Everything has a beginning. For me, the Big Bang occurred just after I had finished a lecture in the Washington DC area in 1988 and a tall, thin woman strode up to me from the back of the room, put both her hands on my shoulders, and said, “Pete... I’m Theo Colborn, and we have to talk.” Within two years she became a Senior Fellow at the W. Alton Jones Foundation where I had just become Director, and six years after that, in 1996, along with Diane Dumanoski we published Our Stolen Future.¹

Our Stolen Future (OSF) was the first major public exploration of endocrine disruption—how chemicals interfere with hormone action—and now, twenty years later, it is still for sale, still used in classrooms, still read widely. More than a few people, often researchers or physicians, have come up to me and said, “I chose my career because of that book.” A friend reported seeing a Congressional aide last year reading OSF on Washington DC’s Metro.

Later in 1996, Congressman John Porter (R, IL) held budget hearings. At the time he was chair of the House Subcommittee on Health Appropriations. He invited me to attend, where I watched him hold up OSF and suggest to then-National Institutes of Health (NIH) director Harold Varmus that he should read it.

I doubt that Varmus did, but I do know that Porter repeatedly found ways to add resources to NIH and the Centers for Disease Control and Prevention (CDC) that strengthened their abilities to fund research on, and monitoring of, the health effects of chemicals in the environment, including endocrine disruptors. Since then, the governments of the U.S., Japan, the European Union and elsewhere have put literally hundreds of millions of dollars into fund research on, and monitoring of, the health effects of chemicals in the environment, including endocrine disruptors.

The research funding noted above unleashed a torrent of scientific results that together have solidified a series of overarching conclusions:

• Exposure to EDCs can have biologically adverse effects at doses well beneath those typically considered in toxicological experiments;⁹

• Exposures during fetal life can set in motion consequences that play out over the lifetime of the individual, and which often are not clearly evident at birth;¹⁰

• Exposures to EDCs are ubiquitous, in part because of the pervasive distribution of persistent compounds like polychlorinated biphenyls (PCBs) and many pesticides that volatilize and are distributed by air currents; in part because these chemicals have been incorporated into consumer products used worldwide in homes and offices and hospitals, etc.; and in part because EDCs are key components of industrialized agriculture and thus are found abundantly in the human food supply;¹¹

• People are exposed to mixtures of EDCs continuously, never one chemical at a time;¹²,¹³ and

• Risk assessment as practiced by public health agencies like the U.S. Food and Drug Administration, the U.S. Environmental Protection Agency, and their counterparts around the world use tools that are incomplete, out-of-date and delegitimized by thoroughly falsified assumptions.³
The tools used by regulatory agencies are incomplete for EDCs because at best they consider only chemical effects on the estrogen and androgen system. All other EDC mechanisms are ignored, including thyroid, which is evaluated solely by hormone levels in the blood. They are out-of-date because they use assays that date, in the most extreme case, back to the 1930s, and fail to incorporate our current understanding of the complexity of these systems using the plethora of assays that have been developed by NIH-funded scientists over the past three decades. They are delegitimized because at least two assumptions core to regulatory testing have been extensively falsified: (1) Standard protocols test the effects of high doses and assume those tests can be used to estimate the adverse effects of lower doses. Nonmonotonicity (U-shaped or inverted-U-shaped) is a common feature of EDC dose-response curves. Doses well below those caused by high exposures can even cause exactly the opposite effect observed at high doses (see figure 1). (2) All tests are done one chemical at a time. The real world is very different. Even pesticides are tested one chemical—the ‘active’ ingredient—at a time, not the complex mixture that is the pesticide as it is sold. That is farcical: the mixture is designed to enhance the effectiveness of the active ingredient. Finally, this testing also assumes that high-dose short-term exposures are generalizable to low-dose, life-long exposures despite empirical evidence in humans that this assumption is not true.

The research community responded strongly to the availability of research funding to study EDCs. In retrospect, it was very important that many of the researchers recruited to the issue were not trained in toxicology but instead were steeped in a wide range of other biological arenas, and they brought new tools and new thinking to bear upon the issue of EDC hazard. More than a few of the scientists new to EDCs literally stumbled into systems. All other EDC mechanisms are ignored, including thyroid, which is evaluated solely by hormone levels in the blood. They are out-of-date because they use assays that date, in the past three decades. They are delegitimized because the data requirements requisite by procedures adopted from the U.S. Institute of Medicine and the World Health Organization could be met by only fewer than five percent of known EDCs.

In 2015 and 2016, a team of EDC specialists and economists estimated the annual economic costs of adverse effects resulting from EDC exposures were in excess of one hundred eighty billion dollars in the European Union, and in excess of three hundred forty billion dollars in the U.S. The team describes the results as very conservative because the data requirements requisite by procedures adopted from the U.S. Institute of Medicine and the World Health Organization could be met by only fewer than five percent of known EDCs.

In 2016, the National Institutes of Health honored, for the first time ever, twelve “Champions of Environmental Health Research.” Four of those twelve have feet partly or completely planted in the field of EDCs (including me). While everything has a beginning, this saga does not yet have an end. The scientific basis for concern has grown massively since we wrote OSF. Public awareness is creating markets for companies that want to reduce EDC use in their products. Some chemists and companies have responded strongly, and even collaborated to produce an intellectual framework for how chemists could avoid EDC hazard in the synthesis of new molecules. But there have been missteps in this process, with regrettable substitutions of poorly known replacements for bad actors like BPA regrettable because some of the replacements being sold as “BPA-Free” are likely as bad or worse than BPA.

Sadly, policy responses lag significantly. The recently passed Lautenberg Chemical Safety Act does little to advance regulations of EDCs, consigning the next several generations of America’s children to more EDC exposures. More progress is evident in the European Union because of several laws that have been passed there over the past decade. However, their implementation is being fought bitterly by private interests ‘manufacturing doubt’ to forestall regulations. Hopefully, the growing public awareness of EDC risks, engagement by scientific and medical societies and market opportunities for chemists to make money by avoiding these hazards will over time reduce the burden of EDC related diseases. It’s all possible, as we continue to get traction with the science, and the public demands safer products.