Chapter 6
Pesticides and Reproduction

There were 3.6 million babies born alive in the U.S. in 2001, and about 15,000 born dead. Estimates of spontaneous abortion, or early fetal deaths before viability, range from 50 to 65% of human conceptions. About 3% of babies have a major birth defect, which the Centers for Disease Control and Prevention (CDC) defines as "a structural or chromosomal anomaly present at birth and recognized before age six". Birth defects are the leading cause of infant mortality, accounting for about 20% of deaths. Many who desire children cannot have them because of primary infertility, estimated to affect 11% of couples in the U.S. Known risk factors, include infectious disease, advanced maternal age, tobacco, alcohol and drug use, among others, which account for less than half of adverse reproductive outcomes in humans.

Many pesticides are known to be toxic to the embryo (embryotoxic) and fetus (fetotoxic) in laboratory animals. Studies of pesticides in humans show adverse reproductive effects in males and females, and in the developing child.

Most pesticide-related reproductive disorders are not a result of acute poisoning. In one of the most severe incidents ever reported, factory workers made sterile by the soil fumigant DBCP (dibromochloropropane), had no signs or symptoms of a pesticide-related health problem during the months and years that the pesticide was destroying their ability to produce sperm. It was not until men who had fathered children noticed that they were not having the additional children they wished, that the problem was discovered.

A study in New York City found widespread pesticide use and exposure during pregnancy among a cohort of African-American and Dominican women. Pest control measures were used in the home by 85% of the women. Eight pesticides were found in 45% or more of the women . The four pesticides found in the highest concentration in personal air samples of women monitored during the third trimester were diazinon, chlorpyrifos, propoxur, and ortho-phenylphenol[1].

Birth defects (Tables 1-A, 1-B)
Studies in North America found a 166% to 249% increase in risk if the mother lived in a pesticide use area in Canada , California, or Minnesota, or if the father was a pesticide applicator. No associations with pesticide exposure were found in the malathion medfly spray program in California, or from heptachlor contaminated milk episode in Hawaii.

Studies in South America found a 210% increase for female flower workers in Colombia with the highest pesticide exposure , which decreased 40% in the low exposure group. The wives of male flower workers had a 30% increase in risk A study in Chile attributed more than half of the risk of birth defects to the mother's occupational pesticide exposure .

In Europe a 316% increase was found in Spain if the mother was an agricultural worker, but a non-significant 50% increase if it was the father. If the father handled pesticides a 49% borderline significant increase was found. The father's exposure to paraquat increased risk by 280% ; a 245% increase for glufosinate exposure was not significant. A 40% increase was found in Finland if the mother was an agricultural worker.

In Asia, a 456% increase was found in conventional Filipino farmers compared to those using IPM (Integrated Pest Management), a 429% increase in women in India who worked in pesticide sprayed cotton fields, and a non-significant increase in Chinese women exposed during pregnancy. No association with methylisocyanate exposure was found for Indian women in Bhopal[9], or in New Zealanders exposed to 2,4,5-T .

Case Reports
Pesticides associated with severe birth defects from exposure during pregnancy include 2,4-D, deet, malathion lice treatment, and metasystox-R/Phosdrin/Lannate treated crops..

Central Nervous System (excluding neural tube defects).
An increased risk of unspecified neurodevelopmental /neurobehavioral defects was reported for Minnesota applicators using glyphosate/Roundup, and the fumigant phosphine, and for hydrocephalus (spinal fluid build up in the brain) in Norway. A study in Texas found no association with maternal exposure to pesticides and Down's syndrome, which accounts for over half of all central nervous system defects.

Cleft Lip and/or Cleft Palate
The highest risk reported is a 380% increase in California for home use before and after conception . Agricultural workers were at increased risk in Finland but a 90% increase in those exposed to pesticides was not significant. An increase was found for agricultural chemical exposure in Iowa, and Michigan , in Georgia farmers, and in female farm workers in England and Wales. Living in a high pesticide use area increased risk in Arkansas and Canada . A 20% decrease was found if the father was exposed at work in England and Wales . No cases were found in 2,4,5-T applicators in New Zealand..

Cryptorchidism
Undescended testicles is one of the most common urogenital defects in males. A study in China found a 1,280% increase if the father was occupationally exposed to pesticides. A 67% increase was found in Denmark if the mother was a garden worker, but no increase if the father.

Eye Defects
Increased risk of cataracts was found if the father worked in forestry or logging in Canada, or was exposed to wood preservatives. In 1993, media reports in England alleged that clusters of anophthalmia (no eyes) and microophthalmia (eyes abnormally small), might be linked to exposure to the pesticide benomyl/Benlate®, a fungicide widely used in agriculture. Higher rates were found in rural areas of England compared to urban areas, but no association with pesticides was reported[2]. A study in Italy found a 37% decrease in risk if the parents were exposed to benomyl at work . A child born to a poisoned farm worker, had ocular hypertelorism, microopthalmia, and optic nerve colobomas among other severe defects.

Gastrointestinal Defects
Few studies report a significant association with pesticide exposure. A 232% increase was found in Norway if the parents were farmers. A 52% increase risk of tracheo-esophageal fistula was found in female farm workers in Canada; an increase in a community sprayed with malathion based on three cases was not significant.

Heart Defects
The highest risk reported is a 470% increased risk of transposition of the great arteries (TGA) if the mother exposure to rodenticides at home in the first trimester; a 280% increase found for home exposure to herbicides, and a 220% increase for any home pesticide exposure[49]. Home use increased the risk of total anomalous pulmonary venous return[51]. A California study found a 310% increased risk of cono-truncal defects for home garden pesticide use , and a 220% increase if them other used insect repellent[34]. A 203% increase was found the father worked in forestry or logging n Canada[41], and a 70% increase if the father was a licensed applicator in Minnesota[20]. Studies in Finland found no association with the mother's occupational exposure to pesticides for hypoplastic left heart syndrome[46], atrial septal defect[47], or ventricular septal defect[48].

Hypospadias
A study in Norway found a 294% increase in risk the parents were farmers[27].No association with pesticide exposure found. in male and female garden workers in Denmark[38.]

Kidney Defects
A study in Canada found a 249% increase in risk for pregnant women living in a high pesticide use area

Limb Reduction Defects
Several studies report an association of limb reduction defects with pesticide exposure. The highest risk found was a 700% increase in Australia for home use more than once in the first trimester (RR7.0), which decreased 390% if

used only once a month (RR 3.1). A 350% increased risk was found in California for periconceptual home use. A 260% increased risk was found in Washington State if the mother was a farm worker Washington State, a 231% increase in California if both parents were farm workers , and a 250% increase in Norway if the parents were farmers. In California, the risk was found to be higher if there were additional defects besides limb reduction. If the mother lived in a high pesticide use area, a 311% increase was found for multiple defects which decreased 120% if limb reduction was the only defect. If the mother lived in a high agricultural production area, a 240% increase for multiple defects decreased 70% if limb reduction was the only defect. If either parent was a farm worker a 10% decrease was found for the single defect; a 60% increase for multiple defects was not significant[53]. Of the 237 infants in the study, 67% had only limb reduction without additional defects. In the 23% with additional defects, a trend of increasing risk with increasing exposure to agricultural production was found[53].

Neural Tube Defects

Spina bifida and anencephaly result when the neural tube, which develops into the brain and nervous system, fails to close properly in the first 28 days after conception. There has been a world-wide decrease in the incidence of neural tube defects with the recognition of the importance of folic acid for normal neural tube development, and the implementation of food enrichment and vitamin supplementation programs. The studies described did not account for possible effects of folic acid. For example, the highest reported risk is a 780% increase in China if the mother was exposed to pesticides in the first trimester. Recent studies in China report a dramatic decrease in neural tube defects associated with folic acid supplementation.

If the mother was a farm worker, increased risk was found in The Netherlands (OR 5.6, OR 3.4), Finland (OR 1.9), Texas, and England; and for Swedish women living on a farm , and in Norway if the parents were farmers (OR 2.76). An increase in farm worker women in Spain was not significant, nor were increases if the father was a farm worker in The Netherlands. Non-significant increases were found if the father was exposed at work in England and Wales, with a 20% decrease in risk of spina bifida (RR 0.8), but no association with anencephaly. A 9% decrease in risk was found in Texas for gardeners and landscapers (OR 0.91).

A California study found a 290% increased risk if the mother applied pesticides in the home , which was borderline significant if the home was commercially treated. Living two and a half miles or less from a crop production area in California increased risk by 150% . An increase was found in mothers living in a high forestry/agricultural use area in Canada. A study of a cluster of neural tube defects in a suburban area of Northern California found no association with pesticide exposure.

When a high rate of neural tube defects was found in Matamoros, Mexico, across the border from Brownsville, Texas, pesticides were suspected. A study of Mexican-American women on the Texas/Mexico border found no association with the parents' exposure to pesticides.

Urogenital Defects

The most common urogenital defects are hypospadias and cryptorchidism in males. Defects in this category also include ambiguous genitalia among others. Two studies reported an increase in urogenital defects without specifying the type– a 170% increase if the father was a licensed pesticide applicator, and an increase if the father was exposed to wood preservatives and chlorophenols.

Spontaneous Abortion (Table 2)

Spontaneous abortion (miscarriage) is fetal loss in the first 20 weeks of pregnancy. The highest reported risk is a 760% increase in wives of pesticide applicators in Italy. Increased risk was found in farm wives in Minnesota and Canada , in wives of cotton field workers in India, and a 583% increase in Indian couples working in pesticide treated vineyards. A 617% increased risk was found in Filipino farmers using conventional methods compared to those using the less pesticide intensive IPM (Integrated Pest Management).

Chinese women exposed during pregnancy had a 390% increased risk of threatened abortion, and for spontaneous abortion if worked during pregnancy. A 240% increased risk was found in Canadian agricultural / horticultural workers, 220% in Colombia flower workers, 80% in Minnesota female pesticide applicators, and increased risk in Spanish greenhouse sprayers. A 90% increased risk was found in Norwegian farmers, which increased to 240% in

grain farmers with a poor yield. The authors postulate that a later harvest results in higher levels of mycotoxins which may increase risk.

Exposure of the father to pesticides can increase the risk of spontaneous abortion. A 300% increase was found in DBCP (dibromochloropropane) exposed banana workers in Israel, and in Canadian farmers. A case report from Canada found a non-significant 500% increased risk in a farm couple when the husband applied 2,4-D without using protective clothing or equipment, which decreased 250% when he used protection.

Increased risk was found in survivors of the 1984 toxic release in Bhopal, India compared to unexposed controls (PR 4.29), and in women who were pregnant at the time of the accident. Women poisoned by hexachlorobenzene in Turkey in the 1950s had a lifetime increase in risk compared to the unexposed.

Ethylene oxide (ETO) is a gas used to sterilize hospital and dental equipment. An increased prevalence of spontaneous abortion was found in Finnish hospital workers exposed during pregnancy, and a borderline significant increased risk found in California dental assistants.

Several studies found no association between pesticides and spontaneous abortion including female farm workers in Bulgaria, workers exposed to chlorinated hydrocarbon insecticides in Germany, and Italy, in a California community sprayed with malathion for medfly control, with DDE and DDT serum levels in Florida women, in crop duster pilots compared to their siblings, and in a 17 year follow-up of DBCP workers in Israel. A 10% decrease in risk was found in female greenhouse workers in Denmark (OR 0.90), and an 11% decrease in the wives of herbicide sprayers in New Zealand (RR 0.89).

Stillbirth (Table 3)
Unlike early fetal loss, which may go unrecognized and unreported, deaths of infants prior to delivery are documented and recorded. An 840% increase in Hispanics living near a pesticide factory in Texas is the highest reported. A 553% increase was found in female agricultural workers in Canada, and lower increases in California (OR 1.6), and Washington State (OR 1.5). A 329% increase was found in wives of pesticide exposed cotton field workers in India. A California study found a 240% increased risk from occupational exposure to unspecified pesticides, a 170% increase from home use, both borderline significant, and Canadian women studies. A U.S. national study of self-reported use found a 50% increase in risk if the mother used pesticides at home, a 30% increase if the father, and a 20% borderline significant increase in risk if the father was exposed at work. U.S. Navy personnel were at increased risk if either parent was exposed.

Living in a high pesticide use area in Canada increased risk 250%. A 30% increase in women living in pesticide use areas in California was borderline significant. No increase was found for community malathion spraying, in wives of Minnesota pesticide applicators, or in women in Bhopal, India who were pregnant at the time of the accident. In Colombia flower workers an 11% decreased risk was found in wives of male worker and a 1% decrease in female workers.

Fertility (Table 4)
Past Incidents:
Two pesticides are known to cause sterility in humans, both in male factory workers. The first episode occurred in a single facility, affecting about 100 severely poisoned workers and was soon recognized. Primary effects were on the nervous system, but testicular dysfunction was also found. In the second episode there were no warning signs of acute poisoning, allowing exposure to continue for months and years before recognition of the problem. Testicular failure was the primary effect with no other apparent disorders. Both pesticides, now banned in the U.S., are still being monitored as environmental contaminants of surface and ground water in California and Virginia.

The first pesticide is kepone (chlordecone), a persistent chlorinated hydrocarbon insecticide produced at a substandard facility in Hopewell, Virginia, a small town on the James River. The plant was opened in 1974 and closed by the Commonwealth of Virginia in 1976.

The second pesticide is DBCP (dibromochloropropane), a soil fumigant that was widely used in agriculture as a

nematicide. The first reports of sterility were from an Occidental Chemical Company plant in Lathrop, California. Further investigations found testicular dysfunction in men exposed to DBCP in Dow and Shell Chemical Company workers in Colorado, Alabama, Arkansas, and Michigan, and other workers in the U.S. and throughout the world. Many more workers were affected by DBCP because of its high production volume, widespread use, and the long silent period of damage to the testes until exposure ceased.

Recent Developments:
Because no recent incidents comparable to kepone and DBCP have occurred does not mean that other environmental and workplace exposures are not affecting human fertility. The major known risk known factor for infertility in otherwise healthy people is infections from sexually transmitted diseases; alcohol, tobacco, and drug use also contribute. Studies of infertility in human often compare medically diagnosed infertile couples with fertile couples. Such studies are useful but have shortcomings when determining the role of environmental contaminants and occupational exposures in healthy people. The infertile couples are not representative of the general population; the controls are usually drawn from hospital based clinics. It is necessary to determine which partner is infertile and analyze the data based on male or female factor infertility. The studies require a large number of cases and controls, who are often difficult to recruit and follow over time.

A more recent development is an easily collected measure of fertility that includes both partners, and can be done in any randomly selected population. It is called 'time to pregnancy', or fecundability. Fecundability is the number of months or menstrual periods it takes to conceive a child when not using birth control. The resulting fecundability ratio is a measure of the fertility rate in exposed couples compared to non-exposed couples. A ratio of one means there is no difference in fertility between them. A ratio greater than one means the exposed couples get pregnant in a shorter period of time, that they are 'more fertile'. A ratio less than one means the exposed couple takes longer to get pregnant, that they are 'less fertile'. The lower the ratio the less fertile (fecund) the couple.

Fecundity:
Several studies report decreased fecundity in pesticide exposed workers. In the Netherlands, pesticide exposed fruit growers had an 18% lower fertility rate (FR 0.82), with an additional 40% decrease in spray season (FR 0.42) . A 22% decrease in the fertility rate was found in female greenhouse workers in Denmark who sprayed pesticides (FR 0.78), with an additional 11% decrease if they did not use gloves (FR 0.67). A 17% decrease in the fertility rate was found in pesticide exposed greenhouse workers in Denmark and France (FR 0.83)[106]. In Canada the amount of time females spent in farm pesticide activity decreased the fertility rate 20 to 49% across several use categories. No association was found with sterility. Male greenhouse sprayers in Italy had a 240% increased risk of a 3 months or more delay in conception, compared to other greenhouse workers who had a 60% increase that was not significant. Another study of pesticide exposed Italian greenhouse workers found a 5.4 month delay in conception compared to 3.9 months for unexposed workers.

Increased fecundability ratios have also been found in pesticide exposed workers. Danish farmers who had used pesticides for 11 to 15 years had a 61% increased fertility rate (FR 1.61, and a 30% increase if exposed 6 to 10 years, (FR 1.30 borderline significant). Pesticide exposed farmers in Denmark and France had 9% increased rate (FR 1.09), and vineyard workers in France a 17% increase.

No significant difference in fecunability was found between traditional and organic farmers.

Infertility:
Several studies comparing fertile couples to medically diagnosed infertile couples have considered pesticides as a risk factor for infertility. In Austria that working in agriculture greatly increased the risk of male infertility. Iowa women who ever worked in agriculture had a 700% increased risk of infertility which was 430% higher if prior to diagnosis; living on a farm increased risk 80%. In California no association with infertility was found in male factory workers exposed to carbaryl, or field applicators of DBCP.

Table 1-A
Birth Defects and Pesticide Exposure - Increased Risk
(See Appendix F for explanation of the table)

Any Major Defect(s)	
Canada NB living in high pesticide use area[(a)2]	SRR 2.49
Chile mother exposed pesticides at work[3]	AR[(b)] 54%
China pesticides at work during pregnancy[4]	OR 1.8 ns[#]
Colómbia female flower workers high exposure[5]	RR 2.1
Female flower workers low exposure	RR 0.6
Wives male workers employed 1-15 yr[6]	OR 1.3
Finland mother an agricultural worker[7]	OR 1.4 [*]
Hungary cluster trichlorfon in fish farming[©)8]	11/15 births
India exposed cotton field workers female[(d)9]	OR 4.29
India Bhopal MIC[(e)] exposure[10]	No assoc.
New Zealand live 2,4,5-T application area[11]	IR > 1[#]
New Zealand 2,4,5-T sprayers[12]	RR 1.19[#]
Norway agricultural workers[13]	No assoc.
Philippines conventional farming vs IPM[(f)14]	RR 4.56
Spain mother an agricultural worker[15(g)]	OR 3.16
Father an agricultural worker	OR 1.5[#]
Father a pesticide handler	OR 1.49[*]
Spain father exposed to paraquat[16]	OR 2.80
Father exposed to glufosinate	OR 2.45[#]
US California mother lives pesticide use area[(h)17]	OR 1.4[#]
Halogenated hydrocarbon pesticide use	OR 2.2
US California malathion medfly spraying[18]	No assoc.
US California placental p,p'-DDE levels[19]	No assoc.
US Hawaii heptachlor milk contamination[(I)20]	No assoc.
US Minnesota licensed applicators[21]	OR 1.96
General population wheat crop area	OR 1.86
Living in high pesticide use area	OR 1.66
Case Reports	
England 2,4-D leaky sprayer multiple defects [(j)22]	-
France mother exposed urogenital defect[23]	-
Germany deet use multiple severe defects[(k)24]	-
Netherlands malathion for lice 1st trimester[25]	-
US California farm worker reentry poisoning[(l)26]	-
Central Nervous System	
Finland agriculture/horticulture 1st trimester[27]	OR 1.0
Norway farmer parents hydrocephalus[28]	OR 3.49
US Colorado father agriculture/forestry[(m)29]	OR 2.3 [*]
US Minnesota glyphosate herbicide users[(n)30]	OR 3.60
Phosphine applicators	OR 2.48
US Texas maternal exposure[(o)] Down syndrome[31]	No assoc.
Cleft Lip/Palate	
Canada NB living in high pesticide use area[(a)1]	SRR 2.49
England/Wales gardener/farm worker females[32]	Increase
England/Wales father exposed[33]	RR 0.8
Finland mother agricultural worker[6]	OR 1.9
Pesticide exposed vs unexposed	OR 1.9[#]
Finland agriculture/horticulture 1st trimester[26]	OR 3.3
New Zealand 2,4,5-T sprayers[11]	Not found
US Arkansas live 2,4,5-T application area[34]	Increase
US California periconceptual home use[35]	OR 3.8
Father occupational exposure	OR 1.7 [*]
US Georgia farmers periconceptual exposure[36]	OR 3.3 [*]
US Iowa agricultural chemical use[37]	OR 2.85
Michigan agriculture chemical use	OR 1.68
Cryptorchidism[(p)]	
China father occupational exposure[38]	OR 12.8
Denmark female garden workers[39]	OR 1.67
Male garden workers	No assoc.
US CPP[(q)] maternal DDE levels[40]	OR 1.3[#]
Eye Defects	
Canada father exposed WPs[®)], chlorophenols[41]	Increase
Canada father's occupation forestry/logging[42]	OR 2.28
England severe cases eye defects[(s)43]	OR 2.4
All cases	OR 1.8
Italy parental occupational exposure benomyl[44]	OR 0.63
Gastrointestinal Defects	
Canada agriculture/horticulture females TEF[(t)45]	OR 1.52
Norway farmer parents[27]	OR 2.32
US California malathion spraying TEF[(t)46]	RR 2.66[(u)#]
US CPP[(q)] maternal DDE levels[39]	OR 1.9 [*]
(Continued in Table 1-B)	

* borderline significance
\# not statistically significant

(a) Fenitrothion, aminocarb, phenoxy/other herbicides. **(b)** Attributable Risk is the percentage accounted for by pesticide exposure. **[c]** Cluster ceased when trichlorfon banned. **(d)** 80% had symptoms of moderate poisoning. **(e)** Methylisocyanate, toxic gas released from an explosion in Dec. 1984 at a factory manufacturing carbaryl (Sevin). **(f)** Integrated Pest Management. **(g)** During the month before conception and first trimester. **(h)** During weeks 3-8 pregnancy in the sq. mile of residence area. **(I)** From 1980-1982 pineapple waste contaminated with heptachlor was used in feed for dairy cows. **(j)** Both parents applied 6 days/week, 7 hoursr/day before conception to 5 weeks after last menstrual period; mother had severe burn from spill on leg. **(k)** Applied daily entire pregnancy (also took chloroquine). **(l)** From exposure to metasystox-R, Phosdrin, and /Lannate on cauliflower first month of pregnancy. Child died with severe multiple defects. **(m)** In craniosynostosis the sutures of the skull fuse prematurely resulting in an abnormally shaped head. **(n)** Neurodevelopmental and neurobehavioral defects. **(o)** 3 months before to after last menstrual period. **(p)** Undescended testicles. **(q)** Collaborative Perinatal Project, a cohort 1959-1965 study of pregnant women and their children at 12 medical centers (about 56,000 pregnancies). **[r]** Wood preservatives. **(s)** Anophthalmia/microophthalmia, alleged clusters linked to the fungicide benomyl. ORs are for rural vs urban areas. **(t)** Tracheo-esophageal fistula. **(u)** Based on three cases.

Table 1-B
Birth Defects and Pesticide Exposure
(See Appendix F for explanation of the table)

Heart Defects	
Canada father's occupation forestry/logging[41]	OR 2.03
Finland mother work exposure HLHS[(a)47]	No assoc.
Finland mother work exposure ASD[(b)48]	No assoc.
Finland mother work exposure VSD[©)49]	No assoc.
Finland agriculture/horticulture 1st trimester[26]	OR 0.3
New Zealand live in 2,4,5-T application area[10]	IR > 1#
US California periconceptual home use CT[(d)34]	OR 3.1
Maternal use insect repellent CT[(d)]	OR 2.2
US Maryland BWIS[(e)] any home use 1st trim.[50]	OR 2.0
Mother home rodenticide exposure	OR 4.7
Mother home herbicide exposure	OR 2.8
Mother home insecticide exposure	OR 1.5 *
Any home pesticide exposure[51]	AR[(f)] 5.5%
US Maryland BWIS[(e)] TAPVR[(g)] pesticides[52]	OR 2.74
US Minnesota licensed applicators[20]	OR 1.7
Hypospadias[(h)]	
Denmark male and female garden workers[38]	No assoc.
New Zealand live 2,4,5-T application area[10]	IR > 1#
Norway farmer parents[27]	OR 2.94
Kidney Defects	
Canada NB living in a high pesticide use area[(a)1]	SRR 2.49
Limb Reduction Defects	
Australia home use more than once 1st trim.[53]	RR 7.0
Home use once during 1st trimester	RR 3.1
Norway farmer parents[27]	OR 2.50
US California periconceptual home use[34]	OR 3.5
US California mother high pesticide use area[(l)54]	RR 3.1
Mother lives high pesticide use area[(j)]	RR 1.9
Mother high agricultural production area[(l)]	RR 2.4
Mother high agricultural production area[(j)]	RR 1.7
Either parent an agriculture worker[(l)]	RR 1.6#
Either parent an agricultural worker[(j)]	RR 0.9
US California one/both parents farm workers[55]	RR 2.31
US National crop dusters vs siblings[56]	No assoc.
US New York State parent pesticide exposed[57]	OR 0.9
Farming occupation	OR 1.1#
US Washington State mother exposed at work[58]	PR 2.6
Musculoskeletal Defects	
Finland mother works in agriculture[6]	OR 1.9
US Minnesota licensed applicators[20]	OR 1.5

Neural Tube Defects[(k)]	
Canada father exposed WPs[(l)40], chlorophenols	Increase
Canada NB living in a high pesticide use area[(m)1]	SRR 2.49
China mother exposed 1st trimester[59,60]	OR 7.8
England/Wales gardeners/agriculture females[31]	Increase
England/Wales father exposed[32]	RR 1.2#
Spina bifida	RR 0.8
Farmers (anencephaly)	RR 1.0
Gardeners	RR 2.3#
Finland mother works in agriculture[6]	OR 1.9
Finland agriculture/horticulture 1st trimester[26]	OR 1.0
Hungary maternal work in agriculture[61]	OR 1.1#
Netherlands mother works in agriculture[62]	OR 5.6
Netherlands agricultural job females[63]	OR 3.4
Netherlands father exposed[64]	OR 1.7#
Norway farmer parents[27]	OR 2.76
Spain mother agricultural worker[(n)14]	OR 2.2#
Sweden women living on a farm[65]	OR 2.2
US California home use mother applied[34]	OR 2.9
Commercial home application	OR 2.5 *
Mother lives within 2.5 miles crop area	OR 1.5
US California Antioch cluster investigation[66]	No asso.
US Texas father farm /ranch work[67]	$\chi^2$1.8 p<.001
Mother farm/ranch work	$\chi^2$1.3 p<.05
Parental pesticide exposure occupation	OR 1.28#
Hired farm workers/ranch workers	OR 1.73#
Gardeners and landscapers	OR 0.91
US/Mexico parental exposure pesticides[68]	Noassoc.
Urogenital Defects[(o)]	
Canada father exposed WPs[(l)40], chlorophenols	Increase
New Zealand 2,4,5-T sprayers[11]	Not found
US CPP[(p)] maternal DDE levels[39]	OR 1.2#
US Minnesota licensed applicators[13]	OR 1.7

* borderline significance
\# not statistically significant

(a) Hypoplastic left heart syndrome. **(b)** Atrial septal defect (secundum). **[c]** Ventricular septal defect. **(d)** Cono-truncal defects. **(e)** Baltimore-Washington Infant Study, a population-based case- control study of cardiovascular defects. **(f)** Attributable Risk, the percentage accounted for by pesticide exposure. **(g)** Total anomalous pulmonary venous return. **(h)** The opening of the urethra (where urine comes out) is not at the tip of the penis but on the underside. **(l)** Had other defects in addition to limb reduction. **(j)** Limb reduction the only defect. **(k)** Neural tube defects (spina bifida, anencephaly). **(l)** Wood preservatives. **(m)** Focus on fenitrothion, aminocarb, phenoxy/other herbicides. **(n)** During the month before conception and the first trimester. **(o)** Not specified. Cryptochidism and hypospadias most common. **(p)** Collaborative Perinatal Project, a 1959-1965 cohort study of pregnant women and their children at 12 medical centers (about 56,000 pregnancies).

Table 2
Spontaneous Abortion and Pesticide Exposure
(See Appendix F for explanation of the table)

Bulgaria farm workers female[69]	No assoc.	Israel DBCP wives exposed banana workers[(m)81]	PR 3.0
Canada Ontario late[(a)] thiocarbamate use[70]	OR 1.8	Only wives conceived before & after expos.	PR 4.66
Late[(a)] glyphosate use[(c)]	OR 1.7	Italy wives pesticide applicators[82]	OR 7.6
Early[(b)] phenoxy herbicide use[(c)]	OR 1.5	Spontaneous abortion/pregnancy ratio[(l)]	OR 3.8
Early[(b)] any herbicide use[(c)]	OR 1.4	Italy hexachlorobenzene, DDT compounds[83]	No assoc.
Early[(b)]triazine herbicide use[(c)]	OR 1.4*	New Zealand wives herbicide sprayers[49]	RR 0.89
Canada preconceptual use 2,4-D by husband[71]	OR 2.5*	Norway late[(n)] farmer parents[84]	OR 1.9
Using 2,4-D without protective equipment	OR 5.0#	Parents grain farmers	OR 1.8
Canada Ontario farm pesticide use males[(d)72]	Increase	Poor grain harvest[(o)]	OR 2.4
Canada Quebec agric/horticulture females[(e)73]	OR 2.4	Philippines conventional farming vs IPM[(p)9]	RR 6.17
China mother pesticide work pregnancy[(f)3]	Increase	Spain female greenhouse sprayers[85]	Increase
China mother work exposure (threatened)[42]	OR 3.9	Turkey HCB[(q)] females 40 years later[86]	Increase
Colómbia female flower workers[5]	RR 2.20	US California malathion medfly spray[45]	No assoc.
Wives of male workers[(g)]	RR 1.79	US California ETO[(h)] dental assistants[87]	OR 2.5*
Denmark greenhouse female pesticide users[(i)74]	OR 2.0#	US Florida DDT, DDE blood levels[88]	No assoc.
Outdoor garden workers	OR 1.3#	US Minnesota wives Cheyenne[(r)] users[89]	RR 2.9
Female greenhouse workers female	OR 0.9	Wives imidizolinone users	RR 2.6
Finland ETO[(h)] hospital workers[75]	PR 1.1	Wives sulfonylurea users	RR 2.1
Exposure during pregnancy	PR 2.98	Female pesticide appliers	RR 1.8
Germany CH[(j)] repeated exposure[76]	No assoc	Wives fungicide appliers	RR 1.6
India Bhopal MIC[(k)] vs unexposed[9]	PR 4.29	Wives herbicide appliers 1st trimester[(s)]	Increase
India Bhopal MIC[(k)] pregnant during accident[77]	Increase	US National crop dusters vs siblings[55]	No assoc.
India wives exposed cotton workers[8]	OR 1.74		
India couples exposed in vineyards[78]	PR 5.83		
Israel DBCP production 17 year follow-up[79]	No assoc	* borderline significance	
Israel DBCP production workers[80]	No assoc	# not statistically significant	

(a) 12-19 weeks gestation. (b) 12 weeks gestation. **[c]** 3 months before to month of conception. **(d)** Thiocarbamates, carbaryl, other pesticides. **(e)** Fetal death after 27 weeks, employed > 30 hrs/wk for > 2 wks any time during pregnancy. **(f)** Risk increased with increasing number pesticides used. **(g)** Employed at least 6 months for one of 58 floriculture companies. **(h)** Ethylene oxide, a gas used to sterilize equipment. **(I)** Union members. Only 9% authorized to spray pesticides. **(j)** Chlorinated hydrocarbon pesticides: pentachlorophenol, lindane, hexachlorobenzene, DDT group. **(k)** Methyliso-cyanate, toxic gas released December 1984 after explosion at factory making carbaryl (Sevin). **(l)** 0.27 in appliers versus 0.07 in controls. **(m)** Whose wives conceived before and after DBCP exposure. **(n)** 16-27 weeks gestation. **(o)** Later harvest results in higher exposure to mycotoxins. **(p)** IPM = Integrated Pest Management. **(q)** Hexachlorobenzene. 1950s outbreak of porphyria cutanea tarda from eating treated seed wheat not meant for consumption. **[r]** Thifensulfuron/tribenuron, fenoxaprop-P-ethyl/MCPA. **(s)** During the time when herbicides are applied.

Table 3
Stillbirth and Pesticide Exposure
(See Appendix F for explanation of the table)

Canada Quebec low-level exposure females[(a)90]	OR 3.1
Canada Quebec pesticides/germicides females[91]	OR 2.06
Canada Quebec agric/horticulture females[(b)72]	OR 5.53
Canada NB living in high pesticide use area[1]	SRR 2.5
Colómbia flower workers female[5]	RR 0.99
Wives of male workers[(c)]	RR 0.89
Colómbia female flower workers[4]	OR 0.99[#]
Wives of male workers	OR 0.89[#]
India Bhopal MIC[(d)] survivors vs unexposed[9]	OR 2.49
Perinatal/neonatal mortality	OR 1.37
India Bhopal MIC[(d)] pregnant during incident[76]	No assoc.
India wives of exposed cotton field workers[8]	OR 3.29
Sudan farmers hospital group females[92]	AR[(e)]34.5%
Farmers community group female	AR[(e)]22.6%
Not farmers community group females	AR[(e)]15.7%
Turkey 40 years after HCB[(f)] incident females[85]	No assoc.
US California mother lives carbamate use area[(f)93]	OR 1.3[*]
Lives halogenated hydrocarbon use area[(g)]	OR 1.3[*]
Lives estrogenic pesticide use area[(g)]	OR 1.4[#]
US California pesticide exposure at work[94]	RR 2.4[*]
Home pesticide use	RR 1.7[*]
US California placental DDE levels[18]	No assoc.
US California malathion medfly spraying[45]	No assoc.
US Massachusetts Boston chlorinated water[95]	OR 2.6[*]
U Minnesota wives of pesticide applicators[20]	No assoc.
US National mother home pesticide use[96]	OR 1.5
Mother pesticide exposure at work	OR 1.6
Father home pesticide use	OR 1.3
Father pesticide exposure at work	OR 1.2[*]
US Navy pre-term birth either parent exposed[97]	Increase
US Texas near pesticide factory Hispanics[(h)98]	OR 8.4
US Washington state farm workers[99]	OR 1.5

* borderline significance
\# not statistically significant

(a) After 28 weeks gestation. **(b)** Fetal death after 27 weeks, employed > 30 hrs/wk > 2 wks any time during pregnancy.**[c]** Worked at least 6 months for one of 58 floriculture companies. **(d)** Methyisocyanate, toxic gas released in December 1984 after an explosion at a factory making carbaryl (Sevin) **(e)** Attributable Risk, percentage accounted for by pesticide exposure. **(f)** Hexachlorobenzene. 1950s outbreak of porphyria cutanea tarda from eating treated seed wheat not meant for consumption. **(g)** During weeks 3-8 of pregnancy within a square mile of residence, or in one of adjacent 8 sq mi. areas. **(h)** Making arsenicals. High exposure group >100 ng/m^3

Table 4
Fertility and Pesticide Exposure
(See Appendix F for explanation of the table)

Austria male infertility work in agriculture[100]	OR 11.34
Canada DDE IVF[(b)] fertilization failure[101]	Increase
Canada CHI[(c)] rate/time to cleavage 1st egg[102]	No assoc.
Canada 6/13 pesticide exposure farm females[103]	FR[(d)]0.51-0.8
Farm pesticide use males only	FR 0.75-1.50
Sterility	No assoc.
Canada occupational chlorophenol exposure[104]	No assoc.
Canada pesticides female infertility[(e)105]	OR 3.02
Denmark female greenhouse pesticide sprayers[106]	FR[(d)] 0.78
Not using gloves	FR 0.67
Handling cultivars many hours/week	FR 0.69
Denmark tradition pesticide use 11-15 yrs[107]	FR[(d)] 1.61
Tradition pesticide use 6-10 yrs	FR 1.30[*]
Traditional vs organic farmer	FR 1.03[#]
Pesticide user vs non-user	FR 1.18[#]
Denmark/France pesticide exposed farmers[108]	FR[(d)] 1.09
Greenhouse workers	FR 0.83
England pesticide spray crew[(f)] impotence[109]	80% (4/5)
Recovery time in months	3-13 months
France vineyard workers fertility[98]	FR[(d)] 1.17
France infertile farm couple lindane use[(g)110]	Case report
Italy delayed conception[(h)] male sprayers[111]	OR 2.4
Greenhouse workers males	OR 1.6[#]
Italy greenhouse mean time to pregnancy[112]	5.4 months
Unexposed controls	3.9 months
Netherlands pesticides fertilization rate[113]	OR 0.38
Moderate pesticide exposure	OR 0.52 ns [#]
Heavy pesticide exposure	OR 0.22
Netherlands fruit growers spray season[114]	FR[(d)] 0.42
Fruit growers before spray season	FR 0.82
US California carbaryl fathering children[115]	No assoc.
US California DBCP applicators infertility[116]	Not found
US Iowa females agriculture work infertility[(i)117]	OR 7.0
Females agriculture work prior to infertility	OR 11.3
Residing on a farm	OR 1.8

* borderline significance
\# not statistically significant

(a) Fertility, the ability to conceive a child. **(b**) In vitro fertilization. Levels in serum, follicular fluid. **[c]** Chlorinated hydrocarbon insecticides: chlordane, DDE, heptachlor epoxide, HCB, oxychlordane **(d**) Fecundability Ratio: the likelihood of pregnancy for exposed couples vs unexposed control couples. A ratio less than 1 means a decrease in the fertility rate. The higher the ratio the more fertile (fecund) the couple. Also called 'time to pregnancy', it is the number of menstrual cycles/months to conceive when not using birth control. **(e**) Infertility: inability to conceive after 1 year of unprotected intercourse; failure to deliver a live-born child. **(f)** Using 5 organophosphates, dinoseb, paraquat, dieldrin, 3 phenoxy herbicides, simazine, linuron, mancozeb. **(g**) Successful pregnancy after protective measures taken. **(h**) 3 months or more. **(i)** All male partners diagnosed as fertile.

In vitro Fertilization (IVF):
In the Netherlands, a 62% decrease in rate of IVF (OR 0.38) was found if the couple was exposed to pesticides. The rate decreased 16% further if heavily exposed. In Canada, an increased failure rate of IVF was associated with levels of of DDE in serum and ovarian follicular fluid[100]. Another Canadian study found no association between chlorinated hydrocarbon pesticides in ovarian follicular fluid and rate or time to cleavage of the first egg.

Sperm Parameters (Table 5)
Men with sperm counts lower than 20 million sperm per milliliter of semen (m/ml) have a condition called oligospermia. Some oligospermic men conceive children. Men with a sperm count of zero have a condition called azoospermia, and cannot father children. The major cause of azoospermia is vascetomy The following discussion describes pesticide related sperm parameters in men without vasectomies.

Sperm Counts:
Many studies have been done of DBCP exposed workers since the first report in 1977 of sterility in the Occidental workers in California. DBCP is a prototype against which other suspected reproductive toxins are measured, since the studies show a strong dose response–the greater the amount and duration of exposure the more severe the damage to the testes, and the effect on sperm production.

Sperm Count
Low Normal - 20-39 million
Oligospermia - 20 million or less
Azoozpermia - no sperm

Sperm Characteristics
Motility
Necrospermia (dead sperm)
Teratospermia (abnormal shapes)

Another study was done at the Occidental plant of 154 DBCP exposed workers, and 52 exposed to other pesticides manufactured there (diazinon, dinoseb, endosulfan, malathion, maneb, methyl parathion, parathion, toxaphene, and zineb). The median sperm count in DBCP exposed was 46 m/ml versus 79 m/ml in unexposed. Azoospermia was found in 13% of exposed versus 2.9% of unexposed.; oligospermia in 6.8% of exposed and none of the unexposed. Low normal counts (20-30 m/ml)were found in 15.8% of exposed versus 5.7% of unexposed workers. A summary of findings in fourteen U.S. studies found a mean sperm count of 107.1 m/ml, a median of 83.0 m/ml;, and sperm counts less than 20 m/ml in 8.7%.

An international study found azoospermia in 64% of DBCP applicators exposed three years or more. In the Phillippines, oligospermia or azoospermia was found in 90% of exposed workers. A follow-up of DBCB workers in Israel found recovery in 30% of azoospermics and 50% of oligospermics over a period of three to four years.

Ethylene dibromide (EDB), a fumigant similar to DBCP also causes sterility. EDB exposed papaya workers in Hawaii had a mean of 80.99 m/ejaculate compared to 139.8 m/ml in unexposed controls.

In Denmark, a report that a self-selected group of organic farmers attending a convention had higher sperm counts than traditional farmers created quite a stir. Press coverage was extensive, assuming that pesticides used by traditional farmers were harming the testes. A well designed study using a random sample of a larger number of farmers did not support the earlier findings. The mean sperm count of 64 m/ml found in organic farmers was 10% higher than the 58 m/ml in traditional farmers, but the difference was not significant. A study of pesticide exposure in Danish farmers found 197 m/ml before pesticide exposure, decreasing 22% to 152 m/ml after exposure, but the difference was not significant. Decreased counts were found in farmers in Argentina using 2,4-D or for any farm pesticide exposure.

Biomarkers of Testicular Dysfunction

Increased
LH: Leutenizing hormone
FSH: Follicle stimulating hormone
SHBG: Sex Hormone Binding Globulin

Decreased
Testosterone
Inhibin B

Sperm Characteristics: **Motility**
In Argentina, a 50% decrease in sperm motility was found in farmers using 2,4-D, and a 95% decrease in an infertile French farm couple using lindane without protection, which improved to a 20% decrease with protection.

Necrospermia:
A 225% increase in necrospermia (dead sperm) was found in Argentinian farmers using 2,4-D. An infertile French farm couple in which the husband used lindane without protection had a 60% prevalence of necrotic sperm which resolved completely once protections were used. No changes were found in ethylene dibromide exposed workers in Hawaii

Teratospermia:
A 216% increase in teratospermia (abnormal shapes) was found in Argentinian farmers using 2,4-D, and a 69% increase in ethylene dibromide exposed workers in Hawaii. Danish farmers with heavy pesticide exposure had a 60% decrease in normal sperm; those with low exposure a 14% decrease. Farmers exposed ten years or more had a 40% decrease compared to farmers exposed for five years. A 60% prevalence in an infertile French farm couple using lindane without protection, persisted even after protective measures were taken.

Aneuploidy:
Chinese pesticide factory workers had a 36% higher number of sperm with abnormal number of chromosomes than unexposed workers. No associations were found with fungicide exposure in Finnish farmers.

Hormone Levels
Hormone which increase or decrease with testicular dysfunction are useful biomarkers of male infertility and sterility. Hormones that increase with testicular dysfunction are LH (leutenizing homone), and FSH (follicle stimulating hormone). Testosterone decreases with testicular dysfunction. Sex hormone binding globulin(SHBG) is a serum protein that binds testosterone. Biologically active testosterone is free and unbound. A high SHBG means less free testosterone so the higher the SHBG level, the lower the testosterone level. Inhibin B is a glycoprotein secreted by Sertoli cells in the testes that correlates with sperm count. The higher the sperm count, the higher the inhibin B level.

High LH and FSH levels were found in Chinese pesticide factory workers, in German workers with short term exposure to pesticides, in Israeli DBCP workers 17 years after exposure ceased, and in lindane factory workers. No increase was found in Minnesota herbicide applicators, vinclozolin factory workers, and molinate factory workers.

Decreased levels of testosterone were found in Chinese pesticide factory workers, Danish farmers, lindane factory workers, and black farmers in North Carolina exposed to DDT. No significant association with pesticide exposure was found in Minnesota herbicide applicators, vinclozolin factory workers, or molinate factory workers. High inhibin B levels and a high testosterone/SHBG ratio was found in Danish organic farmers.

Sex Ratio (Table 6)
More boys are conceived than girls, but more boys die, resulting in a roughly equal sex ratio in surviving infants. A shift in the sex ratio where more females are conceived can be an indicator of adverse reproductive effects. The most striking evidence of a pesticide effect on the sex ratio was found in Israel among the children born to men exposed to DBCP. Long term follow-up studies found the proportion of males conceived prior to exposure was 52.9%, during exposure 35.2%, and after recovery from testicular failure, 16.6%.

Excess female births were found in Minnesota pesticide applicators, especially fungicide users; but male infants predominated in those with birth defects. Excess female births were found in a high level compared to a low level boron village in Turkey, and in California borate workers, but the differences were not significant. In Turkey, women exposed to hexachlorobenzene at the peak of the episode from 1955 to 1957, had a lower lifetime proportion of males than those exposed at a later date. The women's lifetime spontaneous abortion rate predicted the percent males per subject. No effect of pesticides on the sex ratio was found in a national study in Canada, in DBCP water contamination areas in California, and in a national cohort of molinate factory workers.

Reproductive Fluids and Tissues
Two recent developments are dramatically changing the assessment of risks to human reproduction from environmental contaminants. The first is the increasing ability to detect extremely low levels of chemical contaminants in biological samples. The second is a rethinking of the most basic concepts in toxicology–"the dose

makes the poison"[a] when the exposure is to the developing fetus. The dose- response model dominates risk assessment, and is the basis of setting thresholds, or allowable levels of exposure to toxic chemicals. A recent review suggests that the hormetic model, characterized by low-dose stimulation and high-dose inhibition is more prevalent and may be more appropriate for some exposures than the widely used threshold model[b].

Table 5
Sperm Parameters , Male Hormone Levels and Pesticide Exposure
(See Appendix F for explanation of the table)

Sperm Counts[(a)]	
Argentina farm 2,4-D users vs nonusers[118]	OR 0.49
Argentina farm pesticide exposure[119]	Decrease
Denmark farmers pre-pesticide exposure[120]	197 m/ml
Farmers post-pesticide exposure[(b)]	153 m/ml
Pre-exposure controls	223 m/ml
Post exposure controls[(b)]	178 m/ml
Denmark traditional farmers[121]	58 m/ml
Organic farmers	64 m/ml[#]
Internat. DBCP applicators[©] azoospermia[122]	64%
Israel post DBCP azoospermia - recovery[(d)78]	33%
Oligospermia - recovery[(d)]	50%
Philippines DBCP azo/oligospermia[121]	90%
US California DBCP median in exposed[123,124]	46 m/ml
Median in unexposed	79 m/ml
Azoospermia exposed[(e)]	13.1 %
Azoospermia unexposed	2.9 %
Oligospermia exposed	6.8 %
Oligospermia unexposed	0 %
Low normal count (20-39 m/ml) exposed	15.8 %
Low normal count (20-39 m/ml) unexposed	5.7%
US Hawaii papaya workers exposed EDB[(f)125]	81 m/ejac.
Unexposed	140 m/ejac.
US National molinate workers[(g)126]	No assoc.
US National DBCP workers (14 studies)[127]	107.1 m/ml
Median sperm count	83.0 m/ml
Men with sperm counts < 20 m/ml	8.7 %
US Virginia Kepone production workers[128,129]	Decrease
Sperm Characteristics	
Argentina farm 2,4-D motility (improved)[117]	OR 0.50
Necrospermia[(h)] (improved)	OR 2.25
Teratospermia[(h)] (persisted)	OR 2.16
China factory workers aneuploidy[(I)130]	RR 1.51
Pesticide exposed number/1,000 sperm	3.03
Unexposed number/1,000 sperm	1.94
Denmark farmers high expos. normal sperm[131]	60% decr.
Exposed 10 years vs less than 5 yrs	40% decr.
Low exposure, normal sperm	14% decr.
Finland fungicide use sperm aneuploidy[(j)132]	No assoc.
France infertile farm couple lindane use[106]	Case report
Sperm motility without/with protection	5% / 20%
Necrospermia[(h)] without/with protection	60% / 0%
Teratospermia[(h)] without protection	60%
Teratospermia[(h)] with protection	Persistent
US Hawaii EDB[(f)] viable sperm[124]	No assoc.
Teratospermia[(h)]	OR 1.69
US Iowa farmers semen parameters[133]	No assoc.
Hormone Levels	
Argentina farming area estradiol level[115]	Increase
China pesticide factory workers serum FSH[(k)134]	Increase[#]
Serum testosterone	Decrease[#]
Denmark organic farmers inhibin B[(I)120]	Higher
Organic testosterone SHBG[(m)] ratio	Higher
Denmark greenhouse testosterone:SHBG[(m)130]	Decrease
Germany testosterone chronic exposure[135]	Decrease
LH[(k)] after exposure	Increase
Germany vinclozolin[(n)] testosterone ng/m[136]	5.6 I[#]
Serum LH[(k)] IU/ml	9.0[#]
Serum FSH[(k)] mIU/ml	0.42 -4.9[#]
Israel 17 yr later FSH, LH[(k)]severely affected[78]	Increase
Testosterone	Decrease[#]
US California FSH[(k)] level azoospermics[121]	> 5.7mIU/ml
US lindane factory serum LH[(k)] gm. mean[137]	OR 1.53
Serum FSH[(k)]	Increase[#]
Serum testosterone	Decrease[#]
US MN herbicides post season FSH[(k)138]	Decrease
Herbicide use post season testosterone	Increased
Herbicide, insecticide, fumigant use	No assoc.
US National molinate workers[(g)] hormones[125]	No assoc.
US NC farmers hi DDE levels[(o)] testosterone[139]	22% lower

Not statistically significant

(**a)** Azoospermia is complete absence of sperm; oligospermia 20 million or less per milliliter of semen (m/ml). (**b)** Farmers not significantly different from controls. Post exposure drop was significant within each group **[c]** Exposed three years or more. (**d)** Recovery took 3-4 years after exposure ceased. (**e)** 2 had no DBCP exposure for 9 and 13 years; the first exposed for 4 yrs, the second for 2, both fathering children prior to exposure. (**f)** Ethylene dibromide, a fumigant similar to DBCP, and which also causes sterility. (**g)** 272 manufacturing /formulation workers at 3 plants. Mean exposure ranged from 12.7 to 210.9 ug/m^3 (**h)** Necrospermia: dead sperm. teratospermia: malformed sperm. (**I)** Abnormal number chromosomes. (**j)** Disomy /diploidy frequencies for chromosomes 1, 7. (**k)** Increased levels of LH (leutinizing hormone) and FSH (follicle-stimulating hormone) indicate testicular dysfunction. (**I)** Inhibin B is a sperm protein that correlates with sperm count. The higher the sperm count the higher inhibin B levels. (**m**) Sex hormone binding globulin binds testosterone.The higher the level of SHBG, the lower the amount of free (unbound) testosterone that is biologically active. (**n)** Factory workers exposed 1-13 years manufacturing/formulation operations.

[a] Attributed to Auroleus Phillipus Theostratus Bombastus von Hohenheim, known as 'Paracelsus,' a 16th century Dutch alchemist.
[b] Calabrese EJ, Baldwin LA. 2003. The hormetic dose-response model is more common than the threshold model in toxicology. Toxicolog Sci 71:246-250.

(**o)** Blacks who farmed about 30 years; 27% reported having used DDT. Testosterone level declined 1.9% per year of work.

Endocrine Disruptors:
Challengers to the "old toxicology" aver that it is not relevant to the fetus during critical periods of development in the first days and weeks of pregnancy. The fetus is especially vulnerable to low level contaminants that can adversely affect the endocrine system (glands which secrete hormones, including the pituitary, thyroid, adrenal, ovary, and testis). A recent book describes such contaminants as "endocrine disruptors", warning of potential harmful effects on the fetus of low level xenobiotics[c] such as plastics, phthalates, aklylphenols, pesticides, and other chemicals[d] . Many of these xenobiotics are estrogenic, mimicking the female hormone estrogen. See Appendix A for pesticides classified as endocrine disruptors.

Chlorinated Hydrocarbon Pesticides: Chlorinated hydrocarbons insecticides include DDT, DDE, aldrin, endrin, dieldrin, chlordane, heptachlor, hexachlorobenzene, lindane (hexachlorocyclohexane), and toxaphene. DDT was banned in 1972, aldrin and dieldrin in 1974, and chlordane in 1988. All were widely used in the 1950s and 1960s. They are still found in human tissues, especially those high in fat, because they degrade slowly, and are persistent in the environment. Even after two decades or more of non-use, many are still detectable. DDE is the metabolic breakdown product of DDT most commonly found in biological tissues.

Table 6
Sex Ratio and Pesticide Exposure
(See Appendix F for explanation of the table)

Canada National pesticide exposed[(b)] men[70]	No assoc.
Israel males conceived pre-exposure DBCP[(c)]	52.9 %
During exposure	35.2 %
After recovery	16.6 %
Turkey HCB[(d)] born to ♀ exposed 1955-57	Decrease [(e)]
Turkey boron high level villages[(f)] decrease	0.89
Low level villages no difference	1.04#
US California borate workers proportion males	Decrease#
US California DBCP water	No assoc.
US Minnesota pesticide applicators[2]	[0]0.75
Fungicide users	0.57
Subset of children with birth defects	1.75
US National molinate workers[(g)] births[125]	No assoc.

Not significant.
(a) A decrease in the ratio means more girls are born. **(b)** Thiocarbamates, carbaryl, other pesticides. **[c]** Dibromochloropropane, soil fumigant banned in 1979, known to cause sterility in men. **(d)** Hexachlorobenzene. 1950s outbreak of porphyria cutanea tarda from eating treated seed wheat not meant for consumption. **(e)** Lifetime spontaneous abortion rate predicted % males. **(f)**Two villages with high boron levels in drinking water (8.5-29mg/l, 2.05-2.5mg/l); three with low levels (0.03-0.40 mg/l). **(g)** Factory workers

Maternal and Paternal Pesticides (Table 7a)
Adipose Tissue (fat): The highest reported level of DDT in fat tissue of mothers at delivery (5900 ppb) was found in Kenyan women who also had high levels of β-HCH (30 ppb). Very high levels of DDE (4510 ppb) and DDT (1270 ppb) were found in adipose tissue of Mexican women at delivery.

Maternal Blood: The studies listed in Table 7 represent a selection from many studies of pesticides levels at delivery. The highest levels reported are 4450 ppm of DDE and 780 ppb of DDT in Mexican women.

In India, mothers who delivered stillborn babies had a 71% higher level of DDT in their serum than mothers with live births. There was no difference in lindane levels.

DDE was 260% higher in Israeli women who delivered premature babies than full term babies (71.1 vs 2.51 ppb), lindane 340% higher (15 vs 4.3 ppb), dieldrin 763% higher (8.4 vs 1.1 ppb), and heptachlor 303% higher (9.1 vs 3 ppb).

Low levels of DDE, trans-nonachlor and hexachlorobenzene were found in mothers in Greenland, and DDE and β-HCH in Japanese mothers.

Endosulfan and mirex were detected in 50% or more of

Pesticides Known to Cross the Placenta
(Selected List)

Acephate	DFP	Methyl paraoxon
Aldrin	Diazinon	Methyl parathion
Dieldrin	Dichlorvos	Methoxychlor
Apholate	Diquat	Mexacarbate
Benomyl	Formaldehyde	Mirex
Bromophos	Heptachlor	Nitrofen
Carbaryl	Hexachlorobenzene	Nicotine
Carbofuran	Imidan	Paraquat
Chlordane	Kepone	Parathion
Chlorpyrifos	Lindane	Pentachlorophenol
2,4-D	Malathion	Phosfolan
DDT	Mecarbam	2,4,5-T
DDE	Mephosfolan	TEPA
DDD	Methamidophos	Trichlorfon
Deet	Methiocarb	

Source: Salama AK, et al. 1993. A review article on placental transfer of pesticides. J Occ Med Toxicol 2(4):383-397.

[c] Pronounced zenobiotic, any biologically active substance not normally present or produced in the human body, and therefore foreign to it.
[d] Colburn T, Dumanoski D, Myers JP. *Our Stolen Future*. Dutton, New York. 1999.

samples from Canadian women undergoing in vitro fertilization. In the U.S., low levels of DDE were found in mothers in upstate New York, and in a national study, which also found a 500-310% increase in risk of pre-term births, and 90-260% increase in small for gestational age babies associated with DDE levels.

Two case reports from Poland in which women ingested pesticides to provoke abortion, found very high levels of carbofuran, and endosulfan in maternal blood. The mothers survived but the fetuses did not.

Ovarian Follicular Fluid:
Trace amounts of chlordane, DDE and hexachlorobenzene were found in follicular fluid from Canadian women undergoing in vitro fertilization (IVF). Another study found DDE and mirex in 50% or more of samples. Hexachlorobenzene and lindane (HCH) were found in the fluid of German women undergoing IVF.

Placenta
High levels in placenta were found in 1964-1965 samples from women living in a high agricultural production area of Californian . In India placental levels of DDT and lindane (HCH) from stillborn babies were not significantly different from live births. DDE and β-HCH were found in Japanese women in samples from the 1970s. A study in Mexico found that pesticide exposure increased the prevalence of atypical placental villi.

Semen:
Five ppb or more of 2,4-D was found in 50% of seminal fluid samples in Canadian farmers. Detectable levels of hexachlorobenzene, lindane, DDT, and dieldrin were found in German men, with the highest levels in chemistry students[162]. DDE, aldrin, endosulfan, and isomers of hexachlorocyclohexane (α-β-,γ-,δ -), were detected in men in India[163], and DDE and ϵ-HCH in Poland**.**

Testes:
In Greece, autopsies of suicides who died from ingesting pesticides found 21 ppb of paraquat, 5.8 ppb of fenthion and 0.8 ppm of methadithion in the testes.

Testicular Pathology:
Testicular biopsies in DBCP exposed workers show the seminiferous tubules to be the site of damage. Biopsies of ten severely affected California workers found seminiferous tubules devoid of spermatagonia and spermatogenic activity, resembling a Sertoli cell only syndrome. In the less severely affected, a decrease in cellularity was found within the seminiferous tubules without inflammation and only minimal evidence of an increase in fibrosis and interstitial changes. An early report of biopsies in severely affected Israeli DBCP workers found selective atrophy of the germinal epithelium, with the great majority of tubules lined only by Sertoli cells, and no evidence of active spermatogenesis. Large groups of leydig cells were present in the interstitial tissue surrounding the damaged tubules[168]. Biopsies done eight years later in Israeli workers exposed to DBCP for 100 to more than 6,000 hours, not all of whom had clinical signs of testicular dysfunction, found selective atrophy of the germinal epithelium, intact Sertoli cells, and a normal appearance of a relatively increased number of Leydig cells

Endometriosis:
A study of infertile women in German found an association between endometriosis and elevated levels of chlorinated hydrocarbon pesticides[140]. No association was found in wives of pesticide applicator in Minnesota, or with levels of chlorinated hydrocarbon pesticides in infertile women in Canada[141]. A review of chlorinated hydrocarbon pesticides includes a discussion of endometriosis[142].

Amniotic Fluid: A study done in Florida at a time of heavy agricultural DDT use found 14 ppb in black babies and 6 ppb in whites. A recent study found low levels of DDE and α-hexachlorocyclohexane in California women in their second trimester of pregnancy.

Meconium: Meconium is the intestinal contents of a newborn baby, and is the first “bowel movement”. It is an accumulation of intestinal epithelial cells, mucus, and bile. A 1971 study in Japan found DDE, DDT, dieldrin, and α-β-γ-HCH isomers. Two recent studies in Australia and the Phillipines tested for chlorinated hydrocarbon and organophosphate pesticides in the same laboratory, and is one few reporting pentachlorophenol. The amounts were

Table 7-A
Maternal and Paternal Fluids and Tissues
and Pesticide Contamination / Exposure

Adipose Tissue (Fat)	
Kenya total DDT mother at delivery[143]	5900 ppb
β-HCH[(b)]	30 ppb
Mexico DDE mother at delivery[144]	4510 ppb
DDT mother at delivery	1270 ppb
Maternal Blood at Delivery	
Canada IVF[(a)] DDE , endosulfan, mirex[100] detected	>50%
Greenland DDE[145]	4.8 ppb
Trans-nonachlor	1.6 ppb
Hexachlorobenzene	1.2 ppb
India 1983 DDT total stillbirth[146]	96.8 ppb
DDT total live birth	26.2 ppb*
HCH[(b)] stillbirth	17.3 ppb
HCH[(b)] live birth	18.3 ppb#
Israel 1982 DDT premature births[147]	71.10 ppb
DDT term births	26.51 ppb
Lindane premature births	15.0 ppb
Lindane term births	4.30 ppb
Dieldrin premature births	8.40 ppb
Dieldrin term births	1.10 ppb
Heptachlor premature births	9.10 ppb
Heptachlor term births	3.00 ppb
Japan 1976 β-HCH[148]	1.9 ppb
DDE	11.5 ppb
Japan 1974 samples β-HCH[(b)149]	7.7 ppb
DDE 1974 samples	4.7 ppb
Mexico 1994 DDE at delivery[148]	4450 ppb
DDT	780 ppb
Poland ingestion[©)] poisoning carbofuran[150]	9710 ppb
Endosulfan ingestion[©)] poisoning	470 ppb
US California farm workers cholinesterase[151]	Decrease
US CPP[(d)] DDE median (range 3-178)[152]	25 ppb
DDE and pre-term births	OR 1.5-3.1
DDE and small for gestational age	OR 1.9-2.6
US New York State DDE[153]	3.8 ppb
Ovarian Follicular Fluid	
Canada chlordane, DDE, HCB[ed)] IVF[(a)101]	Trace
Canada DDE, Mirex IVF[(a)] samples detected[100]	≥50%
Germany IVF HCB[(d)154]	0.08-1.87ppb
HCH[(b)]	0.05-1.0 ppb
Placenta	
India DDT total stillbirth[150]	60.8 ppb
DDT (total) live birth	39.8 ppb
HCH[(b)]stillbirth	13.4 ppb
HCH[(b)] live birth	17.1 ppb
Japan 1976 samples β-HCH[(b)152]	6.8 ppb
DDE	4.3 ppb
Japan 1974 samples β-HCH[(b)153]	5.7 ppb
DDE samples	3.2 ppb
Mexico atypical placental villi[155]	Increase
US California 1965 samples fat basis DDE[156]	5000 ppb
Semen	
Canada Ont. farmers 2,4-D 50% of samples[157]	≥5 ppb
Germany HCB[(e)], HCH[(b)], DDT, dieldrin[158]	Detected
Chemistry students	Elevated
India general population OCl[(f)159]	Detected
Poland general population ϵ-HCH[(b)160]	500 ppb
DDE in 40% samples	3 ppb
Testes	
Greece poisoning paraquat (autopsy)[161]	21 ppb
Greece poisoning fenthion(autopsy)[162]	5.9 ppb
Methidathion	0.8 ppb
Testicular Biopsy	
Israel DBCP exposed workers 1986 samples[163]	Abnormal
Israel DBCP severe effects 1978 samples[164]	Abnormal
US DBCP severe effects 1978 samples[122]	Abnormal

* Difference statistically significant .
\# Difference not significant
ppb parts per billion = ug/kg, ug/l, ng/g, ng/ml, pg/ml

(a) In vitro fertilization.**(b)** Hexachlorocyclohexane. Lindane is the gamma (γ-) isomer. **[c]** With the intent to provoke an abortion. Women survuved, fetuses died. **(d)** Collaborative Perinatal Project, a cohort study from 1959-1965 of pregnant women and their children at 12 medical centers (about 56,000 pregnancies). **(e)** Hexachlorobenzene. **(f)** OCl: organoclorine pesticides includies DDT, dieldrin, lindane, hexachlorobenzene, chlordane, and others.

much higher in the Filipino babies for all the pesticides found. The organophosphate pesticides diazinon and parathion were found only in the Filipinos. See Table 7a for the specific pesticides and amounts found. A study done in Germany found DDE in 5% of samples collected in 1997. A study in New York found several biomarkers of organophosphate pesticides. The findings represent recent exposures since organophosphate pesticide are not persistent and are rapidly eliminated from the body. Widely used organophoshates include chlorpyrifos, diazinon, malathion, methyl parathion, acephate and others[180].

Umbilical Cord Blood:
The presence of pesticides in cord blood is evidence of transplacental passage. Most tests of maternal/fetal pairs are for persistent pesticides in the DDT family. The highest reported level of DDE was found in Mexican babies born in

1997 (4700 ppb), and DDT levels were also high (880 ppb). The highest level of hexachlorobenzene (HCB) was reported in 1985 from Tunisia (37 ppb) where use was widespread in agriculture; much lower levels were found in babies in Nicaragua (6.39 ppb), in Spanish babies born 1997 to 1999 (1.1 ppb) and in German babies born in 1994 (0.5 ppb). High levels of DDT and lindane were found in stillborn babies in India but the levels were not were not significantly from full term births[150]. In the U.S., low levels of DDE were found in infants born from 1993 to 1998 in Massachusetts[191], and trace amounts of DDE, DDT and endosulfan in Texas farm workers[192]. In Canada, low levels of DDE were found in infants born in 1994 to 1999, and significant decreases in DDT, DDE, hexachlorobenzene and chlordane from 1993 to 2000.

There are racial differences in DDE levels in cord blood. A 1970 study in Florida found a 23% higher level of DDE in the whole blood of black infants than white, and a 1972 study a 22% higher level of DDE in cord blood of full term black infants than whites.

No reports of organophosphate pesticides in cord blood were found. Studies in California and Florida found decreased cholinesterase activity, a biomarker of organophosphate exposure. Since these pesticides are not persistent, the findings reflect recent exposure.

Another pesticide found in cord blood is the widely used insect repellent deet. In a study in Thailand deet was found in 8% of babies whose mothers used the repellent in the second and third trimester of pregnancy.

Umbilical Cord Tissue: A study in the Faroe Islands found DDE and hexachlorobenzene in umbilical cord tissue[193].

Sudden Infant Death Syndrome (SIDS)
A study in Germany found no difference in pentachlorophenol levels at autopsy in children who died of SIDS compared to controls, or in children from rural versus urban areas[165]. Another German study found no significant differences in the levels of hexachlorbenzene, or α-,β-,γ-isomers of hexachlorcyclohexane, heptachlor epoxide, dieldrin, and total DDT in the subcutaneous fat of children who died of SIDS compared to children who died of known causes[166].

Developmental Disabilities
There are no studies of pesticides as a risk factor for developmental disabilities in children such as autism, cerebral palsy, and severe mental retardation, although research interest is increasing.

Table 7-B
Fetal and Infant Fluids and Tissues and Pesticides Residues

Adipose Tissue (Fat)		
Germany fetus, autopsy DDT[167]		700 ppb
HCH[(a)] fetus, autopsy		140 ppb
Heptachlor fetus, autopsy		30 ppb
Germany HCB[(b)] children cryptorchidism[168]		Increased
Heptachlor epoxide		Increased
Great Britain DDT premature infants[169]		960 ppb
DDE 1 day to 3 months		720 ppb
Amniotic Fluid		
US California 2nd trimester[(c)] DDE[170]		0.21 ppb
α-HCH[(a)]		0.15 ppb
US Florida DDE black infants[171]		14 ppb
DDE white infants		6 ppb
Meconium[(d)]		
Australia[172]	Detected	Mean
Lindane wt < 2500 grams	100%	0.2 ppt
Weight > 2500 grams	57%	0.06 ppt
Pentachlorophenol	43%	8.0 ppt
Chlordane	16%	0.23 ppt
DDT	52%	0.04 ppt
Chlorpyrifos[(e)]	59%	0.52 ppt
Malathion	35%	0.052 ppt
Diazinon, parathion	Not detected	
Germany DDE samples detected[173]	5%	-
Japan DDE, dieldrin[174]		9 ppb
DDT		12 ppb
Total HCH[(a)]		30 ppb
Philippines Manila[175]	Detected	Mean
Lindane	73.5%	2.0 ppt
Pentachlorphenol	16.1%	90.0 ppt
Chlordane	12.7%	22.48 ppt
DDT	26.5%	12.56 ppt
Chlorpyrifos	11.0%	8.26 ppt
Malathion	53.0	6.80 ppt
Diazinon	34.3%	12.96 ppt
Parathion	32.0%	2.30 ppt
US New York OP[(f)] metabolites[176]	Detected	Mean
DEP	95%	0.8-3.2 ug/g
DETP	100%	2.0-5.6 ug/g
DMP	5%	16.0 ug/g
DEDTP	5%	1.8 ug/g

Umbilical Cord blood	
Canada Que. DDE 1994-99 samples[177]	0.412 ppb
Canada Que. chlordane decrease 1993-2000[178]	25%
DDT/DDE decrease 1993-2000	66%
HCB[(b)] decrease 1993-2000	69%
Germany HCB[(b)] 1994 (90% decrease)[179]	0.5 ppb
India 1983 total DDT stillbirths[150]	33.6 ppb
Total DDT live births	30.9 ppb
HCH[(a)] stillbirths	14.8 ppb
HCH[(a)] live births	13.6 ppb
Japan 1976 samples β-HCH[152]	5.7 ppb
DDE	5.5 ppb
Japan 1974 samples β-HCH[153]	1.9 ppb
DDE	1.8 ppb
Kenya 1976 total DDT[147]	1.9 ppm
Mexico 1997-98 samples DDE[148]	4700 ppb
DDT	880 ppb
Nicaragua 1990s samples DDE[180]	6.39 ppb
Spain 1997-99 samples HCB[(b)][181]	1.1 ppb
DDE, β-HCH[(a)]	Detected
Thailand deet use 2nd, 3rd trimester[182]	8% samples
Tunisia 1985 samples HCB[(b)][183]	37 ppb
US California RBC cholinesterase[(g)] decrease[184]	53%
US Florida 1972 DDE premature black[(h)][185]	19.0 ppb
DDE premature white[(h)]	22.1 ppb
DDE full term black[(h)]	6.1 ppb
DDE full term white[(h)]	4.9 ppb
US Florida 1970 DDE black infants[174]	5.9 ppb
DDE white infants	4.8 ppb
US Florida low cholinesterase activity[(g)][186]	0.59ΔpH[(i)]
US Massachusetts DDE[187]	0.48 ppb
HCB[(b)]	0.03 ppb
US New York State DDE[157]	1.9 ppb
US Texas DDE/DDT, endosulfan farm wkrs[188]	Detected
Umbilical Cord Tissue	
Faroe Islands DDE whole weight[189]	0.17 ppb
HCB[(b)] whole weight	1.19 ppb

* statistically significant difference # difference not significant
ppm parts per million = mg/kg, ug/g, mg/l, ug/ml
ppb parts per billion = ug/kg, ug/l, ng/g, ng/ml, pg/ml
ppt parts per trillion = ug/ml, ng/kg, ng/l, pg/m l

(a) Hexachlorocyclohexane. Lindane is the gamma isomer. **(b)** Hexachlorobenzene **[c]** One in three samples tested positive for at least one contaminant. **(d)** The intestinal contents of a newborn baby. Te first "bowel movement" after delivery it is dark green and consists of intestinal epithelial cells, mucus, and bile. **(e)** Not detected in 3 babies from Cairns and Mackay, despite heavy agricultural use in those regions **(f)** Organophosphate metabolites: biomarkers of recent exposure to several widely used pesticides including chlorpyrifos, diazinon, malathion, acephate, merhyl parathion, and others. DEP = O,O-diethylphos-phate, DETP = O,O-diethyldithiophosphate, DMP = O,O-dimethylphos-phate; DEDTP = O,O-diethyldithio-phosphate. **(g)** Biomarker of organophosphate pesticide exposure. **(h)** Infant whole blood, not from cord. **(i)** Normal is 1.9 or greater.

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