Pfacts vs Pfear on PFAS – Separating Truth from Fiction

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Biosolids and PFAS

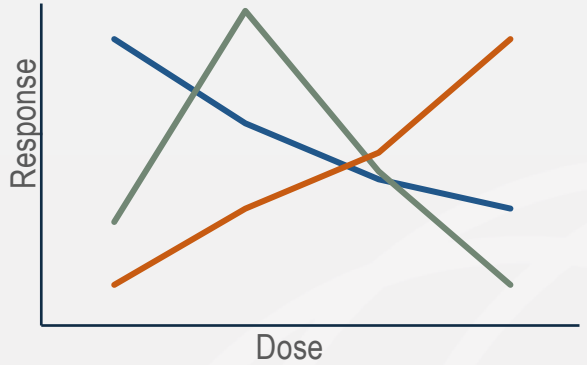
- Data generated from various geographies indicate ppb or ug/kg levels of PFAS in biosolids
  - Like wastewater concentrations, varies depending on input sources and density/location of treatment plant
- Limited field studies and data also indicate that some leaching from applied biosolids to underlying groundwater occurs at ppt or ng/L levels
- Additional uncertainty regarding whether leachate can or has impacted drinking water
- Some PFAS (e.g., PFOS and shorter chain) can accumulate and magnify up the food chain, such as if impacted fertilizer used on cattle grazing fields.
- Limited data collected thus far does not appear to indicate significant bioaccumulation or risk for plants consumed by people (Minnesota study).

Why the Focus?

- Persistent Organic Pollutant
  - Annex B – restriction of PFOS (2009)
  - Proposed: PFOA, PFHxS
- Toxicity Studies
  - Developmental, immune effects
  - Liver/kidney
  - Increased cholesterol, hypertension, thyroid
  - Cancers - liver, testicular, pancreatic, kidney
- Prevalence and persistence in environment and in humans
- Few standards/guidelines available and lots of uncertainty

Toxicity – What do we know thus far?

- Vast majority of studies focused on a limited number of chemicals, primarily PFOA and PFOS, and to a lesser extent, PFNA, PFHxS and PFDeA
- Typically see mixtures vs. single constituent
- "Dose" - mg/kg/day - amount of chemical (mg) taken into receptor per weight (kg) per day via exposure route (largely, oral)



- Drinking water exposure dose >>> soil ingestion/contact/inhalation dose
- Inconsistent correlation between dose and adverse outcome (response) – can be non-linear

Toxicity – What do we know thus far?

- Animal studies in rats and mice were predominant data source for identification of endpoints and dose-response relationships
  - Wide variety of outcomes/effects observed – focus on primary
  - For chronic exposures:
    - Reproductive
    - Endocrine
    - Liver (hepatic)
    - Tumors (liver, pancreas, testicular)

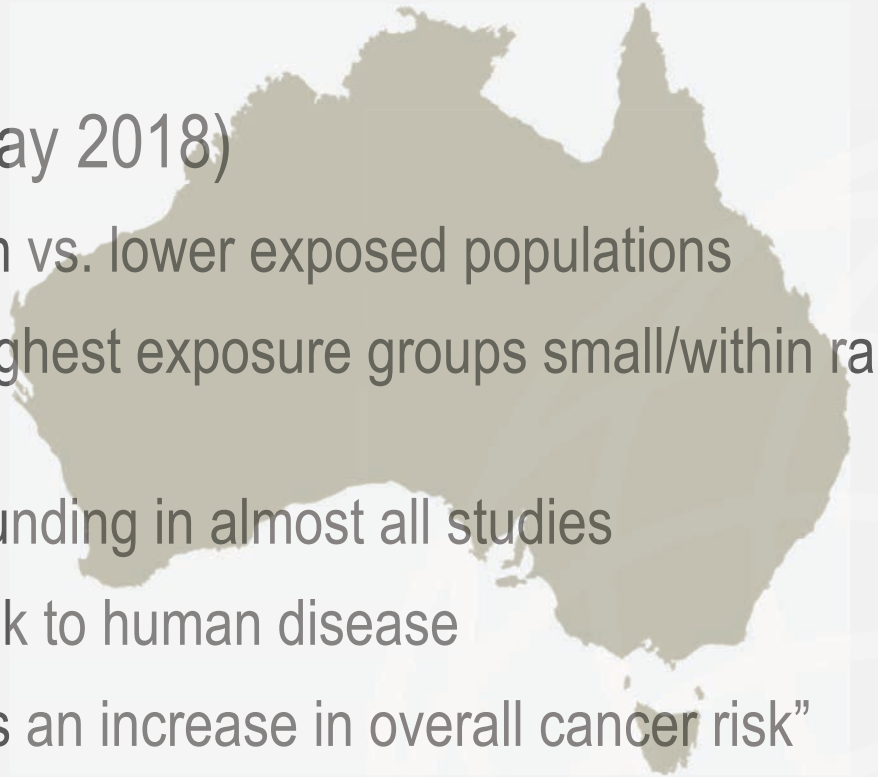
  - BUT, evidence of carcinogenicity is inconsistent or inconclusive
  - Doses in lab studies were often 10,000-1,000,000 higher than expected human environmental doses AND uncertainty factors applied to extrapolate to humans

Toxicity – What do we know thus far?

- Epidemiological studies generally did not have monitoring data but "suggest associations" for some non-cancer effects including:
  - Liver damage (increase in enzymes/decreases in bilirubin levels)
  - Endocrine effects
    - Thyroid/brain neurodevelopment
    - Obesity/diabetes/cholesterol (serum lipid)
  - Reproductive
  - Immune
  - Developmental

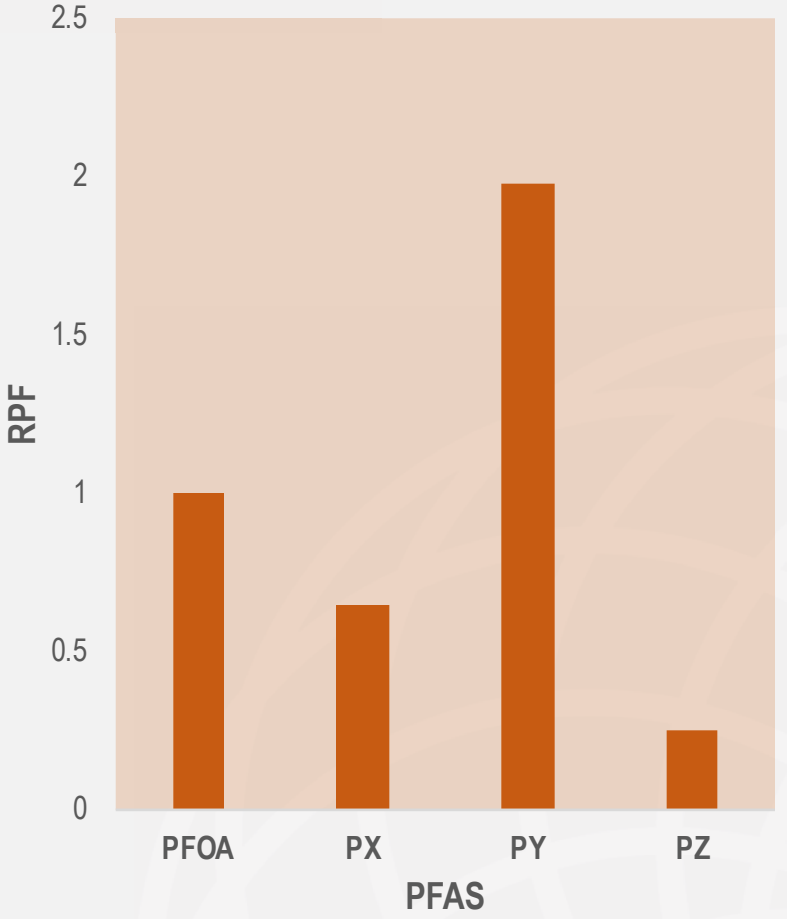
Toxicity – What do we know thus far?

- Local health studies that were ***empirical*** did not demonstrate compelling evidence that PFAS cause cancer – studies were either negative or inconsistent.
- Australian Expert Health Panel (May 2018)
  - Little difference in outcome for high vs. lower exposed populations
  - Level of effect observed in even highest exposure groups small/within range of "normal" distribution
  - Significant potential for bias/confounding in almost all studies
  - "Limited or no evidence" for any link to human disease
  - "No current evidence that suggests an increase in overall cancer risk"



Toxicity – What do we know thus far?

- Netherlands NIPHE proposed “Relative Potency Factor” (RPF) Approach for PFAS mixtures
  - Similar to approach used for PCB and Dioxin Congeners and PAH Mixtures
  - Uses PFOA as “Index Compound” since well studied
  - Assess risks for 19 other chemicals based on their toxicity relative to PFOA and then sum for total PFAS risks (for 20 substances)



Nexus of Biosolids and Exposure to PFAS

- Biosolids Beneficial Reuse:
  - Application in agricultural settings
  - Fertilizer in parks, gardens
  - Landfill cover component for vegetating
  - Reclamation of mines, quarries

- The exposure route of concern in people is drinking water

Primary Exposure Routes for PFAS

- Food and water ingestion
  - Includes packaging/wrapping transport into food
- Interior dust ingestion
- Hand to mouth transfer from treated carpets/fabrics
- Other routes of exposure anticipated to be much lower due to either intake levels or intensity of exposure (e.g., soil contact, air inhalation)

Primary Exposure Routes

- Data indicates concentrations of PFAS in virtually all media going down over time (since phase-out) as well as blood levels in humans – but new/replacement chemicals increasing (“GenX” “ADONA”)
- Leads to questioning of “relative source contribution” component of health advisories/drinking water standards (often assume 20%)
- Biosolids
  - Direct soil intake/exposures low
  - Plant uptake for longer chain PFAS, human exposure risk appears limited, but some bioaccumulation/magnification observed for grazing cattle and their milk
  - Leaching to groundwater is focus but field studies indicate little impact to water supplies likely
  - Need more field test data to better understand movement/migration at current biosolid concentrations – regional preferred (concentrations/weather/water table depth/hydrogeological parameters)

Basic Risk Calculations

- Key Exposure Assumptions
  - Adult weight – 70 kg
  - Ingestion rate of water – 2 Liters/day
  - Upper bound residence time at a single home – 30 years
  - Upper bound occupational tenure (at single job) – 25 years
  - Frequency for residential – 350 days/365 days (year) – 2 weeks vacn!
  - Frequency for worker – 250 days/365 days – five day work week
  - Duration for inhalation exposures for worker – 8 hours/day
- Toxicity Values – Example for Oral (drinking water)
  - Non-cancer = Reference Dose (RfD) in mg/kg/day
  - Cancer = Slope Factor (SF) in $(mg/kg/day)^{-1}$

Basic Risk Calculations

- Ingestion of Drinking Water – Key Variables
  - Dose = $\frac{\text{(Concentration * Intake rate* Frequency*Duration*Exposure Period)}}{\text{(Body Weight*Averaging Period)}}$
- Non-cancer Risk (Hazard Index) = Dose/RfD – compare ratio to benchmark or "limit" of 1
- Cancer Risk (Excess Lifetime Cancer Risk) = Dose*SF – compare to applicable risk limit (e.g., in MA, 1×10^{-5} or 1 excess cancer per 100,000 people exposed)

Risks & Criteria for PFAS and Some Other Chemicals with Stringent Toxicity Values – Assuming Lifetime Drinking Water Use

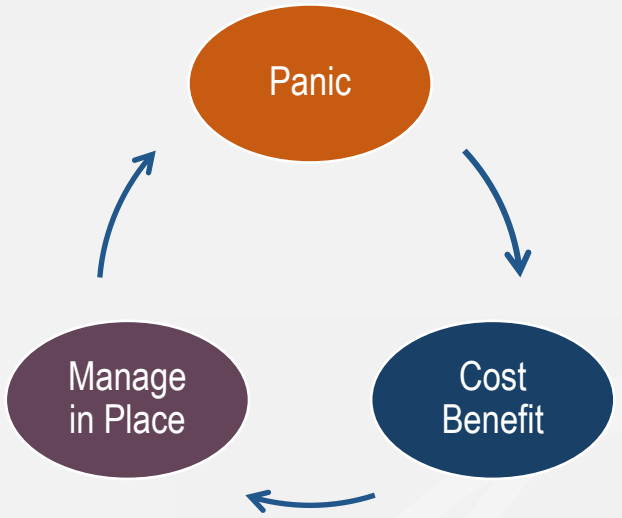
	USEPA HA/MCL (ug/L)	RISKS (HI)	MMCL (ug/L)	RISKS (HI)	Waste Water LOW (ug/L)	RISKS (HI)	Waste Water HIGH (ug/L)	RISKS (HI)
PFOA	0.035	0.048	0.014	0.019	0.006	0.008	0.050	.068
PFOS	0.035	0.048	0.014	0.019	0.004	0.006	0.022	.037
TCE	5	0.274	5	0.274				
PERCHLORATE	15	0.587	2	0.078				

Notes:

MMCL = Massachusetts Maximum Contaminant Level
HA = Health Advisory
MCL = Maximum Contaminant Level
HI = Hazard Index

Some other food for thought …

- Asbestos
  - Class “A” Human Carcinogen
  - Initial AHERA Regulations in 1986



	OSHA (TWA) PEL	Risk (Worker)	Abatement Clearance Standard	Risk (Resident)	EPA RBC Resident (10^{-6})	EPA RBC Worker (10^{-6})
Asbestos	0.1 f/cc	2.5×10^{-3}	0.01 f/cc	8.8×10^{-3}	0.000006 f/cc	.000063 f/cc

Some other food for thought …

- Benzene
  - Class "A" Human Carcinogen
  - Federal MCL = 5 ug/L

	Federal MCL (ug/L)	Risk (Resident) HI	Risk (Resident) ELCR	EPA RSL (Tap Water) NC (1; child only)	EPA RSL (Tap Water) C (10^{-6})
Benzene	5	0.54	1.9×10^{-5}	33	0.46

Notes:

RSL= EPA Regional Screening Level

NC = Non-Cancer

ELCR = Excess Lifetime Cancer Risk

C = Cancer

How Do Other Countries Advise/Regulate? (ppt)

	PFOA	PFOS	Notes
USEPA	70	70	Combined
Australia	70	560 (inc. PFHxS)	
Canada	200	600	
BC, Canada	200	300	
Denmark	100	100	Ind. & Summed (12)
Italy	500	NA	
Sweden	90	90	Summed (7)

Source: ITRC, Table 4-1, Standards and Guidance values for water, 9/18

Understanding your Data (and Audience!)

- Evaluation of Hazard
  - In light of:
    - Conceptual Site Model
    - Standards/criteria/guidance
    - Toxicity Information
    - Site-specific risk assessment

- Communication of Results

RISK = HAZARD + OUTRAGE

(Peter Sandman)

So now what?

- Further research into actual health impacts to people such as Phase II Australian study
- Peer review and public comment on toxicity studies and methods to derive advisories and standards
- Cost-benefit evaluations for proposed testing and standards
- Evaluation of current biosolid concentrations and leaching field studies on regional level
- Balanced and fact-based Risk Communication is key to mitigating misperceptions and fear!

NAMASTE

THANK YOU!!
Questions?

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