T R A N S C R I P T

ENVIRONMENT, NATURAL RESOURCES AND REGIONAL DEVELOPMENT COMMITTEE

Inquiry into the CFA training college at Fiskville

Melbourne — 19 October 2015

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Professor Brian Priestly, director, Australian Centre for Human Health Risk Assessment.

The CHAIR — Thank you, Professor Priestly, for coming to speak to us today. I will go through some of the formalities. On behalf of the committee I welcome Brian Priestly from the Australian Centre for Human Health Risk Assessment. All evidence taken at this hearing is protected by parliamentary privilege as provided by the Constitution Act 1975 and the provisions of the Parliamentary Committees Act 2003 and is protected from judicial review. Any comments made outside the precincts of the hearings are not protected by parliamentary privilege. All evidence today is being recorded, and you will be provided with a proof version of the transcript to review for factual corrections. We welcome you to give us a 15-minute presentation, and then, if you do not mind, we will probably ask you many questions.

Prof. PRIESTLY — Thank you, Chair. I have not prepared any particular presentation. I thought I would give you a quick summary of my experience in this area and talk about the toxicology of the perfluoroalkyl compounds, PFOS in particular, which is relevant to this inquiry, and then I am happy to try to answer the questions you have.

The CHAIR — That sounds very good, thank you.

Prof. PRIESTLY — I will say a few words about my career and experience. I graduated from Sydney University with a degree in pharmaceutical science and a PhD in 1968. Most of my career I have been practising the discipline of toxicology, both in academia and in government. I am currently a fellow of the Australasian College of Toxicology and Risk Assessment. My current appointment is as director of the Australian Centre for Human Health Risk Assessment at Monash University. I took up that appointment in 2003, but it has been part time since 2009, when I allegedly retired from full-time work, although I have been keeping quite busy doing a variety of things.

The experience I have with the perfluoroalkyl compounds is that I have peer reviewed several of the reports on Fiskville for the CFA. About five years ago I did a literature search on the potential health effects of the perfluoroalkyl compounds, and I have recently updated that with the current literature just to get a feel for what has happened over the last five years. I am currently on the program advisory committee for the CRC CARE project, which is developing soil and water criteria for PFOS and PFOA. That is a brief history of my experience with this thing.

What I might do now is give you a summary of the toxicology of the perfluoroalkyl compounds. I presume I do not need to say too much about what they are. Effectively they are heavily fluoro-substituted alkyl compounds with surfactant and stain-resistant properties. PFOS — the particular one we are interested in here — has been used in aqueous film-forming foams used in firefighting to assist with the process of foam formation and stabilisation.

The long-chain PFCs, of which PFOS is a good example, are environmentally persistent and bioaccumulative, which means that they can accumulate through the food chain. Exposure has been pretty ubiquitous for humans. You will find low levels of PFOS and other PFCs in the serum of most populations around the world because of their quite extensive use. The short-chain and branch-chain congeners of PFCs tend to be less persistent and are probably less toxic, although I think it is fair to say that we know less about the toxicity of those other compounds. Most of the work has been done on PFOS and PFOA. Those two compounds have been largely phased out since around about 2001, but some of the PFC telomeres, which are less substituted compounds, are still in use, so you will find some of these now in the blood samples that have been taken.

If I can summarise the toxicological profiles, as I say, we know most about PFOS and PFOA, and the database is pretty extensive. As normally, it consists of studies in animals — mainly rats, mice and monkeys — and that is the usual database that we use for determining the health effects or trying to predict the health effects in humans. I will talk about that in a moment.

But also we have a fairly extensive suite of epidemiological studies directly in humans, and those tend to be of three types. The first type is studies in workers who have been exposed — probably the most highly exposed groups — in the production and manufacture of these PFCs, so they are a group that has probably the highest level of exposure. The next group is studies that have been done in what is called the health study of residents around a PFC factory in the mid-Ohio valley, a DuPont plant where PFOA leaked into the groundwater, so there was reasonably extensive exposure to PFOA in those cohorts, and they have been quite extensively studied. But the majority of the epidemiological studies have been done with what I would call normally exposed humans — people who have had their exposure through the food chain or through house dust or

whatever may be the case — and they have quite low levels of PFOS and PFOA and other perfluoroalkyl compounds in their blood.

The NHANES database in the United States is a very good example of the databases that are available. It is quite an extensive database of studies on blood levels of various pollutants in the serum of people, and these are attempted to be matched to various types of adverse health outcomes. The usual approach is to look at the incidence of disease in the highest exposed quartile and compare that with the incidence of disease in the lowest exposed quartile, and then you draw conclusions about any possible associations. As I say, the United States NHANES database is probably the major one that has been used, but there have been a number of other studies using databases from the United Kingdom, from various parts of Europe and from Japan, so the epidemiological database is now reasonably extensive.

As I said, animal studies are used to identify the target organs for the toxicity of these compounds. These are primarily in the liver, but they also have effects on hormone systems and on lipid homeostasis, in particular a reduction in cholesterol and serum lipids. There have also been some studies looking at the effects on foetal development and on immune dysfunction, but in mice, and possibly some effects on the nervous system, although it is not clear whether these are direct effects or whether they are secondary effects on things like thyroid hormones.

Normally we use these studies — these controlled studies — where we know what the exposure is over time, to establish health-based guideline values and to establish safe level of exposure in humans. But this is complicated in the case of these compounds because of the very marked differences in the way in which they are cleared from the body. In fact in rats and mice, which are the primary database for these sorts of studies, these substances are cleared very quickly. In humans they are cleared much more slowly. So for the same level of intake you will find that they will accumulate to a higher blood level in humans than they will in animals. If we are going to use the animal data, which is in fact the case, then adjustments are made for these differences of clearance and differences in the way in which they are handled.

If I go to look at the epidemiological studies, as I said, this has now been a vastly expanded literature, and I would summarise the overall position as being still rather confusing. Many of the findings are inconsistent. Associations may be found for some of the PFCs, but they are not always the same effect across different studies, nor are they the same individual compound across the studies. I think that complicates the epidemiological picture because we now have measured anything up to eight different congeners in blood and tried to link that to adverse health effects, and, as I said, the situation is really quite confusing.

If you look at the earlier studies that we have done with the occupationally exposed workers, where the blood levels are orders of magnitude higher, there is very little in the way of adverse effects which are linked to those studies. Mainly they are lipid changes, which paradoxically seem to be in opposite directions to what you would predict from the animal studies. In the cohorts with the lower levels, as I said, the general approach is to compare disease incidence in the highest quartile or tertile blood levels compared to the lowest ones. The problem here, and one thing that needs to be very carefully considered, is that the associations that are found may indicate some sort of causal relationship, but they may also indicate some sort of reverse causation, where the disease under study may have changed the way in which the PFCs are cleared from the body, so that the highest blood levels are the result of the disease causing a reduction in their clearance and therefore perhaps what is called reverse causation.

The various diseases that have been studied in these epidemiological studies have included a look at thyroid dysfunction; changes in serum lipids, particularly looking at cholesterol; changes in blood uric acid; altered foetal and postnatal development — birth weight and things like that; altered hormone levels, particularly the thyroid hormones; and also some changes in reproductive function, with sperm abnormalities in males, altered female fecundity — that is, the ability to get pregnant — and onset of menopause. There have also been studies looking at effects on immune responses, and again these are particularly inconsistent. It is very difficult to tease out whether they are associated with a particular PFC, whether they may be confounded by exposures to other persistent organic pollutant compounds, which are also known to have effects on the immune system.

Finally, there is one recent study that I have seen looking at the exposure of Australian firefighters; it was the only study that I have really seen where firefighters' blood levels have been measured. Certainly there was found to be an increased level of PFOS in the serum — about 20 times less than that in the occupationally exposed cohorts — and the levels tended to correlate with years of foam use. But in that particular study they

also looked at serum lipids and uric acid and found no association with PFOS. With that brief summary, I think I might finish and then perhaps try to respond to questions.

The CHAIR — Thank you. You were saying about PFOS that the workers manufacturing some of these products would have the greatest exposure, but does it matter the way in which we are exposed and what sort of protection that you receive when you are talking about levels of exposure? For example, I think one of our previous presenters, or it was in some of the literature, spoke about if it is burnt as opposed to manufactured there may be a different type of exposure. Do you agree with that?

Prof. PRIESTLY — It is a difficult question to answer because if it is burnt — I presume you are talking about the use of these things in firefighting foams where they are used on fires — I am not really sure what sort of chemical changes would occur. These compounds are pretty persistent and difficult to degrade, but I am no expert on that area so I do not know. But in terms of whether personal protective equipment and so on would prevent exposure, it often does. But given the fact that it has been fairly consistently shown that people who work in environments where these things are manufactured do accumulate quite high blood levels, so they must be getting an exposure through either hand-to-mouth oral ingestion or possibly inhalation of some fumes — although these things are not terribly volatile — or dusts, or something like this. They may be the major routes of exposure.

The CHAIR — In this area there seem to be different opinions within the scientific or the health field. Do you know why that is the case? I am thinking about industrial disease, whether it is miner's lung or asbestos or tobacco connections to cancer. There have always been things that initially have been there, but there has been a lot of disagreement and a lot of difference of opinion. So at what point does a consistent view become apparent?

Prof. PRIESTLY — Again, a very difficult question. Certainly you will find scientists will tend to interpret the same study in slightly different ways. If I look back at the way in which the animal studies, for example, have been used to develop health-based guidelines to protect humans, most of the agencies around the world have tended to use a rather small set of studies to set those guidelines, but the numbers that are generated are often different. The differences relate to the way in which you extrapolate from the dose used in animals; whether or not you take into consideration the differences in blood levels associated with those doses — and that seems to be the more contemporary approach, to extrapolate the blood level — and then, more importantly, there are policy-driven differences in the way in which adjustment factors are applied.

The standard practice in setting a guideline of value for humans is to try to establish a dose in a controlled setting in animals at which there is no effect on even the most sensitive animals in the cohort, apply a series of safety factors or adjustment factors to that to account for differences in extrapolation between species and differences in sensitivity in the human population. So the guideline value that is set is often an order or two of magnitude below the dose which is actually producing effects or not producing effects in animals.

The CHAIR — Do you think there is a link between exposure to these compounds and the health of humans?

Prof. PRIESTLY — The easiest way I can answer that is that the animal studies and the extrapolation from those to human exposures suggest that there are quite significant margins of exposure between what we are actually seeing in the normal human and what would be expected to cause adverse health effects based upon the animal studies. When you look at the epidemiological data, that is when it starts to get a bit more confusing because the studies in the most highly exposed groups do not show all that much. There are some effects that are there, but these are in orders of magnitude higher levels of exposure than you get in the normal population. It is when you start to look at the normal population where the blood levels are much the same across the world, then you can start to pick up these small associations which may or may not have any biological or clinical significance and they may not be causative and they are certainly not consistent. So it is very difficult to draw any firm conclusions from the current epidemiological database.

The CHAIR — There is a lot of money being spent on confining and decontaminating. Do you think that is a waste of money if it is not causing any health problems, or can we not actually say for sure?

Prof. PRIESTLY — I really would not like to comment on the economic side of these sorts of things. I think it is fair to say that we always try to be cautious when we are dealing with the potential linkage between

human health effects and an environmental chemical, so we do use quite conservative methodology to assess potential risks and that tends to drive, if you like, a conservative approach to clean-up.

The CHAIR — But in a lot of other areas we are not taking the same conservative approach when it comes to environmental pollutants, I would not have thought.

Prof. PRIESTLY — I am not so sure that that is the case. I think where environment pollutants are fairly well studied we tend to use a similar sort of conservative approach to assessing the health risks and determining what level of clean-up is required. I do not see anything particularly different about the perfluoroalkyl compounds.

Mr McCURDY — But it is fair to say Australia seems to be dragging the chain a bit when it comes to PFOS levels in humans. It seems like we have standards for water and standards for soil, but we have not jumped on the bandwagon for humans. Is there a reason for that?

Prof. PRIESTLY — Let me try to think if I can answer that question. Guideline values for assessing human health tend to be a federal responsibility. I am not sure of the agencies that would necessarily be involved in doing that. I am aware that there are particular agencies that set guideline values for water quality, and these have been addressed in relation to PFOS. In relation to occupational health and safety there are other agencies that look at those particular aspects and often you find that the occupational safety levels are higher than for the normal population.

In terms of where we are in relation to other countries, the resources we have in this country to do the sort of work that is required are probably less. Although we take the lead in many cases from agencies like the United States EPA, where there have been quite extensive recent reviews of PFOS and PFOA, my understanding is that those reviews are currently going through a consultation process in the United States and they may well lead to a change in the sorts of levels that are being used as health guidance values.

The CHAIR — Higher or lower?

Prof. PRIESTLY — I suspect they will probably go lower, based on what I have read. But again, these are out for consultation at the moment so they are not confirmed.

Mr McCURDY — Can you just clarify what I thought you said before about retention of PFOS in humans? If fireman A and fireman B are both exposed to the same amounts of PFOS, one's body might retain it more than the other person?

Prof. PRIESTLY — Yes. It will depend to some extent on the length of time of exposure because continued exposure will cause these compounds to accumulate in blood. Because of the very long what we call half-life — the time it takes to get rid of half the body burden, which is in the order of four or five years for PFOS — it means that you keep on accumulating these things with continuous exposure reaching a steady state after maybe 15 to 20 years. It is a long, drawn-out process. We do not fully understand why it is that humans tend to accumulate a lot more than animals at the same dose levels. It is probably something to do with the way that they are reabsorbed in the kidney, because they tend to be eliminated in the kidney unchanged and then extensively reabsorbed back into the body.

Mr RICHARDSON — Thank you, Brian, for coming in today. Just a few questions about the standards and the Stockholm convention. This was brought to our attention in a transcript of Dr Roger Klein which stated:

> PFOS is classified under the Stockholm convention as very persistent, bioaccumulative and toxic.

Given those references, how does that accord with some of the Australian standards or standards that are applied by certain remediation agencies? Can I get your views on those findings or those references from the Stockholm convention?

Prof. PRIESTLY — Certainly I agree with the idea that these are bioaccumulative and persistent in the environment. From a toxicity point of view, toxicity depends upon the level of exposure and the intrinsic toxicity. We know a reasonable amount about that because of the animal studies. I do not believe these compounds are necessarily highly toxic but they are toxic and they are bioaccumulative and persistent, so that is enough to worry about. In terms of the Stockholm convention, I am aware that PFOS is on the schedule which

is intended to be phased out. My understanding is that some of the actions to phase out PFOS in Australia have started to occur. NICNAS, the industrial chemical regulator, has already suggested that some uses be withdrawn, but as far as I am aware — and perhaps I cannot be quoted on this — I do not believe Australia has actually ratified that action in the Stockholm convention as yet.

Mr RICHARDSON — Taking that point further, what is the nature of your work with CRC CARE? We heard from Professor Ravi Naidu today. What is your involvement with CRC CARE?

Prof. PRIESTLY — I have been involved in a number of the CRC CARE projects relating to guideline development for chemicals that they are interested in. PFOS and PFOA are a current project they are doing looking at emerging toxicants. I think the primary aim of CRC CARE is to work out ways of remediating the environment associated with those chemicals, but to do that you have to have some idea of what level of clean-up you need to get to. Part of the project, which I think is being done by consultants for CRC CARE, is to develop some of these clean-up guidance values based upon the toxicology, based upon the sort of health-based guidance values that have been established elsewhere. My role is really to be part of the project advisory group that reviews those particular recommendations.

Mr RICHARDSON — This morning we were given particular figures that they are striving for and examples where the clay-based technology is extracting PFOS. Where do those standards come from? What are those levels that they are trying to achieve based on? Why are they not similar standards that are being looked at across other jurisdictions?

Prof. PRIESTLY — I am not sure that I can answer that question, because that is not really part of the process that I have been involved in. My advice really has been more the interpretation of the toxicological data which then is used to derive those sorts of standards for soil and so on. There is quite a bit of assumption that one needs to make between determining an appropriate or safe level of exposure and how that might translate to what is actually in that environment and the pathways by which exposure may occur.

Mr RICHARDSON — In the absence of that information, given that whether it is emerging or whether the health impacts are still being assessed, particularly on humans, what steps should organisations be taking now to mitigate future risk or future potential harm?

Prof. PRIESTLY — Probably the best answer I can give you to that is to use the best available science at the time to try to guide that process. You cannot always predict where the science may go in future so I think you have to use the best available science at the time.

Mr RAMSAY — The question I really want to ask is a question that may well have to wait until later, so I am going to ask you to dance around your peer review of another well-known toxicologist the CFA used in relation to sampling. I am trying to get an understanding of the samples that were taken, which were conducted by the CFA, in relation to soil, water and human, which provided different levels of PFOS within the samples. The question I am asking is: is there a relationship between samples of water in relation to PFOS activity or levels and soil and human? Can you give me an idea of the interaction? I assume they are quite different, so if you get a high reading in water it does not automatically relate to the reading that you might have in humans; is that right?

Also, in an article you wrote in relation to toxicology you talked about it being under-recognised, and I am just wondering is there the expertise now in relation to dealing with impacts on health of PFOS contact sampling and the relationship between animals, water, soil and human health?

The CHAIR — Professor Priestly, just before you answer that, in asking you to come today it was in your capacity at Monash University. We do not want to put you on the spot. I think we all do have questions about the peer review, but it is our understanding that that may come after we have spoken to Dr Roger Drew and so on. I am just raising that — —

Mr RAMSAY — I was not asking him to comment on the peer review. I was asking him about the relationship — —

The CHAIR — I am trying to clarify that for Professor Priestly just so that he understands that.

Prof. PRIESTLY — I will try to give you a generic answer if I can.

The CHAIR — I am not trying to stop discussion, but I just feel that we have asked you to come on one basis and I wanted to make it clear that that was the basis on which you are here. Do not feel you are under pressure to answer.

Prof. PRIESTLY — As I say, I will try to give you a generic answer. When you are trying to assess the potential health impacts with a localised source of exposure, you take measurements of the chemical of concern in various environmental sources of exposure, and they could be water, they could be food. They are the most likely sources of exposure I think under those circumstances. With soil, you have to consider the pathways by which the intake could come from soil to humans. Humans, apart from maybe small children, do not eat a lot of dirt, so most of the pathways for exposure from soil to humans may involve estimates of leaching from the soil into water and then into another directly ingested material.

Yes, you have to take consideration of the analytical precision associated with those measurements. It needs to be from a good laboratory so that you can get reliable data, and then the general approach is to estimate what would be the likely exposure in milligram per kilogram per day or some other measurement which is adjusted for body weight in order to calculate what the blood levels may be in people and then to compare that with the blood levels or intakes in animals which might be associated with those adverse effects.

In the case of Fiskville, as far as I am aware, there were those measurements done in soil, there were measurements done in water, there were measurements done in fish and some off-site animal — sheep, I believe — so that there were a number of those sorts of input data available to do a risk assessment, and on top of that my understanding is that there were also some blood levels that were taken from people who had consumed certainly the fish anyway. This all helped to inform the risk assessments that were done for that particular scenario. Did it answer your question or not?

Mr RAMSAY — Yes. It might be a question that I will ask at another time. Can you just tell me why we do not have an Australian standard in relation to PFOS levels for safety in human health? Why are we having to refer to a quasi-international standard?

Prof. PRIESTLY — If you are talking about standards in food, it is really the responsibility of FSANZ, Food Standards Australia New Zealand, to set those levels. I am not aware as to whether or not they are doing that work or whether they are waiting for some clarity from overseas work. I just do not know. In relation to water quality standards, my understanding is yes, there have been some standards done for Australia. In terms of health guideline values such as a tolerable daily intake, we have not, as far as I am aware, set one of those. We tend to adopt standards that have been set overseas, using the extensive database that has been used overseas. But as to why it has not been done by some responsible Australian authority I really cannot answer for that authority.

Ms WARD — We know that in the 1990s manufacturers became aware that there could be issues with PFCs in the environment, and we know that over the last probably seven to eight years literature in this particular area has become quite large. It has got bigger and bigger and bigger. Do you see a narrowing in the way that people are writing about PFOS and PFOA in terms of how they affect human health? Does the science seem to be getting wider or is it narrowing in terms of what they are finding?

Prof. PRIESTLY — Certainly the number of studies is getting much larger and the range of health effects which have been examined is also getting broader. When I first reviewed the literature five years ago there was a relatively narrow range of health effects that were being examined in the epidemiological studies. That has now expanded quite substantially, and with some of the health effects there are anything up to five or six or more epidemiological studies on the same health outcome. There has been a lot more work, particularly in the last five years. I find that if I go back into the literature almost every week there is something new coming out, so it is very difficult to keep up to date with exactly where the front is moving.

Ms WARD — Are we finding consistencies with the findings with all of this literature that is coming through?

Prof. PRIESTLY — I think I made that point in my original statement — that no, there is not a lot of consistency.

Ms WARD — Even in terms of immunity levels in children, because there does seem to be quite a lot of literature around this area? There is a Danish study, there is a whole variety of work that does support the idea that there is decreased immunity in children with high incidence of PFOS in the blood.

Prof. PRIESTLY — Yes, that is true. But when you look closely at the studies you find that the associations are not always with the same PFC. They are not always with the same type of vaccine. For example, I have seen studies where the effects were limited to one vaccine and not to another. The other point that I make is that when you are looking at multiple types of chemicals that are studied in these studies and some of them appear to have an association but others do not, it is very difficult to tease out whether the associations you are seeing are actually really related to that particular chemical or whether they are confounded by other chemicals. The researchers tend to have to use very sophisticated statistical techniques to try to tease out what are the contributions of each of the chemicals that have been measured. As I pointed out, one of the groups in particular that has studied this phenomenon of resistance to vaccines has also studied a number of other persistent organic pollutant compounds and found similar findings. So what you tend to find is where there are higher levels of some of the persistent pollutants, you find them for other compounds as well.

Ms WARD — So you would be of the view that no matter what the PFOS level in your blood was, whether it was 2, 20, 200, that you would not be concerned about those levels in your body?

Prof. PRIESTLY — No, I did not say that. At the moment the evidence is suggesting that the current levels of exposure, even for some of the occupationally exposed people, which are orders of magnitude higher, do not appear to be clearly associated with any well-defined adverse health effects. I think that is what is coming out at the moment.

Ms WARD — So there is a consistency then in the literature that is coming out, which is that there are not definitive health effects? That is consistent?

Prof. PRIESTLY — No, I am not saying that necessarily. What I am saying is that the situation is confused and inconsistent at the moment. There is no clear association. There are studies which have demonstrated weak associations for some of the PFCs but not others, and so I do not think it is appropriate to draw conclusions at this stage.

Mr RICHARDSON — Just following up on the point that Vicki made, in your view do you think that the use of PFOS and PFOA should have been discontinued in Australia?

Prof. PRIESTLY — I think it is reasonable given their biopersistence and bioaccumulation. We tend to be a little more wary with compounds that have that sort of property, because we know they will tend to occur throughout the population. They will occur perhaps at very low levels, but over time if we continue to use them they will probably accumulate to higher levels. Particularly with the PFCs, when we started to recognise that they were biopersistent and so on we did not have a great deal of information about the toxicology of the compounds. That information was accumulated through, I think, the 1990s, the early 2000s and with the epidemiological studies it has been more in the last decade, perhaps the last five years in particular. I think it was appropriate to take precautionary approaches to these compounds in the early 2000s at a time when we may not have known as much about them as we know now.

Mr RICHARDSON — From a human health risk assessment perceptive what level would be satisfactory before precautionary measures are taken from a risk management assessment point of view, or what threshold do you have to reach before that is the case? How many peer-reviewed assessments need to take place before a more precautionary approach is taken with regard to human health?

Prof. PRIESTLY — I am not sure that I can answer that question directly, really. The quality of the data and the quality of the studies is important. The severity of the health effect is important. All of those need to be balanced before you take a decision. But as I said earlier on, you take decisions based upon the best available science at the time. Sometimes we are a little more precautionary than at others in regard to that, but we try to be as precautionary as we can as the data emerges.

Mr RICHARDSON — So in a position where we have the Stockholm convention putting forward some great concerns about that, have they erred in going that far, given that at this stage you have referenced

bioaccumulation but not the toxicology side? Have they erred in putting PFOS and PFOA forward as a high-risk category?

Prof. PRIESTLY — I do not think I would be so brave as to criticise the Stockholm convention. I think they have looked at it pretty carefully and taken a fairly precautionary view of it. My understanding is that most countries are looking to phase out these chemicals because of those particular properties. The different stages of where they are at may depend upon local situations, but I certainly would not criticise the Stockholm convention for taking that action.

Mr RICHARDSON — So where does it fall down then? Is it bioaccumulative or is it the toxicology element of it?

Prof. PRIESTLY — The fact that you can bioaccumulate and these things are very persistent means that it is appropriate to take measures to limit exposures. It may take a long while before you reach a toxic level, but I think the more appropriate way is to start to take action before you get to that level of intake.

The CHAIR — It seems there are a lot of countries that are banning the use of the product, its manufacture and so on, and, as I was saying before, it seems that has not been the case in other things. I suppose what comes to mind is asbestos-related products. They still have not been banned by a lot of countries and we know there is a clear link between asbestos and terminal disease. I cannot quite understand why there seems to be more action around PFOSs and firefighting foams than, say, asbestos and yet there is no conclusive information that they really do have any adverse health effects. It just does not make any sense.

Prof. PRIESTLY — I guess the issue you are talking about is the question of whether a ban on use is the most appropriate management technique. Sometimes you can get quite effective reductions in exposure through things other than bans. You can restrict usage, and my understanding is that that is essentially the approach being taken by NICNAS, the industrial chemical regulator, which said, 'We should restrict this type of usage to minimise exposure'.

Mr RAMSAY — Some of the active ingredients of PFOS we use regularly in our daily lives; is that not true? There are many products that have an active constituent within their food chain that we use regularly — for example, toothpaste. The absorption of some of the active ingredients happens on a daily basis through the food we eat. I am trying to put it into perspective. We all have some PFOS in us to some degree or level. It gets to a point where we are unclear about where the danger levels are or where the health impact of those levels kick in. There is talk about banning an active ingredient of PFOS, but given we all have some and we all use it daily, it is probably unlikely. Can you put it into perspective?

Prof. PRIESTLY — Certainly most of the uses of PFOS and PFOA have been phased out as far as I am aware. There are some of the other compounds which are telomers which are less fluorinated, shorter chain, branch chain compounds, which are still in use because they have useful properties. They tend to be less biopersistent than the longer chain compounds. We are certainly monitoring the presence of those compounds in blood serum and we are finding them fairly ubiquitously, as we do with PFOS. They tend to be at much lower levels because the use patterns have been different over the years and they are less bioaccumulative. But it is also fair to say that we probably have less information about the toxicology of some of these alternative surfactants. That is something that will probably be a gap that will have to be filled in coming years.

The CHAIR — Thank you so much for coming in and answering all our questions.

Prof. PRIESTLY — I will tidy up and get out of your hair.

The CHAIR — It has been very helpful. That concludes the public hearings for today.

Witness withdrew.

Proceedings in camera follow.