# ALTERING OREGON'S OREGON'S DESTINY: HORMONE-DISRUPTING PESTICIDES IN THE WILLAMETTE RIVER

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## **INTRODUCTION**

As we wrestle with the question of how much chemical contaminants are contributing to the trends and societal patterns we see — in breast cancer, prostate disease, infertility, and learning disabilities — it is important to keep one thing in mind. Scientists keep finding significant, often permanent effects at surprisingly low doses. The danger we face is not simply death and disease. By disrupting hormones and development, these synthetic chemicals may be changing who we become. They may be altering our destinies.

- From Our Stolen Future (1996:197)

Zoologist Theo Colborn and the other authors of *Our Stolen Future* pose a bold hypothesis: low doses of certain synthetic chemicals in the environment can mimic hormones and disrupt natural growth and development in animals and humans (Colborn, Dumanoski, and Myers 1996). A wide array of animal and human studies link these "hormone-disrupting chemicals" to a profusion of problems, including: infertility; genital deformities; low sperm counts; hormonally-triggered human cancers (e.g., breast, prostate gland); neurological disorders in children (e.g., hyperactivity); and reproductive problems in wildlife. In these ways, human alteration of the environment appears to be profoundly transforming the fundamental life processes of birth, growth, and death.

A number of widely-used pesticides are among the chemicals associated with hormone disruption. The term pesticide includes, for example, herbicides to control weeds, fungicides to kill fungi, insecticides to control insects, and so on. Pesticides are the only class of toxic chemicals intentionally introduced into the environment to kill or damage living organisms. Yet, pesticides rarely stay where the applicator intends them. In fact, researchers at Cornell University estimate that over 99% of the pesticide applied does not reach the target pest and instead moves into ecosystems to contaminate the land, air, and water (Pimentel and Levitan 1988).

Here in Oregon, the widespread use of pesticides over the past 50 years has led to contamination of the Willamette River and its tributaries. Recent government studies of the Willamette River Basin found pesticides in the water with alarming regularity. Could these pesticides be altering Oregon's destiny in ways that the authors of *Our Stolen Future* suggest?

NCAP produced this report in order to take a closer look at this question. Specifically, we wondered which of the pesticide pollutants most frequently detected in the Willamette River are known to be associated with hormone disruption in general, and breast cancer and male fertility problems in particular.

## **PESTICIDES IN THE WILLAMETTE**

As the largest river within the boundaries of Oregon, the Willamette holds a special place in the hearts of many Oregonians. Draining an 11,500 square-mile area in the northwestern portion of the state between the Coast Range and the Cascade Mountains, the basin includes about 12% of Oregon's land area. Arguably the most important river in the state, the Willamette hosts some of the best agricultural and forest lands. Furthermore, with our three largest cities — Eugene, Salem, and Portland — situated along the banks of the river, nearly 70% of the state's population lives, works, and plays within the basin.

Recognizing the river's importance and a variety of lingering pollution problems within the basin, the Oregon Legislature initiated the multiphase Willamette River Basin Water Quality Study in 1990. As part of this study, the U.S. Geological Survey (USGS) conducted an analysis for trace elements, volatile organic compounds, organochlorine compounds, and pesticides during 1992-1994 (Anderson, Rinella, and Rounds 1996). The USGS analyzed samples from 40 sites for 94 different dissolved pesticides or their metabolites. (A metabolite is a compound derived by chemical action from a parent compound.)

Released at the end of 1996, the USGS study reports some startling findings of contamination from pesticides:

\* Overall, 48 of the 94 pesticides that the USGS looked for were found at the 40 sites studied in the basin.

\* At each sampling site, the median number of pesticides detected was eight.

\* At ten of the 40 sites where samples were gathered, between 16 and 34 pesticides showed up in the analyses.

\* Eight organochlorine compounds were detected at 14 sites. These included lindane, dieldrin, and DDT which were in about 30% of the samples.

\* Atrazine, metolachlor, simazine, and diuron were the four most commonly detected pesticides in the basin.

"Overall, 48 pesticides were found at the 40 sites studied in the basin."



### BARRIERS TO UNDERSTANDING PESTICIDE USE IN THE BASIN

Significant amounts of a wide variety of pesticides are used within the Willamette River Basin in crop production, on forest land, along rights-of-way, and in urban areas. But no one knows for sure how much. That's because there is no comprehensive mechanism to keep track of and publish information on where those pesticides are applied, for what purposes they are employed, and how much of them are used annually. We do know that in 1996 chemical companies registered nearly 8,700 pesticide products for use in Oregon, according to the Oregon Department of Agriculture.

Also, Oregon State University researchers collected voluntary survey data in 1987 to estimate annual pesticide use. This was the last inclusive look at the range of pesticide uses in Oregon (although some pesticides were not included). They tabulated 199 pesticide ingredients totalling over 16 million pounds of pesticides used that year across Oregon (Rinehold and Witt 1987; Rothlein 1996). Approximately 5 million pounds or 26% of that total estimate were used in the nine counties in the basin in 1987 (Anderson, Rinella, and Rounds 1996:10).

In conducting its analysis of the Willamette, the USGS relied on the 1987 pesticide usage estimates to try to identify a correlation between the frequency of detection and the estimates of pesticide usage. In relying on such old information, the USGS scientists recognized that these "best available" data are "neither current nor comprehensive" and that they do not provide site-specific information (Anderson, Rinella, and Rounds 1996:7).

The paucity of good data on pesticide use means that it is difficult to draw useful conclusions about the relationship of particular pesticide practices to the contamination problem. More importantly, it is difficult to put our emphasis where it should be — on preventing water contamination in the first place.

## HORMONE-DISRUPTION AND THE RIVER'S TOP 25 PESTICIDES

Convincing toxicological evidence now exists that a number of pesticides and industrial chemicals have disruptive effects on the endocrine system (see Colborn, Dumanoski, and Myers 1996; Colborn, vom Saal, and Soto 1993). The endocrine system is composed of glands and hormones that act as messengers in the body in order to regulate growth, development, behavior, and sexuality. When the body mistakes synthetic chemicals for natural hormones, it reacts to them in ways that can cause irreversible damage, especially when exposure occurs during the critical period of development before and immediately after birth. Colborn and her colleagues have reviewed a wide variety of studies that link hormone-disrupting chemicals to problems like infertility; genital deformities; low sperm counts; hormonally-triggered human cancers (e.g., breast, prostate gland); neurological disorders in children (e.g., hyperactivity); and low reproductive rates in wildlife.

Did any of the pesticides that have been associated with hormone disruption show up in the Willamette River Study? That study documents the 25 pesticides that were most frequently detected in their analyses of the water in the Willamette and its tributaries (Anderson, Rinella, and Rounds 1996:34). NCAP compared the "top 25" list to information on selected health problems that have been associated with those chemicals. Specifically, we were interested in identifying which of the top 25 pesticides have been associated with disruption of the hormone system in general, and with breast cancer and male fertility problems in particular.

Table 1 summarizes the results of our comparison. The first two columns indicate the top 25 pesticides (or their break-down products) and the percent of samples that were found to be contaminated with the analyte. Analysis of the table reveals findings that may alter Oregon's destiny in disturbing ways:

#### \* Five of the top 25 pesticide pollutants in the Willamette River are found on a list of chemicals reported to have reproductive and hormone-disrupting effects, as shown in column 3 (see Colborn, vom Saal, and Soto 1993).

In order of frequency of detection, these include: atrazine, carbaryl, metribuzin, trifluralin, and 2,4-D.

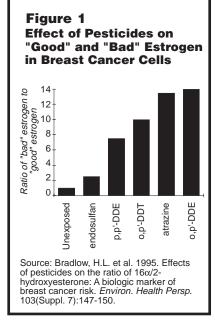
#### \* Three of the top 25 have been linked to breast cancer.

Breast cancer is a disease whose frequency has been increasing in the United States over the last forty years. Sixteen currently used pesticides have been linked with breast cancer in laboratory tests (see Cox 1996a for a review). Many of these are triazine herbicides, some of the most commonly used weed killers in the United States. Disturbingly, three of these triazines showed up with great frequency in the Willamette:

Atrazine. Feeding atrazine to rats caused increases in benign and malignant mammary gland tumors in females (US EPA 1994a). One study found that it increased the incidence of typically rare benign mammary tumors in males (Pinter et al. 1990). A series of studies has shown that women who have been exposed to higher levels of the organochlorine insecticide DDT have a risk of breast cancer that is between two and four times the risk of cancer in women with lower exposures. Laboratory studies indicate that DDT and its breakdown products promote the formation of a hormone, nicknamed "bad" estrogen, that is associated with breast cancer (see Cox 1996a for a review of these studies). As Figure 1 illustrates, atrazine is nearly as potent as the DDT breakdown product DDE in promoting the formation of the "bad" estrogen,  $16\alpha$ -hydroxyestrone, that has been linked with breast cancer (Bradlow et al. 1995).

*Simazine*. Feeding rats simazine caused an increase in the incidence of malignant mammary gland tumors in females (US EPA 1994a).

*Prometon.* In feeding studies with rats, prometon causes mammary tumors in females (US EPA 1997a).



Endosulfan, atrazine, and DDT all promote the formation of "bad" estrogen. In this experiment, concentrations between 2 and 4 parts per million were used.

## Table 1. Selected health problems known to be associated with the25 most frequently detected pesticides and metabolites in theWillamette River Basin, 1992-1994

Analyte	Percent of samples <sup>b</sup> contaminated in the Willamette <sup>1</sup>	Reported to have hormone- disrupting and reproductive effects <sup>2</sup>	Linked to breast cancer <sup>3</sup>	Linked to male fertility problems⁴
Atrazine	90	YES <sup>2*</sup> *	YES <sup>6,7,8</sup> * *	YES <sup>9</sup> * *
Simazine	82	* *	YES <sup>6</sup> * *	YES <sup>10</sup> * *
Metolachlor	81			
Desethylatrazine <sup>a</sup>	61	* *	* *	* *
Diuron	54			
Hexazinone	48	* *	* *	* *
Diazinon	47	* *	* *	YES <sup>11</sup> * *
Cycloate	43	* *	* *	* *
Desisopropylatrazine <sup>a</sup>	40	* *	* *	* *
Terbacil	37	* *	* *	* *
DCPA (Dacthal)	35	* *	* *	* *
EPTC	32	* *	* *	* *
Napropamide	29	* *	* *	* *
Prometon	29	* *	YES⁵ * *	* *
Chlorpyrifos	26	* *	* *	YES <sup>12,13*</sup> *
Ethoprop	26	* *	* *	* *
Fonofos	26	* *	* *	* *
Carbaryl	23	YES <sup>2*</sup> *	* *	YES <sup>14,15</sup> * *
Carbofuran	23	* *	* *	YES <sup>16,17*</sup> *
Tebuthiuron	23			
Metribuzin	21	YES <sup>2*</sup> *	* *	* *
Pronamide	20			YES <sup>18</sup>
Trifluralin	17	YES <sup>2</sup>		
Trichlopyr	13	* *	* *	* *
2,4-D	12	YES <sup>2*</sup> *	* *	YES <sup>19-22</sup> * *

Despite limited testing, 11 of the top 25 pesticides in the Willamette Basin have already been associated with some form of hormone-disruption.

<sup>a</sup> Both desethylatrazine and desisopropylatrazine are metabolites of atrazine. A metabolite is compound derived by chemical action from a parent compound. A metabolite is sometimes more toxic than the original compound.

<sup>b</sup> Detection data based on 58-93 samples depending on the chemical analyzed.

\* \* These pesticides have not been tested according to EPA's current scientific standards for product registration (see page 7).

### \* Eight of the top 25 pesticides found in the Willamette have been linked to male fertility problems (see Table 1, Column 4).

Around the world, sperm counts in healthy men have fallen about 50% in the last 50 years (Carlsen et al. 1992). Over 50 currently used pesticides have caused problems related to male fertility in laboratory and clinical tests. Some of these are among the most commonly used. Estimated annual use of these chemicals totals nearly 200 million pounds, about 25% of total agricultural pesticide use (see Cox 1996b for a review). Those pesticides frequently found in the Willamette that are putting male fertility at risk include the following:

Atrazine. This herbicide has been found to interfere with testosterone (sex hormone) metabolism and binding in rats (Kniewald et al. 1995).

*Simazine.* One study found that the herbicide simazine atrophied testes in sheep (Dshurov 1979).

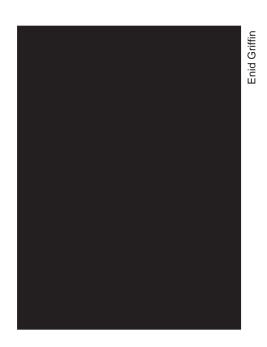
*Diazinon.* This insecticide has atrophied testes and arrested sperm production in dogs (US Dept. of Health and Human Services 1994).

*Chlorpyrifos.* This insecticide caused damage to semen-producing structures in testes in rats (Mikhail 1979), and caused undescended testicles in boys exposed prenatally (Sherman 1995).

*Carbofuran.* This insecticide decreased libido (sexual drive) and sperm number in rabbits (Yousef 1995), and decreased number and motility of sperm in rats (Pant et al. 1995).

*Pronamide.* A weed killer, pronamide has been found to increase testicular tumors in rats, with some effects on the concentration of sex hormones (US EPA 1994b).

2,4-D This herbicide has decreased sperm number and motility, and it has increased the proportion of abnormal sperm in exposed farmers (Lerda and Rizzi 1991). 2,4-D has also inhibited the synthesis of DNA (genetic material) in testes of rats (Seiler 1979). In addition, 2,4-D is contaminated with 2,3,7,8-TCDD which reduces sperm number in prenatally exposed rats (Gray et al. 1995; US EPA 1994c).



## **A FAILED REGULATORY PROCESS**

## \* Only 5 of the top 25 Willamette pesticide pollutants have met current federal testing requirements for EPA registration. These include: metolachlor, diuron, tebuthiuron, pronamide, and trifluralin.

The national pesticide law, the Federal Insecticide, Fungicide and Rodenticide Act or FIFRA, says that all pesticides must be "registered" by the U.S. Environmental Protection Agency (EPA). But the regulatory process has never been able to cope with the huge number of products used as pesticides, so that many of the pesticides in use today do not meet the requirements of the law.

In 1972, Congress instructed EPA to "reregister" the 50,000 products that were already on the market at that time (Bosso 1987; US GAO 1986). Reregistration involves bringing all health and safety testing up to current standards and considering concerns about chronic toxicity and environmental pollution.

By the late 1980s, little progress had been made. As a result, Congress passed FIFRA amendments in 1988 that required a strict time line for industry submission of registration tests. When all the required tests have been evaluated by EPA and the agency determines that the pesticide does not cause unreasonable adverse effects when used according to the label, EPA issues a Reregistration Eligibility Decision (US EPA 1991).

However, EPA has fallen behind in meeting Congress's scheduled goals set in 1998. As of March 1997, 380 of the 604 active pesticide ingredients requiring reregistration were still being supported by pesticide manufacturers. (Supported pesticides are those for which the manufacturer pays registration fees and submits reregistration data to EPA.) Of these 380, only 148 had completed the reregistration process. EPA now estimates that the reregistration process will take until 2002 (US EPA 1997b).

Although there are many flaws in the registration process, the point is that EPA is hopelessly behind in its effort. Clearly, the federal government has failed to protect public health and the environment from the hazards posed by widespread pesticide use. While we wait for the EPA to do its job, the Willamette remains polluted with a host of pesticides which are not tested by current standards and which threaten Oregon's future.

## **POLICY RECOMMENDATIONS**

The Willamette River Study is a wake-up call for Oregon to take action to halt pesticide contamination. NCAP has these three recommendations:

1. The state of Oregon should improve public information on pesticide use. Some states require pesticide applicators to report the types and quantities of pesticides they use. Here in Oregon, we need better information on pesticide use — whether on farms, at schools, or suburban lawns — to protect our health and safeguard our water from contamination. To reverse the water contamination trends discussed in this report, we need accurate information about the sources of pesticide pollution. Without it, we are unable to form rational plans based on prevention of contamination. And we cannot focus on finding the alternatives for the pesticide uses that cause the greatest problems.

Also, without accurate information on the types and amounts of pesticides that people are exposed to, health researchers find it very difficult to understand the relationship between exposure and illness. As a result, we cannot design good laws to avoid the risks, and are unable to evaluate efforts to reduce pesticide use in agriculture and other settings.

2. Pesticide users should adopt alternatives to pesticides that pollute our water and that threaten humans and wildlife with hormone-disruption. Oregon pesticide applicators need to stop using the pesticides that are most frequently detected in the Willamette River. An increasing number of innovative pest management strategies prevent pest problems so as to avoid the need for pesticides. Indeed, across the country and around the globe farmers are finding that sustainable production practices allow them to be successful economically and to be good stewards of their land.

**3.** Consumers should support farmers and other pest managers who adopt alternative management strategies. Oregonians can support farmers and others who are using the successful and cost-effective alternatives that are available. Sales of organic foods have sky-rocketed growing by more than 26% to 3.5 billion dollars in 1996 (Natural Foods Merchandiser 1996).

## **REFERENCES** FOR TABLE 1

- Anderson, C. W., F. A. Rinella, and S. Rounds. 1996. Occurrence of selected trace elements and organic compounds and their relation to land use in the Willamette River Basin, Oregon, 1992-1994. Water-Resources Investigations Report 96-4234. Portland, OR: U.S. Geological Survey.
- Colborn, T., F. S. vom Saal, and A. M. Soto. 1993. Developmental effects of endocrine-disrupting chemicals in wildlife and humans. *Environ. Health Persp.* 101(5):378-383.
- Cox, C. 1996. Pesticides and Breast Cancer: Prevention is crucial. *Journal of Pesticide Reform* 16(1):2-7.
- 4. Cox, C. 1996. Masculinity at risk. *Journal of Pesticide Reform* 16(2):2-7.
- U.S. Environmental Protection Agency, Office of Pesticide Programs. 1997. List of chemicals evaluated for carcinogenic potential. Washington, D.C. (February).
- U.S. Environmental Protection Agency. 1994. Atrazine, simazine, and cyanazine: Notice of initiation of special review. *Federal Register* 59(225):60412-60443.
- Pintér, A. et al. 1990. Long-term carcinogenicity bioassay of the herbicide atrazine in F344 rats. *Neoplasma* 37(5):533-544.
- Bradlow, H.L. et al. 1995. Effects of pesticides on the ratio of 16/v/2-hydroxyesterone: A biologic marker of breast cancer risk. *Environ. Health Persp.* 103(Suppl. 7):147-150.
- Kniewald, J. et al. 1995. Effect of s-triazine compounds on testosterone metabolism in the rat prostate. J. Appl. Toxicol.15:215-218.
- Dshurov. A. 1979. Histological changes in organs of sheep in chronic simazine poisoning. [German with English abstract.] *Zbl. Vet. Med.* A26:44-54.
- US Dept. of Health and Human Services. Public Health Service. Agency for Toxic Substances and Disease Registry. 1994. Toxicological profile for diazinon. (Draft.) (August.) p.41.
- Mikhail, T.H. et al. 1979. Acute toxicity of organophosphorus and organochlorine insecticides in laboratory animals. Z. Ernährungswiss 18:258-268.
- Sherman, J.D. 1995. Chlorpyrifos (Dursban)-associated birth defects: A proposed syndrome, report of four cases, and discussion of the toxicology. *Intern. J. Occup. Med. Toxicol.* 4:417-431.
- Wyrobeck, A.J. et al. 1981. Sperm shape abnormalities in carbaryl-exposed employees. *Environ. Health Persp.* 40:255-265.
- Shtenberg, A.I. and M.N. Rybakova. 1968. Effect of carbaryl on the neuroendocrine system of rats. *Fd. Cosmet. Toxicol.* 6:461-467.
- Pant, N. et al. 1995. Effect of oral administration of carbofuran on male reproductive system of rat. *Hum. Exp. Toxicol.* 14:889-894.
- Yousef, M.I. 1995. Toxic effects of carbofuran and glyphosate on semen characteristics in rabbits. *J. Environ. Sci. Health.* B30:513-5343.
- US Environmental Protection Agency, Office of Prevention, Pesticides and Toxic Substances. 1994. Reregistration Eligibility Decision (RED): Pronamide. Washington, D.C. (May.) p.11.
- Lerda, D. and R. Rizzi. 1991. Study of reproductive function in persons occupationally exposed to 2,4-dichlorophenoxyacetic acid (2,4-D). *Mut. Res.* 262:47-50.
- Seiler, J.P. 1979. Phenoxyacids as inhibitors of testicular DNA synthesis in male mice. *Bull. Environ. Cont. Toxicol.* 21:89-92.
- U.S. Environmental Protection Agency, ORD. 1994. Estimating exposure to dioxin-like compounds. Vol. II. Properties, sources, occurrence and background exposures. Washington, D.C. (June.) p.3-58.
- Gray, L.E. et al. 1995. Exposure to TCDD during development permanently alters reproductive function in male Long Evans rats and hamsters. *Toxicol. Appl. Pharmacol.* 131:108-118.

## **REFERENCES FOR REPORT**

- Anderson, C. W., F. A. Rinella, and S. A. Rounds. 1996.
  Occurrence of selected trace elements and organic compounds and their relation to land use in the Willamette River Basin, Oregon, 1992-1994. Water-Resources Investigations Report 96-4234.
  Portland, OR: U.S. Geological Survey.
  Bosso, C.J. 1987. Pesticides and politics: The life cycle
- Bosso, C.J. 1987. Pesticides and politics: The life cycle of a public issue. Pittsburgh, PA: Univ. of Pittsburgh Press.
- Bradlow, H.L. et al. 1995. Effects of pesticides on the ratio of 16/a/2-hydroxyesterone: A biologic marker of breast cancer risk. *Environmental Health Perspectives* 103(Suppl. 7):147-150.
- Carlsen, E. et al. 1992. Evidence for decreasing quality of semen during the past 50 years. *British Medicine Journal* 305:609-613.
- Colborn, T., D. Dumanoski, and J. P. Myers. 1996. *Our* Stolen Future: Are We Threatening Our Fertility, Intelligence, and Survival? — A Scientific Detective Story. New York: Penguin Books.
- Colborn, T., F. S. vom Saal, and A. M. Soto. 1993. Developmental effects of endocrine-disrupting chemicals in wildlife and humans. *Environmental Health Perspectives* 101(5):378-384.
- Cox, C. 1996a. Pesticides and breast cancer: Prevention is crucial. Journal of Pesticide Reform 16(1):2-7.
- Cox, C. 1996b. Masculinity at risk. Journal of Pesticide Reform 16(2):2-7.
- Dshurov. A. 1979. Histological changes in organs of sheep in chronic simazine poisoning. [German with English abstract.] *Zbl. Vet. Med.* A26:44-54.
- Gray, L.E. et al. 1995. Exposure to TCDD during development permanently alters reproductive function in male Long Evans rats and hamsters. *Toxicol. Appl. Pharmacol.* 131:108-118.
- Kniewald, J. et al. 1995. Effect of s-triazine compounds on testosterone metabolism in the rat prostate. *Jour*nal of Applied Toxicology. 15:215-218.
- Lerda, D. and R. Rizzi. 1991. Study of reproductive function in persons occupationally exposed to 2,4-dichlorophenoxyacetic acid (2,4-D). Mut. Res. 262:47-50.
- Mikhail, T.H. et al. 1979. Acute toxicity of organophosphorus and organochlorine insecticides in laboratory animals. Z. Ernährungswiss 18:258-268.
- Natural Foods Merchandiser. 1997. Sales of natural products soar over 25% in 1996. News release dated June 23. Boulder, CO.
- Pant, N. et al. 1995. Effect of oral administration of carbofuran on male reproductive system of rat. *Hum. Exp. Toxicol.* 14:889-894.
- Pimentel, D. and L. Levitan. 1988. Pesticides: Amounts applied and amounts reaching pests. *Bioscience* 36(2):86-91.
- Pintér, A. et al. 1990. Long-term carcinogenicity bioassay of the herbicide atrazine in F344 rats. *Neoplasma* 37(5):533-544.
- Rinehold, J. W. and J. M. Witt. 1989. Oregon Pesticide Use Estimates for 1987. Corvallis: Oregon State University Extension Service.
- Rothlein, J. 1996. Oregon Chemical Surveillance Project. Portland, OR: Center for Occupational and Environmental Toxicology, Oregon Health Sciences University.
- Seiler, J.P. 1979. Phenoxyacids as inhibitors of testicular DNA synthesis in male mice. Bull. Environ. Cont. Toxicol. 21:89-92.
- Sherman, J.D. 1995. Chlorpyrifos (Dursban)-associated birth defects: A proposed syndrome, report of four cases, and discussion of the toxicology. *Intern. J. Occup. Med. Toxicol.* 4:417-431.
- Shtenberg, A.I. and M.N. Rybakova. 1968. Effect of carbaryl on the neuroendocrine system of rats. Fd. Cosmet. Toxicol. 6:461-467.
- U.S. Dept. of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. 1994. Toxicological profile for diazinon. (Draft.) (August.).
- U.S. Environmental Protection Agency, Pesticides and

Toxic Substances. 1991. Pesticide reregistration. 21T-1004. Washington, D.C. (March).

- U.S. Environmental Protection Agency. 1994a. Atrazine, simazine, and cyanazine: Notice of initiation of special review. *Federal Register* 59(225):60412-60443.
- US Environmental Protection Agency, Office of Prevention, Pesticides and Toxic Substances. 1994b. Reregistration Eligibility Decision (RED): Pronamide. Washington, D.C. (May.) p.11.
- U.S. Environmental Protection Agency, ORD. 1994c. Estimating exposure to dioxin-like compounds. Vol. II. Properties, sources, occurrence and background exposures. Washington, D.C. (June.) p.3-58.
- U.S. Environmental Protection Agency, Office of Pesticide Programs. 1997a. List of chemicals evaluated for carcinogenic potential. Washington, D.C. (February).
- U.S. Environmental Protection Agency, Prevention, Pesticides and Toxic Substances. 1997b. 1996 Food Quality Protection Act: Implementation Plan. Washington, D.C. (March).
- U.S. General Accounting Office (GAO). 1986. Pesticides: EPA's formidable task to assess and regulate their risks. Washington, D.C. (April).
- U.S. General Accounting Office (GAO). 1991. Better data can improve the usefulness of EPA's benefit assessments. RCED-92-32. Washington, D.C. (December).
- Wyrobeck, A.J. et al. 1981. Sperm shape abnormalities in carbaryl-exposed employees. *Environmental Health Perspectives* 40:255-265.
- Yousef, M.I. 1995. Toxic effects of carbofuran and glyphosate on semen characteristics in rabbits. *J. Environ. Sci. Health.* B30:513-5343.