The Guest That Cannot Finish

PFAS retention, harm and cancer read as a closure problem

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Abstract. Per- and polyfluoroalkyl substances are described in the public and regulatory literature as forever chemicals: compounds whose carbon–fluorine bond resists degradation and which therefore accumulate in the body. This essay argues that the standard account misplaces the mechanism twice, and that the misplacement explains both why the compounds are hard to remove and why the epidemiology around contaminated sites is so persistently inconclusive. First, PFAS is not retained because it cannot be destroyed; indestructible solutes are ordinarily excreted. It is retained because the body actively recaptures it, through the same renal, hepatic and intestinal transporters that conserve bile acids and organic anions. Retention is a conservation mechanism operating correctly on a guest it cannot distinguish. Second, PFAS is not genotoxic: the International Agency for Research on Cancer places PFOA in Group 1 on the strength of epigenetic alteration and immunosuppression, not DNA damage. On the reading developed here, PFAS does not generate a malignancy but lengthens the return time of the pathway that would have closed one off. Both conclusions follow from a single structural claim, stated in the vocabulary of the Vacuum.Net framework: closure is a property of a whole path and of the host–guest pair, and is never readable at a point. Two measurements that have not been made follow directly, and both are specified: a removal trial blocking two recapture limbs simultaneously, and a dose-gradient analysis of the Dutch Chemours cohort using serum data already collected in 2017.

1. Two questions that do not fit their answers

The literature on perfluoroalkyl substances contains two statements that are each repeated constantly and that do not sit well together.

The first is that PFAS persists in the human body for years because the carbon–fluorine bond is the strongest single bond in organic chemistry and no mammalian enzyme cleaves it. The elimination half-life of perfluorooctanoic acid in humans is given in years; for perfluorohexane sulfonate it is longer still. The explanation offered is indestructibility.

The difficulty is that indestructibility does not by itself produce retention. The kidney does not require a solute to be metabolised before excreting it; it filters, and what is not reclaimed leaves. A small, highly water-soluble, chemically inert anion is in principle the easiest thing a kidney ever handles. Many such compounds are cleared within hours. If PFAS stays for years, something must be holding it, and inertness is not that something.

The second statement is that PFAS causes cancer. The International Agency for Research on Cancer classified PFOA as carcinogenic to humans, Group 1, in 2023. Yet where this has been tested in a resident population around a European point source — the DuPont and later Chemours plant at Dordrecht in the Netherlands — three separate studies covering three decades found no excess of any cancer, and specifically none of the three sites the investigators went looking for.

These are not minor discrepancies to be absorbed by better data. Each of them points at a place where the standard mechanistic picture is carrying the wrong shape. This essay sets out what shape fits instead, and what follows from it in the form of experiments and analyses that can be performed now.

2. The material

2.1 Binding, distribution and the failure of the lipophilicity model

PFAS is routinely grouped with the classical persistent organic pollutants — dioxins, polychlorinated biphenyls — and the grouping carries with it an assumption about where the compound goes. Those compounds are lipophilic and partition into adipose tissue. PFAS does not. More than ninety-nine per cent of the serum burden is bound to protein: to albumin for the shorter chains, and increasingly to globulins for chains of seven carbons and longer. The longer homologues additionally associate with membrane phospholipids. Octanol–water partitioning, the standard descriptor of lipophilicity, predicts PFAS distribution poorly; protein–water and membrane–water partition coefficients predict it well.

This is not a technical footnote. A compound that sits in fat is sequestered and comparatively inert in situ. A compound that sits on albumin is in circulation, presented continuously to every receptor, transporter and membrane the blood reaches. The two produce entirely different exposure histories at the cellular level from the same body burden.

2.2 The documented effects

The toxicological literature records a consistent set of effects across models. PFAS binds the nuclear receptors that govern lipid and bile-acid handling: PPARα, PPARγ, CAR, PXR, FXR and LXR. It impairs mitochondrial β-oxidation of fatty acids, which is the best-supported mitochondrial effect; it uncouples oxidative phosphorylation and lowers membrane potential, most strongly for chain lengths C8 to C11. It generates reactive oxygen species beyond the capacity of glutathione and superoxide dismutase, with lipid peroxidation and mitochondrial DNA damage following. It triggers endoplasmic reticulum stress through the unfolded protein response. It raises bile acid synthesis and transport while inhibiting CYP7A1, with cholestasis, steatosis and fibrosis as downstream findings.

Listed this way the effects appear as an inventory of separate insults, and that is how they are usually presented. Section 3 argues that they are better read as one condition observed at successive positions along a single path.

2.3 The transporters

Three organs recapture PFAS from fluids that would otherwise carry it out of the body. In the kidney, the organic anion transporters OAT1, OAT3 and OAT4 together with the urate transporter URAT1 reabsorb filtered PFAS from the tubular lumen; urinary excretion of the protein-bound fraction falls below one tenth of one per cent. In the liver, OATP1B1, OATP1B3 and the sodium–taurocholate cotransporting polypeptide NTCP take PFAS up from portal blood. In the intestine, the apical sodium-dependent bile acid transporter ASBT and OATP2B1 return biliary PFAS to the circulation, completing an enterohepatic loop.

A state-of-the-science review of these interactions identified nine human and eight rat renal transporters already studied for PFAS handling, proposed seven further untested candidates, and linked the well-documented sex- and species-specific differences in elimination half-life to differences in transporter activity. The review frames this as population variability. Section 3.3 argues that it is not variability around a true value but the quantity itself.

2.4 The carcinogenicity classification

The grounds of the IARC Group 1 classification repay close reading, because they are not what the classification is usually taken to mean. Evidence in exposed humans for specific cancers was rated limited, for renal cell carcinoma and testicular cancer. Evidence in experimental animals was sufficient. The classification as carcinogenic to humans rests on the combination of that animal evidence with strong mechanistic evidence in exposed

humans, and the mechanistic evidence rated strong concerns two key characteristics of carcinogens: epigenetic alteration and immunosuppression.

Genotoxicity is not among them. PFOA is not classified as a mutagen, and the Group 1 placement does not assert that it damages DNA. This single fact, taken seriously, reorganises everything downstream of it, and section 4 does so.

2.5 The Dordrecht record

The DuPont, later Chemours, works at Dordrecht emitted PFOA for decades. The Dutch National Institute for Public Health and the Environment estimated that residents were exposed through air above the chronic exposure limit, in the worst case for twenty-five years, with the limit no longer exceeded after 2002; drinking water was not elevated. Liver effects could not be excluded. Blood PFOA was measured in residents in 2017.

Cancer incidence in the postcode areas around the plant was counted for 1989 to 2013 in Dordrecht-Noord, Sliedrecht-Zuid-West and Papendrecht-Oost, with kidney, testis and liver singled out because of the PFOA literature. No site was elevated in any of the three areas; the total in Dordrecht-Noord fell below expectation. A comparable study in Molenwaard in 2018 reached the same conclusion. In Sliedrecht for 1998 to 2017 the total was raised, attributable mainly to skin cancer, while kidney and testicular cancer were not.

2.6 The removal trials

Two controlled studies have tested whether the body burden can be reduced. A randomised crossover trial in Denmark with forty-five participants gave cholestyramine, a bile acid sequestrant, at three times four grams daily for twelve weeks, and recorded a fall in serum PFOS of sixty-three per cent, with smaller reductions for PFHxS, PFOA, PFNA and PFDA. A smaller crossover study in Ronneby, Sweden, using cholestyramine and colesevelam in ten participants, found reductions up to forty per cent. A randomised trial of 285 firefighters in Victoria, Australia, found that plasma donation every six weeks reduced levels by approximately thirty per cent over twelve months, an effect still present three months after the intervention ended, with blood donation every twelve weeks also significantly below control.

Against these, an observational analysis within the C8 Health Project compared serum PFAS in twenty-three adults taking probenecid, an organic anion transporter inhibitor, and thirty-six taking cholestyramine, among 56,175 participants. Cholestyramine lowered serum PFAS significantly, PFOS most. Probenecid produced modest increases that did not reach significance. On that basis the independent PFAS Scientific Advisory Panel for Jersey recommends against probenecid and makes bile acid sequestrants the first-line intervention, with phlebotomy second.

3. The reading

3.1 Entry by resemblance into a path with no terminus

A fatty acid entering the body traverses a path that ends. It is taken up, recognised, carried, β-oxidised, consumed; what remains leaves. The path has a terminus, and the terminus is what releases the position.

PFAS enters that same path on the strength of its shape. It has the geometry of a fatty acid and the behaviour of a bile acid, and the handling machinery treats it accordingly. What it cannot do is complete the traverse, because the oxidative step cannot run: no mammalian enzyme cleaves a perfluoroalkyl carbon–fluorine bond. PFOA and PFOS are terminal; precursors are metabolised to them, and they are metabolised to nothing.

So the position is occupied and the path has no end for its occupant. This is the structural statement from which the rest follows, and in the vocabulary of the framework it is a path that is entered and cannot close.

Read this way, the inventory of section 2.2 ceases to be an inventory. Receptor occupancy, impaired β-oxidation, uncoupling, bile acid dysregulation and ER stress are not five attacks delivered by one agent. They are what a non-terminating occupancy looks like when it is sampled at five successive positions along one path. The agent does nothing at any of them; it fails to leave all of them.

3.2 Retention as conservation operating correctly

The second displacement concerns the transporters. In the PFAS literature the renal and enterohepatic transporters are described as reabsorption, and the word carries an implication of leakage: a defence that is failing to hold.

They are nothing of the kind. Bile acids are metabolically expensive, and the body conserves them deliberately. Of the bile acid pool, the great majority is recovered from the ileum and returned to the liver, several times per meal. OAT1, OAT3, URAT1, OATP1B1, OATP1B3, NTCP, ASBT and OATP2B1 constitute the return limbs of a conservation circuit, and for a genuine bile acid the circuit is sound. It is sound precisely because there is a forward limb with a terminus: what is recaptured is eventually used, modified and voided, so the circuit drains.

Place in that circuit a guest that cannot complete the forward limb and the identical machinery becomes a trap. The kidney reclaims it from the filtrate. The liver reclaims it from portal blood. The intestine reclaims it from bile. Each organ performs its function correctly, and because nothing terminates, nothing drains.

This inverts the usual account. The years-long half-life does not measure the toughness of the molecule; it measures the efficiency of the conservation machinery. Retention is not a defence failing. It is a conservation mechanism succeeding, on the wrong guest.

The strongest evidence for this reading is not observational but interventional. If retention were a property of the compound, interrupting bile acid recapture would do nothing to it. Interrupting bile acid recapture removes sixty-three per cent of the PFOS burden in twelve weeks. The sequestrant does not touch the molecule; it removes one return limb, and the burden drains through the opening. That number is a direct measurement of what one limb was contributing.

3.3 Closure belongs to the pair, not to the compound

In the framework used here, closure is a property of the host–guest pair and not of either member taken alone. Applied to PFAS the consequence is immediate and testable: the elimination half-life of PFOA is not a property of PFOA. It is a property of a compound–host pair, set by how many return limbs that host runs and how vigorously.

This is already half-confirmed in the data without having been read as such. Mice clear PFOA in hours and humans take years. Male and female rats differ by more than an order of magnitude, and the difference tracks renal transporter expression. In the human population the variation is wide, and the state-of-the-science review maps nine human renal transporters onto it with seven more candidates untested. In a modelling exercise, glomerular filtration rate influenced serum half-life more than transporter expression did.

The field calls this population variability, which treats it as noise around a compound-level constant. The reading offered here says there is no compound-level constant to be noisy around. The host-specific return capacity is the quantity, and reporting a single human half-life for PFOA is a category error of the same kind as reporting a single protection correlate for a host–pathogen pair.

4. Cancer without a signature

The standard narrative of chemical carcinogenesis runs: an agent reaches DNA, a mutation arises, clonal expansion follows. For benzene or aflatoxin this is the mechanism, and it has two convenient properties. It leaves a signature in the tumour, and it supports a threshold argument. Neither property is available for PFAS, because PFAS is not genotoxic. The Group 1 classification rests, in exposed humans, on epigenetic alteration and immunosuppression.

In the vocabulary of this essay those two are one. Epigenetic alteration means that the reading settings of a cell do not return to their prior values after a perturbation. Immunosuppression means that an immune response does not run to completion. Both are statements about return, not about damage: the transient component fails to subside within its characteristic time, and the registering component carries an offset. That is the operational definition of a closure deficit, stated in an earlier essay of this series for host–pathogen pairs, now applied to a chemical guest.

From this the role of PFAS in carcinogenesis follows, and it is not the role usually assumed. Transformed cells arise continually in everyone. What determines outcome is whether the surveillance and repair pathway closes on them. PFAS does not produce the transformed cell. It lengthens the return time of the pathway that would have closed it off.

Three consequences follow, and they are what the epidemiological record actually looks like.

There is no signature. A multiplier on an existing background leaves no mark in the tumour. There is no PFAS mutation, no PFAS-specific histology, no fingerprint to find on examination.

There is no threshold. A rate multiplier acts at every level of exposure, scaled. The regulatory apparatus is built to find a dose below which an effect does not occur; for this mode of action there is no such dose, only a progressively smaller multiplier.

There is no individual attribution. A raised probability cannot be reduced to a single case. The question a resident asks — did the plant cause my cancer — has no answer in principle, and not for want of study.

5. Why Dordrecht could not find it

5.1 Count against gradient

The Dutch investigations counted incident cancers in defined postcode areas and compared the totals with national expectation. That design asks: is there an excess here. It is a cross-sectional question, one number per area.

The quantity being sought, on the reading above, is a multiplier on a background dominated by smoking, age and chance. In a count of roughly two thousand cases accumulated over twenty-five years, a modest multiplier is not distinguishable from the variance of the background. The design has almost no power to detect what it was deployed to detect. A null result from it is close to uninformative, in either direction — and it is being used by both parties to the dispute as though it were decisive.

This is the same structural point made throughout this series in a different setting. A closure deficit is not readable at a point; it requires at least two values, because what is being measured is a return or a slope. A single incidence figure per area is a point measurement, and a multiplier is invisible in it.

5.2 What the American studies did differently

The contrast with the Mid-Ohio Valley investigations is instructive, and it is not principally a contrast of size — a correction worth making explicitly, since the comparison is often drawn that way.

The C8 Health Project survey of 2005 and 2006 enrolled approximately 69,000 people, but that was a single cross-sectional health survey with blood sampling. The cohort assembled for cancer follow-up was substantially smaller: 32,254 individuals, comprising 28,541 community members and 3,713 plant workers, with median follow-up from age twenty of 32.9 years but only 4.4 years following the survey itself. The study that produced the clearest cancer signal was a third design again, a case-control analysis of 25,107 cancer registrations across thirteen counties for 1995 to 2005.

That case-control study reported an adjusted odds ratio for testicular cancer of 5.1, with a confidence interval from 1.6 to 15.6 — resting on six cases in the highest serum exposure category. Six. The interval is wide and the estimate correspondingly fragile, which is part of why IARC rated the human evidence limited. But the relevant observation for present purposes is that a signal was obtained at all, on six cases, where three Dutch studies on far larger counts obtained nothing.

Two things differ. The first is exposure contrast: at Washington Works the route was drinking water over decades, with serum concentrations in some water districts two orders of magnitude above background, whereas at Dordrecht the route was air and the drinking water was not elevated. The second, and the decisive one, is that the American studies had a dose axis. Individual cumulative exposure was reconstructed from modelled water concentrations, residential histories and measured serum, and the analysis asked whether risk rose with the amount received. The Dutch studies asked whether a geographic area had more cases than the country.

For a rate multiplier these two questions are not equivalent, and the second is far the weaker. The signal of a multiplier lies in the ordering, not in the total. A gradient analysis can recover it from a handful of cases; a geographic count can miss it in thousands.

6. What follows

Three measurements follow from the reading, and none of them has been made. They are stated here in the order of how readily they could be performed.

6.1 Block two recapture limbs at once

Every intervention tested to date removes one return limb. Cholestyramine and colesevelam remove the intestinal limb and work. Probenecid removes the renal limb and, alone, did not.

On this reading the probenecid null is exactly what should have been expected, and it carries no verdict on the drug. Closing one of three limbs while the other two run does not evacuate the burden; it redistributes it. What the kidney declines to reclaim is still taken up by liver and intestine and returned, and serum may even rise as tissue stores mobilise into a compartment that still has no exit. That is what the C8 analysis observed: modest, non-significant increases.

The prediction is therefore specific. Probenecid combined with a bile acid sequestrant should contribute more than its standalone effect, and more than additively, because the sequestrant supplies the terminus that the renal blockade then stops the system from circumventing. The sequestrant opens the circuit; the transport inhibitor prevents the return. Neither alone constitutes an exit.

No study has blocked two limbs simultaneously. The Danish, Swedish and Australian trials each address one. Given that the single-limb intervention already achieves sixty-three per cent, and that the drugs involved are long-established and inexpensive, this is a small trial

standing between the current standard of care and a materially better one. It is the most actionable item in this essay.

6.2 Link the 2017 serum data to the cancer registry

Serum PFOA was measured in residents around the Dordrecht plant in 2017. The Netherlands maintains a complete national cancer registry. These two datasets have not been joined.

Joining them converts the Dutch question from a count into a gradient, which is the form in which the quantity is detectable at all. Individual serum values, supplemented by residential history for cumulative reconstruction, would permit precisely the analysis that produced a signal in the Mid-Ohio Valley on six cases. It would not require new sampling, new consent for collection, or new follow-up time — only linkage and the governance to permit it.

Until that is done, the dispute cannot resolve, and it is worth being plain about why. The company is correct that no excess has been demonstrated. The residents are correct that exposure above the limit occurred for up to twenty-five years. Both are correct because they are describing different quantities, and the measurement that would adjudicate between them has not been performed on data that already exists.

6.3 Measure return, not burden

The third follows from section 4 and is the most general. PFAS produces no acute illness. There is no fever, no crisis, no moment at which the body visibly responds. The transient compartment is, to a first approximation, empty; there is only register.

That is why cross-sectional serum concentration correlates so weakly with outcome, and why the epidemiology has been so unrewarding relative to its effort. A closure deficit is not present in a single measurement. What is measurable is return: deliver a defined perturbation and record how long the system takes to come back, and what offset it retains.

The notable thing is that the field has already done this once, without describing it that way. Vaccination is a defined perturbation; antibody titre over time is a return measure; and reduced vaccine response is the one human effect of PFAS at ordinary exposure levels that is found reliably and is accepted as a valid endpoint for immune impairment. This is not a coincidence about immunology. It is the only endpoint that happens to have been measured in the right form.

The prediction that generalises it: PFAS burden lengthens return time across multiple systems, not immunity alone, and does so at burdens at which cross-sectional measures show nothing. Any system admitting a defined, ethically deliverable perturbation with a measurable recovery trajectory — glucose tolerance, inflammatory resolution after a standardised challenge, wound closure — is a candidate test. Each requires at least two timepoints, and none of them is a blood level.

7. What this is and is not

This essay does not claim that PFAS is harmless, nor that the residents of Dordrecht were wrong to be concerned. Exposure above the chronic limit for up to twenty-five years is established by the national institute, liver effects could not be excluded, and the compounds carry a Group 1 classification on grounds that this essay accepts and reinterprets rather than disputes.

Nor does it claim that the Dutch cancer studies were incompetently performed. They answered the question they posed, correctly. The argument is that the question posed could not have revealed the quantity at issue, and that treating its null result as evidence of absence is an error made by both sides of the dispute.

What the essay does claim is structural, and it is falsifiable at three points. If a combined sequestrant and transport inhibitor regimen proves merely additive rather than more than additive, the recapture-circuit account is weakened. If individual-level dose-gradient analysis of the Dordrecht cohort shows no relation between cumulative serum PFOA and cancer incidence across a genuine exposure range, the multiplier account is weakened. If return measures across non-immune systems show no dependence on body burden where cross-sectional measures also show none, the generalisation in section 6.3 fails.

The wider point is worth stating separately, because it does not depend on PFAS. The regulatory and epidemiological apparatus of environmental health is built to detect agents that do something: that bind a receptor, damage a base, cross a threshold. It is correspondingly poor at detecting agents that fail to leave, or that slow a return. For the second kind, every instrument in standard use — the body burden, the incidence count, the threshold dose — is a point measurement, and the quantity is not located at a point. PFAS is unlikely to be the only member of that second class. It is simply the one whose production volume made it impossible to overlook.

References

Forever Chemicals, Finite Defenses: PFAS Burden the Liver, Break Mitochondria, and Outpace Modern Regulation. International Journal of Molecular Sciences, 2026. https://www.mdpi.com/1422-0067/27/6/2723

Source for the binding and distribution data in section 2.1 and the mechanistic inventory in 2.2, including the primacy of protein and membrane partitioning over octanol–water partitioning, and the identification of impaired β-oxidation as the best-supported mitochondrial effect.

A state-of-the-science review of interactions of per- and polyfluoroalkyl substances with renal transporters in health and disease: implications for population variability in PFAS toxicokinetics. https://pfascentral.org/science/a-state-of-the-science-review-of-interactions-of-per-and-polyfluoroalkyl-substances-pfas-with-renal-transporters-in-health-and-disease-implications-for-population-variability-in-pfas-toxicokinetics

Source for the transporter inventory in 2.3 and for the attribution of sex and species differences in half-life to transporter activity. The review's own framing of this as population variability is the framing contested in section 3.3.

Roles of Organic Anion Transporters in the Renal Excretion of Perfluorooctanoic Acid. Basic & Clinical Pharmacology & Toxicology. https://pure.fujita-hu.ac.jp/en/publications/roles-of-organic-anion-transporters-in-the-renal-excretion-of-per/

Primary source for renal OAT-mediated reabsorption of PFOA and the sex difference in rats.

Carcinogenicity of perfluorooctanoic acid and perfluorooctanesulfonic acid. IARC Monographs evaluation, summarised in The Lancet Oncology, 2023. https://pfascentral.org/science/carcinogenicity-of-perfluorooctanoic-acid-and-perfluorooctanesulfonic-acid

The basis of section 2.4. Establishes that the Group 1 classification rests on sufficient animal evidence plus strong mechanistic evidence in exposed humans for epigenetic alteration and immunosuppression, with human cancer evidence rated limited for renal cell and testicular carcinoma, and genotoxicity not among the strongly evidenced characteristics. The whole of section 4 depends on this reading.

IARC Monographs evaluate the carcinogenicity of perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS). International Agency for Research on Cancer, 30 November 2023. https://www.iarc.who.int/news-events/iarc-monographs-evaluate-the-carcinogenicity-of-perfluorooctanoic-acid-pfoa-and-perfluorooctanesulfonic-acid

The agency's own announcement of the classification, used to confirm the Group 1 and Group 2B placements directly.

Risicoschatting emissie PFOA voor omwonenden locatie DuPont/Chemours, Dordrecht, Nederland. RIVM. https://www.rivm.nl/publicaties/risicoschatting-emissie-pfoa-voor-omwonenden-locatie-dupontchemours-dordrecht-nederland

Source for the exposure estimate in section 2.5: air exposure above the chronic limit for up to twenty-five years in the worst case, no elevation in drinking water, limit not exceeded after 2002, liver effects not excludable, additional cancer risk assessed as limited on the basis of animal data.

Betekenis resultaten bloedonderzoek PFOA omwonenden DuPont/Chemours. RIVM rapport 2017-0101. https://www.rivm.nl/bibliotheek/rapporten/2017-0101.pdf

The 2017 serum measurements in residents. This is the dataset whose linkage to the national cancer registry is proposed in section 6.2.

Incidentie van kanker in het gebied rond chemische fabriek Chemours, Dordrecht (1989–2013). Dienst Gezondheid & Jeugd Zuid-Holland Zuid. https://www.dienstgezondheidjeugd.nl/assets/uploads/2016-03-23-Rapportage-incidentie-kanker-rondom-Chemours.pdf

The principal Dutch incidence study. Observed against expected for kidney, testis and liver in three postcode areas, with no site elevated in any area and the Dordrecht-Noord total below expectation. The design discussed in section 5.1.

Het optreden van kanker in Sliedrecht (1998–2017). Dienst Gezondheid & Jeugd Zuid-Holland Zuid, 2019. https://www.dienstgezondheidjeugd.nl/assets/uploads/2019-11-25-Rapport-Het-optreden-van-kanker-in-Sliedrecht-in-de-periode-1998-2017.pdf

The later Sliedrecht analysis, in which total incidence was raised through skin cancer while kidney and testicular cancer were not elevated.

PFAS en kanker. GGD Zuid-Holland Zuid. https://www.ggdzhz.nl/gezondheid-en-milieu/pfas-en-gezondheidsadviezen/veelgestelde-vragen/kanker

The public health summary confirming the Molenwaard 2018 result and the regional position that recent-emission cancer risk for residents is negligibly small.

Winquist, A. et al. Design, methods, and population for a study of PFOA health effects among highly exposed Mid-Ohio Valley community residents and workers. Environmental Health Perspectives, 2013. https://escholarship.org/content/qt54q143pp/qt54q143pp.pdf

Source for the cohort figures corrected in section 5.2: 32,254 participants, 28,541 community and 3,713 workers, median follow-up from age twenty of 32.9 years and 4.4 years after the 2005–2006 survey. Distinguishes the follow-up cohort from the approximately 69,000-participant C8 Health Project survey, a distinction commonly elided.

Vieira, V. et al. Perfluorooctanoic acid exposure and cancer outcomes in a contaminated community: a geographic analysis. Environmental Health Perspectives, 2013. Summarised at https://foodpackagingforum.org/news/high-exposure-to-perfluorinated-compound-linked-to-kidney-testicular-cancer-in-humans

The case-control analysis of 25,107 cancer registrations across thirteen counties for 1995–2005. Source for the testicular cancer odds ratio of 5.1 (1.6–15.6) resting on six highly exposed cases, and for the approximate doubling of kidney cancer odds in the high and very high categories. The decisive example in section 5.2 of a gradient design recovering a signal that a count design cannot.

Substantial decrease of per- and polyfluoroalkyl substances with anion exchange resin treatment: a randomised crossover trial. Environment International, 2024. https://www.sciencedirect.com/science/article/pii/S0160412024000837

The Danish cholestyramine trial, forty-five participants, twelve weeks, sixty-three per cent reduction in PFOS. The central interventional evidence for the recapture account in section 3.2 and the baseline against which the combination trial of section 6.1 is proposed.

Certain medicines accelerate elimination of PFAS from the body. University of Gothenburg, reported in Environment International. https://manufacturingchemist.com/certain-medicines-accelerate-elimination-of-pfas-from-the

The Ronneby crossover study of cholestyramine and colesevelam in ten participants, with reductions up to forty per cent. A second, smaller, independent confirmation of the intestinal-limb result.

Effect of Plasma and Blood Donations on Levels of Perfluoroalkyl and Polyfluoroalkyl Substances in Firefighters in Australia: A Randomized Clinical Trial. JAMA Network Open, 2022. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8994130/

The Victorian firefighter trial, 285 participants. Source for the approximately thirty per cent reduction over twelve months with six-weekly plasma donation. Read in section 3.2 as the creation of a fourth, external exit rather than the blockade of a return limb.

Perfluoroalkyl substance excretion: effects of organic anion-inhibiting and resin-binding drugs in a community setting. https://pfascentral.org/science/perfluoroalkyl-substance-excretion-effects-of-organic-anion-inhibiting-and-resin-binding-drugs-in-a-community-setting

The observational C8 analysis of twenty-three probenecid and thirty-six cholestyramine users among 56,175 participants. The source of the probenecid null reinterpreted in section 6.1, and confirmation that the two agents were analysed separately and never in combination.

Third Report of the Independent PFAS Scientific Advisory Panel for Jersey. Government of Jersey, 2025. https://www.gov.je/SiteCollectionDocuments/Health%20and%20wellbeing/Draft%20Third%20Report%20of%20the%20Independent%20PFAS%20Scientific%20Advisory%20Panel%20for%20Jersey.pdf

The current clinical position: bile acid sequestrants as the single best intervention, colesevelam recommended from defined serum thresholds, phlebotomy second-line, probenecid advised against. The guidance that section 6.1 proposes be tested rather than accepted as settled.

Named and not read

Barry, V. et al. (2013), Perfluorooctanoic acid (PFOA) exposures and incident cancers among adults living near a chemical plant, Environmental Health Perspectives. Repeated attempts to retrieve the full text failed at the host. The cohort figures used in section 5.2 are taken from the Winquist design paper rather than from Barry, and no hazard ratio from Barry is cited anywhere in this essay.

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