

# PFOA: Another Toxic Chemical Regulatory Science Ritual Battle



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The PFOA risk assessment released by the Agency for Toxic Substances and Disease Registry in June, and the surrounding regulatory dispute, treads familiar ground. DDT, PCBs, MTBE, PCE, or whatever — in important ways, disputes over toxics regulation are all the same. Predictable actors play predictable roles, make predictable arguments about predictable toxics science (and also regulation, politics, law, and whatnot), with predictable outcomes. It is like watching the Roadrunner and Wile E. Coyote have at it — the details differ each time, but the plot is always basically the same, the endpoint inevitable.

PFOA is the best known of a group of closely related chemicals, the perfluoroalkyl acids. They are commercially valuable because of their surface properties, which makes them useful in non-stick frying pans, carpets, and firefighting foam.

Predictably, the best science concerns the harmful health effects at high doses. Predictably, epidemiologists documented those effects by studying factory workers and surrounding communities exposed to high doses. The harms are typical for toxics, and include cancer, reproductive damage, liver damage, and immunological damage. Predictably, the most prominent PFOA tort cases have sought redress for the high exposure of factory workers and those exposed by groundwater contamination in nearby communities. In one such case the award exceeded \$500 million.

Those tort cases, and ensuing action by federal and state regulators to reach agreements with commercial manufacturers and users of perfluoroalkyls, resulted in effective and quick (by toxics regulatory standards anyway) action to dramatically reduce these types of

exposures. Consequently, over the last couple decades, exposure to PFOA and its chemical relatives has dropped dramatically. That is real progress.

Yet those successes with the high-dose problem left a residual problem of exposure of the general population to low doses of PFOA. Critically, the residual low-dose problem that remains is both qualitatively different and much harder. The ritual combat over low doses entails the combatants just repeating old arguments and stating their predictable positions, since real progress toward understanding toxic harms at low doses is either extremely difficult, or just flatly impossible.

Why the lack of progress at low doses? Though the analogy is not entirely apt, being exposed to a high dose of a toxic chemical is like being clobbered with a hammer — the toxin just kills or harms outright. Conversely, at low doses, toxic chemicals typically act more like a malicious computer program, where the harm is not the physical chemical per se, but rather the information it encodes.

Tiny amounts of information can have immense downstream repercussions, especially in complex living systems. The importance of the Declaration of Independence derives not from the physical paper it was written on, but rather from its words, which information surely got King George III riled up and precipitated a system crisis all out of proportion to the Declaration's minuscule information content.

The perfluoroalkyls' harmful effects derive, in important part, from their binding to the PPAR $\alpha$  receptor. They thus mimic chemicals found naturally in humans, rats and mice that bind to the PPAR $\alpha$  receptor. In a very real sense, the perfluoroalkyls and many other low-dose toxins are analogous to

a door key, and the receptor is a lock. Just as a key has information content, so too does a toxin.

Herein lies the conundrum of extremely low doses of toxic chemicals. We know toxins typically bind to receptors of various sorts, and thereby disrupt important paths of information flow in living humans. We know there are hundreds of different types of receptors (each "keyed" differently). Yet predicting the full downstream repercussions of receptor binding is well beyond the ken of science, now or in the foreseeable future.

For PFOA, as with many toxins, essentially everyone alive is exposed to minuscule doses. Not only has the dispute over high doses of PFOA played out predictably, but the endpoint of that dispute is the same all-too-familiar low-dose conundrum.

The large evolutionary biology literature on ritual combat goes back to Darwin's work on sexual selection. As described in Elizabeth Penissi's article on ritual combat in ants and birds, it evolves in animals so as to reduce losses from actual combat. Better for a peacock to display and strut, than to risk injury in a physical encounter.

With PFOA, and other low-dose toxins, it is not just the cost of a battle over low-dose toxic regulation. No amount of money spent on research will allow scientists to know what is fundamentally unknowable. In this circumstance, I just imagine the protagonists in toxics regulatory dramas as strutting peacocks.

**High exposures are hazardous; are there harms from low doses in general population?**