

# OUR STOLEN FUTURE

## Twenty Years Later

Pete Myers, PhD

**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*.<sup>1</sup>

*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 answering research questions we raised in OSF, and much more. These investments have yielded a revolution in our understanding of Endocrine-Disrupting Compound (EDC) science.

Importantly, none of the core themes we explored in OSF have been scientifically rebuked. Instead, the research that has emerged has deepened and widened concerns. For example, while writing OSF we did not anticipate obesogens or metabolic disruptors and now it is well understood that chemicals can interfere with metabolism and body weight regulation.<sup>2</sup> We must acknowledge now that virtually any hormonal signaling is theoretically vulnerable to disruption, and that we still likely have barely scratched the surface because of the tens of thousands of chemicals now in commerce that are novel molecules in the human body. Because of the impact of EDCs on fertility and reproduction, it is quite possible that molecular evolution is currently underway.<sup>3</sup>

Like any other scientific revolution, endocrine disruption stands on the shoulders of giants, notably research on diethylstilbestrol’s effects in the U.S. by Arthur Herbst, John McLachlan, Retha Newbold, Howard Bern and others.<sup>4,5</sup> McLachlan’s insights led him to convene two prescient conferences (1979 and 1985) on ‘estrogens in the environment’ and then a highly influential series

of annual meetings, e.hormone, at Tulane University beginning in 1999.<sup>5</sup> Another important early body of work came out of efforts to understand what Niels Skakkebaek and colleagues have termed ‘testicular dysgenesis syndrome’ (TDS).<sup>6</sup> TDS is characterized by testicular cancer, hypospadias, cryptorchidism and poor semen quality. Considerable evidence now exists linking these conditions to fetal exposure to EDCs.<sup>6</sup>

What might be called the ‘modern’ era of EDC research was launched at the 1991 Wingspread Conference, where Colborn gathered twenty-one scientists whose research provided extremely diverse insights into how chemical contaminants might interfere with hormone action, and what some of the consequences might be.<sup>7</sup>

My role at Wingspread was to guide the workshop toward a consensus statement patterned after the Intergovernmental Panel on Climate Change (IPCC)’s executive summary statement of 1990. That document’s genius structure provided policy makers and journalists a sense of the broad agreement existing among scientific experts about the reality of climate change, but satisfied scientists because it also allowed disagreement and debate over details that remained uncertain. Fresh off a writing project about the complexities of climate disruption, I suggested at the workshop that ‘endocrine disruption’ captured the complexity of what we were discussing.<sup>8</sup> The term has since taken root.

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;<sup>9</sup>
- 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;<sup>10</sup>
- 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;<sup>11</sup>
- People are exposed to mixtures of EDCs continuously, never one chemical at a time,<sup>12,13</sup> 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.<sup>9</sup>

**The final point warrants elaboration.** 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.<sup>9</sup> Doses well beneath 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.<sup>14</sup> 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 them because they discovered their animal or cell models were contaminated by seemingly inert plastics. Moreover, they were not hobbled by one of the limitations of many scientists (but by no means all) in toxicology, close financial association with an industry that seeks to protect its products and works to diminish concerns about chemical safety.

While researchers leapt in, the evidence had to build before it reached mass critical enough to attract the attention of practicing physicians. What might be considered a tipping point arrived in June 2005 with a symposium in San Diego organized by Andrea Gore for The Endocrine Society (TES), the world’s largest professional association of endocrinologists. Roughly seventy percent of its membership is practicing physicians. Gore has since become an editor-in-chief of *Endocrinology*, the society’s flagship journal.

TES has since become a major international player in promoting understanding of EDCs and encouraging development and adoption of public policies designed to reduce EDC exposures. They have published two major reviews on EDCs, a ‘statement of principles’ and formed a Global EDC Task Force, which has been particularly active in bringing EDC science to bear upon policy decisions currently underway in the European Union.<sup>3,15</sup>

In 2012, a report from the World Health Organization and the United Nations Environment Program concluded EDCs are a global public health threat, and that (1) disease risks due to EDCs may still be significantly underestimated and (2) that significant opportunities for disease prevention by reducing exposures may

be within reach.

Other medical and scientific associations have also engaged, including: the American Public Health Association, the American Chemical Society, the American College of Obstetricians and Gynecologists, the American Society of Reproductive Medicine, and the Royal College of Obstetricians and Gynecologists.<sup>3</sup>

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.<sup>16</sup> 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.<sup>17</sup>

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.<sup>18,19</sup> 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.



*Figure 1. Neonatal exposure to 1 ppb/day body weight of diethylstilbestrol, an estrogenic EDC, for 5 days causes morbid obesity in adulthood (experimental animal on right; control on left). In contrast 1000 ppb causes weight loss compared to control (not shown).<sup>20</sup> Photograph courtesy of Retha Newbold.*



*Pete Myers is founder, CEO, and Chief Scientist of Environmental Health Sciences. He holds a doctorate in the biological sciences from UC Berkeley and a BA from Reed College. A full list of references is available at [www.sfms.org](http://www.sfms.org).*