

FOCUS ON PESTICIDES

#1 Male Reproductive Disorders

"Pesticides and Parkinson's Disease" is the first in a series of fact sheets summarizing topics regarding pesticide health risks, exposure, and policy. Copies of the original studies cited in this review are available from Environmental Advocates at a cost of \$2.00 each for postage and handling.



The Pesticide Right-to-Know and Reduction Project is a joint initiative of Environmental Advocates and the New York Public Interest Research Group with funding from the Pew Charitable Trusts. To learn more about this project please contact:

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Chemical exposures are well known to adversely affect reproductive health.¹ While it may be comforting to think of this phenomena as limited to certain chemicals and certain high exposure scenarios (such as manufacturing or drug ingestion) – because to do otherwise requires a fundamental reassessment of the chemical culture in which we live – a spate of information in recent years is forcing us to confront the fact that the constituents of our daily lives may be putting us all at risk. In much of this recent research, male reproductive health has risen to the fore. Many chemicals are implicated in the latest warnings regarding male reproductive health. Among the most commonly cited are numerous pesticides, whose pervasive use makes their potential role in this problem worthy of the scrutiny it is beginning to receive in the scientific and public health communities

Trends in Recent Years

One of the most striking aspects of imperiled male reproductive health is the array of disorders being examined and the consistency of changes in their incidence patterns. The most widely publicized of these disorders is falling sperm counts. Riven with controversy, the question of whether sperm counts have dropped in recent decades has been the subject of heated debate in the medical literature following the 1992 publication of a landmark paper that analyzed 61 existing studies of semen quality and revealed a worldwide pattern of declining sperm counts.² After publication, these findings were echoed by some and disputed by others. In the ensuing years, a few widely publicized studies that did not find such declines were offered as evidence that if this effect were indeed occurring at all, it was not a geographically uniform phenomena.³ But studies showing declines, some specifically in younger men, also continued to appear.⁴ In 1997, the continuing controversy led to a reanalysis of the 61 studies previously examined that reaffirmed the earlier finding of a real drop in sperm counts, although with regional variations.⁵

This latest analysis cannot be considered to have settled the question, but the debate should be viewed within the context of other afflictions that both compromise male fertility and signal underlying problems. Abnormal male genital development, for example, influences reproductive capacity, and may reflect subtler damage as well. The most dramatic example of this effect is the case of the male alligators of Lake Apopka, Florida, whose large exposures to

a brew of pesticides is the presumed cause of their abnormally small penises, malformed testes, and subsequent inability to mate successfully.⁶ Rather than being simply a newsworthy anomaly, the Lake Apopka alligators may well be sentinels, warning of exposures that endanger humans. In fact, we need not look very far afield for indications that this could be the case. During the past few decades there has been a steadily rising incidence of the male genital abnormality hypospadias (a defect of the urethral opening on the penis).⁷ Cryptorchidism (undescended testes) may also be on the rise.⁸ Both anomalies have been associated in the past with maternal exposure to the synthetic hormone diethylstilbestrol – DES – given to pregnant women in the 1950s and 1960s,⁹ because it disrupts the delicate hormonal balance within the womb.¹⁰ There is no definitive word on why these more general increases are occurring (or even if they definitely are, in the case of cryptorchidism), but studies showing an association of these defects with the endocrine mimicking drug DES, and the emerging reports in the literature regarding associations between endocrine-disrupting chemicals and other pesticides (see below) with these defects, are telling coincidences, worthy of detailed investigation.

In tandem with rising genital abnormalities, the incidence of testicular cancer has surged in recent decades and it is now the most common malignancy in young men in many countries throughout the world.¹¹ Primarily a disease of adolescent and young men, testicular cancer is

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speculated to have its origins in utero, or in early life, during testicular development, and to begin burgeoning at or after adolescence in the presence of greater sexual hormone levels.¹²

Intersections

Reciting such a laundry list of list of adverse health effects, while the simplest way to order the record, obscures a key fact: these disorders are inextricably connected. It is well established, for example, that males born with genital abnormalities experience higher rates of testicular cancer later in life than males with normal genitalia at birth.¹³ Infertile men have higher rates of testicular cancer than fertile men, and problems with sperm production are often associated with genital abnormalities and testicular cancer.¹⁴ The relationships between these effects form a complex web of interactions, although it is not yet possible to make blanket statements about whether they spring from similar causes, whether one effect causes another, or whether the association results from some combination of these.

Because these disorders are connected, the confluence of their incidence patterns is more compelling than any of the component studies could be on their own. And while much of the course of scientific debate by necessity focuses on the merits of individual studies – design, assumptions, and statistics – many scientific observers are also stepping back to view this larger picture. There is a growing chorus of voices asking why.¹⁵

Endocrine Disruption and the Particular Case of Pesticides

Topping the list of culprits are chemicals, known as endocrine-disruptors, that interfere with the hormone system by either mimicking or impeding natural hormones, thereby disturbing the finely-tuned balance of hormones necessary for virtually aspects of maturation and daily functioning.¹⁶ Such disturbances, particularly at critical developmental stages in utero, can have profound and life-long repercussions – DES furnishes a tragic example of this effect. The prospect that widespread exposure to

chemicals that disrupt the endocrine system may underlie the general decline of male reproductive health as well as other adverse health effects (such as rising breast cancer rates and wildlife pathologies) has garnered serious attention and led to a major new federal program, established under the federal Food Quality Protection Act of 1996, to test all chemicals for this effect.

Though we have yet to examine the bulk of pesticides for endocrine disruption capability, many are al-



ready known to do so – both banned pesticides such as DDT, and those in widespread current use, such as atrazine and related herbicides, and pyrethroids such as permethrin.¹⁷ Still other pesticides exert direct toxic effects on various aspects of the male reproductive system.

Whether through endocrine disruption or other mechanisms, pesticides have been repeatedly linked to the major disorders of male fertility delineated above. The soil fumigant dibromochloropropane (DBCP) banned because it rendered male workers infertile, is the best example of a pesticide that targets sperm quality,¹⁸ but a partial list of other pesticides shown to cause deterioration in sperm quantity or quality, or testicular pathology (which can interfere with normal sperm production) includes: the insecticides malathion,¹⁹ carbaryl (the active ingredient in Sevin) and carbofuran,²⁰ the herbicides glyphosate

(the active ingredient in Roundup),²¹ isoproturon,²² molinate,²³ and 2,4-D;²⁴ and the fungicide Thiram.²⁵

General examinations of pesticide exposure and male fertility reinforce these pesticide-specific findings. Studies have found that men who have had long-term or high exposures to pesticides in the agricultural setting have abnormal sperm production,²⁶ higher rates of miscarriages and premature birth in their offspring,²⁷ and their mates require longer times to achieve pregnancy.²⁸

Pesticides have also been implicated in birth defects in general, both as the result of maternal exposure while pregnant and paternal exposure prior to conception,²⁹ and genital defects in particular. An examination of the birth defects registry for Norway over a span of more than two decades, found that both cryptorchidism and hypospadias were associated with a number of indicators of pesticide exposure, most notably finding that hypospadias was more prevalent in infants conceived during the growing season.³⁰ A Spanish study found a geographical association with overall pesticide use in a community and higher incidence of cryptorchidism.³¹ And closer to home, Minnesota researchers found an excess of several birth defects, urogenital anomalies (a blanket term for related anomalies that includes hypospadias and cryptorchidism) among them, in the children of pesticide applicators. The incidence of these and other anomalies was higher for infants conceived during the pesticide application season, for male children overall, and particularly higher for male children of pesticide applicators relative to the general population.³²

Whether or not pesticide exposure contributes to development of testicular cancer is less clear. Studies examining testicular cancer incidence relative to pesticide exposure have resulted in conflicting conclusions.³³ Since at least some portion of testicular cancer cases are theorized to originate in utero (and possibly only during certain stages of development in utero), examining only adult exposures

to pesticides and subsequent testicular cancer rates may well miss a significant portion of risk. To date, only a handful of studies have looked at parental exposure and later development of testicular cancer in offspring, and only one of those specifically looked at pesticide exposure and testicular cancer risk in offspring. That study found an increased risk of testicular cancer for farmers' sons but those risks did not also correlate with the study's

“EPA believes that most pesticides – despite having an EPA registration – have not been adequately studied to determine their effects on people or the environment.”

measures of pesticide exposure.³⁴ With such limited data, however, the dual connections between testicular cancer and other male reproductive abnormalities and between those other abnormalities and pesticide exposure render the connection a plausible one that awaits further examination.

Sex-ratio

A final note of interest: the proportion of male to female births, known as the sex ratio, has been called an “unambiguous measure of the reproductive health of large populations.”³⁵ Sex ratio is not a constant, but a shifting balance subject to alteration by many factors, including toxic exposures (which have been shown to decrease the number of male births relative to female births).³⁶ Although not strictly a measure of male reproductive health in the same manner as sperm counts and abnormalities in male reproductive systems, skewed sex ratios represent an even greater peril: threatening the very existence of males. As with other male reproductive disorders, several recent studies and review articles, with profound implications for the health of our species, have found evidence that sex ratios across the globe may be declining.³⁷ Revelations about sex-ratios are the latest warning shot across the bow.

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What You Can Do

Global trends indicate the need for global solutions, and it may seem at first glance that localized efforts to stem this tide would be ineffective. But many of the toxic exposures being called into question with relationship to male reproductive health are themselves quite local, particularly in the case of high pesticide exposures. While national and international efforts to control suspected endocrine disruptors are essential, there is ample reason to demand a response at the state and local levels as well.

As noted above, we already know that certain pesticides are both endocrine disruptors and widely used, and as the federal testing program proceeds we will have information on many more. We also have some information on pesticides that directly affect the reproductive system (California, for example, maintains an ever-expanding list of these for its own regulatory purposes). The New York State Department of Environmental Conservation (DEC) has the authority to revoke the availability (through its pesticide registration process) of any pesticide in the State. Citizens concerned with their exposure to endocrine disrupting materials can let their State legislators, the Governor, and other State policy-makers know that they do not want these pesticides allowed in commerce in the State. DEC should not allow pesticides containing endocrine disruptors from being sold and used in the State, and if they are reluctant to act, the State legislature should step into the void and enact legislation to accomplish this.

In addition, local initiatives to phase-out the use of the of pesticides on municipal property have already been enacted in several communities across New York State. By doing so, municipalities not only reduce actual exposure to toxic chemicals with a wide range of adverse health and environmental effects, reproductive risks among them, but

also demonstrate to other communities and the public at large that toxic chemicals are not necessary to control pest problems.

For more information on specific chemicals, contact information for policy-makers, and enacting municipal pesticide phase-outs in your community, please contact Environmental Advocates (518-462-5526) or NYPIRG (518) 436-0876.

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