3. Human Effects

3.1 Clinical and Case Reports

Human poisoning by DDT has been reported to have occured only through ingestion. The earliest symptom of poisoning is hyperesthesia of the mouth and lower part of the face. This is followed by paresthesia and tremor of the extremities, confusion, malaise, headache, fatigue, and delayed vomiting. The vomiting is probably of CNS origin and not due to local irritation. Convulsions occur only in severe poisoning (Hayes 1959, 1975; Gosselin et al 1976; WHO 1977).

Onset may be as soon as 30 minutes after ingestion of a large dose or as late as 6 hours after smaller but still toxic doses. Recovery from mild poisoning is essentially complete in 24 hours, but recovery from severe poisoning may require several days. In two instances, there was some residual weakness and ataxia of the hands 5 weeks after ingestion. The human acute oral LD_{50} has been estimated at about 250 mg/kg (Gosselin et al 1976). Table 2.1.3 gives a summary of information on human responses to various doses of DDT.

A few persons apparently have been killed by uncomplicated DDT poisoning, but none of these cases has been reported in detail. Death has been caused much more frequently by the ingestion of solutions of DDT, but in most instances the signs and symptoms were predominantly or exclusively those of poisoning by the solvent (Hayes 1959). This does not mean that the toxicity of the solvent always predominates. For example, the recurrent convulsions in a case reported by Cunningham and Hill (1952), though more characteristic of poisoning by one of the

cyclodiene insecticides, was certainly not typical of solvent poisoning. A 2-year-old child drank an unknown quantity of fly spray containing 5% DDT, but the nature of other active ingredients or the solvent was unknown. About 1 hour after drinking the material, the child became unconscious and had a generalized, sustained convulsion. Convulsions were present when the child was hospitalized 2 hours after taking the poison, but the convulsions were controlled by barbiturates and other sedatives. Convulsions recurred on the 4th day and again on the 21st day but were stopped each time by treatment with sedatives. On the 12th day, it was noted that the patient was deaf. Hearing began to improve about the 24th day and was normal as were other neurologic and psychologic findings when the patient was examined about 2.5 months after the accident (Cunningham and Hill 1952).

Other neurologic effects of DDT poisoning have been reported. Freemon (1975) stated that prolonged exposure resulted in neurologic dysfunction. Others have reported clinical manifestations, including polyneuropathy, paresthesias, tremors, and convulsions (Campbell 1946, Hsieh 1954). Peripheral neuropathy has been occasionally ascribed to DDT, usually as a result of occupational exposure. One syndrome consists of numbness and paraesthesias, hypotonia, and asymmetric weakness or paralysis, with a slow spontaneous recovery when exposure is terminated (Jenkins and Toole 1964, Mackerras and West 1946, Onifer and Whisnant 1957).

The signs of intoxication in a 10-year-old girl who died after exposure to DDT were reported by Jacobzinger and Raybin (1963). They

included red blotches on the skin, hands, and arms; petechiae, hemorrhagic bullae around the lips; cellulitis; lymphangitis; lymphadenitis; nosebleed; hematuria; and uncontrollable fever. Primary skin irritation is rarely if ever due to DDT, and allergic dermititis has been reported only infrequently (Higgins and Kindel 1949). Purpura with marked thrombocytopenia has occurred in exposed children (Karpinksi 1950). An isolated case of agranulocytosis (Wright et al 1946) and postmortem findings resembling periarteritis nodosa (Hill and Damiani 1946) suggest more serious allergic manifestations. Sánchez-Medal et al (1963) presented circumstantial evidence that implicated DDT as a cause of aplastic anemia and thrombocytopenia. However, the association of these conditions with BHC and other chlorinated hydrocarbons is on firmer ground (USDHEW 1969).

Causes of accidental and suicidal poisoning in which the effects were clearly caused by DDT are summarized in Table 3.1.1.

3.2. Volunteer Studies

A number of small-scale studies involving controlled exposure of volunteers to technical DDT were conducted in the 1940's (Hayes 1959, USDHEW 1969, WHO 1977). Table 3.2.1 summarizes the reported results of controlled oral exposures.

Two chronic exposure studies with penitentiary volunteers given DDT orally by capsule have been reported by Hayes et al (1956, 1971). The first study involved 51 men. Of these, three completed 1 year of dosage at 3.5 mg/man/day, and seven completed 1 year at 35 mg/man/day.

TABLE 3.1.1

SUMMARY OF THE EFFECTS OF THE ACCIDENTAL OR SUICIDAL INGESTION OF DDT

Individual Dose, Formulation, Number of Persons	Effects
300-4,500 mg, in food, 1 man	Onset in 1 hr; vomiting; restlessness; headache; heart weak and slow; recovery next day
Unknown dose, in tarts, 25 men	Onset in 2-2.5 hr; all weak and giddy; 4 vomited; 2 hospitalized; 1 confused, incoordinated, weak; 1 with palpitations and numbness of hands; recovery in 24-48 hr
5,000-6,000 mg, in pancakes, 3 men	Onset 2-3 hr; throbbing headache; dizziness; incoordination; paresthesias of extremities; urge to defecate; wide nonreacting pupils; reduced vision; dysarthria; facial weakness; tremor; ataxic gait; reduced sensitivity to touch; reduced reflexes; positive Romberg; slightly low blood pressure and persistent irregular heart action; partial recovery in 2-3 days, but slight jaundice appeared 4-5 days after ingestion and lasted 3-4 days; all normal 19 days after poisoning except irregular heart action in one
Up to 20,000 mg, in bread, 28 men	Onset in 30-60 min in those most severely affected; men first seen 2-3 hr after ingestion; in spite of severe early vomiting that reduced the effective dose, severity of illness and especially intensity of numbness and paralysis of extremities proportional to amount of DDT ingested; recovery in all but 8 men in 48 hr; 5 others fully recovered in 2 wk, but some weakness and ataxia of the hands in 3 5 weeks after ingestion

TABLE 3.1.1 (Continued)

SUMMARY OF THE EFFECTS OF THE ACCIDENTAL OR SUICIDAL INGESTION OF DDT

Individual Dose, Formulation, Number of Persons	Effects
Unknown dose, in flour, about 100 women	Onset about 3.5 hr after ingestion; total of about 85 cases of which 37 were hospitalized; symptoms mild and similar to those in earlier outbreaks except gastrointestinal disturbance in most severe cases included abdominal pain and diarrhea as well as nausea; most fully recovered in 24 hr
Unknown dose, 14 cases	Symptoms in established cases similar to those reported earlier
286-1,716 mg, in meatballs, 8 cases, 11 exposed	Except in one man who was already sick when he received a dosage of 6 mg/kg, no poisoning at dosages of 5.1-10.3 mg/kg (Doses calculated from known consumption of meatballs and estimated concentration of DDT in mixture--Hsieh 1954); excussive perspiration, nausea, vomiting, convulsions, headache, increased salivation, tremors, tachycardia, and cyanosis of the lips after ingestion of 16.3-120.5 mg/kg; onset in 2-6 hr depending on dosage; as much as 2 days for recovery
Unknown dose, 1 case	Death 13 hr after suicidal ingestion
Unknown dose, 22 unrelated cases	Twenty-two separate cases, including 15 attempted suicides; some complicated by solvents; 3 deaths

Adapted from WHO 1977, Hayes 1959, Committee on Pesticides 1951, USDHEW 1969

TABLE 3.2.1

SUMMARY OF THE EFFECTS OF ONE OR A FEW ORAL DOSES OF DDT ON VOLUNTEERS

Dose (mg)	Formulation	Result	Reference
1,500	Butter solution	No effect, but lice killed when fed 6 and 12 hr after dose	MacCormack 1945
500	Oil solution	No effect	Neal et al 1946
700	"	"	"
250	Suspension	None execpt slight distrubance of sensitivity of mouth	Velbinger 1947a,b
250	Oil solution	Variable hyperesthesia of mouth	"
500	"	"	"
750	"	Disturbance of sensitivity of lower part of face; uncertain gait; peak reaction (6 hr after ingestion) characterized by malaise, cold moist skin, and hypersensitivity to contact; reflexes normal	"
1,000	"	Same as above; no joint pains, fatigue, fear, or difficulty in seeing or hearing	"
1,500	"	Pricking of tongue and around mouth and nose beginning 2.5 hr after dose; disturbance of equilibrium; dizziness; confusion; tremor of extremities; peak reaction (10 hr after ingestion) characterized by great malaise, headache, and fatigue; delayed vomiting; almost complete recovery in 24 hr	"

The latter dosage was about 200 times the average daily rate of dietary exposure in the general population at that time. The second study involved 24 men, whose exposure to DDT lasted 21.5 months. Four men were controls, and daily oral doses of technical DDT were given at 3.5 mg/man to six men and at 35 mg/man to six others. Another eight men received p,p'-DDT at 35 mg/man/day. Twenty exposed men were kept under observation until 4 years after the beginning of the study, and 16 of these completed an additional year of observation. No volunteer in either study complained of any symptom or showed any sign of illness, in the tests used, that did not have recognizable cause unrelated to the exposure to DDT. At intervals, the men were given a systems review, physical examination, and a variety of laboratory tests. Particular attention was given to the neurologic examination and liver function tests. No adverse changes were detected, although two men were removed from the 1956 study because of illness. One had contracted hepatitis and the other suffered a myocardial infarction. However, their illnesses were not considered to be caused by exposure to DDT.

In another study which was reported by Morgan and Roan (1971), four volunteers were given oral doses of technical DDT at 10 or 20 mg/day, p,p'-DDE at 5 mg/day, or p,p'-DDD at 5 mg/day for 81-183 days. A battery of hematologic and clinical biochemical tests were conducted before, during, and after exposure. No abnormalities were detected in the four volunteers.

3.3 Studies of Occupationally Exposed Workers

Three studies have been reported of workers with prolonged heavy exposure to DDT. Ortelee (1958) carried out clinical and laboratory examinations of 40 workers, all of whom were exposed to DDT and some of whom were exposed to a number of other pesticides. The men had been employed at this work for up to 8 years, with "heavy" exposure in some cases for up to 6.5 years. Exposure was so intense that during working hours many of the men were coated with a heavy layer of DDT dust. By comparing their excretion of DDA with that of volunteers given known doses of DDT, it was possible to estimate that the average amounts of DDT absorbed by three groups of the workers with different degrees of occupational exposure were 14, 30, and 42 mg/man/day. With the exception of the excretion of DDA and the occurrence of a few cases of minor irritation of the skin and eyes, no correlation was found between any abnormality and exposure to the insecticide. Special attention was given to a complete neurologic examination and to laboratory tests for liver function. Although a few abnormalities, such as hypertension and hearing loss were revealed, the author considered them unrelated to DDT exposure. One worker with a previous history of malaria had a palpably enlarged liver, three had hyperactive deep tendon reflexes, and five had slight tremors of the outstretched hand at rest.

Laws et al (1967) studied 35 men employed for 11-19 years in a plant that had produced DDT continuously and exclusively since 1947 and, at the time of the study, produced 2,722 metric tons/month. Findings from medical histories, physical examinations, routine clinical

laboratory tests, and chest X-rays revealed no ill effects attributable to exposure to DDT. Storage levels of DDT and metabolites in the men's fat ranged from 38 to 647 ppm, versus an average of 8 ppm for the general population. Based on their storage of DDT in fat and excretion of DDA in urine, the average daily intake of DDT by the 20 men with high occupational exposure was estimated to be 17.5-18 mg/man.

Rabello et al (1975) studied lymphocytes from 42 workers who worked in direct contact with DDT in 3 insecticide plants. The frequency of chromatid aberrations was not significantly higher than that in controls from the same plants but not in direct contact with DDT. However, there was evidence that one of the control groups had high exposure to DDT, as evidenced by high residue levels in plasma. When this group was included with the directly exposed workers, there was a significant increase in chromatid aberrations in the exposed workers compared to the control groups. The frequency of chromatid aberrations was 12.0% in exposed workers, 8.8% in workers from the same plants not directly exposed to DDT, and 2.2% in a control group from the general population. The corresponding concentrations of DDT and metabolites in blood plasma were 0.993 ppm, 0.275 ppm, and 0.03 ppm, respectively. The authors suggested that exposure to DDT may cause chromatid lesions.

A number of other studies of occupationally exposed workers have been published, although in most cases no quantitative measures of exposure are available and the workers were exposed to other pesticides in addition to DDT. Studies by several investigators (Long et al 1969;

Morgan and Roan 1969, 1973, 1974; Warnick and Carter 1972; Sandifer et al 1972; Embry et al 1972;) have failed to reveal effects of clinical significance in workers with prolonged, moderate exposure to a wide variety of pesticides. The possibility of adaptive changes (other than enzyme induction) has been suggested (Tocci et al 1969), but the World Health Organization has dismissed these effects as unproven (WHO 1977).

Other reports give some evidence of toxic effects. The reports under discussion tend to fall into two sets, those involving general debility and those involving a single organ or system. Reported conditions representative of general debility include dermatitis, subtle blood changes, general weakness, palpitations, functional angiospasm, headache, dizziness, inappetence, vomiting, lower abdominal pain, chronic gastritis, benign chronic hepatitis, insomnia, a sympathetic "vascular/asthenic syndrome," "vegetative dystonia," and confusion (Kostyuk and Mukhtarova 1970, Bezuglyi et al 1973).

The largest number of heavily exposed workers whose health has been investigated are those associated with malaria control in Brazil and India (WHO 1973). In Brazil, periodic clinical examinations were made of 202 spraymen exposed to DDT for 6 or more years, 77 spraymen exposed for 13 years ending in 1959, and 406 controls. In the first examination, carried out in 1971, differences between exposed and unexposed groups were observed in some neurologic tests, but this result was not confirmed by the second examination in the same year or in subsequent examinations. During 3 years, a survey of illnesses

requiring medical care during the 6 months preceding each periodic medical examination failed to demonstrate any difference between exposed and control groups. A relatively small number of analyses indicated that the concentration of DDT in the blood of spraymen was about three times that of controls.

In India, the blood levels of 144 spraymen were 7.5-15 times those in controls and were at least as high as those reported for workers who make and formulate DDT elsewhere. When the spraymen were examined the following differences from controls were found: knee reflexes were brisker, slight tremor was more often present, and a timed Romberg test was more poorly performed by the spraymen. These apparently positive results led to the selection of 20 men for examination by a neurologist, who concluded that either the differences found initially were not real or that the men's conditions had returned to normal in the few months between the two examinations. The signs were apparently not dose-related, since they showed no correlation with serum levels of DDT (WHO 1973).

Persons have been reported to have experienced headache, dizziness, nausea, vomiting, pain and numbness of the limbs, and general weakness beginning 1-1.5 hours after entering a field treated with DDT (Kolyada and Mikhal'Chenkova 1973). This has been attributed to possible food poisoning or hysteria (WHO 1977). A small number of workers experienced mild narcotic effects (vergito and nausea) when working in confined spaces with DDT (Hayes 1959). Gil and Miron (1949) reported that some persons suffered temporary irritability, fatigue, and other

ill-defined symptoms after exposure in the dusty atmosphere of a delousing station, but the relation of these findings to DDT was not clear. The relationships of these reported symptoms to DDT, to solvents or carriers or to both, are not clear from the circumstances of the reports.

Effects on reproduction have also been reported. One study of the course of labor and puerperium in 390 vineyard workers exposed to DDT, sulfur, methyl parathion, and copper sulfate reported a higher frequency of miscarriage, toxicosis, and asthenia in women exposed to these pesticides than in women not so exposed. Histologic changes in placentas, CNS disturbances, and low birth weight in their children were also reported (Nikitina 1974). The mean concentration of DDT and metabolites in the exposed women were 0.12 ppm in milk and 0.19 ppm in placentas (4.8 and 5.4 times those in controls, respectively). Peck (1970) suggested that the interference with the synthesis of steriod hormones by DDT and other insecticides might be a cause of impotence reported in farmworkers.

Some cardiovascular effects have been reported. In a study of workers occupationally exposed to a combination of organochlorine and organophosphrous pesticides, the incidence of myocardial dystrophy was 56%, versus 9.3% in a control group, and abnormalities in EKG's and vascular effects were noted together with elevated levels of cholesterol and beta-lipoproteins in the blood and decreased phospholipid levels (Bezuglyi and Gorskaya 1976). Carlson and Kolmodin-Hedman (1977) reported that eight men exposed for 6 hours to a number of chlorinated

pesticides (mainly lindane, but including DDT) showed a fall in alpha-lipoprotein levels after their exposure had ceased. Kolmodin-Hedman (1973) had previously reported that these same workers had hyper-high-density alpha-lipoproteinemia.

Several investigators have reported toxic effects on the liver associated with exposure to DDT or other pesticides. Chronic liver damage (cirrhosis and chronic hepatitis) has been reported on the basis of liver biopsies from eight workers heavily exposed to BHC, DDT, or both for periods ranging from 5-13 years. Other factors such as alcoholism, were reportedly excluded as the cause of the cirrhosis (Schuttmann 1968). Bezuglyi et al (1976) reported a case of chronic toxic hepatitis progressing to liver cirrhosis in a pest control worker with 24 years of exposure to DDT, BHC, trichlorphon, ronnel, zinc phosphide, warfarin, and diphacinone. Elevated levels of C-reactive protein were found in workers with chronic exposure to organochlorine pesticides (Takahasi et al 1976). Morgan and Roan (1974) were unable to find a significant correlation between worker exposure to DDT (as determined by serum concentrations of DDT and DDE) and urinary glucaric acid excretion (a measure of microsomal enzyme activity). However, they found a small but significant increase in serum lactic dehydrogenase (LDH) activity and a more substantial and significant decrease in serum creatinine phosphokinase (CPK) in workers with higher levels of DDT and DDE in their serum (Table 3.3.1). Levels of other enzymes were unchanged.

Increased liver microsomal enzyme activity, as reflected by decreased plasma half-life of phenylbutazone, was found in 14 workers

TABLE 3.3.1

SERUM ENZYME ACTIVITIES AND URINARY GLUCARIC ACID EXCRETION IN RELATION TO SERUM CONCENTRATION OF p,p'-DDT IN 127 SUBJECTS

	Subject Quartiles, Based on Serum p,p'-DDT (ppb)				Analysis of Variance
	I	II	III	IX	Among Means
No. of subjects	32	32	32	31	
Mean serum p,p'-DDT (ppb)	2	4	9	52	
Ranges	0-3	3-5	5-16	16-167	
Urinary glucaric acid excretion, umoles/g urinary creatinine	23	18	20	17	NS
Serum GOT	29	26	23	25	NS
Serum GPT	33	35	35	39	NS
Serum LDH	172	174	183	194	P <0.05
Serum alkaline phosphatase	64	63	63	70	NS
Serum CPK	7	6	6	4	P <0.05

Adapted from Morgan and Roan 1974

exposed to DDT and lindane. However, the workers' exposure to lindane was the only one that was clearly significant, as indicated by its concentration in plasma (Kolmodin-Hedman 1973). In a previous study (Kolmodin et al 1969), workers exposed to DDT and lindane metabolized the drug antipyrine more rapidly than unexposed persons, a phenomenon which also was attributed to induction of liver microsomal enzymes.

Poland et al (1970) studied a group of workers in a DDT manufacturing plant. They found that the half-life of the drug phenylbutazone in the blood plasma of the workers was significantly reduced below that in unexposed controls. They also found that excretion of 6-beta-hydroxycortisol in urine was increased by 57% relative to that in controls. Both effects were considered to result from a DDT-induced increase in the activity of microsomal enzymes. The average level of p,p'-DDT in the blood of the exposed workers was 0.573 ppm, corresponding to an average daily intake of about 18 mg/man/day (Poland et al 1970).

Electroencephalograms were obtained from 73 workers exposed to DDT, BHC, and benzilan for periods ranging from 7 months to 20 years. Just over 78% of the records were normal and 21.9% were abnormal. The most severe changes were in persons exposed to the three compounds for 1-2 years; less severe changes were seen with either shorter or longer exposure. The changes were not correlated with age. Some of the EEG records showed bitemporal sharp waves with shifting lateralization combined with low voltage theta activity. Other records showed spike complexes, paroxysmal discharges composed of slow and sharp waves most pronounced anteriorly, and low-voltage rhythmic spikes posteriorly.

None of the persons examined showed abnormal clinical neurologic findings (Israeli and Mayersdorf 1973, Mayersdorf and Israeli 1974).

Extensive experience and numerous medical studies of groups of workers have been reviewed (Hayes 1959), with the finding that dermatitis was common in men who used DDT solutions. The rashes were apparently due primarily to the solvent, especially kerosene. As often happens with rashes caused by petroleum distillates, they were most severe in men when they first started work and cleared in a few days unless contamination was exceptionally severe. Ortelee (1958) also reported eye and skin irritations in his study of 40 workers with intense exposure to DDT.

3.4 Epidemiologic Studies in the General Population

A number of epidemiologic searches for health effects associated with general uses of DDT have been conducted in various parts of the United States, with generally negative results (USDHEW 1969). However, such studies are difficult to conduct in the general population, ie., in nonoccupationally exposed persons, because residues of DDT and its metabolites are widespread in the environment. Virtually everyone is exposed to traces of DDT in food, and residues of DDT and metabolites have been found in the tissues of almost every person examined (USEPA 1975). Accordingly it is impossible to identify unexposed groups for rigorous comparison with exposed groups.

However, since residues of DDT and its metabolites are retained in tissues for months or years after exposure, it is possible to use

these residues as indirect measures of the intensity of past exposure. In a number of published epidemiologic studies, correlations have been sought between various pathologic conditions and tissue levels of DDT and its metabolites.

Maier-Bode (1960) found no essential differences between the concentrations of DDT and DDE in 21 persons who died of cancer (unspecified sites) and those in persons who died of other diseases. Robinson et al (1965) detected no significant differences in levels of DDT and metabolites between 50 biopsy and 50 necropsy samples of fat. Among the necropsy samples, there were no differences between mean storage levels in groups classified by cause of death (neoplasm, cardiovascular disease, infection, or accident). Hoffman et al (1967) measured the concentrations of DDT and DDE in the lipids of abdominal wall tissue taken from 995 persons at autopsy and found no significant associations between residue levels and pathologic changes in any body tissues. For example, DDT and DDE at an average total concentration of 9.6 ± 6.5 ppm in abdominal wall fat was reported in 292 patients with cancer (unspecified sites). This did not differ significantly from an average of 9.4 ± 6.5 ppm in 396 patients with other diseases. Another study dealing with autopsy material from 38 persons over 36 years in age revealed that most of the patients with malignant tumors had above-average levels of DDT in their tissues. High concentrations of DDT were associated with the combination of emaciation, carcinoma, and extensive focal or generalized pathological conditions of the liver (Casarett et al 1968). In another investigation, the average concentrations of DDT

and metabolites in fat tissues at autopsy were 21.96 ppm in 40 cases of carcinoma, 21.37 ppm in five cases of leukemia, 13.66 ppm in 5 cases of Hodgkin's disease, and 9.75 ppm in 42 control cases. Each of the differences from controls was statistically significant. Samples of fat and brain from six patients with brain tumors contained DDT residues comparable to those of controls. Mean concentrations of DDE in tissues of patients with portal cirrhosis were 1.7 times higher than those in controls. Levels in patients with other liver diseases were difficult to analyze because of high variability. However, DDT levels were significantly higher (by a factor of 2.7) in tissues of patients with hypertension than in controls (Radomski et al 1968). (Table 3.4.1)

Oloffs et al (1974) found that liver specimens from cirrhotics contained significantly higher concentrations of DDT than specimens from controls. However, this effect was probably due to an increased concentration of lipid in the liver, as there were no differences between the concentrations of DDT in the adipose tissues or brains of cirrhotics and those in controls.

Wassermann et al (1976) reported that concentrations of DDT and metabolites in malignant tissues from patients with breast cancer were significantly higher than those in adjacent normal tissues in the same patients. In another study, significantly higher concentrations of DDT and metabolites were found in lung tissues from patients with lung cancer than in lung tissues from patients who died of other diseases (Dacre and Jennings 1970).

TABLE 3.4.1

PESTICIDE CONCENTRATIONS IN THE FAT TISSUE OF PATIENTS WITH VARIOUS TERMINAL CONDITIONS

Diagnosis	No. of Cases	Level of Pesticide (ppm SD)		
		p,p'-DDE	p,p'-DDD	p,p'-DDT
Normal	42	6.69 ± 4.07	0.28±0.38	2.77±1.42
Infectious diseases	20	8.89 ± 7.67	0.12±0.27	3.94±5.01
Atherosclerosis	54	12.01 ± 2.53	0.27±0.75	5.10±7.57
Hypertension	8	17.91 ± 6.28	0.40±0.42	6.54±3.64
Carcinoma	40	15.97 ± 3.78	0.34±0.51	5.65±5.61
Leukemia	5	16.10 ± 5.53	0.58±0.45	4.69±4.28
Chronic renal disease	8	8.11 ± 4.25	0.21±0.25	2.11±1.10
Pancreatitis	3	11.57±10.81	0.09	1.29±1.05
Hodgkin's disease	5	10.06 ± 4.43	0.38±0.27	3.22±1.20

Adapted from Radomski et al 1968

A statistical association between serum cholesterol and p,p'-DDE has been reported (Rashad et al 1976). This may have been due to stimulation of liver cholesterol synthesis by p,p'-DDE.

A study of six patients (five with pancytopenia and one with chronic lymphocytic leukemia) who had been exposed to pesticides revealed significant lymphocyte sensitization to DDT in two patients, as measured by the degree of inhibition of leukocyte migration (Traczyk et al 1976). As discussed in Section 3.3, cases of aplastic anemia and other blood dyscrasias have been associated circumstantially with exposure to DDT, but the association is stronger with BHC and other organochlorine pesticides (USDHEW 1969).

Appreciable serum levels of DDT and its residues have been reported in premature infants, although no other toxic effects were detected in these infants (D'Ercole et al 1976). In an independent study, DDE levels in whole blood were much higher in premature infants than in full-term infants. This difference was found independently in white and nonwhite ethnic groups (O'Leary et al 1972; Table 3.4.2).

In most of the studies in which elevated tissue levels of DDT have been associated with cancer or other diseases, levels of other chlorinated hydrocarbons, including dieldrin, heptachlor epoxide, and BHC) have also been elevated in the diseased patients (Casarett et al 1968, Radomski et al 1968, Wassermann et al 1976, Dacre and Jennings 1970).

TABLE 3.4.2

FETAL WHOLE BLOOD DDE VALUES (ppb) IN PREMATURE AND MATURE INFANTS

Race		Term	Premature
White	Mean	4.9	22.1
	Range	2-13	18.7-26.8
	Median	5	21
Negro	Mean	6.1	19.0
	Range	3-12	6.6-34.4
	Median	5	17
Total	Mean	5.8	19.5
	No.	44	23

Adapted from O'Leary et al 1972