Our Stolen Future

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Abstract

The book Our Stolen Future by Theo Colborn, Dianne Dumanoski, and John Peterson Myers espouses the adverse effects that endocrine system disrupting chemicals are have on various wildlife populations and groups of humans as well. The hypothesized effects of suspected chemicals include abnormal reproductive organs, decreased sperm production, altered behavior, weakened immune systems, decreased intelligence, and other problems that altogether are thought to decrease the reproductive capacity of the affected individuals. The evidence that the authors present to support their conclusions was briefly reviewed and critiqued. Conclusions and recommendations based on the book as well as other supporting resources are presented.

Introduction

Numerous technological advances have drastically altered our world, especially over the last half century. Mankind has learned how to synthesize chemicals for use as medicines to improve human and animal health, pesticides to increase the quantity and quality of crops produced, as well as countless other chemicals for many uses that generally improve our quality of life. We are also able to produce these chemicals in extremely large quantities. As a result of this synthetic activity, these chemicals have been released into the environment. Some, such as pesticides, are deliberately released in to the environment to accomplish their task, while others are released as a means of waste disposal or were inadvertently released as a result of accidental spillage. In most cases, the only properties of these chemicals that were known were those that were relevant to the application for which they were produced. Very little consideration was given to the properties of the chemicals that were undesirable and might cause them to cause problems in the environments in which they were released. Some of these problems included persistence in the environment, chronic and acute toxicity to non-target organisms, migration over great distances from the sites of release, and exposure of humans through unexpected routes, such as through ground water. Society mistakenly believed that “dilution is the solution” to these chemical problems or did not imagine the adverse effects these chemicals could have. Our society is slightly more enlightened than it was at the beginning of this chemical revolution, recognizing that many of the chemicals that have been historically produced and released into the environment degrade very slowly and have many undesirable effects. For this reason, some chemicals are no longer produced or their environmental

release is tightly controlled. One of the undesirable characteristics that has been discovered in some of these environmentally contaminating chemicals is that, when present at sufficiently high concentrations in humans and animals, they are similar enough to naturally occurring endogenous biochemicals to disrupt the normal chemical communication that occurs in complex animals. This toxic effect is contrasted with the acute toxicity response observed when an animal is poisoned by a chemical and becomes sick or dies or a chronic toxic response such as the development of cancer in the exposed animal at anytime following exposure. Disruption of the animals endocrine chemical communication system is usually observed in the form of an effect on the offspring of the animals actually exposed to the toxicant. The book *Our Stolen Future* by Theo Colborn, Dianne Dumanoski, and John Peterson Myers was written to bring the hazard of environmental contaminating chemicals to the endocrine system to the attention of the general public by showing examples of some of the effects that can or may result from exposure of humans and animals to these chemicals.

Background

The book's authors appear to be well qualified to write a book such as this. Although it appears that none of them actually does toxicology research, Colborn and Myers both hold PhDs in zoology. The book was well researched with many original research articles in peer-reviewed journals cited in the "Notes" section of the book. However, to its credit, the book certainly was not an exhaustive review of the effects of endocrine disrupting chemicals on animals (which would have been extremely boring to most audiences).

This whole idea that environmentally dispersed chemicals affect the endocrine systems of non-target animals in ecosystem was not new either when research began on the book or when it was published. As the authors pointed out, the estrogenic effects of DDT were reported in 1950 (Burlington and Lindeman, 1950). The estrogenic effect of kepone was discovered as a result of worker exposure in manufacturing plants and was well known in the 1970's (Deobald, 2000). It isn't clear what the timing of the publication of this book is about. One could speculate that they believed that this phenomenon is not well known among the general public, that they believe there is increasing injury as a result of ongoing endocrine disruption, or that there is more new, subtle forms of injury that have been recently attributed to endocrine disruption.

Discussion

The authors begin their argument for the seriousness of endocrine disrupting environmental contaminants with some anecdotal statements regarding observations of different populations of wildlife displaying abnormal reproductive behavior, such as two female herring gulls sharing a nest, or the presence of abnormal reproductive organs in alligators. The hypothesis is stated that environmental contaminants may be responsible for these abnormalities. Polychlorinated biphenyls are implicated in the abnormal behavior in the gulls and dicofol (a hydroxylated form of DDT) is suggested to be involved in alligator defects. Because these animals are displaying abnormal reproductive behavior or anatomy, it is reasonable to suspect chemicals that mimic estrogen in these processes. It is known that an excess of estrogen during gestation can effect the expression of male characteristics in adult males. The authors summarized some of the research done in rats and mice that demonstrates this

phenomenon. The authors reviewed some of what is known about diethylstilbesterol (DES). This compound was a drug prescribed to pregnant women in the 1950s and early 1960s and has been linked to a number reproductive tract abnormalities in the daughters of women who took DES. This compound has been demonstrated to be a synthetic estrogen mimicking compound. Exposure of people to DES is dramatically different from exposure to estrogenic environmental contaminants. Women that took DES were exposed to much larger amounts of active material at a time when their developing children were especially vulnerable to teratogenic effects. In contrast, the authors hypothesize other exposure to result from persistent chemicals that had accumulated in the mothers adipose tissue as result of consumption of contaminated foods, especially fish, which results in a much lower dose to a developing fetus or child. This discussion of DES is a good example of some of the well-documented effects on humans due to exposure to an estrogenic compound. In addition to effects of a synthetic estrogenic compound on offspring, the authors reviewed some experimental results where naturally variable amounts of estrogen in the uterus and exogenously applied estrogen affect the characteristics of mice and rats. These discussions were useful to introduce some of the effects that might be observed in animals exposed to hormone mimicking or blocking activity. Some of these effects were also demonstrated with chemicals that are common environmental contaminants.

There are many interesting examples mentioned in this book of wildlife populations displaying either abnormal behavior or having a high rate of reproductive anomalies, such as the appearance of anatomical characteristics intermediate between male and female. There are too many examples to recite, but generally, I think it is not

possible to draw the conclusions for cause and effect relationships that the authors drew. In those examples, the environments were generally highly contaminated with many chemicals, any one of which could be having effects. It is reasonable to suspect that some of the chemicals had estrogenic properties.

This book is a useful and valid warning of the adverse effects that environmental contaminants can have on wildlife and humans, especially to developing individuals. I am however, skeptical that all of the observed effects stated in the book are the result of endocrine disruption. There is no question that an animal's endocrine system is very complex and prone to disruption at many points. And, I think the evidence is compelling that some of the observed effects of chemicals on wildlife, such as feminization of male alligators and vitellogenin induction in male fish, are the result of estrogenic chemicals mimicking natural hormones. However, I think that some of these effects may result from mechanisms other than endocrine disruption. There are some unequivocal attributions of certain toxic effects due to exposure to various hormonally active synthetic chemicals, but I believe that the authors are attempting to ascribe all observed toxic effects to an endocrine disrupting mechanism. For example, the authors mention the prospect of chemicals acting on "orphan" receptors whose function is not known. It is not uncommon in pharmacology for a receptor to be discovered only after a drug has been found that acts on that receptor. Effects of some of these contaminants may be on receptors that aren't part of the endocrine system. It has been suggested that alternative forms of suspected receptors are expressed at different levels within a species and between species (Crews, et al., 2000). This may explain variation of sensitivity of organisms to endocrine disrupting chemicals. Because the effects

described are most commonly observed in animals exposed during developmental periods in their life histories, such as during gestation and early life, I would consider these toxic end points to be a type teratogenesis. Although teratogenesis is defined as the toxic endpoints observed in offspring when a mother is exposed to a toxicant during gestation, birth of the offspring is an artificial boundary for this definition because development of the organism continues into adulthood. When one thinks of teratogenesis, one thinks of a grossly deformed juvenile, but these authors have expanded my vision to include many less profound abnormalities that can have serious consequences for the reproductive capacity and quality of life of the individuals suffering with them. We as a society should be no less concerned about the toxic effects described in the book suggested to be caused by the environmental contaminants on wildlife and humans whether the mechanism of these toxicoses is through endocrine disruption or some other unknown mechanism.

The most convincing argument for concern about endocrine disrupting chemicals affecting human and animal populations are the results of tests on babies and children born to mothers who had eaten Great Lakes fish. Assuming that conduct of the tests done on the children and the statistical analysis of the results are scientifically defensible, these results raise serious concern. The results clearly suggest that something passed from the mothers to their developing children has an undesirable effect on learning capability and behavior of these children. The authors suggest that PCBs and dioxins are responsible for these effects. However, I wonder if this group of compounds has been proposed because they are obvious suspects that can be readily detected in lake sediment. Anyone even slightly knowledgeable in analytical chemistry

would recognize that PCBs are easily extracted and concentrated using nonpolar solvents and can be analyzed with great sensitivity by gas chromatography with electron capture detection. It is possible that one or more other compounds in the lake sediments that are not as easy to analyze for or that have not been looked for may be the actual culprit. Immune system compromise, increased testicular and breast cancer rates, and decreased sperm counts are some of the other effects that the authors have attributed to endocrine disrupting environmental contaminants. While this might be true, there wasn't sufficient evidence provided in the book to accept this conclusion.

The saddest realization from reading this book is that some animal species, especially those already endangered, may become extinct as a result of decreased fecundity from exposure to endocrine disrupting chemicals. Some of this exposure results from contaminants that have migrated throughout the earth from sites of chemical release thousands of miles away from sensitive organisms. It appears that the animals most at risk, such as polar bears, are those unlucky enough to be at the top of a food chain, that includes aquatic and marine benthic organisms or filter feeders that bioaccumulate and biomagnify the toxins. Ironically, some of these chemicals, such as PCBs, are no longer manufactured. One can expect this problem to become more pandemic as more concentrated deposits of chemicals, such as PCBs in the Hudson River, disperse throughout the world as a result of thermodynamics.

Animals inhabiting ecosystems contaminated by endocrine disrupting chemicals appear to adapt somewhat to these chemicals. It has been suggested that hormone receptors increase in their discrimination of natural hormone and endocrine disrupting chemicals (Crews et al., 2000). If what Darwin tells us is true, an animal population

should have the capability to evolve increased resistance to endocrine disruptors or can produce enough offspring in unimpacted ecosystems to repopulate impacted areas.

Conclusions

The authors of this book may have overstated the impact of endocrine disrupting chemicals on too many problems observed in wildlife and human populations. There is, though, a lot of good evidence presented to suggest that certain wildlife problems are a result of exposure to endocrine disrupting environmental contaminants, especially in locations where chemical concentrations are high. Some other observed deficits, such as behavioral and learning problems in children born of mothers who had consumed Great Lakes fish strongly suggests that a contaminant in the lakes has a trangenerational effect on the children consistent with the hypothesis stated in the book. Whether this effect is due to endocrine disruption is not certain, but this definitely warrants further investigation. The information in the book should cause society concern, but we should not be alarmed because unequivocal proof is still lacking.

Recommendations

Assertions were made in this book regarding the cause and effect relationship between endocrine disrupting chemicals in the environment and toxic effects observed in humans and wildlife. While I do not agree there is compelling evidence to support all of these assertions, I do believe there is enough evidence to suggest there is a serious threat to human and animal health. Because this threat is so serious, a prompt effort should be made to ground these hypotheses in scientific fact. The various compounds implicated in this book, along with other widely used chemicals, should be tested with various animals and appropriate endpoints determined. For those found to be

endocrine disrupting, or that have other previously unknown toxicological properties, their use and release into the environment should be restricted or other appropriate regulatory actions taken to minimize human and animal exposure. For example, there is good evidence that bis-phenol A is an estrogenic compound. Although it may not be persistent, its use in packaging materials that could result in food contamination should be discontinued. The book's authors recommend that all chemicals demonstrated to have endocrine disrupting activity should be banned, while others consider that this is an overstatement of the risk (Fumento, 1998). The example Fumento gives for this position is the widespread occurrence of natural estrogenic compounds in plants in a typical human diet. Many of these, he claims are more active than synthetic estrogenic compounds and are consumed at higher concentrations. It is essential for society to expend some research effort to resolve this question of why synthetic chemicals cause problems and plant chemicals do not before economic hardship is forced on industry as a result of a chemical ban. I also recommend that toxicologists and regulators look beyond endocrine disruption as a mechanism for exertion of toxic endpoints. By limiting our concern only to endocrine disruption, tests may be developed and come into widespread use that may be limited in scope and not detect other subtle, but equally concerning, toxic endpoints of chemicals. For example, while it has been previously hypothesized that estrogenic compounds act by interacting with estrogen receptors, triazine herbicides have been found to exert their estrogenic activity by inducing aromatase enzyme (Renner, 2000). This enzyme converts androgens to estrogen (Renner, 2000). Some proposed assays for endocrine disruption activity would miss these compounds. Interest in endocrine disruptors has intensified dramatically in the

last 5 years with a plan by the U.S. EPA to screen chemicals for endocrine disrupting activity. It is recognized that new and better assays need to be developed and several are now in use of have been proposed (Sadik and Witt, 1999). I have also found that the authors, as well as many others, refer to estrogenic compounds as estrogens. This is an unfortunate use of words because, while many chemicals can have properties of estrogen, only estrogen is estrogen. Anyone with any credibility in the field should recognize this mistake.

PCBs are a special problem. If these are confirmed to be endocrine disrupting and are found to cause the effects suggested in the book, there is little that can be done to prevent exposure. These have already been released into the environment and their manufacture has ceased. Efforts should be made to accelerate the biodegradation of these compounds in the environments in which they accumulate. Because their threat is so serious, cleanup of areas with the highest concentrations of PCB contamination should be considered. It may have appeared uneconomic to clean up some of these sites in past analyses. If the risk from these contaminants is much greater than previously estimated, it should be worth more to clean them up. After all, how much is it worth to save polar bears from extinction?

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