a copy ourselves.

CHAIRMAN: Do you want a copy?

MR LEE: Yes.

CHAIRMAN: Have you ever seen this document first?

MR LEE: Online, yes. Because to us, they are totally unimportant. Nothing to do with this case.

CHAIRMAN: So you want a copy?

MR LEE: I want to show you and the members --

CHAIRMAN: Why don't you show me online first?

MR LEE: Okay, online. Can we do it here? I don't know whether we can do it now, because we do not have ...

CHAIRMAN: Let me make this suggestion, since now it's almost 4.15. Why don't you or your junior go back to your chambers and look at the relevant paragraphs and identify the relevant paragraphs, and then I don't think

you really need to print an entire copy of these documents. So print the relevant paragraphs or pages, or indicate to us which particular paragraph or pages that you want, then we can print it ourselves.

MR LEE: My junior read them. Perhaps I can consult with her.

CHAIRMAN: Yes. Or you can tell me now, actually.

MR LEE: It may be possible for us not to go into these pamphlets, but I would like to discuss with the team in the meantime.

CHAIRMAN: All right. So if --

MR LEE: I won't be long, but I would like to have some time to make sure I won't be long.

CHAIRMAN: I will give you the evening to prepare your case. So if you think a particular paragraph or pages are relevant to our hearing, then inform my secretaries and the other legal teams.

MR LEE: Yes, we will do that.

CHAIRMAN: So that's it for the day, Prof Bellinger. I would be grateful if you could come back at 10 o'clock tomorrow morning.

WITNESS: Certainly.

CHAIRMAN: Thank you.

(4.15 pm)

(The hearing adjourned until 10.00 am the following day)

Tuesday, 15 December 2015

(10.05 am)

MR KHAW: (Via interpreter) Good morning, Chairman. Before the professor gives evidence, earlier on there was a witness, Mr Chan Siu Wah, a former employee of Ho Biu Kee. We issued a letter to him, asking for a witness statement, and at that time, there was no reply. Then, around about the 7th or the 8th of this month, we issued a witness summons and Mr Chan has now arrived to attend our Inquiry.

CHAIRMAN: (Via interpreter) Mr Chairman, good morning. I am the chairman of the Commission of Inquiry, Justice Chan.

Earlier on, we have issued a witness summons for you.

MR CHAN SIU WAH: (Via interpreter) Yes.

CHAIRMAN: (Via interpreter) This is because in relation to the Inquiry, we do have some questions for you.

MR CHAN SIU WAH: (Via interpreter) Yes.

CHAIRMAN: (Via interpreter) You are not represented by any lawyers; right?

MR CHAN SIU WAH: (Via interpreter) No.

CHAIRMAN: (Via interpreter) It doesn't matter. It's fine for you to represent yourself. Basically, we would like to ask you some questions, and that's in relation to the

time when you were working for Ho Biu Kee, in particular in relation to the purchase of solder materials. Do you understand?

MR CHAN SIU WAH: (Via interpreter) Yes.

CHAIRMAN: (Via interpreter) For the witness summons, you have been asked to attend the hearing today. You are asked to attend but then you haven't been told as to when you should give evidence. First of all, our lawyers' team will make an appointment with you, either today or sometime later, at your convenience. We would like to meet with you so we can get a witness statement from you. Do you understand?

MR CHAN SIU WAH: (Via interpreter) Yes, I do.

CHAIRMAN: (Via interpreter) In other words, it will be at a time convenient to both parties. After you have given the witness statement, then later on you will be asked to attend the hearing as a witness.

As to exactly which day, which month, we don't know, but of course we don't want you to be here waiting all the time, so later on, when we do know when you will be called, then we will give you notice a few days before the date of attendance. Is that fine?

MR CHAN SIU WAH: (Via interpreter) Yes.

CHAIRMAN: (Via interpreter) Thank you. You may now be excused, and our team of lawyers will make

an appointment with you, when you are outside, so as to get a statement from you.

MR CHAN SIU WAH: (Via interpreter) All right. Thank you very much.

MR KHAW: (Via interpreter) I now invite Prof Bellinger.

CHAIRMAN: (Via interpreter) Thank you.

PROF DAVID CHUDLEIGH BELLINGER (sworn)

(All questions and answers were in English)

CHAIRMAN: Thank you. Take a seat please, Professor.

Examination-in-chief by MR KHAW

MR KHAW: Prof Bellinger, first of all, thank you very much for coming all the way to Hong Kong to provide assistance, as an expert appointed by this Commission for the purpose of this Inquiry.

We understand that you have prepared a report, dated 1 December this year. This report can actually be found in our e-bundle at V1. It is now shown on the screen. Your report has also been downloaded to the website of this Commission, so that in fact the public could have access to the same.

I don't intend to read out the contents of your report verbatim, as I don't think people here can bear my voice for so long. Instead, I will try to go through and summarise most of the issues that you have addressed in your report.

Unlike the chairman, my science knowledge is rather limited, so perhaps, in the course of going through your report, I will ask you to further explain some of the matters which might call for further discussion.

If I may now first turn to internal page 3 of your report, page 47 of the bundle, where you have set out the areas on which you have been instructed to provide your opinion for the purpose of this Inquiry. Perhaps I will just go through those items:

"(1) to explain the short, medium and/or long-term health effect(s) ... 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 ... ('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 ... for heavy metals and make recommendations, if necessary."

If we first look at the introductory part of your report, at internal page 4, page 48 of this bundle -- I think here, before you deal with the first question that we have just seen, you have provided some introductory comments on the availability of information on the adverse effects of lead on human health. It seems that you have come to a conclusion, at I think around the fifth line under "Introductory comments", that:

"... 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."

May I just ask you to briefly explain what you mean by "dose-response and dose-effect relationships"?

A. Yes. That's a distinction made in toxicology, referring

to how an adverse effect is related to exposure.

A dose-response relationship, if one exists, means that the higher the dose an individual is exposed to, the greater the risk of some adverse effect such as cancer. And a dose-effect relationship pertains to an outcome that is not dichotomous, such as do you have cancer or don't you have cancer, but something like an intelligence quotient score. So a dose-effect relationship would mean that the higher the dose of some chemical an individual is exposed to, the IQ goes down, as dose increases.

Q. Thank you. Then you go on to say:

"In my response to the queries" -- ie the questions set out above -- "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."

If I may just ask you to explain this observation.

A. My understanding is that the highest water lead concentration that was measured in the Hong Kong estates was 83 micrograms per litre, but most of the levels were in the range of 10 to 20 micrograms per litre.

According to our current knowledge, regularly consuming water with water lead in the concentration of 10 to 20 would be unlikely to give a large enough dose that someone would show clinical signs of lead toxicity, which tend to occur at much higher blood lead levels, in the range of -- it varies from individual to individual, but it's rare to see clinical symptoms in someone with a blood lead below 40, 45 micrograms per decilitre, and higher than the blood lead levels observed in the residents of those estates.

Q. Under the same paragraph, you then continue to talk about the overt signs of acute intoxication.

CHAIRMAN: Before you ask that question, can I ask you, Prof Bellinger, how long can one be regarded as chronically exposed?

A. That's a very difficult question to answer. It typically refers to a period of months to years, would be chronic exposure. Anything else shorter would be considered more of an acute exposure.

CHAIRMAN: Thank you.

MR KHAW: Under the same paragraph, you then continue to talk about the acute -- the overt signs of acute intoxication, acute lead intoxication, and also chronic lead exposure.

Then, if we turn to internal page 5, the second

paragraph, about the second sentence, you mention that:

"The major classes and 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 ([including the] use of contaminated folk or herbal medicines)."

Then further down, in about the middle, you talk about the places in a person's body where lead resides. I think, in short, just to summarise what you have set out here, in adults lead in bone accounts for about 90 per cent of the total body burden, whereas in children, lead in bone accounts for about 70 per cent of the total body burden, because in the case of children lead will move in and out of bone more rapidly, due to the changes in bone structure.

Then there is a discussion regarding re-equilibration process. If I may just quote what you have stated here, about the last ten lines on this page, starting from the word "Therefore" -- can you see that?

"Therefore, the blood lead level measure for an individual at any time reflects the equilibrium between an individual's current exposure to 'new' lead and the 'legacy' lead" -- ie the old lead which has accumulated in our body for a relatively long period of

time -- 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 ..."

May I just ask you to elaborate a bit more on the re-equilibration process that you have just mentioned here.

A. Certainly. Over time, the lead that is distributed into the three major compartments, the mineralised tissue, the soft tissue -- by which I mean organs such as the brain, kidneys and the liver -- and the blood is the third compartment, will come to a balance that reflects the amount of lead someone is taking in and the amount that they are excreting.

Now, if there's some intervention that changes an individual's exposure at a given time, such as removing exposure to water lead or administering -- in

cases of much more severe exposure, giving drugs that bind to the lead and promote its excretion from the body -- then the amount of lead that's distributed in those three major compartments will find a new balance. And what happens is if blood lead is reduced because of cutting off an exposure pathway, blood lead will decline reflecting that, but lead from the other two compartments, the hard and the soft tissue, will come into the blood compartment and a new balance will be achieved between those three compartments.

How long that takes will reflect how much the exposure is decreased and how much past lead an individual has been exposed to and is storing in those other compartments.

Q. Thank you. Then you continue to talk about a study, one study of children -- at internal page 6, page 50 of the bundle -- who had a blood lead level of 25 to 29 micrograms per decilitre and were placed in a case management system; it took an average of two years for the blood lead to drop below 10.

Can you provide any reasons for this slow drop in that study?

A. Yes. That's because children who had a blood lead of 25 to 29, in this particular study were living in homes that had deteriorated lead paint, and they had been

living there for some time, so they had been continuously exposed and had built up a considerable amount of lead in their bones and in their soft tissues, so that when the children were removed from those homes and placed into the case management system, which means that their current exposure was drastically reduced, because they were removed from the hazardous environment they had been in, then their blood lead fell quite slowly, because of this equilibration process, because they had been exposed to so much lead in the past that during the re-equilibration it was being drawn out of the hard tissues and the soft tissues into the blood, so that the blood lead fell very, very slowly, certainly longer you would expect from the simple fact of a half-life of lead being 30 days. That's why you can't expect blood lead to drop by a half in 30 days. It depends on the entire exposure history of an individual.

Q. Thank you.

I think most people would be interested in the next topic, because that goes to the IQ score of children. Most parents want their children to obtain a high IQ score and they would like to see whether the lead content in water --

CHAIRMAN: You have to speak up, Mr Khaw.

MR KHAW: Sorry. I think most parents want their children

to achieve a high IQ score, and they also want to know the effect of lead content in water on this particular aspect.

You have started your discussion by saying that young children are considered to be most vulnerable subgroup of the population. Then you went on to say that children with blood lead levels below 25 micrograms per decilitre generally do not show any signs or symptoms that bring them to medical attention. But studies also show 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 wellbeing.

Then you talk about what you call the most complete and compelling evidence in respect of children. That is I think the last ten lines on this page:

"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 [microgram per decilitre] increase in blood lead concentration), was steeper over the range below 10 ...

than it was over the range between 10 and 30 ... Although the biological mechanism of this is not known, the finding has now been replicated in several independent studies."

Professor, just one question here. We all know that children's IQ score may be affected by various factors -- environmental factors, behavioural factors, et cetera -- so to what extent can these studies on lead content demonstrate the extent of the impact of lead on children's IQ scores?

A. That's a very important question, and it's always -- whether or not investigators have addressed and measured and taken adequately into account the other factors that influence a child's IQ is always the major point of contention in interpreting a study such as lead, because we can't do an experimental study with children like we can with animals. We have to observe samples of children who are exposed to different amounts of lead and see if we can see an association with their cognitive outcomes.

So in each study -- and as I say, it's very important to measure the parents' socioeconomic status, their education, if possible the parents' IQ, because that's very strongly predictive of a child's IQ. It's important to take account of a child's nutrition, how

stimulating the home environment is, whether they are exposed to other things that can pose a threat to their cognition.

So the statistical models that have to be implemented in these studies can be quite sophisticated and very detailed. And what we find is if we take account of all of these other factors to the best that we can, is there still a relationship between lead and children's IQ, and what these studies indicate is yes, there is.

We can also get a sense of how important lead is in the context of all these other factors by asking what per cent of the variability in children's IQ scores can be explained by their lead exposure. Typically, what we find is that it's a relatively small amount. It's statistically significant and consistent across studies, but it explains usually less than 5 per cent of the variation in children's IQ, whereas something like socioeconomic status captures about 30 per cent of the variation in children's IQ.

So lead is important but it's not the most important predictor of children's outcomes, and this is something that I counsel parents about when they call me, when they are concerned that their child has a blood lead higher than we would like to see. I tell them, yes,

that's very important and we certainly want to prevent that lead exposure, but it's important to put it in the perspective of what else may be influencing how their child does, and I try to help them relax a little bit in this regard; that yes, we should prevent lead exposure, but it's also important to do these other things that parents do that maximise their child's endpoints.

CHAIRMAN: Can I ask you a more basic question first: why is lead so toxic?

A. That's a good question. It works at a very general biological level, and so it impairs processes that are fundamental to many aspects of the way our bodies work. Chemically, it's what's called a divalent cation; it has a 2-plus charge, and many important things in the body like calcium have a 2-plus charge. So lead interferes with a lot of the biological processes that depend upon calcium, and many of our body processes do depend on calcium.

So how the nerve cells in our brain communicate with one another is dependent on calcium, so lead interferes with the communication between cells in the brain. It also interferes with processes that are important in long-term memory and storage of information. In fact, lead interferes with just about everything that anybody has looked at in the brain. So that explains his

pervasive and widespread toxicity.

CHAIRMAN: Thank you. So that's why you say the central nervous system is considered to be the most vulnerable organ among all the organs within our bodies?

A. That's right, in particular the developing central nervous system. As a colleague of mine says, a child gets only one chance to develop a brain, and so we want to prevent lead from interfering with the complex spatial and temporal choreography that's involved in putting a brain together, that involves billions of nerve cells that have to be in the right place at the right time in order for a brain to be normal.

CHAIRMAN: So when you say the central nervous systems, you are not confining yourself to simply the brain; you are also talking about the nerve cells, the nerves that can be found in our bodies, the whole body as well?

A. Well, the central nervous system typically is used to refer only to the brain and the spinal cord, and the connections of the central nervous system to, like, the hands and sensory organs in other parts of our bodies is the peripheral nervous system. That is also sensitive to lead but less so. Often the effects in the peripheral nervous system are reversible, once exposure stops. But we are concerned that the effects are more persistent in the central nervous system.

CHAIRMAN: Is that because the lead displaces the calcium in the process of transmission of nerve messages; that actually adversely affects the development of children?

A. Yes, that's part of it. These chemicals that lead interferes with are involved not just in the function, the transmission of information, but also they are involved in the formation, the laying down of the structure, the fine structure of the nervous system. So lead is both a developmental neurotoxicant and a functional neurotoxicant. That's why the developing nervous system is of particular concern, because lead can interfere with actual structural formation of the brain.

CHAIRMAN: Thank you.

MR KHAW: Still at internal page 6, the last sentence is perhaps quite important:

"The details of this dose-effect relationship suggest 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 [micrograms per decilitre]."

The reason why I ask you in relation to this particular sentence is since your report has been released, there has been some media coverage which has taken this sentence to mean that the IQ of a child with

a blood lead level of 10 micrograms per decilitre would be one-third lower than that of a child with zero blood lead content, but in fact you are talking about one-third standard deviation here. Can you explain a bit more on the standard deviation point?

A. Yes, that would be an incorrect interpretation.

Standard deviation is a property of a bell curve distribution. It refers to how much variability there is in a particular characteristic. So, for instance, with regard to IQ, we expect that the average person will have an IQ of 100, and the standard deviation of the IQ distribution is 15 points.

So this is just a way of saying how many people we expect to have IQ scores within certain ranges, so between the mean of 100 and plus 1 standard deviation, that is a score of 115, we would expect 34 per cent of the population to be between 100 and 115. Similarly, because the distribution is symmetrical, we would expect 34 per cent to have a score between 85 and 100.

So the one-third standard deviation just simply means that the effect that has been observed in studies that's attributable to lead, the effect on IQ, amounts to five points, for an increase in blood lead from 0 to 10, which is one-third of that standard deviation. It doesn't mean that the child with a blood lead of 10

would have an IQ that's only one-third as large as the child with a score or a blood lead of 0.

Q. Thank you. In the next paragraph on the same page, you have referred to a series of neuroimaging studies of young adults, and those studies actually show some structural or ischemic changes of various parts of the brain, including the grey matter volume and also the white matter volume.

Can you just briefly explain the importance of these parts of our brain?

A. The grey matter is basically what we think with, so it's the cerebral cortex; we have a frontal lobe, occipital lobe, parietal lobe on both sides of our brain, and that's where most of our thinking goes on; and there are also some subcortical structures that are part of the grey matter that are also important, the basal ganglia and so on.

The white matter are basically the information highways in our brain. The white matter tracts are quite long and they connect different parts of our brain, so they are the way that our different parts communicate with one another.

So they are all important in terms of having a healthy brain. We want both grey and white matter to be functioning as they were intended.

Q. In these studies which actually show persistent changes in brain structure and function, are we talking about blood lead content which is quite high?

A. We are. The neuroimaging studies come from a particular study that was conducted in the United States in Cincinnatti, among mostly minority African-American children who were living in fairly dire situations of poverty; inner city, in poor housing, exposed to lots of lead. They had blood leads that were -- most of the children in the study, there were about 250 children, had at some point in their life a blood lead above 25, and some of them had blood lead so high that they were actually hospitalised and given chelation therapy to reduce their lead burden.

So these were children with exposures that were not that uncommon at that time -- this study was begun in around 1980, in the US -- but fortunately they're fairly rare these days, and it's uncertain the degree to which these studies apply to children with lower blood lead levels. They are simply a lesson that lead is actually doing observable damage to the brain.

Q. Then the last paragraph on the same page, where you are addressing the issue regarding children and teenagers between 6 and 18 years. I think you have also referred to various studies which provide evidence regarding the

inverse association between early-life lead exposure and neurodevelopment.

Then at page 8 you talk about the US National Toxicology Program evaluation, which concluded that the evidence is sufficient for the association between blood lead level under 10 micrograms per decilitre and both decreased hearing, delayed puberty, and reduced postnatal growth.

Again, I just want to ask whether these studies showed relatively high blood lead content?

A. Well, this conclusion pertains to a blood lead range of less than 10 micrograms per decilitre. So, again, these are findings that are relatively consistent across studies, sufficiently consistent that the NTP was willing to consider that in aggregate the evidence was sufficient to draw this conclusion.

But again I would say that finding a statically significant association is one thing, and finding an association of a large magnitude in the effect is another thing.

Q. Yes.

A. So, for instance, this reduced postnatal growth association, it may be relatively modest and something that you would not notice in an individual child, but if you are looking at large groups of children then you

might find that those children with higher blood lead within this range were a centimetre smaller than children with blood lead levels lower than this range. Statistically significant but not really very meaningful for an individual child.

Q. Then in the next paragraph you talk about the association between lead exposure and ADHD, attention deficit hyperactivity disorder. Then you have referred to various studies in this respect.

May I just ask you how these studies were conducted, regarding the association between lead exposure and ADHD?

A. A variety of study designs were used. In some of the studies, which are called cross-sectional studies, a sample of children were recruited for participating in some large survey, and as part of the survey a blood sample was taken, and that was measured for blood lead content.

Then the parents were interviewed and asked, "Has a medical professional ever told you that your child has ADHD?" In other studies, the parents were given a structured interview that was linked to the criteria for diagnosing ADHD, and in yet other kinds of studies, called case control studies, two groups of children were studied. One group had been identified and diagnosed

with ADHD, and the other had not, and then blood samples were collected to see whether or not the group carrying the diagnosis of ADHD had a significantly higher blood lead level than the group that did not.

Q. In the last paragraph on this page, you talk about various studies which show the association between childhood lead exposure and propensity for violence and aggression. Then you went on to say that in fact these studies are difficult to conduct and they are subject to a variety of biases. Why do you say that?

A. Well, in order to try to see if there's an association between early childhood lead exposure, which is the primary concern, and the propensity to engage in these kinds of behaviours 20 years later, it's very difficult to identify a situation in which you have those data.

If you do the study by identifying individuals who already have committed offences, it's difficult to look back and get reliable information about their early childhood lead exposure. Typically, you don't have access to those data. So one of the biases would be -- it's called a retrospective bias; whereas people or parents of individuals who are offenders, as adults may have different levels of recall. They may be more likely to say, "Oh, yes, he was lead poisoned as a child" and you don't really know whether that's true

or not or if they are just looking for an explanation for their child's behaviour.

So it's difficult to find exactly the right situation in which you can address this question in a very compelling way.

The study that I mentioned where the neuroimaging was conducted, a study in Cincinnatti, has provided the best evidence, in my mind, of this association, because they enrolled these children during pregnancy. As the children grew older, they collected periodically blood lead histories, and then they followed the children until they reached their 20s, and they went to administrative county records where these individuals now resided and obtained information from those objective records as to the number of times these participants had been arrested for some offence or another.

So that study, which is very difficult to carry out over that period of time, is less subject to these biases because the blood lead information was collected prospectively, long before the offending occurred, and so there's no concern about bias there. Also because this study had collected a lot of information about factors that I mentioned earlier that are important to take account of -- family socioeconomic status,

nutrition, stimulating-ness of the home environment, and so on -- the associations that they saw between early childhood lead exposure and risk of offending and being arrested later in life I think are much more solid; they provide a much more compelling base of evidence for drawing a conclusion that lead is linked to a propensity for aggression and violence.

But I would add, again, that participants in the study had lead exposures that were quite high by contemporary standards and many of these children actually got chelated because of their lead poisoning.

Q. Then in relation to pregnant women, page 9, in the third line you mention that:

"... the lead exposure of a foetus is essentially the same as that of the pregnant woman."

Then in the next paragraph you refer to research on the potential effects of lead exposure on the health of pregnant women, in particular reproductive health, the course of pregnancy, the health of the foetus at birth, et cetera. Then you have also referred to the US Centers for Disease Control, which "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."

Then you go on to discuss various aspects regarding pregnant women, including fertility, hypertension, et cetera.

In relation to gestational hypertension, at page 10, there's just one question I have. Here, you say:

"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 ..."

Just one question: would such limited magnitude affect the validity of the findings in those studies?

A. No, it doesn't affect the validity of the findings, because the findings have been reported in multiple studies. So I think that they are real. But the modest magnitude does affect the interpretation of the clinical significance.

Again, as I indicate, the correlation, which is a measure of the degree of relatedness of blood pressure to blood lead, is quite small, meaning that most of the variation among pregnant women in their blood pressure or risk of hypertension is not due to lead, it's due to

other factors. Again, we know how to prevent lead exposure and so it's worthwhile to do everything we can, but it won't solve the problem of pregnancy hypertension entirely.

Q. Then, in relation to spontaneous abortion, you first mention that:

"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 [micrograms per decilitre]."

Then you refer to one high-quality prospective study conducted in Mexico City which actually reported that "the risk began to increase, and increased consistently thereafter, when maternal blood lead level exceeded 5 [micrograms per decilitre]".

Are there any special features in this particular study conducted in Mexico City which show this result?

A. Well, I say it's high quality because of the care that the investigators took to try to identify potential confounding factors that may also be related to risk of spontaneous abortion. As I say, this study did suggest that the risk of spontaneous abortion begins to rise once maternal blood lead exceeds 5 micrograms per decilitre. I have no reason to think that the study is wrong, but in this kind of work one never draws a strong

conclusion based on one epidemiological study, because it's always possible there's something unique about Mexico City and the women and their practices that may have contributed to this result. So it's always helpful to be able to have evidence that's been collected in multiple studies, that all point towards the same direction.

So I think this study is important in telling us that there may be something, an important signal here, but until we have additional evidence I would be reticent to draw a very strong conclusion.

Q. Then in relation to foetal growth, that is the next passage, you have mentioned that the evidence regarding the association between lead exposure and foetal growth is perhaps not that consistent.

A. Right.

Q. Can you explain a bit more on this?

A. Well, I just mean that some studies find that increased blood lead during pregnancy is associated with a reduced length of gestation. Other studies don't find that. The same with the other endpoints, the birth weight, head circumference, birth length. So I find it very difficult to know what the truth is. Until we get a heavier weight of evidence pointing in one direction, it's difficult to draw a strong conclusion, in my mind.

CHAIRMAN: But logically, it should have a greater adverse effect on the baby's growth, because the baby's brain is developing at the time?

A. I'm sorry?

CHAIRMAN: Because the baby's brain is developing at the time, so logically it should have a greater adverse effect on foetal growth?

A. Yes, you would think so. Interestingly, the evidence has not borne that out.

CHAIRMAN: I see.

A. And I don't have an explanation for it. It appears to be that the exposure of a postnatal child is a stronger predictor of their IQ and educational achievement than is their prenatal exposure. It may be that there are some protective features in the foetal unit that protect them, protect the foetal brain, from the lead.

It's an important question, and it is a little counter-intuitive, but so far the evidence just doesn't bear it out.

CHAIRMAN: Thank you.

MR KHAW: Perhaps there is just one minor typo, in the last paragraph on this page:

"The NTP concluded that the evidence was ..."

I think the word "is" is perhaps redundant.

A. Yes. I apologise for that.

Q. If we turn to the next page, in relation to lactating women, in the first paragraph, about the sixth line, you have mentioned:

"... the concentration of lead in breastmilk is low, comparable to that in the plasma fraction of blood (which accounts for only about 1 per cent of the lead in whole blood). Thus, this pathway of exposure likely contributes relatively little to an infant's lead exposure."

Then you went on to say:

"Water can be a very important pathway of lead exposure for infants who consume formula" -- milk powder -- "made up with water that contains lead. Balancing the known benefits of breastfeeding and the slight risks of substantial lead exposure from breastfeeding, the US CDC encourages mothers with a blood lead level [not exceeding 40 micrograms per decilitre] to breastfeed."

So I take it that so long as the blood lead level of the mother is not higher than 40 micrograms per decilitre, it is still safe for her to breastfeed. Is that the case?

A. That's correct.

Q. Regarding elderly people, we have the same discussion in relation to legacy lead, the old lead that accumulates

in the body.

If we turn to page 12, internal page 12, you have mentioned one recent development in lead toxicology which is generating considerable interest. That is the study regarding the association between overexpression of genes involved in the production of a protein, which is a constituent of the plaques that are found in the brains of patients with Alzheimer's Disease with lead exposure.

Can you explain a bit more on this study, on whether it is applicable to human beings?

A. Well, the simple answer is we don't know. The studies have only been done in rodents and monkeys at this point. This is another case where it's very difficult to do this study in humans because the finding in the animals is that it's the exposure to lead in very early life that predicts this late overexpression of these genes involved in neurodegeneration.

To do this study in humans, we would have to have a prospective cohort that we had early-life lead exposure measures on, and follow them up to age 60 and beyond, and unfortunately that's very difficult to do and usual people have to take advantage of other data that had been collected, in which this question can be asked. But there are relatively smaller opportunities

to do that.

There is one study in humans that I was involved in where, at age 30, we looked to see whether, on children for whom we did have good lead exposure histories, we looked to see whether or not there was a relationship to the level of expression of genes that are involved in this amyloid protein synthesis pathway.

Now, what we are really concerned about is whether or not the genes are being over-expressed in the brain, but we obviously can't do that, so we were limited to seeing whether they were over-expressed in the blood, and we did see some suggestive relationships between gene expression of individuals around 30 years of age in relation to actually their cord blood lead levels. That's the only study that we have in humans at the moment.

So this hypothesis remains highly speculative, but it is of concern because the current elderly population grew up in a time when we were exposed to much more lead than our current cohorts of children, and so we do know that the incidence of Alzheimer's Disease is rapidly increasing as our population ages, so it's reasonably -- and it's biologically plausible to think that lead might be involved in this process, but again it's too soon to draw very strong conclusions.

Q. Thank you.

Further down this page, at 1h, you talk about long-term patients with chronic illnesses. Then you have referred to kidney function, cardiovascular function and also cognitive function.

There's just one aspect regarding cognitive function that I would like you to elaborate. That is, in the middle of this particular passage regarding cognitive function, internal page 13, you have mentioned at line 4:

"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' operationalised as poorer reading achievement, an outcome that presumably pre-dated the onset of occupational exposure to lead."

Can I just ask you to explain a bit about the low cognitive reserve point that you have mentioned here?

A. Yes. That's a concept that pertains to how readily an individual can be protected from an adverse effect of an exposure such as lead. It's thought that if an individual who is cognitively functioning at a higher level, as reflected in a higher reading achievement or other scholastic achievement, may be less affected by

a given exposure, such as lead, and so show fewer decrements in performance as a result. So, basically, they are buffered, and it's people whose cognitive performance is more fragile who may suffer a greater impact.

Q. Then, regarding the WHO standards that we have talked about very often at this Inquiry, you first discussed the lead content in tap water and then you have gone through the changes in relation to the WHO Guidelines. May I just first ask you this: can you briefly let us know your personal participation in the formulation of the WHO Guidelines over the years?

A. I have not been involved in the guidelines pertaining to water lead. I currently chair a committee at the WHO that's developing guidelines for the diagnosis and treatment and prevention of lead poisoning in children and adults. I've been involved in the lead evaluations for the WHO/FAO joint expert committee on food additives and contaminants that has set provisional tolerable weekly intake for lead, that is the basis for the water lead guidelines. Then I have also been involved in the Foodborne Disease Epidemiology Reference Group, which is developing estimates of the global burden of foodborne disease, and I have been involved in the chemical aspect of that process and lead is one of the chemicals that we

have developed estimates for.

Q. Thank you. We all know that the current reference value regarding lead in water is 10 micrograms per litre. At internal page 14, the first paragraph, perhaps the last six or seven lines of this particular paragraph, you have mentioned that:

"... this is designated as 'provisional' on the basis of treatment performance and analytical achievability."

Then you went on to say:

"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 emphasised that all other practical measures to reduce total exposure to lead, including corrosion control, should be implemented."

Do you have anything further to add in this respect?

A. Not really. Unlike many chemicals which are present in the source water, where you can take measures at that point in the system, lead is different, in that it is introduced into the system at places closer to the point of consumption. So I think the WHO is recognising that to remove plumbing and fittings is costly, and so they

recommend that as a first preliminary step, to take measures to reduce corrosivity of the water it, to reduce leaching of lead, and presumably they would endorse, if that doesn't work, then to take the more costly step of replacing the fittings.

CHAIRMAN: So does that mean that that was known to the WHO back even in 1958, when they first developed the allowable limit?

A. I don't know the answer, what they knew back in 1958. Certainly lead in water and the contribution of lead in plumbing and actually the use of lead to make pipes was known back then. But I don't have specific knowledge of that.

CHAIRMAN: But certainly it was known back in 1974, when the United States passed this Safe Drinking Water Act (1974)?

A. Again, that was before my time. I don't know what the --

CHAIRMAN: Okay. Thank you.

MR KHAW: In relation to blood lead content, we know that the current reference values are 5 micrograms per decilitre for children and 10 for adults. You have also mentioned at internal page 15 --

CHAIRMAN: Before you move to page 15, I note on page 14 the United States adopts sort of a higher value,

15 micrograms. Is there a rationale behind that?

A. Well, that's not the only respect in which the EPA's guideline is different than the WHO's, because the EPA does allow up to 10 per cent of violation, of 15, before some action is mandated.

CHAIRMAN: I see.

A. They will allow up to 10 per cent to be above --

CHAIRMAN: So 16.5?

A. Yes. So all of these guidelines really try to balance health-based considerations with practicality, and I think, you know, that's why they are all provisional, because the health information and the technologies improve, and so over time permit the implementation of more stringent guidelines. But this is where they are at the moment for the EPA.

CHAIRMAN: I understand the ideal value of course is zero. Does that mean that 16.5 is still acceptable?

A. According to the EPA --

CHAIRMAN: But what about according to you?

A. You know, it's hard to take one number out of context.

CHAIRMAN: Right.

A. It would depend upon -- because, as I've said, lead is a multimedia pollutant, so we have to take a look at the entire profile of exposure sources -- the paint, the air, the soil, the diet, hobbies, occupations. For some

people, having a 16.5 might be relatively trivial because they are not exposed to lead in any other aspects of their life, but for other people, who have a higher baseline exposure, that may be enough to put them into a range where we need to be concerned.

So I think it really is hard to give one answer.

CHAIRMAN: I see, that's what you mean. So it's the blood lead level that matters, rather than the lead in water?

A. In my view, that's what we use to make decisions, management decisions, as to whether or not we need to help an individual reduce their exposure. It's what's in their body, that to me is the index of likelihood of an adverse effect.

CHAIRMAN: Thank you.

MR KHAW: In relation to the blood lead level, you have come to a conclusion at internal page 16 that the reference values selected by the Hong Kong government for prioritising individuals for follow-up based on blood lead level are appropriate and consistent with those identified by the international bodies.

A. That's right.

Q. You can confirm that?

A. Yes, I do.

Q. If we then take a look at the recommendations for follow-up actions in respect of both children and

adults, as suggested by the CDC of the United States, at internal page 17. If we talk about children first, the first table, we can see in the left-hand column, blood lead concentration less than 5 micrograms per decilitre, and then we can see that the recommendations for follow-up actions are more limited. They consist of lead education, environmental assessment, and then follow-up blood lead monitoring.

In the next column, if we are talking about blood lead concentration which is in the range of 5 to 45 micrograms per decilitre, then there have been more recommendations for follow-up actions. For example, we have things like environmental investigation, lead hazard reduction, neurodevelopmental monitoring, abdominal X-ray.

First of all, in relation to environmental investigation, and neurodevelopmental monitoring, can you tell us what steps are needed for these follow-up actions?

A. Yes. Usually, the environmental investigation would involve querying a parent about possible lead sources in the environment, finding out where a child spends time, in addition to the home. That of course would be age-dependent. Then, depending on the blood lead level, actually going into the home to collect samples of

different media that the child might come in contact with, such as collecting air samples or dust samples, samples of the soil outside the home, the paint, and noting whether or not the paint is chipping, and asking the parent whether or not they have seen the child put things in their mouth, the child put things in their mouth that are not food. In the extreme it's called pica; asking about occupations of the parents, whether they might be involved in an occupation that involves exposure to lead and so may bring lead home on work clothes; asking whether the parents engage in hobbies that might involve the use of lead and so could contaminate the home environment; asking about foods that the family eats, the medicines they take, whether they take herbal medicines, which unfortunately have been reported, in some instances, to have fairly high lead content; looking at the materials that the family use to prepare food and to serve food -- sometimes, at least in the United States, they are glazed with lead glaze which can leach lead and so get into the food and be consumed -- looking at the toys the child plays with and the furniture, whether it's painted with potentially lead paint.

Really, it's taking a comprehensive look at the environment that the child lives in, to identify

potential sources, because it can be very hard to tell.

Q. What about neurodevelopmental monitoring?

A. This could vary in intensity as well, depending upon a child's blood lead concentration. It could be as simple as asking the parent about a child's developmental milestones, when they first sat unaided, when they first took their first step, said their first word, said their first sentence; or it could involve an in-person assessment using a screening tool by a medical professional; or, if there are red flags that are raised by any of these screening methods, actual administration of more comprehensive neurodevelopmental assessments for diagnostic purposes.

So it really is based on the level of lead exposure and a clinical indication.

Q. Thank you.

In relation to adults, that is the last table on the same page, we can see that for blood lead concentration which is in the range of 10 to 29 micrograms per decilitre, then various follow-up actions are recommended.

Then we can see things like exposure assessment. I take it that that is similar to the environmental investigation that you have just mentioned?

A. That's correct, yes.

Q. Thank you.

Then, talking about the test results that we have got here in Hong Kong, the next part of your report actually addresses the adequacy of the care plan as devised by the Department of Health. Before you talk about the adequacy of the care plan, you have provided a summary of the results, and I understand that at the fifth line you have mentioned that the results of repeat blood lead tests are reported for 28 individuals.

I understand that at the time when you were compiling the report, the information set out here was based on the first list of test results that you gathered at the time?

A. That's right, the 22 October 2015 line listing.

Q. Because in fact subsequently there have been two updated lists, one dated 19 November 2015, and also the latest one is as of 3 December 2015.

I think now you have had a chance to look at those two updated lists, and just for reference sake, the second list, as of 19 November this year, can be found at E2/770 to 773, and the latest one is at E2/850 to 853.

I understand, Professor, that you have had a chance to now look at these updated lists of blood test results. Do you have anything to add to your original

summary of the test results?

A. Well, I didn't have a chance to carry out the same calculations that I carried out on the 22 October list.

I do note that there is one additional follow-up test on a pregnant woman.

Q. Yes.

A. I reported on the four pregnant women, so there are now five. I had calculated an average change, a decrease in blood lead level, over the one month follow-up interval, of 13.7 per cent for those four women for whom I had the data at the time.

The fifth woman had a decline of 14.3 per cent between her initial and follow-up blood test. So her data are very consistent with those that I saw for the other four.

Looking at non-pregnant individuals, I think what I had seen in the initial group of 24 non-pregnant individuals was a decline between the baseline or first blood test and the follow-up blood test of about 30 per cent. Without having carried out the calculations, by just estimation, it looks like the additional data that are now represented in the line listing are quite consistent with that.

There is one individual that was of some concern to me: a two-year-old female whose first blood test was

6.48 micrograms per decilitre, and her follow-up three months later was 27.32 micrograms per decilitre, and then a blood lead conducted one month after that, it was 21.37, and three weeks after that it was 15.3.

So I'm very happy to see that this child was followed up so assiduously and it's good that her blood lead was coming down. It does suggest, however, that there was something in her environment, certainly not the water because that had been stopped, but she was exposed to something between the first and the follow-up -- first follow-up blood lead -- that led to quite a spike in her blood lead level.

But apart from her, everyone else seems to be a nice decline in their blood lead; as I say, averaging about 30 per cent.

Q. Thank you.

Just for record purposes, the two-year-old girl that you have just mentioned, I think the result of her blood test can be shown at page 851 of E2. I think her result is the one just above the middle of this page. The first follow-up blood test result, it's about 27.32.

I will now move on to discuss your recommendations in relation to the care plan devised by the Department of Health.

I am just wondering if this is an appropriate time

for a break?

CHAIRMAN: Yes. Let's first take a 20-minute coffee break and then resume later. Thank you.

(11.28 am)

(A short adjournment)

(11.49 am)

MR KHAW: Regarding the care plan which is discussed at internal page 19 of your report, you have provided your opinion that "the general components of the care plan proposed for the residents are appropriate, although some are not described in detail".

Then you go on to say:

"... 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 ... outside activities [et cetera] ..."

Then you have also mentioned that:

"The care plan also stipulates a 'health evaluation' ... for individuals with a blood lead level of 5 [to] 44 ... and a 'medical assessment' for individuals with a blood lead level [exceeding] 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."

So there you talk about what is actually lacking or what was then lacking in the care plan that you saw at that time.

Now, I understand that the lawyers representing the Department of Health issued a letter to us yesterday, providing more information regarding the care plan. Perhaps we can take a look at such information and then I'll probably ask for your views on whether you find those plans, those existing plans, adequate or not.

If we can first take a look at E2, tab 71, at page 846. Page 846 talks about the updated tables and also the updated blood test results. I think we have just clarified that point earlier.

Then if we can focus on the next page, that is page 847. Again, we have dealt with the issues arising from the first paragraph, regarding the difference between 28 individuals and 29 individuals, because according to the first list there were 28 individuals, as mentioned in your report, and if we look at the updated list, there are actually 29 individuals.

If we can then move on to look at the second

paragraph, from the second paragraph onwards it says:

"As for the 'exposure assessment' mentioned in page 19 of [your report], a lead exposure assessment ... is conducted by a nurse of the Department of Health ... for every individual with elevated blood lead level. For selected circumstances, [lead exposure assessment] will be supplemented by a home visit. Relevant documents including the questionnaire and reference materials used for conducting lead exposure assessments, protocol and flowchart for home visits of cases with elevated blood lead level are included in the above-mentioned updated 'Master List of documents published'."

If we can now perhaps take a quick look at the issue of the home visit, at page 870. In fact, this is a document entitled, "Investigation protocol for elevated blood lead level", published by the Department of Health. If we take a look at "Home visit", page 870, paragraph 13, it says:

"Home visit provides a good opportunity for health education to the case and household members. Also, any probable source(s) of lead exposure of the case may be identified through home visit so as to guide risk reduction measures. The visiting team should take photo(s) of any highly suspicious item(s) in the

household and discuss with the Government Laboratory for the standard and the methodology of analysis prior to collection of any specimen(s).

14. Home visit should be conducted under the following circumstances:

-- Single venous [blood lead level exceeding or equal to 20 micrograms per decilitre] ...; or

-- Any suspicious source(s) of lead exposure is identified ...

-- Follow-up BLL(s) is persistently elevated ..."

So these are the circumstances which call for home visits, according to the measures now taken by the Department of Health.

Do you find this suggestion for home visit sufficient for this specified category of residents?

A. I do. I am very impressed, actually. I particularly like the second bullet point, "Any suspicious source(s) of lead exposure is identified", as cause for a home visit.

Now, I assume it's identified by means of an initial interview and survey of the informant about what potential sources in the home might be. The reason I like this very much is because it really focuses on prevention. It doesn't require that someone be exposed before the home visit is done to reduce exposure and it

holds the potential of actually preventing the exposure.

So I think this is very well thought-out, yes.

Q. If we go back to the letter from the Department of Health, page 847, there are a few more suggestions here. If we take a look at the third paragraph, starting from the words:

"Regarding the 'developmental assessment' mentioned in page 19 of the expert report, all children with elevated blood lead level will receive preliminary developmental assessment at [Department of Health's] Child Assessment Centres ... or Student Health Service Special Assessment Centres ... by a developmental surveillance team composed of [doctors] and nurses. The developmental assessment covers major developmental areas including gross and fine motor, language and communication, cognition, learning, behaviour and emotion, self-care, vision and hearing. This is conducted through history taking (including birth, developmental and medical history, daily and school functioning and parents concern on development and behaviour), reviewing school reports and school work, physical examination (including soft neurological signs), clinical observation and use of questionnaires and assessment tools."

Then finally it says:

"Children identified with developmental or behavioural problems will have follow-up evaluation at Child Assessment Service according to individual needs. Children with largely normal development would receive continuous monitoring through enhanced developmental surveillance at Maternal and Child Health Centres ... during pre-school years and annual health visits at student health centres during school years. Parents are provided with anticipatory developmental guidance and information on children's development in the form of pamphlets."

If we can then take a look at the table which I think basically describes the details of this particular plan, at page 871, we can see various -- the table under paragraph 17, we can see various care plans devised for different groups of people with different ranges of blood lead results.

Basically, it summarises what has been set out in the letter that we have seen.

Also, I take it that you have had a chance to look at the checklist, at page 873, which contains all the items that need to be examined during the home visit.

Now you have had a chance to see the details of such care plans, are you happy with these suggestions now, or do you have any further comments on the adequacy of the

care plan?

A. Again, I am very impressed with the level of detail and the thought that's been invested in the different contingencies.

What I particularly like about the clinical management or developmental assessment plan is that children with blood lead levels of 5 to 20 will receive a preliminary developmental assessment by the Child Assessment Centre or the Maternal and Child Health Centre or the Student Health Services. I like that because it actually is more -- it's providing more services than the US CDC developmental management guidelines provide, which suggests that only children with levels above 20 be surveilled in this way. But I think this is going to provide an opportunity to identify problems that children may be experiencing that have nothing to do with lead, but a child would benefit from receiving some kind of supportive services.

So I think this is generally even beyond the scope of responding to the water lead issue, this is going to benefit children and parents.

I also had a chance to look at the lead exposure assessment questionnaire that will be administered. That's page 877 and beyond. I find that it covers all of the topics that I listed in my report that would be

important to include in an exposure assessment.

So, again, I am very pleased to see this level of detail. I think it's going to serve the purpose for which it was designed.

Q. Thank you. I think finally, we have come to the last question that you have answered in your report, ie the efficacy and suitability of the acceptance criteria laid down by the Water Supplies Department for heavy metals, and if necessary to make recommendations. That's internal page 19, paragraph (5). I think here, apart from lead, you have also discussed the acceptance criteria in relation to three other heavy metals, ie cadmium, chromium and nickel. I think you have come to the conclusion that the acceptance criteria specified by the Water Supplies Department are based on sound reasoning. Can I just for the purposes of general understanding of these heavy metals -- I am sure that you have a lot of experience in dealing with heavy metal poisoning. If we are talking about heavy metals such as cadmium, chromium and nickel, what are the general harm that they can do to human beings?

A. Well, it differs from metal to metal. Cadmium is primarily a renal toxicant. It damages renal function, it reduces the ability to kidneys to filter out poisons in the body. So it can lead to chronic renal failure

and require transplant, at very high doses, mainly occupationalists, people who encounter cadmium working in their jobs.

Chromium is a little more complicated, because there are different forms of chromium. Trivalent chromium or chromium (III) is actually an essential nutrient, so we need a little bit of that. Chromium (VI), hexavalent chromium, is one of the most toxic chemicals known, and it's well known, it's recognised by the International Agency for Research on Cancer as a human carcinogen.

Nickel, there's actually not that much evidence about nickel. Nickel is, like chromium (III), essential, but too much is thought to be an irritant. So that's the reason for establishing exposure limits for nickel.

Q. In general, how can these heavy metals -- we're talking about cadmium, chromium and nickel -- how can they actually enter water?

A. Sometimes they are just in the groundwater. They are natural constituents of the earth's crust. So, depending upon where an aquifer from which drinking water is taken, they may be naturally present, or they could be present as a contaminant. Cadmium is used industrially a lot. Hexavalent chromium is also an industrial pollutant. And so, depending on the care

taken in its disposal, it could contaminate water supplies.

Q. Thank you very much. That leads us to your conclusion at page 22. If I may just quote what you have said here, "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 micrograms per decilitre. Therefore, the ideal blood lead concentration for a human is 0 ... 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 per cent 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."

Just as a sort of general comment, now you've had a chance to look at all the blood test results of the residents involved and the updated test results -- just as from a broad comparative angle, in view of the other studies and cases that you have seen in other countries, what is your observation about the magnitude of this particular incident, in view of the test results that we have seen?

A. As I say, certainly the fact that once the residents were not drinking the lead-contaminated water their blood lead levels came down -- they were being exposed, there's no question about that, and as I say, it is prudent to interrupt any pathways of exposure that one feasibly can. I do look at incidents such as this in

terms of the context of other scenarios that I have been involved in. When I first got involved in lead work, this was back in the late 1970s, at that time the mean blood lead level of US pre-school children was 15 micrograms per decilitre, and 90 per cent, it was 88 per cent, of children had a blood lead level above 10 micrograms per decilitre.

I was thrilled at that time when my two sons had blood lead levels of 6 micrograms per decilitre. In the context looking back now, even that's too high, but at the time it was pretty good.

Looking at the 3 December line listing, I see there were only two individuals who had a blood lead level above 15. One was a 89-year-old who had a 16.7 -- it perhaps may have been an occupational exposure at some point in the past -- and the other was a 15.8 in a one-year-old.

So from that perspective, these levels don't seem very high. They are certainly higher than we would like to see, and it's good that they are coming down.

Another episode that I was just dealing with at WHO was an episode of lead poisoning in Zamfara, Nigeria, where more than 400 children died from lead poisoning and one child had a blood lead level of 700 micrograms per decilitre.

So there are certainly still cases where children are very, very seriously poisoned, and I don't think that's the case here. I don't think any children are going to be showing signs or symptoms of lead toxicity. On a population basis, I think we can conclude that there probably will be some shifts, minor shifts, in mean level of performance, shifts that you would never probably notice at the level of the individual child.

So my overall position is that this is a very unfortunate situation; I don't think the health consequences are going to be terribly serious, and I think that the authorities are taking totally appropriate steps to support the affected people and taking steps to make sure that exposure from water is not a major contributor to the overall exposure.

Q. Thank you. Finally, may I just confirm a few things with you, just as a matter of formality. May I just ask you to confirm that all the facts and matters that you have set out in your report and you have discussed today, which are within your personal knowledge, are true and correct? Can you confirm that?

A. Yes, I do.

Q. You also confirm that the opinions expressed in your report and also expressed today at the hearing are honestly held?

A. Yes, they are.

Q. Can you please also confirm that the contents of your declaration as an expert, as attached to your report, and also your CV attached to your report, are true and correct?

A. Correct, yes.

Q. You are willing to adopt what you have set out in your report and what you have discussed orally at this hearing today as your expert evidence?

A. Yes.

MR KHAW: Thank you very much.

CHAIRMAN: Thank you. Anyone wants to ask any questions? Yes, Mr Pennicott.

MR PENNICOTT: I think there are others as well, but I am happy to go first if nobody else wants to go first.

Cross-examination by MR PENNICOTT

Q. Prof Bellinger, I represent one of the contractors who was responsible for building two of the eleven affected estates. One of those affected estates is an estate called Kai Ching, and as it happens it is Kai Ching in respect of which we have most information in relation to, amongst other things, blood lead levels, and I expect you have picked that up during the course of your reading of the materials for the purposes of your report.

You will know, if you have read that material, that Kai Ching Estate comprises of six separate residential blocks. Is that something you are aware of?

A. I wasn't aware of that level of detail.

Q. It is six residential blocks. There are 5,204 individual units within those six blocks, and what I want to do, I hope fairly briefly, is ask you some questions about the various matters you have been discussing with Mr Khaw, but specifically in relation to the Kai Ching Estate, if I may.

Before I do that, I am going to hand you some documents in a moment that are going help me and hopefully help you as well.

Can I ask you this: so far as the blood lead level tests are concerned, have you established how those tests were actually carried out on the individuals who went for those tests?

A. Only indirectly. I notice that some of the values are reported as less than 3.3 micrograms per decilitre, which leads me to think that a portable LeadCare analyser instrument was used.

Q. Right, and do you know how the blood was taken? Have you made an assumption as to whether it was a venous approach or just a prick on the end of a thumb or a finger?

A. You can use either type of sample with the LeadCare analyser, and I don't know whether venous or capillary blood was used. If the sampling protocol is adequate, it shouldn't matter very much.

Q. That leads me to ask you this: if you are going blood lead level tests in a laboratory, is there any margin for error that you would impose, or are they sort of absolute figures?

A. Oh, absolutely, it does -- the different methods that are used do have different levels of precision.

Q. Can you explain what level of precision, for example, there is on a venous approach?

A. Well, the precision would be more specific to the method, whether it's isotope dilution mass spectrometry or atomic absorption spectrometry or anodic stripping voltammetry. It's not so much whether it's capillary or venous blood that's collected. The same level of precision would apply depending upon which analytical method is used, not the sample collection method.

Q. Can you give an approximation as to the margin of error?

A. Well, for the ICP-MS method, it can be plus or minus only a half of a microgram per decilitre or even a tenth. Yet for the atomic absorption, it may be plus or minus 1 or 2 micrograms per decilitre. Again, it depends on whose hands are applying the method.

Q. Right, the skill of the operator?

A. Right. The US CDC was resisting lowering the action level from 10 to 5 because they were claiming that many labs didn't have the ability to get closer than several micrograms per decilitre in their measurement.

Q. Prof Bellinger, one point that you discussed briefly with Mr Khaw earlier arises on page 58, internal page 14, of your report. It's the last sentence of the paragraph, headed "USA Environmental Protection Agency", and it's this point about "If more than 10 per cent of the tap water samples collected exceed the action level of 0.015 [micrograms per litre] (15 [micrograms per litre]), a water system is required to take steps such as corrosion control treatments", and so forth.

If the percentage is less than 10 per cent -- let's say, for example, 7.5 per cent, just by way of example -- is there anything to be done in those circumstances, or does this only kick in when you get to 10 per cent?

A. Yes, my understanding is it only kicks in when you get to 10 per cent. But there are some -- I'm no water expert, I have to say that right upfront. I believe that if a water system is supplying more than some number of individuals or units, they are required to take appropriate steps to reduce corrosion. And this

10 per cent applies to I think smaller systems, that are providing service to a smaller number of units.

Q. Have you, for the purposes of your report, studied in any detail the water tests that have been done in relation to the various affected estates?

A. No. I have focused on the health side of things.

Q. So you haven't looked at them at all, the results, the test results?

A. Not in detail, no.

Q. Okay. We will come to it a little bit in a moment.

I wonder if I could now hand out some documents that are going to help me to ask a few questions. I hope it won't take too long. (Handed).

If everybody has those, Prof Bellinger, for the first document, it's titled "Table A". You will recall, in your discussion with Mr Khaw earlier, you had identified I think 24 results as at 22 October 2015.

A. Right.

Q. Where there were in fact 28 results, you have taken off four pregnant women and then got an average of 30.8 per cent, I think.

A. (Nodded head).

Q. What we see on table A is in fact 25 results, so similar to you, what we have done is we have taken out the four pregnant women but we have added back the one result

that you didn't have, so we've now got 25 rather than 24. As you can see, we have calculated the average and it's changed very marginally to 30.27.

Can I ask you this first: this table includes two women, lactating women. Why do you think you would include those rather than exclude them for this purpose? You have taken out the pregnant women, I understand, and we see the reasons for that, but why do you include lactating women?

A. Well, to be honest, I didn't notice that. It probably would have been better to calculate the average change including and excluding them. Again, because during lactation, bone is still being mobilised to provide calcium, it does complicate the kinetics a bit.

Q. I just wonder whether there was a particular reason for it. I am not pressing you, I just wanted to know whether there was a particular reason for it.

A. Which two were lactating?

Q. If I am using the references on the left-hand column, about halfway down there's a reference "KC00190"; do you see that?

A. Yes.

Q. The 32-year-and-10-month-old lady -- and the cross-reference for anybody who is interested is E1/542, where we will pick up that there's a Y in the lactating

column on that page -- the other one is KC00257, a little bit further down the page. And similarly, it's the same page where we pick up the reference.

I am not pressing. I just want to know that there was a reason that there is one calculation with and one calculation without?

A. Yes. I see they both declined about 18 per cent, and I would have predicted that they would decline a little bit less than non-lactating individuals. So that would probably boost up the 30 per cent drop a bit.

Q. It would clearly push it up?

A. It would.

Q. The other thing on this page, table A, Prof Bellinger, that I just want to ask you a couple of questions about -- we have highlighted the two extremes in the right-hand column which I think you draw attention to in your report. That is the most dramatic percentage change is in the fourth reference down, of minus 55.54 per cent.

A. Mm-hmm.

Q. And the smallest change appears to be in the fifth reference up, of minus 2.74 per cent.

A. (Nodded head).

Q. Presumably you would agree with me, that's a rather large range for this type of exercise?

A. It's hard to say. I mean, it is a large range, but whether it's larger than one would see elsewhere, I can't say.

Q. What I am quite interested in, Prof Bellinger, is really whether taking an average of all these percentages actually tells us anything? Is it very meaningful, given the wide range that we have here, just to say 30 per cent?

A. Well, it is one way of summarising the evidence. As I mentioned in my report, there is a statistical phenomenon known as regression to the mean, which means that if you take repeat measurements of some biological function, or any function, usually those who are most extreme on the first test will regress towards the mean. If they are high on the first test, they will be closer to the mean on the second test because they will decrease. If they are very low on the first test, on the second test they will probably increase towards the mean.

I did show that the mean -- the percentage decline between the first and the repeat blood test was larger among people whose first blood test result was higher. Now, that does suggest to me that there was some regression to the mean going on. But we can't know how much regression there was unless we also tested people

whose -- did repeat testing on people whose first blood test did not exceed the reference value. That would give us a sense.

Q. Yes.

A. But there are biological reasons why we would expect there to be variability among people in the difference between their first and second blood test. Their age would be one thing.

Q. Yes.

A. Because the older people would have more lead in their bones.

It also depends upon what the other exposure, lead exposure sources are for an individual. An individual who's exposed to -- whose exposure is primarily from non-water sources is not going to show much of a decline once the water pathway is eliminated.

So, you know, for all of those reasons, it's impossible in my mind to draw any conclusion from the variability in the per cent change, without having lots more information.

Q. Yes. That's a very fair answer. But just to point out that -- to really underline the point that you have just made -- the two extremes, the 55 per cent and the 2.74 per cent -- the first one appears to be a female child of 10 months old, that's the 55.54 per cent, and

the other one -- and I should emphasise, for a reason we will come to in a moment, resides in Yuet Ching House -- we will come to the reason or the relevance for that in a moment. At the other end of the scale, as it were, the 2.74 per cent, is another child, a female of one year and four months old. So you've got two children of not great difference in age showing a huge difference in the readings between the first and second blood test. Does that tell you anything?

A. It tells me that water lead was probably a much more significant portion of the first child's -- the ten month old child's overall lead exposure than it was the other child, and that's why you would see a larger drop in the blood lead after the water pathway has been remediated.

Q. Just looking at these percentages and the drops, if you like, in levels, again the passage that Mr Khaw read out to you -- I don't want to go back to it again; I certainly don't want to read it out again -- it's in the passage where you refer to the half-life and you had lengthy exposure and acute exposure, and you explain what the differences are and the implications of that.

Can you apply those principles to these results?

A. Not really, and for the reasons that I suggested: because the half-life has a very narrow applicability,

and how it will play out for an individual will depend upon the other sources of exposure and how much past exposure an individual has had. The half-life of 30 days was actually identified by studies where adults had radioactively labelled lead injected into their blood. A known amount was injected and then measurements were made of the amount of radioactively labelled lead that came out in the urine and the faeces, through the bile. That's how it was arrived at, 30 days, and it's tied to the life cycle of the erythrocyte, the red blood cell, to which 95 per cent of the lead is bound.

Q. Yes.

A. So while that is true, 30 days is the half-life of a labelled atom of lead, for the same reason that the study I mentioned in my report about children who had a blood lead between 25 and 29, it took years for their blood lead to fall by half because of these other toxicokinetic factors that go well beyond the half-life of lead.

Q. With regard to the 10-month-old child referable to 55 per cent reduction in percentage terms -- so we know the child lived for ten months when she had the first blood test, no doubt a few more months older when she had the second blood test -- from your experience, that exposure to lead at the levels we have seen, is that

likely to have any long-term effect, do you think, on the child?

A. Well, you know, again, it's hard to say without knowing more information. If this 10-month-old child was nursed for eight months and then had two months of exposure to formula made up with water that contained lead, then that child probably didn't have much lead in the body, and so that's probably -- that would explain a big drop two months later, once the water lead was remediated. So, you know, under that scenario, I wouldn't anticipate very serious impact on the child's life.

Q. Again, I make the point, Prof Bellinger, just simply because it seems to us that this is very much a sort of micro-analysis that one needs to do on a very much individual-by-individual basis, and therefore taking these broad percentages doesn't really assist the analysis a great deal. Do you agree?

A. Well, I agree that things are always complicated. This is one way of organising the data. Certainly a mean can be misleading. I didn't calculate a median, you know, where it's a 50 per cent value. I suspect that it would not be that much different than 30 per cent.

But yes, these are -- if you are talking about individual management of a child, then an average is meaningless.

Q. Yes.

A. But if you are trying to capture an overall impact, I think it's reasonable, with appropriate caveats.

Q. Okay. Can I then ask you please to go to what I hope is called table B, the next chart. Whereas the previous table just listed out all of those people who had been tested and re-tested, what we have done here, Prof Bellinger, is we have just focused on what I would call abnormal, still abnormal, tests. So you will see, in the box on the left-hand side, we have simply again just replicated the abnormal results first test and those that were still abnormal after the second test, so you can see what we have done here.

Just to explain the blank at number 19 on the top graph and indeed the bottom graph: that's the two-year-old child that you referred to, when you were discussing the matter with Mr Khaw earlier, the one who started at 6.83, went up to 21.32 and then dropped back a little bit on the third test, and then we have seen more recently dropped back a little bit further to 15 on the fourth test. We thought it appropriate just to take that particular person, the child, out of the equation, as it were.

A. I agree.

Q. You agree?

A. Yes.

Q. Okay. We see, if we're just comparing abnormal to abnormal, as it were, the average we get is just under 20 per cent. Do you see that?

A. Yes.

Q. What we see in the bottom graph is in fact, at items 12 and 16, some increases in blood lead levels. Had you spotted those?

A. Yes.

Q. Any comment about those particular ones, the increases?

A. The only conclusion I would draw was that water was not a particularly important pathway for those individuals and that there were other sources in the environment that would have remained the same after the water pathway was interrupted. So they continued to be exposed to those sources and had slight increases in blood lead because of it.

Q. Okay. Then if you go to the next table, table C -- again, this is just referable to Kai Ching. And what we now know, leaving aside the results that I think were only received, certainly by us, yesterday or the last couple of days, that on Kai Ching there have been 59 individuals who have been tested and re-tested. And what we have done here is, if you look at the box in yellow on the left-hand side, those above the line, that

is between references 388 and 324, are the ones we saw on the previous table, the normal to abnormal, save that this time we have included, as it were, the rogue item.

A. 321 per cent.

Q. Yes. Those below the line are, as it were, abnormal but now normal. So this depicts the whole range so far as Kai Ching is concerned, and we see that the average is 22 per cent.

So, again, not miles off the 30 per cent exercise that we saw earlier.

But then over the page to a table I hope called D, this is exactly the same as the previous table, but this time we have taken out the two-year-old child that we were discussing earlier.

A. Yes.

Q. One sees what a dramatic effect that can have on the average percentage.

A. Yes.

Q. Lastly, if I could ask you to go to table E. I hope you can read it, because it's a bit small at the top, but again this is a breakdown of a number of statistics, a number of details, that we have in relation to Kai Ching Estate. You see listed across the top the names of the six blocks and then some totals at the far end. Okay?

A. (Nodded head).

Q. So taking the first one as an example, Hong Ching House, there were 165 blood lead level tests done, 149 of which were normal and 16 which were abnormal. Do you see that?

A. Yes.

Q. So that's now 9.7 per cent, for what that's worth.

If we then go down to the next items in the table, we can see how the normal and the abnormal break down. So there were 12 children -- of the 16 abnormal, 12 were children under 6, one between 6 and 8, none between 8 and 18, and then three people between 18 and 65. Do you see that?

A. Yes.

Q. Can I then just ask you to look at Yuet Ching House, which is the one on the far right-hand side before you get to the "Totals" column, and there, there were 147 -- sorry, there were 150 total tests; do you see that?

A. Yes.

Q. Of which three were abnormal; do you see that?

A. Yes.

Q. Underneath all the details regarding the blood lead level tests, we have put some details of the water sample tests. Do you see that?

A. I'm sorry, where are those?

Q. Under this first table, there's just a little table with two lines.

A. Yes, I'm sorry.

Q. It says on the left-hand side, "Water samples taken"; do you see that?

A. Yes.

Q. We can see on the far right-hand side of that little table that there were 93 water samples taken from the 5,204 units that exist on Kai Ching, of which seven showed excessive lead in water, out of 93.

However, if you look at Yuet Ching House, there were no samples that exceeded the criteria, the 10 micrograms per litre criteria that was being used. Do you see that?

A. Yes.

Q. So, on one view, Yuet Ching, so far as water samples was concerned, was clear, or in the clear, and that was the same for another block, Yan Ching House as well?

A. Yes.

Q. Can I ask you this, Prof Bellinger, by way of introduction: if one assumes, for the sake of argument, that the level or the degree of testing that was done on Yuet Ching House was valid and reliable, doesn't that raise a serious question as to whether or not any high lead levels in blood from the tests is related to the

water?

A. No.

Q. Why do you say that?

A. Because there's nothing magic about 10 micrograms per litre, except that it's a standard. If it were 9 or 8 or 7 or 6, there's still lead in the water and it still could be getting into people and be responsible for some fraction of the lead that's measured in the blood. Just because the water lead level doesn't exceed 10 doesn't mean that the water lead isn't contributing at all to an individual's exposure. It just means it doesn't exceed the regulatory standard.

Q. But the problem -- one issue about that, and this is a point that you fairly mention in your conclusion, is that it's a contributing factor or may be a contributing factor, but you simply can't apportion the contribution, can you?

A. Well, you could if you actually did the study where you went in and measured all of the other potential contributing factors; it would be possible to come up with a per cent of exposure that you attribute to water. That's been done in many, many studies. That wasn't done in this case.

But the fact that we see the declines in blood lead following the interruption of the water pathway, what

other explanation is there, other than that water was no longer contributing to blood lead and the blood lead is coming down? Unless you assume that because of the publicity, people were going out and learning about all the pathways of exposure to lead and taking steps to reduce their overall exposure.

Q. Understood.

Just finally then, can I ask this. I know, and you have already said once, you are not a water expert, you are a medical expert, and of course I accept that. Would you expect there to be a correlation, however, between the water sampling on the one hand and the blood lead levels on the other?

A. In general, yes, other things being equal. But if everybody had the same non-water contributions to their blood lead, but water lead concentration varied among people, yes, you would expect a very high correlation and water lead would explain all of the variability in people's blood lead.

But of course all other things are not equal, and so water will contribute different percentages across individuals of their total exposure to lead, which is why I would expect to see variability in the per cent decline once water lead is eliminated.

MR PENNICOTT: All right. Thank you very much, Professor.

CHAIRMAN: Mr McCoy?

Cross-examination by DR McCOY

DR McCOY: Prof Bellinger, is this your first trip to Hong Kong?

A. No. I am fortunate enough to be able to come once a year, to help with grant reviews.

Q. Have you been here previously on formal scientific reasons?

A. The once-a-year trip is to review grant proposals submitted by Hong Kong investigators for funding.

Q. In biology?

A. Biology and medicine, yes.

Q. I noticed in your report that you refer that there are, as far back as April 2012, some 28,900 peer review publications in relation to excessive lead.

A. That's right.

Q. It's obviously gone up since then, and not just because of this incident. But what proportion of those publications do you think pertain to Hong Kong?

A. Probably very few.

Q. Have you ever used Google Scholar?

A. Yes.

Q. If you put in "lead" and "Hong Kong", can you estimate how many articles might come up, academic scientific papers written about lead poisoning in Hong Kong?

A. I could hazard a guess but it's just a guess.

Q. If I tell you that there have actually been published theses in Hong Kong written on lead poisoning, that would not surprise you?

A. No.

Q. I am going to ask you in due course to have a look at one of those published theses, and it examines the many causes and effects of lead poisoning in Hong Kong.

In fact, I could ask even now that it be put up on the screen, with permission. It's G3, item 88.

This is a thesis in the faculty of science at Hong Kong University. It's slightly dated -- it's April 1987, and I noted that it doesn't actually refer to any of your works, but other Hong Kong publications which I will take you to do -- and it's written in the department of chemistry at Hong Kong University.

You can see the abstract. On (i) -- if you roll over one or two pages, please, past the acknowledgments -- keep going, please.

A. Yes.

Q. The next one. Exactly.

The abstract, of course, is the summary of the academic conclusions. I won't read them all out, but the author shows that lead is detrimental, especially to children, and he identifies some reasons why that is

particularly so, not different from what you have said to the Commission of Inquiry.

He points out that petroleum -- so this was 1987 -- gasoline perhaps in your language, was a major contributor, and also that there was lead in consumer products available in Hong Kong.

Consumer products include, plainly, food and items like paint, for example.

A. (Nodded head).

Q. The methodology of the thesis was to actually examine the lead content inside 6,065 Hong Kong schoolchildren. You will see that in the second paragraph, commencing, "In this survey, the massive screening programmes ..."

It was a study of, as I said, of over 6,000 children in Hong Kong, aged from 6 to 17.

If we come down, please, to the next paragraph:

"In the scheme III screening, 700 primary schoolchildren aged from 6 to 16 years old were studied. The mean blood lead level ... of them was found to be 14 [plus or minus 5.1 micrograms]. The spread of [blood lead level] was from 3.7 to 45 [micrograms]. The blood lead levels of Hong Kong children are similar to or lower than those in large cities. They [are] found to conform to the EEC guidelines on the criteria of undue lead absorption."

Now, the EEC guidelines that the author is referral to, are you able to comment as to whether that conclusion is sound; that, for example, levels like 45 micrograms conform to the EEC guidelines in 1987?

A. Well, I'm not sure actually what the author means by saying they conform, whether that is intended to mean that all levels were below whatever criteria the EEC was using at that time. I personally don't recall what the EEC guidelines were in 1987.

Q. Sorry, I wasn't trying to make it a test of absolute memory. But we will read on and perhaps it will become clear.

The conclusions are:

"[There is an] association of some personal, family, behavioural and environmental factors with blood lead levels of children [when] analysed. It was found that sex, mouthing habit, parents' occupation, floor level of residence as well as the time spent on street(s) do have positive association with [lead] levels in children."

Would you agree with that as a generality, Prof Bellinger?

A. Certainly there are studies that indicate that all of those, under certain circumstances, can contribute to children's lead exposure, yes.

Q. Yes. Then we read on:

"With the use of controlled Pearson Correlation Analysis, it was discovered that the more direct personal factors, namely the occupation of parents and mouthing habits, are more important than the environmental factors such as floor level of residence and the time spent on the street."

Then there was a special reference in the thesis to the risk of fishermen's children, a study at Aberdeen, one of the ports on Hong Kong Island, where the children were often literally handling lead weights and there was an ingestion through the body.

Would that surprise you that that generated an obvious risk of potential excess lead?

A. No. That's been reported numerous times.

Q. I am obviously not going to go through all of this thesis, but there are some matters that I would like to raise with you for your comment which may be of assistance to the Commission of Inquiry.

If we come firstly to page 105, please. This is really near the end of the thesis. You find a conclusion -- it's chapter 4, page 105 -- about 30 per cent of the way down the page, you see the words, "Australia et cetera", full stop. Then this:

"Also the increase in use of copper pipings in domestic water supply system should be carefully

monitored because the leaching of lead from the solders has been reported to be the source of undue lead absorption."

That's a well documented empirical fact; I assume you would agree with that?

A. Yes, if the pipes have been connected by solder that contains lead.

Q. Yes, exactly.

The author in particular identifies a large number of environmental factors that can contribute to lead.

Now, in America, what is the current greatest contributor of lead in the environment?

A. It's the lead in dust, house dust.

Q. The lead in house dust?

A. That's usually the strongest predictor of a child's blood lead level.

Q. And it seems naive but how does the house dust get to contain lead?

A. Well, in numerous ways, and that's why it's a significant predictor. It could be lead-based paint that is chipping and crumbling into fine particles that contribute to the dust. It could be people tracking in soil from outside that may contain lead, because of atmospheric deposition or the deterioration of exterior lead-based paint that gets into the soil. It could be

lead blowing in through the windows; back in the days when lead was added to gasoline, that was a major, major problem. It also could be in the air because of the child's house being proximate to a point source such as an industry that uses lead. It could be because people are carrying out activities in the home that involve lead, and little particles get into the dust.

So it integrates lead from many different sources, and we think that's why it's usually the best predictor.

DR McCOY: Yes.

Is that a convenient time?

CHAIRMAN: Let's take the lunch break first. We will resume at 2.30. Thank you.

(1.00 pm)

(The luncheon adjournment)

(2.35 pm)

CHAIRMAN: Yes, Mr McCoy.

DR McCOY: Good afternoon, Prof Bellinger.

A. Good afternoon.

Q. Just before we broke, I had asked you to confirm what is the number one source in America of lead in the environment, in the free environment, and you indicated that it was basically household dust, or dust?

A. Correct.

Q. That is simply a product of other processes, as opposed

to it being naturally occurring, I assume?

A. Yes. It's not a source in itself. It's a pathway.

Q. That's right.

I see from your very lengthy bibliography that you have written widely, and you have studied, for example, the problem of excess lead with children in India --

A. Yes.

Q. -- Bangladesh, Mexico, Palestine, to name just a few of the places.

Have you ever yourself studied the incidence of excessive lead in any part of Asia?

A. Well, Bangladesh and India.

Q. They may be for FIFA.

A. What?

Q. They may be in terms of football classification, but say east of India, have you ever studied any of those Asian regions, including Hong Kong, China, Japan, Malaysia?

A. No, I haven't.

Q. You haven't? The reason I ask that question is whether there are any well-understood regional differences throughout the world in terms of ordinary background lead standards in daily life.

A. By "lead standards" do you mean regulatory exposure standards?

Q. Yes, first of all regulatory, and then second, as

a matter of fact, the incidence of lead.

A. Well, I'm not familiar with the lead standards used in each country. I know many do refer to the WHO standard of 5.

In terms of background incidence of lead exposure, yes, there are very large differences, mostly between developed and developing countries. The WHO, in an analysis they published in 2003, estimated that about 10 per cent of the world's children have a blood lead level greater than 20 micrograms per decilitre, and 99 per cent of them reside in developing countries. So the success we have seen in reducing population lead exposures in developed countries over the last 30 years have not seen the same benefit in developing countries. So they do have more -- continue to have more of a problem with lead.

Q. Lead does not degrade as such, being an element, does it?

A. That's correct.

Q. And you can't transmute it into anything else, unless you believe in alchemy, I suppose?

A. That's correct.

Q. So once the lead has entered the total environment, it remains, unless dispersed by air or water?

A. That's correct.

Q. So it would be important in understanding the background levels of lead, in any urban environment, to understand the history of that land before its current usage?

A. That can be important, yes.

Q. You see, I will show you in due course that there are well-documented peer-reviewed academic studies in Hong Kong that because certain parts of Hong Kong had been heavily industrialised in the past, the lead effectively has sunk into the land and remains. Does that seem a logical proposition, if the assumption is correct about the prior industrialisation?

A. Yes. There have been similar studies in Massachusetts, where I live, actually, where people have looked at historical land use and identified higher blood lead levels among children living in regions which formerly had industries that used lead.

So, yes, that's a well-described phenomenon.

Q. So if you are living in an area that has previously been relatively heavily industrialised, is there a likelihood that the dust in that area will be significantly higher in lead content than otherwise?

A. I would say -- I would presume yes. It would remain to be confirmed, but it's a very reasonable hypothesis. It would add to the background risk and perhaps raise the background blood lead level against which other sources

would add.

Q. And the human ingests lead by, what, touch, inhalation, consumption; every method possible?

A. Except for skin absorption, yes. Inorganic lead is not absorbed on the skin. But ingestion and inhalation are the main routes of exposure.

Q. Can I ask you to come now to page 1 of the thesis that I have partly shown you this morning. Page 1. I see you have a hard copy now.

75 per cent of the way down the page, it says:

"There are many routes by which lead can enter the air, water and soil as a result of the wide use of lead and its compounds."

I won't read all of this out, but in particular the combustion of coal is identified as a source of lead entering the environment.

Are you familiar with that as a contributing factor to environmental lead?

A. No, actually. That's a new one. Mercury I know is a problem with coal combustion, but it's new to me that lead may also be released.

Q. Again if we can just move down to the last two lines on that page:

"The lead level in the food chain (plant, fish, vegetable, poultry et cetera) is also increased by

several orders of magnitude."

Then this:

"The daily intake of lead of the average person in [the United Kingdom]" -- and (2) is the scientific reference for it -- "and USA (3)" -- ditto -- "is estimated to be 18 [micrograms per day and 175 micrograms per day] respectively."

Are you familiar with those figures?

A. Yes. These are quite old numbers, and the estimates for today are much, much less, in the order of 20 or 25 micrograms per day, because of all the reductions in the last 20 to 30 years and dispersion of lead into the environment.

Q. Then if you come down, please, on page 4 to the first full paragraph, and the last two sentences:

"Lead has a very slow turnover rate in the body."

What dictates that turnover rate?

A. Well, I assume that means the excretion of lead from the body. I had earlier talked about the different pools in the body in which lead resides. Each pool has a different turnover rate, so you can't assign one single number. So lead that's in the type of bone that's hard and compact, like the tibia, has a half-life of maybe decades, three decades, whereas the lead in soft, spongy bone, the trabecular bone that is in

contact with circulation has a half-life of years to a decade, and in soft tissues will be somewhat less, and as I mentioned, in blood it's 30 days.

Certainly in bone there's a very slow turnover rate. It's not so slow in the blood.

Q. Halfway down that page, there's a historical reference to lead poisoning from Roman times. It's probably plumbum, being the chemical name for lead, and plumber going together.

A. Exactly.

Q. Plumbers carry lead.

A. Right.

Q. If you turn the page, please, to page 6, you see the first reported environmental exposure from lead was in Queensland in Australia in 1892. That was paint.

Then an issue the chairman raised with you this morning, the issue about young children. The author here states:

"Young children are widely held to be more susceptible to lead poisoning in the environment than adults for the following reasons.

1. Young children have a higher metabolic rate than adults, so that their intake of food and drink, and hence lead, is greater relative to their body weight than that of an adult."

Do you agree with that?

A. Yes.

Q. "2. Young children may absorb and retain more lead from the gut than adults."

A. Yes.

Q. Thank you.

"3. Young children may have a greater biologically active lead burden than adults."

Do you agree with that?

A. I am not exactly sure what the author means. If he means that a greater or a lesser fraction of lead is in the bone in children than in adults, so it's more available to interact with cellular targets, then yes, I would say I agree with that interpretation of the phrase.

Q. "4. The opportunity for oral intake of significant amounts of lead from non-food sources is higher in young children than in adults due to mouthing habits."

I think that's pica, the obscure word P-I-C-A, the non-nutritive intake by children, eating things that they shouldn't be eating.

A. Right, although even children in whom the behaviour is not as extreme to warrant the application of the term pica still -- typically, developing children explore their environment with their hands and their mouth,

so ...

Q. And the fifth one:

"Deficiencies in nutritional status may also be of greater consequence in the enhancement of absorption of lead in young children."

Do you agree with that?

A. Yes. Children are more likely than adults to be deficient in iron and calcium, and those promote absorption of lead in the gut.

Q. So the conclusion is children are more susceptible to lead poisoning. But can I now ask you: are they more likely, because of the nature and size of their bones, to excrete or eliminate that ingested lead than a comparator adult?

A. I am not familiar with evidence that that's the case, that the excretion rate is not different from adults.

Q. There's no difference at all?

A. Not that I am aware of.

Q. Then perhaps if I could ask you to come across to page 8, and I am certainly not going to go through all of this, but there are just a few highlights. If you come to page 8, please, 10 per cent down the page:

"However the public awareness of this problem" -- that's the problem of excessive lead -- "has increased since early 1980s."

And several factors are set out. I won't read all those factors out, but we have at number 4 a reference to the Hong Kong Consumer Council, a prestigious body that by its very name looks after consumer interests. It was reported by the Hong Kong Consumer Council that the lead in petroleum in 1980 was at 0.84 grams per litre, six times more than in the United States. This was staged down to 0.15 grams in January 1987.

Now, the lead from the petroleum, it enters the environment. It stays, I think we agree, unless it is literally blown away out of Hong Kong.

A. Correct.

Q. So, in a small, dense, compact society like Hong Kong, when lead in petroleum was available, the probability is it remains adjacent to where the vehicle burnt the lead, the petrol?

A. Yes, it is emitted in the area proximal to the roadways. So the farther you go from the roadway, the concentration of lead declines.

Q. If you come over to page 10, please, at number 8:

"High level of lead at kerbside soil was reported. Mean concentration of lead was 2,974 [plus or minus] 408 [micrograms] and the range was from 271 to 19,073 [micrograms]."

Extreme readings.

A. It's quite high.

Q. Then at 9:

"Approximately 7.5 per cent of 1,135 adult blood lead levels were found to exceed generally accepted reference value of 35 [micrograms] in a survey carried out in Hong Kong."

And the reference is to 60 in the bibliography.

"The findings suggested that the group studied did not conform to the EEC guidelines."

Now, with the revised limits, they would be well over, would they not?

A. Correct.

Q. Then 10 -- and I would like you just to go to page 113 briefly. At item 61, because D Barltrop and I Thornton in Hong Kong in 1982 wrote a book, "Lead Pollution in Hong Kong -- Report to the Special Committee on Air Pollution", so that's the source material that the author is referring to.

If you come back then to page 10, at paragraph or item 10:

"A report on 'Lead pollution in Hong Kong' prepared by two experts from UK was published. It stated that a comprehensive evaluation of lead in the environment and its impact on the population were required."

Was there comparable efforts, say, in the

United States at that time?

A. Comparable efforts to ...?

Q. To publish a comprehensive evaluation of lead in the environment.

A. Yes.

Q. And in the United Kingdom?

A. I am not as familiar with ...

Q. Have you seen what these two experts in 1982 recommended to the government should happen? Have you seen the outcome?

A. No, I haven't.

Q. Nor have I.

Now, this thesis involves, as I said, the screening of over 6,000 Hong Kong children. If you turn over, at page 12 -- I won't spend time on this for the non-chemists -- but at page 12 you find the interference with the biosynthesis of the haem, the haemoglobin, and that's set out in diagrammatic form.

The author, at page 11, 80 per cent down, is identifying zinc protoporphyrin. You would be familiar with that as a metabolite.

A. It's not really a metabolite but I am familiar with it.

Q. So the author of this thesis, he examined ZPP, as I will more conveniently call it. You see the chemistry at 13. We can go past that.

Then at page 16 you see what was achieved. He went to government or subsidised Hong Kong schools. The children were all of Chinese race and aged from 6 to 17, and the distribution of schools covered the three major areas of Hong Kong: the Island, which we are now on; Kowloon Peninsula; and New Territories together with the Outlying Islands. Are you generally familiar with the geography?

A. Generally, yes.

Q. We can avoid now much of this, but we come to page 20. The blood sample from each of these children was taken from the finger, from a capillary. Now, this morning, Mr Pennicott, senior counsel, did ask you a question directed at whether there was a margin of error between capillary blood and venous blood for the purposes of determination of excessive lead, and you, would I be right, were hesitant in accepting that there was a difference, or is that unfair?

A. No, no. There should not be a difference if adequate cleaning protocol is done. The problem with capillary samples is that there's sometimes lead dust on the finger, and when you lance and get a drop of blood, it takes up some of that lead on the finger, if you haven't cleaned it properly, and so it can give you a false high reading.

But if you follow accepted practices for the cleaning of the finger before you stick, the capillary blood lead level should be very close to the venous blood lead level.

Q. If I suggest to you that a recent peer-reviewed study actually shows that even with proper cleaning, there is a 10 per cent false positive risk with capillary taking of blood for the testing of lead, would that surprise you?

A. Well, what exactly is -- is it 10 per cent --

Q. I can produce the paper.

A. Is it a 10 per cent difference in the blood lead level or a 10 per cent difference in --

Q. In false positives.

A. That doesn't mean anything to me.

Q. All right.

A. I would have to see the paper.

Q. I am happy to do that and I will do it soon.

A. But I have seen other papers where the difference between the venous and the capillary levels were exactly the same. So I don't know how this study would differ from those.

Q. Now, could we then please move way through much of this and come to page 54. The author notes, 30 per cent down the page, that ZPP, the metabolite, as I'm calling it,

"is firmly bound with haem and persists there for the life span of the red blood cell (about 120 days)."

You agree with that?

A. Yes.

Q. So that's relevant to a 30-day cycle in your 25 per cent equation, the cycle of re-testing the blood every 30 days?

A. Yes.

Q. If we come much further across towards the end -- just give me a moment, please -- the author of this thesis concluded that there is a statistical correlation between the lead levels in children and the occupation of their parents. Is that something you have seen in any other study?

A. Yes. It's quite well described that parents involved in occupations involving lead can bring it home and those children will have higher blood lead levels than children of parents who don't work with lead.

Q. May I ask you to please leave that document, and I would like to look at a study that refers to one of your own works, with appropriate approval, I should add.

Could you come please to item G3/90, please. That's right, "Home sweet home? A case study of household dust contamination in Hong Kong", from the University of Cincinnatti and the Chinese University of Hong Kong in

March 2000.

If we just look at the abstract:

"It is well recognised that many heavy metals have chronic effects ... particularly to young children ..."

I will leave words out in the interests of time.

"... house dust ... This research aims at quantifying the concentrations of heavy metals within the home environment in Hong Kong and their relationships with environmental factors. The results of this study seem to suggest that traffic and the age of the building and neighbourhood are more important factors than the types of industry and socioeconomic status in affecting household dust contamination. The metal burdens in Kwun Tong ..."

Now, that's a particular district in Kowloon.

"... an old area with heavy traffic, are significantly higher than other districts."

If we just move past that and come to a second document, and this is the next one, G3/92, called, "The study of metal contamination in urban soils of Hong Kong". This is written between Imperial College London, the Department of Civil and Structural Engineering at Hong Kong Polytechnic University, and the Department of Land Surveying and Geo-informatics at the Hong Kong Polytechnic University, in September 2003.

If we again look at the abstract, it refers to the highly urbanised Kowloon area:

"A significant spatial relationship was found for [nickel, copper, lead and zinc] in the soils using a GIS-based analysis, suggesting that these metal contaminants in the soils of Kowloon ... had common sources. Several hot-spot areas of metal contamination were identified from the composite metal geochemical map, mainly in the old industrial and residential areas ... The [lead] isotope composition of the contaminated soils showed clear anthropogenic origins."

I will try to deconstruct some of that with you very briefly, and I won't spend long on this, I promise you.

If you come to the second page of this article, at page 114, five or six lines down -- first of all, you can see your own name there, Prof Bellinger, and immediately after that:

"The heavy metal concentrations of soils have been widely studied in Hong Kong ..."

And here a list of Hong Kong academics, from 1978, 1982, 1987, 1996, 1997 and 2001, who have all studied them.

According to a survey conducted in 1981 by Lau and Wong ... in which the heavy metals in soils of different sectors (recreational, commercial, industrial and minor

agricultural) were studied, the highest [cadmium] concentration in Hong Kong was found in a recreational area (Chung Pui), where 54 [micrograms per kilo of cadmium] was found in roadside soils. The highest copper concentration ... was found in an industrial area (Aberdeen). The highest [lead] and [zinc] concentrations in Hong Kong ... were found in an agricultural area (Man Uk Pin)."

That's up near the border, Sha Tau Kok area, for those more familiar.

Then please coming over to the conclusion, you'll see at page 116 the results and discussion, and at page 116, the right-hand column, about three lines down into the last paragraph:

"It has been shown that the concentration of [lead] in Hong Kong is related to Hong Kong's high traffic volumes ... Although [lead] has been banned in petrol for a number of years, the concentration of [lead] in urban soils still reflects the significant degree of historical [lead] contamination nation and the long half-life of [lead] in soils."

That would be consistent with your own experience?

A. Yes.

Q. If we turn over, at 119, the left column, halfway down:

"Several hot-spots [of very high levels] are

identified from the composite geochemical map, including" -- and these will make more sense to others in the room like than yourself -- "Lai Keng, Cheung Sha Wan, Shek Kip Mei, Kowloon City, Ngau Chi Wan, To Kwa Wan, Ho Man Tin. These are mainly old industrial and residential areas in Kowloon. Therefore, the history of an urban site can contribute to heavy metals in soils."

Page 119, in the right-hand column, it is pointed out that "soils in the hot-spot areas were generally about 2.5 times more contaminated than the rest of the urban area". There's even a reference to the fact that "the hot-spot areas were generally found at the northeast or east side of a major road", because of the diffusion of pollutants by the prevailing wind in Hong Kong, which is from the southwest.

One last point. At page 120, the left column, five lines from the bottom:

"3.3.3. The effects of buildings and landscapes on heavy metal dispersion.

... It has been shown that high-rise buildings can obstruct air movements, and prevent the particulates in air from dissipating."

Have you seen this conclusion reached before, Prof Bellinger?

A. No, I haven't.

Q. That's a study by Taiwanese academics, under their own regime.

If we turn over, the effect of it is that the shape, height, density and configuration of buildings disallows the lead particulates to ever leave the area where they originally had been, so they simply stay and remain.

Does that appear at first blush to be a logical conclusion? In a sense, they are trapped where they got to?

A. Oh, it does -- yes. If something prevents the particulates from becoming airborne and carried by the prevailing winds, then it's likely to continue to reside in the soils.

Q. So if we have a combination of what had been in the past a heavily or medium heavy industrialised areas, and they have now been built upon in a dense way, high buildings, the logical inference is that what was originally in the soil and has now become dust will therefore remain in that area; it can't escape?

A. That's correct.

Q. There's more -- I won't go through them all, but there are more to the effect -- other studies. In fact, there are a large number of Hong Kong studies on these issues.

Now, Prof Bellinger, perhaps I could bring these

points, as it were, to a head. At page 49 of your expert report, the bottom right-hand corner of page 5, also marked with a large 49. The first full paragraph:

"Lead is often characterised as a 'multimedia' 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 ..."

And you make a reference to herbal medicine; here, of course, it would be Chinese medicine.

Now, lead in food, and I see you have studied, for example, the effect of lead in turmeric, I think was it in Bangladesh or India. It's a root vegetable. Do root vegetables take in the lead?

A. To a certain extent, yes, and leafy greens will accumulate lead that's in the air or in soil and then becomes entrained as air as soil is worked. Then also in the past there have been some lead-based pesticides used in agriculture -- lead arsenic, a great combination of lead and arsenic.

Then lead is also introduced along the way as food is processed and packaged. Lead-soldered cans used to be a big problem.

So it's possible at a number of points in the production system for lead to get into food.

Q. What about seafood?

A. To my knowledge, lead was not a major problem in seafood, although I can imagine, if there are local hot-spots near discharge points from industry, seafood, especially shell fish, that are feeding in that area on the bottom where lead may settle into the sediments, that may be a problem locally. I am not familiar with it being a problem globally.

Q. Now, the next one you have is air. Hong Kong is positioned adjacent or part of the Pearl River Delta, which is on any view reasonably heavily industrialised. What is the effect of air pollution coming from those sources? It transports lead or it may do so?

A. Well, it depends on what those transports burn, if they are still using leaded petrol, which I don't think they are. Then, if lead is used in any of the industrial processes that go on in those locales, and it's not within my area of expertise, I don't know -- I don't know if there have been recent measurements of air-lead concentrations in Hong Kong.

Q. So you've identified for the Commission there are a number of sources of lead which can have contributed to the aggregate total of lead inside an individual's

body?

A. That's correct.

Q. And on the face of it, the lead in any solder that has been used is a potential source of some of that lead?

A. Presumably, yes.

Q. The outcomes that Mr Pennicott showed you this morning and the variations and the differences, is that likely to be attributable to any particular factor in any particular individual? Is there any predetermined allocation that you can say is due to lead solder?

A. No. I presume you are referring to the per cent decline in blood lead observed?

Q. Yes.

A. As I said, every individual differs in terms of what they are exposed to from all these different potential sources, in terms of how frequently they come in contact with it, what foods they eat, whether they play outside in contaminated soil. So my first hypothesis would be that it's the mix of -- the contributions of these different pathways and sources differs from one individual to the next, and as a result of that you would expect to see some variation among individuals in the per cent decline when one of these pathways, specifically water, is interrupted and no longer contributing.

Q. Yes, because even the difference in an individual's diet could well be material?

A. Absolutely.

Q. I indicated I would show you the materials relating to the protocol for taking of blood. Could I have this given to you, please. (Handed). This is a document called "Pediatric lead toxicity work-up", and it is published on 6 September 2015.

I also have, incidentally, a document from the Oregon Department of Human Services, the Oregon Lead Poisoning Prevention Program, which I would like you to look at, but please look at the first document.

One can see, under the cross-heading "Whole blood lead level":

"Whole blood lead level ... is the criterion standard for confirming the diagnosis of lead poisoning. For convenience, a finger-stick capillary lead level has been used for screening. Properly collected capillary samples have a 10 per cent false-positive rate. Once an elevated lead level is detected, a venous lead level is assessed for confirmation."

A. Yes, that's absolutely right. In fact last week, at the meeting of the guideline work group, producing guidelines for the diagnosis and treatment of lead poisoning in Geneva, this is an important part of our

document, that capillary samples are fine for screening but you don't make treatment decisions based on them; you get a confirmatory venous blood lead level. But that's when you are contemplating clinical management.

Q. Do you know, for the Hong Kong results, whether they were obtained by capillary or venous method?

A. I don't know, no.

Q. So it is important because if it was capillary, but had not been audited by a venous test, then there is significant intrinsic doubt as to the reliability of the original capillary reading?

A. But the pattern of findings over time, in the individuals that were identified as having a result above the reference level, are so coherent in terms of the distribution of declines, I think that's very, very unlikely, that there was contamination of the original blood lead levels.

Usually, when there's a contamination from a capillary sample, the value is greatly elevated, because a microgram of lead is microscopic, and much more than that is on the finger from contamination. So, when that gets in the blood, you are going to see a blood lead level that is much higher than 6 or 7.

Q. Can I ask you a likely different matter for a moment. Do you agree that lead absorption in children is

increased when there is insufficient calcium in the child's diet?

A. There are epidemiological studies showing that children whose diets are insufficient in calcium do have higher blood lead levels, and it's based on the underlying biology. The inference is that the absorption of lead is higher in those children. I don't know of any experimental evidence to back that up, but it makes sense in terms of the known biology.

Q. If I can just ask you the same question but changing the variable. Instead of an insufficiency of calcium, if there was an insufficiency of iron in the diet, is that also likely to lead to increased lead absorption in children?

A. Yes.

Q. Why is that?

A. Well, lead and iron also look very similar to -- they look similar to one another chemically, just as lead looks similar to calcium, so they compete for binding sites in the gut. So if iron is not present, then lead will preferentially be attached to those binding sites and be absorbed into the gut and then into the blood.

DR McCOY: I have no further questions. Thank you, sir. Thank you, Prof Bellinger.

CHAIRMAN: Anybody else? Mr Ho.

Cross-examination by MR HO

MR HO: I just have one or two very minor questions.

Prof Bellinger, I am representing the Housing Authority. I have just got one or two minor questions for you.

When you were answering my learned friend Mr McCoy's question, he showed you a paper which I think was in 1987, and there were some figures about food intake, and you said those are outdated because now, normally, the intake of lead from food is much lower. Do you remember that?

A. Yes. Was that -- I thought that was total lead intake, not intake just from food.

Q. Right.

May I just ask you to look at a background paper to the WHO Guidelines for Drinking-water, which is a paper that's been referred to in this Inquiry, and that is at A1. Page 403 is the cover page. Perhaps you can have a look at that first.

You see the title. This is a background document. If you look at the next page, you will see the date of this paper is 2011; do you see that?

A. Yes.

Q. Are you familiar with this paper?

A. Yes.

Q. You are?

A. Yes.

Q. Did you have a hand in the preparation of this paper?

A. No. I had a hand in the process that this was based on --

Q. I see. Thank you very much.

A. And the JECFA re-evaluation of lead earlier in 2011.

Q. If I may ask you to go to page 2, which is at page 410, please.

Paragraph 2 -- of course you see, I believe, these are the sort of pathways that you identified and explained to us earlier: air; 2.2 is water; 2.3, we see food; 2.4, which is over the page, we see "Other routes of exposure", and that refers to household dust as being one of the significant sources.

If I may ask you to come back to food, at 2.3. At the bottom of that page, do you see it gives examples of the daily dietary lead intake from various countries, for example, like Sweden, and over the page, do you see 66 micrograms for Finland; 23 micrograms for the USA; and further down the page we see a figure for England; we also see Canada, which is 53.8 micrograms per day; Belgium, 90 micrograms per day; Sweden has a lower figure, 24; Mexico has, amongst these countries, the highest, 177. Do you see that?

A. Yes.

Q. So there is a great variety of the daily dietary intake of lead between the countries?

A. Right. One caution I would interject here is that it is important to look at the dates that these figures were taken from, because, looking very quickly at the references, I see that the Finnish number comes from a 1980 study, the USA from 1982 to 1984, and Sweden from 1985. So these are quite outdated numbers.

Q. Right.

A. They are much lower now.

Q. Right. But even amongst what we call the developed countries, for example some of the European countries and even Canada, the daily intake from diet could be much higher than what you said, 20 to 25?

A. They could be. You do have to be careful about dietary estimates, because it depends a lot about how you survey the diet.

Q. Certainly.

A. There's a lot of variability of individuals within the same country and what their diet is. So you just have to be careful to not put too much stock in any single number unless you know exactly how the data were --

Q. Exactly. So there can be different variants which may contribute to a higher figure or a lower figure, even

amongst developed countries on the one side and developing countries on the other side?

A. It's very hard to be certain here.

Q. You can't just generalise, as it were?

A. You can, but at your own peril.

Q. All right. Interestingly, the last sentence of that paragraph talks about even you get lead from drinking wine.

I know quite a lot of my colleagues here drink wine, so do be careful.

Moving down the page to 2.5, you see there is a sentence that starts:

"More than 80 per cent of the daily intake of lead is derived from the ingestion of food, dirt and dust."

From water, that is a relatively small proportion of total intake. That's still true, isn't it?

A. I think on average that's a fair statement, but it will vary from setting to setting.

Q. In fact, the same point is made, if we go to the end of this document at page 423. The last paragraph on that page, if we start with the second sentence of the last paragraph:

"Nevertheless, because lead exposure arises from a range of sources, of which water is frequently a minor one, as it is extremely difficult to achieve

a concentration lower than 10 ..."

Again, I believe that's in fact a sentence that is lifted and becomes part of your report, so that must be correct?

A. Yes, as far as I know.

Q. Thank you very much.

I see that apart from what is stated here -- air, water, food, household dust, and so on -- in your own report at page 63 of the bundle, you also identify children's toys as a potential source of lead intake.

A. (Nodded head).

Q. Is that, in your experience, a general problem with lead in children's toys?

A. In the US, it is a problem with toys imported from certain areas. Mainland China is one of the areas of great concern.

Q. Sorry to say.

A. Sorry to say, yes.

Q. Yes, in mainland China there is a danger or a risk, at least, of producing toys that may contain lead content.

A. Yes, but it's true for toys imported from other areas as well. The surveillance is not really what we would like to see, and if a child is mouthing a toy that's painted with lead-based paint, that can lead to very high exposures.

Q. Of course let's put your report into perspective. You are telling us generally about the effect of lead on various groups of people, but of course, in any individual case, if we want to see the effect of lead on a particular individual, we have to do a lot more study, examination and so on in order to find out the cause; is that correct?

A. What do you mean by particular effects on an individual?

Q. For example, if we isolate one particular person in question, then what you are doing in your report wouldn't give the answer?

A. What I said in my report applies to groups of individuals, and the associations that have been observed are population statistics.

Q. Yes.

A. It can be difficult to identify effects in any individual child. All we can say is that, on average, children with higher blood lead levels or adults with higher blood lead levels have this health adversity.

Q. Yes. Thank you. This is a sort of statistical general observation?

A. Right. We can't say with certainty. We might be able to say more likely than not a child's problems are due to lead.

Q. Thank you. But in order to find out as referring to

a particular individual, we have to study a lot more, we have to have a lot more information about his background, his living environment and so on?

A. I see what you mean. Yes, in order to identify the potential contributors to blood lead level.

MR HO: Yes. Thank you, Professor.

Cross-examination by MS WONG

MS WONG: Prof Bellinger, if I may ask a few questions.

This morning, Mr Chairman has raised the issue of the rationale as to why the US was adopting a higher value of 15 micrograms. I wonder if I may assist by producing or showing you a document. (Handed).

Prof Bellinger, I think this is a meeting note dated 11 January 2006, by National Drinking Water Advisory Committee.

Is it correct that this is a federal advisory committee that supports the EPA in implementing its duties and responsibilities?

A. That's my understanding, yes.

Q. We can look at page 2. We see from that table, there are three different contaminants commonly found in drinking water; correct?

A. Yes.

Q. They include the distribution before water enters the distribution system; the second one is at the end of the

filtration process, before distribution system; and lastly, at the consumer's tap.

I can see there are three different regulatory approaches; correct? Different EPA regulatory standards, in that table.

A. Three approaches, one for each of the contaminants?

Q. Yes, for each contaminant.

Can you explain why we have three different standards for the three different sources?

A. A different standard for arsenic and cryptosporidium and lead?

Q. Yes.

A. Because there's a conclusion that we need to worry about different levels of contamination for each of the three, that they can't all be the same -- the same standard would not be appropriate for all three. They need to have their specific standard, based on the hazard identification process and the whole risk assessment that underlies the setting of standards.

Q. And the purpose is of course to eliminate the lead in each of these possible sources, so that you could achieve the so-called lead offset zero in drinking water?

A. Well, I think the EPA says that it's not really feasible to achieve a level of zero.

Q. Yes.

A. So they set a standard that they hope will be protective of the most vulnerable subgroup of the population.

Q. Yes, and if we turn to page 3, which appears to explain why this 15 micrograms was arrived at -- if we look at paragraph 2, it states:

"To establish the action level, EPA reviewed information from representative water systems, efficacy of different treatment technologies, and cost-effectiveness of these technologies."

And it referred to footnote 1, which states:

"EPA gathered data from 39 medium-sized water systems. Approximately 96 per cent of these systems were able to keep in the 90th percentile in the range of 10-20 [micrograms per litre]. Thus, EPA concluded that 15 [micrograms per litre] represented the feasible level for public water systems."

It appears why they have chosen this particular figure. Do you accept that, Professor?

A. Yes, it's not strictly health-based but it takes into account the feasibility as well. It's a risk management standard, not necessarily a health-based standard.

Q. Yes. Thank you. It refers to an action level. This is my last question. So is it correct that this action level is simply a screening tool for determining whether

certain treatment technique actions are required?

A. That's my understanding, yes.

Q. Thank you.

MS WONG: Thank you, Professor.

Cross-examination by MR LEE

MR LEE: Yes, Professor. You have said that a child gets one chance to develop his brain, something like that. If he misses that chance, presumably, when the child grows older, his IQ level would not compare as well as his peers; is that right?

A. Could you say the last part, may not compare?

Q. May not compare as well as his peers.

A. Not as well as it would have been had the child not been exposed to lead. It may still be higher than his peers.

Q. But he has one chance to develop the brain, and if he misses that chance, can he ever catch up?

A. Unfortunately, with brain development, it may not be possible to catch up. As I mentioned, a variety of processes need to happen at the right time, and putting cells in the right place, and if that chance is lost then there may be permanent effects that cannot be overcome. The effects may be reduced if the child has certain advantages after that point, but the brain cannot go back and rewire itself.

Q. But if the lead content, say from whatever source, gets

into the blood, then presumably it could reach the brain?

A. Unfortunately, yes. There is -- as adults, we have a nice system called the blood-brain barrier that has very tight junctions between the endothelial cells and it limits the size of molecules that can pass into the brain.

In children, those adjunctions are not so tight, they are kind of leaky, for the first couple of years. So things like lead can get into the brain more easily in a young child than in an adult. That's why the central nervous system is more vulnerable in a child than the peripheral nervous system in an adult.

Q. When the lead gets into the brain, does it settle there?

A. Yes. Unfortunately, it has an easier time getting in than getting out.

Q. Ah. So what effect would it be on that child?

A. Well, it depends on the dose, how much lead. What we now think is that there are certain areas of the brain that are more vulnerable to the effects of lead than other areas. One of the primary areas is the frontal lobe, the prefrontal cortex, which is where what are called executive functions tend to lie, and those are things like the ability to do long-term planning and organisation, to develop strategies and to adapt the

strategies in the face of new information, the ability to delay gratification. This is why we think that children with too much lead are more prone to ADHD and behavioural problems, because executive functions are the major underlying deficit of children with ADHD.

We also know that lead is particularly dangerous to a small area of the brain called the hippocampus which is where learning and memory take place, a process called long-term potentiation, which is dependent upon the glutamate system of the brain, which is a neurotransmitter, and lead interferes with the function of that.

So this is a child who may have trouble keeping up in school, may get into behavioural difficulties, and those I think also underlie the propensity I mentioned to aggression and getting into trouble and violence, because they can't delay gratification, don't have as much success in school and so drop out and start making poor choices because of executive function problems.

So I think these issues compound as a child get older and play out in ways that are very disadvantageous.

Q. So once the lead gets into the brain, and supposing the cause of the lead in the brain is actually from lead in the water -- let's assume that -- will the child improve

after he moves away from that source?

A. Well, it's always good if exposure is reduced. So if the child is no longer taking in lead from that source, that's certainly of benefit.

In the literature on lead, you do see that the early deficits associated with childhood exposure are permanent. Personally, I don't believe that. I think it's too pessimistic. I think there are studies -- the prospective studies that have followed children do show that the deficits persist, but usually children remain in the same environment, and so they are continually exposed.

My feeling, and there is actually some animal evidence to support this, is if you raise an animal in an enriched environment, which for a rat means a bigger cage, a cage that includes toys to jump around on and other rats to interact with, that actually can prevent some lead-associated cognitive problems.

Q. Provided there is no lead in the toys?

A. No lead in the toys, correct. But we don't know -- we don't have good studies to indicate whether that's true for children, and I believe that it probably is, but we just haven't done those studies.

Q. But would you agree with me that once lead gets into the brain, it might or it might not be permanent? Would you

accept that?

A. It might not. There are animal studies that show that -- that have sacrificed the animals and measured the actual concentration of lead in different areas of the brain, and those studies have shown that if you put -- if you expose an animal, their blood lead goes up, eventually their brain lead goes up. You take the animal out of that, you stop the exposure, their blood lead comes down, but for the foreseeable future the brain lead remains elevated. But the animals have been sacrificed. If they let the animals live forever, perhaps brain lead would come down, but so far, it doesn't look like that.

Q. Let me come back to lead in the brain of a child. Can you get rid of the lead once it gets into the brain?

A. As far as we know, the chelating drugs that are used when a blood lead level in a child gets above 45 primarily takes the lead out of the blood and to a lesser extent some of the soft tissues. But the brain doesn't seem to be one of those soft tissues, and I think it's because the chelating agent is too large a molecule and even it can't cross the blood-brain barrier. So the answer is, as far as we know, no.

Q. So the chelation treatment, is it an intrusive form of treatment?

A. Certainly it has to be considered --

Q. How would it affect the child, say?

A. Well, there are different options among chelation. Until about a decade and a half ago, the chelation required hospitalisation and giving the child very painful intramuscular injections or IV injections of chelating agents.

Q. How frequent would the injections with?

A. Well, for the most common course of chelation, it's a 19-day course of treatment, and then you stop and let the re-equilibration process that I talked about go on. Usually what happens is the child's blood lead drops precipitously when you start chelation, but then a week or so after you compete it, it comes back up because of the re-equilibration, and so it's necessary to give another course of chelation.

Q. So how many courses of 19 days each?

A. As many as needed. In the Zamfara episode, where children had blood lead levels of several hundred -- I talked to the paediatrician who did the chelation and one child required 68 courses of chelation, and the blood lead still was elevated.

There are now oral chelators, dimercaptosuccinic acid, that can be given on an out-patient basis, which is much less intrusive and more acceptable to parents

and children.

Q. Is it also more effective?

A. It is not effective in reducing the cognitive deficits. It does reduce the blood lead and the soft tissue lead, but there was a randomised trial where children of blood lead levels of 20 to 44 were randomised to receive chelation with oral succimer or placebo, no treatment, and the children were followed up after treatment was completed and there was absolutely no difference in the cognitive outcomes of the children.

So waiting for the child to become poisoned, it's too late, apparently, to prevent the adverse effects of lead on the brain.

Q. Professor, you mentioned chelation. Are there other methods of treatment?

A. That's the first choice. But as I say, chelation is only indicated when a child's blood lead is above 45.

Q. That's very high.

A. Yes. For blood lead levels 44 and below, the interventions are the environmental investigation, to identify an ongoing source, the nutritional counselling, to make sure that the diet is adequate. But unfortunately there's really not much else to offer. The chelation is counter-productive in a child with a blood lead below 44 because it chelates not only lead

but chemicals that look like lead, and the calcium and the zinc that we talked about earlier, and there is some renal toxicity. So it's not performed below 45.

Q. I move on to another topic. You have been asked many questions by other counsel about other sources of lead getting into the body, apart from lead in the water; soil, air, dust and all this. Let me pose a number of questions to you.

I will use two examples, scenario A and scenario B. Before I go to those examples, is there any relationship between the level of lead in water and the level of lead in the blood?

A. Yes.

Q. Scenario A: if the lead content in water is low, like 2 micrograms per litre, but the lead content in the blood is relatively high, say 20 micrograms per decilitre, would you say in this example that the excess of lead in the water is an important contributing factor to the rather high presence of lead in the blood?

A. I would say it's unlikely. It's more likely that there's some other source that's of primary importance for that one child.

When I said there is a relationship between water lead and blood lead, that again is looking at a large population, where those sorts of unusual circumstances

would even out.

Q. So I come to scenario B. Now, this time there's also high lead content in the water, say 50 micrograms per litre in the water, and on the blood side it's also high, say also 50 micrograms per decilitre. In scenario B, would you say the lead content, high lead content in the water, is an important contributing factor to the lead in the blood?

A. Probably, but again I would want to know what the other potential sources are, and I would want to know if the child was actually drinking the water.

Q. Of course, or the baby drinking milk powder where leaded water was used.

A. Yes.

Q. But of course I forgot to mention that if you assume the other factors were equal in scenario A and scenario B -- do you follow me?

A. (Nodded head).

Q. Otherwise, if the other factors are totally different, then you cannot make a comparison. So you have to make a comparison on the basis that the other factors were equal in scenario A and scenario B?

A. I agree.

Q. So, if that is the case, then if the presence of lead in the water is high and the presence of lead in the blood

is also high, then you would say the water is an important contributory factor?

A. More likely than not, and you might want to interrupt the water pathway to see what happens to the blood lead and see if it drops.

Q. Yes. Thank you.

Now, you have in your report made some importance on the age of a child: 0 to 1 and then 1 to 6, and then 6 to 18. I think this is more or less what you are directed to do by the Commission.

A. Yes.

Q. But six years is a rational age; is that right?

A. A threshold?

Q. The threshold of six.

A. What do you mean by "threshold"?

Q. Because I see in your own report, at page 7, internal pagination, that is page 51, the first complete paragraph:

"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."

There, you use six years; is that right?

A. Well, in this study, they measured blood lead quarterly

in these children for the first few years of life, and then up to six years of age, but levels beyond that were not available until they became adults.

Q. I see. So you say this: if you call that a threshold at all of six years, it's really optional? You could put it up to eight years or seven years?

A. I was just describing the data that were presented in this paper. They didn't have blood leads at eight.

Q. But the Commission then designated six years as the threshold?

A. Yes, and I think that's because, for instance, the US CDC says that children up to six are the most vulnerable group. There is no bright line.

Q. But there is logic in it?

A. There's what?

Q. There's logic in six years, is there; you agree?

A. Well, I agree that the risk is greater among young children, but I don't think there's a bright line. For pragmatic reasons, we have to choose a cut-off, and six is the one that people generally use. But, you know, lead isn't good for anybody.

Q. And it should be zero?

A. That would be great. I would love to see that.

Q. Yes.

Now, looking at your report, at page 17, the

internal pagination -- it's the care plan, is that right, that has been used in the US?

A. Correct.

Q. For children, what is the age of those children? What is the threshold there?

A. This is children under six.

Q. Also six?

A. Yes.

Q. Very well. You see on the left-hand side, it's "Blood lead concentration [less than 5 micrograms per decilitre]". Then you have a number of things: lead education, dietary and environmental, and then environmental assessment for pre-1978 housing.

That's old housing; right?

A. Yes. That's when leaded paint for interior use was banned in the United States. So it's a useful screening criterion.

Q. Then follow-up blood lead monitoring. If you look at that and compare it to Hong Kong -- it's bundle E2, page 871. I think you just got these documents yesterday or today. It should be there.

We can see the chart. The first one, 1, then it's less than 5 micrograms per decilitre; "Reassurance and no further follow-up".

So, in other words, as far as the Hong Kong

government is concerned, when the lead content in the blood is less than 5 micrograms, then they don't do anything about the child, it would appear? Do you understand that?

A. Yes.

Q. But in the United States, there were these four things that I have mentioned to you -- three things; right?

A. I'm sorry, could you repeat that?

Q. I referred to your own chart just now. So, even less than 5 micrograms, there are three things done in the United States?

A. Well, I think I actually made an error here, because if you look down the table, for the schedule for follow-up testing, the first --

Q. Your table or the Hong Kong government's table? You say there's a mistake. Mistake in --

A. In my table.

Q. Okay.

A. Yes. There isn't a schedule for following up a blood lead level of a child whose concentration is less than 5 micrograms per decilitre. You can see it's only when it's between 5 and 10, the recommendation is to have a follow-up between two and four months after the initial value, and then, if the blood lead is declining, only after six to nine months.

So, in the United States, there is not any universal screening of children for blood lead. There's targeted screening if the child has some risk factors, if someone suspects that they may be exposed to too much lead.

In my own State, there is mandated screening to age 4, but then thereafter only if there are circumstances that make the paediatrician suspicious.

Q. When there are certain symptoms; is that right?

A. Well, not symptoms of lead toxicity, but if the child has risk factors, so that if the child is living in a pre-1978 home, or if a sibling has been lead poisoned, or if it's known the parent is occupationally exposed to lead; factors such as those.

Q. Now, the Hong Kong government set the threshold at eight years old instead of six.

A. Six? That's prudent. It's conservative.

Q. That's more prudent, because instead of six it's now eight?

A. Right.

Q. But these are only children in the affected public estate, as far as the Hong Kong government is concerned, right, not generally?

A. That's right. Actually, they include children under 12, so that's even more prudent.

Q. But the trouble is, if a child lives in a certain public

housing estate which is affected by lead in the water, and he has been living there for a number of years, but there was no discovery of excess of lead in water yet, and then he moves out, and then say he becomes nine or ten years of age, then he goes above the threshold of eight years, and so he will miss out from the government's plan. Do you follow me? Because the government only looks at children under eight years old.

A. I don't know that that's the case.

Q. But if that is the case, what do you think about it?

A. Well, they are doing screening of all individuals who live in the estates.

Q. You understand that the Hong Kong government does screening for all children in these estates which are affected? Is that your understanding?

A. That was my understanding, yes. And I would hope that there is a system in place to continue tracking these individuals over time. So, in the scenario you mentioned of a child moving out of the estates, if that child still has an elevated blood lead, no matter where that child is, they should be in the case management system until the blood lead falls below the reference value.

Q. But the problem is, if a child is already nine or ten when the whole of Hong Kong knew about this excess of

lead in water, then he is already too old for the threshold of eight.

A. Well, looking at the line listing that I have been provided, there are individuals above the age of eight who are being followed and, if they have a blood lead level above the reference, they are being re-tested.

So your scenario doesn't accord with my understanding of the data that I have been given.

Q. Perhaps I should show you a document which we got from the internet, a government document. It is from the Centre for Health Protection, and the title is "Incident of lead in drinking water". (Handed).

These are what's called the frequently asked questions, and then the government providing the answers. The question is, "When can we arrange for a blood test?"

"For residents of the affected public estates who are children under eight years old, pregnant women and lactating women, they can make arrangements for blood testing via the hotline ... The hotline will operate [between certain hours]. It will be diverted to the Government hotline ... after operating hours."

So it only caters to children under eight years old and pregnant or lactating women. So if the children are over eight years old, then they have to consult their

own laboratory or whatever.

That is why, if a child had lived in one of these estates affected by excess lead in the water but then moved out, and is now nine or ten, then there's no such facility provided to this child.

A. I see what you are saying, yes. It appears so.

Q. Is that satisfactory?

A. Well, in the ideal world, no. This document does say, in this particular situation, we need to channel limited resources to children under eight, pregnant women and lactating women. If I were forced to make a choice about which groups to focus on, I would make the same choice.

But I agree, in an ideal world, the coverage would include other subgroups as well.

Q. But would you not mention somewhere, maybe in a footnote or whatever, that if the children have been living in these estates for a number of years but now they are over the age of eight, then also ring the hotline or at least get their parents to ring the hotline?

A. That's a very point.

Q. Thank you. Now, if the government sets the threshold at eight years, which you say is prudent, more prudent than six, but they should treat it as a reference point only, or should they treat it as at a reference level or

an action level? Do you see the difference between reference level and action level?

A. I don't. In the care plan, they do recommend or mandate certain follow-up activities, if a child's level exceeds the reference value. So it looks to me as if action and reference levels are being interpreted as one and the same.

Q. Now, I want you to look at the letter from the government which you have been looking at. This is page 846 of E2. You have been looking at this letter already, right, earlier?

A. Yes.

Q. You are familiar with this letter.

Go to the next page, towards the bottom of the page, 847. The last paragraph says:

"Children identified with developmental or behavioural problems ..."

Perhaps I should go -- sorry, I want to read the paragraph above that, beginning with "Regarding":

"Regarding the 'developmental assessment' mentioned in page 19 of the expert report ..."

That is your report; right?

A. Yes.

Q. "... all children with elevated blood lead level will receive preliminary developmental assessment at DH's

Child Assessment Centres" -- that's Department of Health -- "(for pre-school children below 6 years) or Student Health Service Special Assessment Centres (for schoolchildren 6 to 12 years) by a developmental surveillance team composed of paediatricians and nurses."

Then it talks about what the development assessment covers. Then the last paragraph:

"Children identified with developmental or behavioural problems will have follow-up evaluation at Child Assessment Service according to individual needs."

So once a child is identified to have these problems, developmental or behavioural, then there will be follow-up. Then:

"Children with largely normal development would receive continuous monitoring through enhanced developmental surveillance at Maternal and Child Health Centres ... during pre-school years and annual health visits at Student Health Centres during school years."

So two types, those who are identified with behavioural or developmental problems, there will be follow-up; those with largely normal development would still have enhanced developmental surveillance. Then:

"Parents are provided with anticipatory developmental guidance and information on children's

development in the form of pamphlets. These pamphlets are available online at [so and so]. Due to the large volume of these documents, hard copies will be provided upon [the Commission of Inquiry's] request."

Do you see that? Have you actually gone online to take a look at these pamphlets?

A. No, I didn't.

MR LEE: Can I have some indulgence? I want to discuss with my junior, because it does say if you want hard copies, it's only upon your request. We couldn't get a copy ourselves.

CHAIRMAN: Do you want a copy?

MR LEE: Yes.

CHAIRMAN: Have you ever seen this document first?

MR LEE: Online, yes. Because to us, they are totally unimportant. Nothing to do with this case.

CHAIRMAN: So you want a copy?

MR LEE: I want to show you and the members --

CHAIRMAN: Why don't you show me online first?

MR LEE: Okay, online. Can we do it here? I don't know whether we can do it now, because we do not have ...

CHAIRMAN: Let me make this suggestion, since now it's almost 4.15. Why don't you or your junior go back to your chambers and look at the relevant paragraphs and identify the relevant paragraphs, and then I don't think

you really need to print an entire copy of these documents. So print the relevant paragraphs or pages, or indicate to us which particular paragraph or pages that you want, then we can print it ourselves.

MR LEE: My junior read them. Perhaps I can consult with her.

CHAIRMAN: Yes. Or you can tell me now, actually.

MR LEE: It may be possible for us not to go into these pamphlets, but I would like to discuss with the team in the meantime.

CHAIRMAN: All right. So if --

MR LEE: I won't be long, but I would like to have some time to make sure I won't be long.

CHAIRMAN: I will give you the evening to prepare your case. So if you think a particular paragraph or pages are relevant to our hearing, then inform my secretaries and the other legal teams.

MR LEE: Yes, we will do that.

CHAIRMAN: So that's it for the day, Prof Bellinger. I would be grateful if you could come back at 10 o'clock tomorrow morning.

WITNESS: Certainly.

CHAIRMAN: Thank you.

(4.15 pm)

(The hearing adjourned until 10.00 am the following day)

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