Minutes of public meeting of the PFAS Scientific Advisory Panel on Teams
3pm – 5pm on Thursday 8th February 2024

Panel Members present:	Dr Steve Hajioff – Independent Chair Dr Tony Fletcher – PFAS and Health member Prof Ian Cousins – PFAS and Environment member
Subject Matter Experts present:	Dr Sue Fenton – Director, Center for Human Health and the Environment, North Carolina State University Dr Christel Nielsen – Occupational & Environmental Medicine, Lund University, Sweden
In attendance:	Grace Norman – Deputy Director of Public Health Sarah Tyler – Senior Policy Officer Anita De La Cour – Executive Assistant (minutes)

Welcome

The Chair welcomed everyone and briefly outlined the running order of the meeting as per the agenda.

Introductions

Steve Hajioff is the Independent Panel Chair, with a background as a physician and a public health expert, in health economics, and a retired Director of Public Health in an area with two major international airports and a variety of other environmental hazards. Steve is not a PFAS expert.

Tony Fletcher is the health Panel Member. An Environmental Epidemiologist from London School of Hygiene and Tropical Medicine.

Ian Cousins is the environment Panel Member. A Professor in Environmental chemistry at Stockholm University, an expert on PFAS, whose expertise is on the sources, transport, fate, and human exposure of PFAS.

The Chair mentioned that the meeting is also joined by Grace Norman, Deputy Director of Public Health, who commissioned this work and is a standing observer.

Support staff, for programme management and administration were also in attendance.

The Chair reminded people that the meeting is being recorded and a copy of the recording can be requested by emailing publichealth@gov.je.

Declarations of Interest

None.

Minutes of the last meetings

The minutes of the November meeting are not yet available, as final sign off was needed by those experts by experience who attending that meeting. It was anticipated that they would be available for agreement and sign off at the next meeting on the 4th of March.

The minutes of the meeting of 18th January 2024 were also not yet available due to the wide ranging and detailed presentations by subject matter experts needing to be captured, which was taking longer than normal to complete.

The minutes of the meeting of 7th December 2023 were agreed, subject to adding in a post meeting point of clarification as follows:

- On page 3 of the minutes: To clarify that in areas where there is not a direct source of PFAS contamination, then foods like meat fish and eggs are important for PFAS levels, but where there is a source of contamination from water, then this is the primary factor for exposure

- There are variances between types of PFAS. For example, PFOS in the general population (not specific exposed populations) then PFOS in fish is an important factor

There were no other matters arising.

Additional findings from the last meeting

No additional findings.

Subject Matter Experts

The Chair asked the subject matter experts to introduce themselves to the meeting, after which they would each present to the Panel.

Dr Sue Fenton is a reproductive endocrinologist, having worked for 11 years in the USEPA (*United States Environmental Protection Agency)* specifically focused on PFAS and other environmental contaminants and their health effects. Dr Fenton then moved to the National Institute of Environmental Health Sciences for 14 years and expanded her work specifically looking at development and targeting the effects of chemicals such as PFAS. She is now the Director of the Center for Human Health and the Environment at North Carolina State University.

Dr Christel Nielsen is an environmental epidemiologist from Lund University in Sweden. Dr Nielsen works on health effects in children and pregnant women in Ronneby, Sweden, which has PFAS contamination which also had exposure from firefighting foam.

Dr Fenton's presentation

Dr Fenton gave a presentation entitled "PFAS health effects in a mouse model: Early life exposure and later life effects". The work has looked at 'known' or 'legacy' PFAS and comparing them to new PFAS. Mouse models indicating health effects were used to show direct causations to PFAS exposure.

Dr Fenton outlined that PFAS is detected across the USA, with the northeastern areas having the biggest concentration. The presentation focused on PFAS exposure in North Carolina State coming from industrial sites (including a fluorochemical manufacturer, producing a replacement for PFOA, called GenX), an Airforce base, fire training sites and landfill, which were all across the Cape Fear River basin. It was noted that there are also popular tourist areas in this vicinity.

PFAS was discovered in the water about ten years ago, originally there were permits allowing the discharge of waste into the water and then these were revoked, however PFAS by-products had been discharged into the water for about 40 years. It is now known that 650 personal wells in the area are contaminated with 'novel' (*newly researched compounds)* PFAS.

There are many PFAS exposure pathways in this area (old and new), including:

Ingestion, inhalation, dermal (skin) via:

- industrial sites (point sources)
- fire training / fire-fighting facilities
- landfill
- wastewater treatment plants/biosolids
- consumer products/dust
- food items (e.g., fish/shellfish)
- food packaging

<u>The effects of PFAS on human health</u>
There are health effects associated with PFAS exposure (seen in both animal and human studies), which may include:

- Immune function changes
- Thyroid function/disease
- Liver disease and cancer
- Metabolic dysfunction
- Kidney disease and cancer
- Reproductive and developmental outcomes (a key focus of Dr Fenton's work)
- There is some evidence from several papers on delays or a shortened duration of lactation due to PFAS exposure (and potentially other life factors)

There was a 2023 PFAS Report delivered to Congress, which Dr Fenton contributed to as a subject matter expert, which was a high-level summary focusing of the following areas:

- Removal, safe destruction, or degradation of PFAS from the Environment
- Safer and more environmentally friendly PFAS alternatives
- Sources of PFAS and pathways of human exposure
- Understanding the toxicity of PFAS to humans and animals:
    1. Epidemiology evidence *(a particular exposure causes a particular harm)*
    2. Laboratory animal models *(e.g. mice, rats)*
    3. Ecotoxicology studies *(substances harmful to the environment)*

The report also listed gaps in knowledge of PFAS health effects, which included obesity and metabolic disease, and the assessment of the placenta or pregnancy complications. It was hoped that this focus would enable direct funding into the understanding of the health effects of PFAS, as this is not currently widely understood.

There has been a lot of focus on non-alcoholic fatty liver disease, which cases are rising globally, and has been linked by some to PFAS exposure in both humans and animal models. This is relevant to Dr Fenton's work as liver disease is on the rise in the United States.

A comparison study between PFOA and its replacement GenX was conducted exposing mice, where the foetuses and subsequently the offspring were studied. PFAS was looked at in the blood, in livers, foetuses, and the placenta. The aim of the comparison between PFOA and GenX was to see if GenX was a safe replacement for PFOA, and this study showed that it may not be.

The results of this study showed that exposure to PFOA and GenX was associated with placental lesions, increased maternal weight gain, an increased embryo to placenta ratio, and a constellation of adverse liver features. These occurred in both the mother *(mouse)* and the liver of the foetus, which suggested that what was happening in the mother was also happening in the offspring.

In the offspring, there were sex specific liver effects, and metabolic disease in both males and females. The female mice had worse liver disease, the males became obese, had glucose intolerance and insulin resistance.

The conclusion from the study is that PFOA and GenX are very similar, with almost identical effects, even though GenX has a much shorter half-life than PFOA. *(Half-life is the time it takes for the concentration of a substance in the body or in the environment to reduce to half its initial value).* The GenX half-life is about one day in a mouse compared with a 3-week half-life in a mouse for PFAS.

Dr Fenton explained that there are numerous other PFAS in our environment, but there is no health data for these. In future, it is hoped to be able to predict pathways for new emerging PFAS, to analyse their effects, including adverse outcome pathways for foetal growth restriction (FDR).

Dr Fenton outlined that the full extent of the work cannot be covered in this short presentation, and in summary in utero PFAS exposure sets the stage for a lifetime of increased disease susceptibility, for example this could include:

- Transplacental transfer- PFAS has been found to cross into the placenta and in mouse models found lower birth weight
- Hypertensive disorders of pregnancy
- Low birth weight
- Reduced immune function
- Disruptions in timing of puberty
- Menstrual issues and reduced fertility
- Elevated cholesterol
- Reduced kidney function
- Thyroid hormone disruption
- Cancer

Dr Fenton felt that protecting pregnant women and babies from PFAS effects was important. Finally, Dr Fenton talked about PFAS in AFFF (Aqueous Film Forming Foam) which is found in firefighting foam and concluded that there is not a lot of health data on AFFF. It is difficult to know about changes in formulas from the manufacturer unless they make it known or it is discovered via research. In the future Dr Fenton will be looking at genomic signatures and assays to help understand these novel PFAS components quickly, which is important for understanding health impacts.

The Chair thanked Dr Fenton for her fascinating presentation.

The Chair asked for clarification as to whether socio-economic factors were considered with the lactation studies, i.e. mothers from poorer areas are less likely to breastfeed, and more likely to live in areas where exposure is higher (e.g. in industrial areas) which could be a source of confounding. Dr Fenton confirmed these factors were controlled for in the analysis and that socio-economic factors have long been associated with PFAS levels.

Dr Fenton was asked about the fact that that GenX is considered to be a more toxic substance (given its half-life was much shorter than PFOA) yet the results showed same effect in the lab mice.

She confirmed that appeared to be the case, and other studies had also shown that lower doses of GenX shows same effects in mice too. GenX appears to be causing metabolic disease.

To clarify for members of the public listening, no GenX had been found in Jersey.

A further discussion followed about testing, what has been tested and what is available to test. There are thousands of mixtures in different PFAS substances, for example AFFF is a complex mixture of substances. This can present challenges in testing as it is difficult to know what to test for.

Dr Christel Nielsen's presentation
Dr Nielsen gave a presentation entitled "High exposure to PFAS in Ronneby, Sweden – effects on child health and development".

Dr Nielson gave a brief background summary, notably her interest in public health following PFAS exposure in Ronneby, Sweden. Ronneby is a typical Swedish small town, with a population of 28,000 people, with mostly municipal drinking water from two water plants. Sweden has a strong tradition of drinking tap water, which, in this case, was an important source of exposure.

PFAS contamination was discovered by chance in December 2013 and action was taken as a result of:

- Money was left in the budget for regional environmental monitoring
- There was an awareness of the upcoming EU drinking water directive at the time
- There was new knowledge of PFAS contamination around the airfields

Extended sampling of water was undertaken in Bredåkradeltat, one of Sweden's largest water reservoirs, located in the Kallinge area of Ronneby. High levels of PFAS were found in this reservoir. A municipal water treatment plant was located in the exposure area, and outgoing drinking water was analysed. The results showed the sum PFAS in the contaminated waterworks was 10,380 ng/L (nanograms per litre), compared with Kärragården (47.6ng/L), the other waterworks, and <5ng/L in the neighbouring municipality Karlshamn, which was used as a reference point. The exposure profile was driven by AFFF (Aqueous Film Forming Foam) contamination. The contaminated waterworks was immediately closed.

In terms of the population exposure, the Brantafors reservoir served a third of the households with water in 2013. The start of the exposure is unknown (there were no saved or banked samples) however military purchase records from the mid-1980s suggested that exposure had taken place for over 30 years.

In 2014, after the water source was closed, blood testing was offered to the local population for PFAS:

- Open samplings at around 20 locations
- Tests were free of charge
- The participation rate was 13% of the population
- This showed elevated serum concentrations in everyone tested in Ronneby (including those not living at exposed addresses)
- Other contaminants measured were not elevated

The 2014 results showed blood serum concentrations were much higher in Ronneby than in the reference population; the PFHxS median was 280ng/mL (nanograms per litre), and PFOS 303ng/mL, compared with the median levels of 0.5ng/mL for PFHxS and 2.9ng/mL for PFOS in the reference population.

In 2013, there was no information about how PFAS impacted human health in terms of comparable data for effects of AFFF, so the Ronneby PFAS research Program was formed. This is a joint venture between Lund and Gothenburg Universities, which Panel member Tony Fletcher is a part of. Together they are looking at health impacts of PFAS in the population.

From this work, 6 areas for research needs in children were identified: pregnancy health; birth outcomes; neuro development; cardio-metabolic health; immunologic effects, and puberty.

In Ronneby, they enrolled mothers during early pregnancy to take part in a study. All pregnant women were invited. Follow up was until the child was one year old. The study involved taking biological samples and completing questionnaires. Pregnancy health and PFAS transfer during pregnancy was explored. This showed that, at birth, an infant can have 30-60% of the mother's level of PFOS. Breast milk concentrations and blood concentrations were also reviewed. The study showed a lower transference through breastmilk. Further studies are underway to find out more.

The changes in breastmilk concentrations over time was researched and in conclusion:

- Transfer efficiencies are not affected by the maternal exposure level
- Transfer during pregnancy is more efficient than the transfer into breastmilk
- Breastfeeding is an exposure source for the infant but is also associated with several health benefits
- If/when possible, female PFAS serum levels should be reduced before pregnancy
- There is a further need to understand how early-life exposure affects child health
- Previously there were no studies regarding high AFFF exposure

Sweden has a comprehensive register of population data which supports PFAS studies. The data collected in Sweden was also outlined and they have a long history of keeping various registers which are useful for these studies, including population characteristics, healthcare data and socio-economic data. There is mandatory participation in this data collection and personal identification numbers are used. For example, they could look at the national birth register and see who had background exposure to PFAS and pregnancy complications and birthweight in the county.

In terms of pregnancy complications, the studies concluded there was no increased risk. For birth weight, there was an effect, but the magnitude was small and different in girls and boys.
It should be noted that the data used was before 2013 so breastfeeding decisions were not based on knowledge of PFAS exposure. It was suggested that breastfeeding mothers had a 3 times higher risk of not establishing breastfeeding than in the non-exposed population. Also, more mothers were not breast feeding at 6 months, which may be indicative of delayed mammary gland development in exposed mothers. Dr Nielsen commented that what we can conclude today, is there is not sufficient evidence to recommend that highly exposed mothers refrain from breastfeeding, given the other benefits breast milk and breastfeeding has for infant health.

The final study Dr Nielsen shared was in relation to neuro development, and developmental language disorder. This research included all pregnancies in the county between 1998-2013 and conditions diagnosed by speech and language pathologist after screening by Child Health Services. Hence, the data is based on clinical diagnosis. The results showed that girls from the highly exposed area had a 1.6 higher risk of being diagnosed with a developmental language disorder *(no effect in boys).* Therefore, Dr Nielsen concluded that there may be an effect on neurodevelopment that warrants further investigation.

Overall, her concluding remarks on health effects in the Ronneby population:

- No increased risk of pregnancy complications
- A small and sex-specific effect on birth weight
- Impaired breastfeeding ability was seen
- Increased risk of developmental language disorder in girls (more research needed here)

- Adverse effects on breastfeeding and language development can be intervened on
- A many times higher exposure did not cause many times higher risks (however more research is needed)

Dr Nielsen concluded by letting the meeting know that an English summary of the Ronneby research is published at https://pfas.blogg.lu.se.

The Chair thanked Dr Nielsen for a fascinating presentation.

Questions and discussion

The Chair commented that he had previously worked as part of an Infant Mortality Taskforce in England, which looks at deaths of babies in first year of life, and he wanted to stress that one of the most important factors in first year of life is breastfeeding, and whilst there is some exposure to PFAS by breastfeeding, there is no strong evidence of breastfeeding causing harm. Breastfeeding is extremely important for babies' health and wellbeing. The Chair asked people not to over interpret the studies or change their behaviours with regards to breastfeeding. Dr Fenton echoed this, commenting that breastfeeding gives children the best start in life.

A general discussion then took place, the summary points to note are:

- It is difficult to collect breastfeeding data in terms of volumes of milk consumed (in Sweden they do have biomonitoring data for 1-year olds)
- Studies are ongoing as to whether lower breastfeeding rates could be associated with PFAS exposure, possibly through delayed lactation
- In Sweden, 98% of mothers breastfeed so there are only a small number of mothers who do not breastfeed, meaning that there is greater statistical uncertainty (as displayed by confidence intervals) around non-breastfeeding
- Studies on breastfeeding pathways show higher levels of PFAS intake and levels peak at about 20 months, this maybe when breastfeeding stops
- Transfer during pregnancy is more efficient than transfer into breast milk
- There is not sufficient evidence to suggest that the general health benefits of breastfeeding are significantly offset by PFAS, so it is important to continue to promote breastfeeding because of its additional benefits
- Study design and dose response are important factors in the research and its conclusions
- Ronneby did have a number of immigrants (who don't always have a personal identification number in Sweden yet), however, there is confidence that the majority of people had a personal identification number and therefore have contributed to the evidence
- Thyroid effects of PFAS need further study as this appears important and could be affecting some of the other health areas discussed above
- It is important to understand the threshold effect and dose response in PFAS as there appears to be a saturation point, so it is not necessarily the case that the higher the PFAS exposure leads to a greater health concern, which would normally be assumed to be the case in general toxicology
- There could be differences in the metabolism of peoples or animals, as well as differences in population exposure, which may affect the measurements and data. Some populations therefore would be more vulnerable to internal confounding *(i.e. mixing of effects, which can distort the data)*
- There is a role for further animal studies to explore some of these issues. It is likely that no animal group will have a zero PFAS exposure (which would be used for comparison) due to the presence of PFAS in water
- By looking at gene data a very low level of PFAS can be identified and this has been used in animal models and this can be compared with the serum levels found in human populations

- It could be that wider endocrine disrupting hormones could lead to health effects other than thyroid as there is some evidence of this (thyroid information is being collected from the pregnant women in the Swedish study, so this can be explored further in their research)
- Research showed a glycogen deficit in the placentas of animals exposed to novel PFAS (and their metabolism affected at birth). In animals, if their glycogen deficit is large enough, they cannot survive. This is also important in humans also as it could have implications for insulin resistance, for example. It was noted that it can be difficult to collect this data in humans

In conclusion, the Chair stressed the need to be careful about interpreting numbers, measurements and exposure information quoted in today's presentations and using them out of context. For example, animals have a half-life of about 3 weeks for PFAS and in humans, by comparison, the half-lives are many years, so there are meaningful differences between humans and animals. There has been some PFAS research on larger animals such as sheep and horses to compare exposure, although there are differences such as their food consumption and exposure (noting that animal studies are different to human studies when looking at the effects of PFAS).

The Chair thanked Dr Fenton and Dr Nielsen for their informative presentations, which will be useful for the Panel's report.

Any other business
None.

Date of next meeting
4th March 2024 at 10.00 am.

Thank you and close
The Chair thanked the Panel, the expert guests, and the supporting staff.

A reminder was given that the meeting recording is available on request by emailing publichealth@gov.je.

There being no further business, the meeting was closed.

To note that the Panel can be emailed via PFASpanel@gov.je.

Details of meeting dates and times can be found at PFAS in Jersey (gov.je)