UNIVERSITY OF BIRMINGHAM

Sources and Pathways of Human Exposure to PFAS

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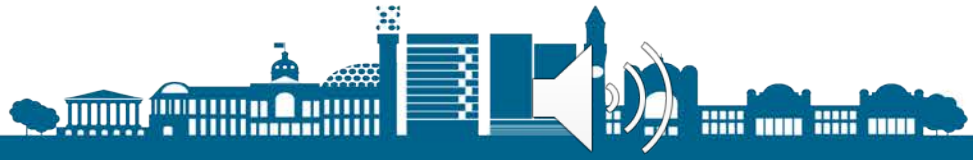
What do we know?

- Biomonitoring data across Europe shows us that humans are exposed!
- Question is how?
- This presentation will use UK (and Irish) data to illustrate state of knowledge and highlight areas for future study
- This presentation addresses non-occupational exposure only

What can we learn from other POPs?

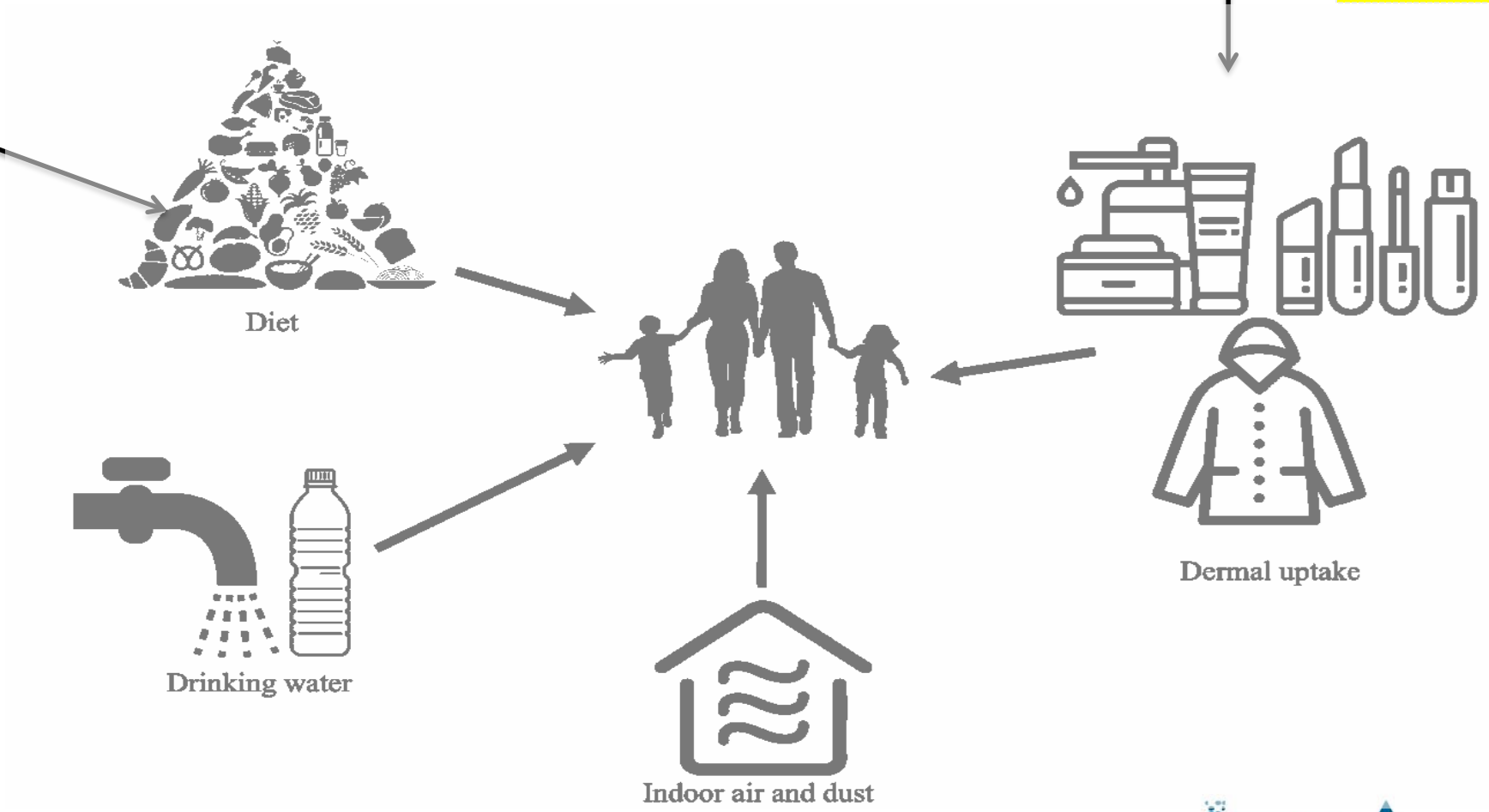
- Dioxins suggest diet – as a result of food chain accumulation (far-field exposure)
- Early 21st century saw recognition of indoor (near-field) exposures for POPs with substantial indoor uses;
- Indoor air for less involatile POPs like PCBs
- Unintentional ingestion of indoor dust for higher Mw POPs like BFRs
- For POPs with substantial use in items with direct skin contact, then dermal exposure could be important – e.g. PBDEs and HBCDD in furniture fabrics



Are these plausible for PFAS?

- Dioxins suggest diet – as a result of food chain accumulation (far-field exposure) **YES**
- Early 21st century saw recognition of indoor (near-field) exposures for POPs with substantial indoor uses;
- Indoor air for less involatile POPs like PCBs - **YES**
- Ingestion of indoor dust for higher Mw POPs like BFRs - **YES**
- For POPs with substantial use in items with direct skin contact, then dermal exposure could be important – e.g. PBDEs and HBCDD in furniture fabrics – **YES**
- IN ADDITION… chemistry of PFAS means **drinking water** could be important
- **AND…PFAS use in food contact materials**, provides another **dietary pathway**

PATHWAYS OF EXPOSURE TO PFAS



What's the evidence for PFAS?

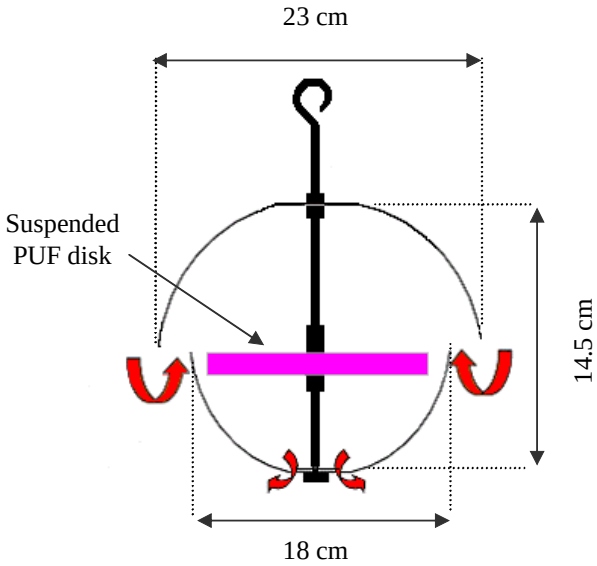
Diet

- Most recent evidence for the UK is that of Junque et al (2025)
- This measured PFAS (including the $EFSA_4$) in 102 UK retail fish samples (also in Spanish retail fish n=50))
- **Typical UK adult exposure from fish consumption calculated at 10.7 ng/kg bw/week**
- This exceeded by fish consumption in Spain (24.6 ng/kg bw/day). The difference largely due to PFNA for which concentrations were 35 x higher in Spain

What's the evidence for PFAS?

Air

- Most complete evidence for the UK is that of Goosey & Harrad (2012)
- This measured PFAS (including PFOS, PFOA, PFHxS but NOT PFNA) in home (n=20), office (n=12), and outdoor air samples (n=10)
- **Typical UK adult exposure from air inhalation calculated at 0.21 ng/kg bw/week**
- High end exposure = 0.70 ng/kg bw/week



What's the evidence for PFAS?

Indoor Dust

- Most complete evidence for the UK is that of Goosey & Harrad (2011)
- This measured PFAS (including PFOS, PFOA, PFHxS but NOT PFNA) in home (n=45), office (n=20), classroom (n=42), and car samples (n=20)
- **Typical UK adult exposure from indoor dust ingestion calculated at 1.2 ng/kg bw/week**
- High end exposure for adults = 17.4 ng/kg bw/week
- **Typical UK toddler exposure from indoor dust ingestion calculated at 13.7 ng/kg bw/week**
- High end exposure for toddlers = 322 ng/kg bw/week

What's the evidence for PFAS?

Drinking water

- Most complete evidence for the UK is that of Gao et al (2024)
- This measured PFAS (including $EFSA_4$) in tap (n=41) and bottled water (n=29)
- **Typical UK adult exposure from drinking water calculated at 0.30 ng/kg bw/week**
- Bottled water less contaminated than tap water in this study

What's the evidence for PFAS?

Human milk

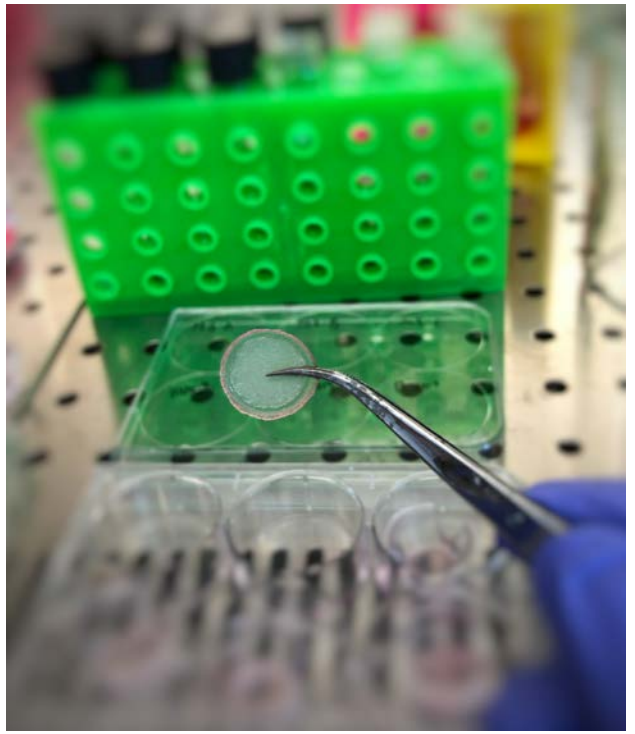
- Unaware of evidence from the UK, so use data from Ireland (Abdallah et al, 2020)
- This measured PFAS (including $EFSA_4$) in 16 pooled samples of human milk consisting of samples collected from 92 individuals from Galway and Dublin in 2018-19
- **Typical exposure of a 1 month old infant via breast feeding calculated at 182 ng/kg bw/week**
- **High end (95th %ile) exposure of a 1 month old infant via breast feeding calculated at 700 ng/kg bw/week**

What's the evidence for PFAS?

Dermal

- Ragnarsdottir et al, 2024 found the following when 3D-HSE models exposed to 500 ng/cm^2 of individual PFAS

PFAS	Absorbed %	Skin Reservoir
PFBS	48.7	19.6
PFHxS	21.9	33.2
PFHpS	6.4	44.3
PFOS	1.3	60.3
PFNA	0.9	36.9



- The importance of the skin reservoir illustrated by a single human subject where $^{13}C_4$-PFOA added to sunscreen applied to skin yielded maximum serum level of 132 ng/L 22 days after exposure (Abraham and Monien, 2022)

Summary

- For the $EFSA_4$, UK adult exposure typically predominantly from diet, likely 10X more than dust ingestion, followed by drinking water and air inhalation
- For infants, breast feeding is a major exposure pathway. For toddlers, indoor dust likely exceeds diet as a pathway, with drinking water and air inhalation minor contributors
- These findings will differ to varying degrees for other PFAS, depending on use patterns and physicochemical properties – for example, combined inhalation exposure to the more volatile Me and EtFOSEs is an order of magnitude greater than for the $EFSA_4$

Future Research Needs

- Definitive assessment of dermal exposure is required. 3D-HSE *in vitro* model data show dermal uptake feasible. This, coupled with the reported presence of various PFAS in cosmetics and clothing, means such assessment is an urgent priority
- This presentation based on data from two countries derived from studies covering ~15 years. For a complete picture, temporally and spatially consistent exposure assessments are needed
- And…in Ireland, concentrations of PFOA (242 cf 63 pg/m^3), PFNA (5.7 cf 1.5 pg/m^3), & MeFOSE (6.9 cf 1.3 pg/m^3) were significantly higher in air of cars containing child seats (n=12) than those that did not (n=17)

Future Research Needs

- Contribution of precursors to PFAA body burdens
- Both PFOS and PFOA are highly stable residues
- Major research gap currently concerns the proportion of PFOS & PFOA body burdens arising from the *in vivo* metabolism of precursor compounds (e.g. FOSEs & FOSAs)
- Under a high-end exposure scenario, 60-80% of PFOS body burden forecast to arise from precursors
- Risk assessments to date have assumed an arbitrary figure (e.g. 100%) for precursor conversion
- Difficulty is in distinguishing between PFAAs that arise due to external exposure and that arising from metabolism of precursors

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