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Levels of dichlorodiphenyltrichloroethane and hexachlorocyclohexane in human adipose tissue of the Indian population.

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Levels of dichlorodiphenyltrichloroethane and hexachlorocyclohexane in human adipose tissue of the Indian population

by Jitesh P Jani, PhD, Jayendra S Patel, MSc, Mahesh P Shah, MSc, Mohmadishak R Variya, BSc, Yogesh H Shah, BSc, Satendrakumar Gupta, PhD, Shrikant Kashyap, MD, PhD¹

JANI JP, PATEL JS, SHAH MP, VARIYA MR, SHAH YH, GUPTA S, KASHYAP S. Levels of dichlorodiphenyltrichloroethane and hexachlorocyclohexane in human adipose tissue of the Indian population. *Scand J Work Environ Health* 14 (1988) 201–204. Concentrations of dichlorodiphenyltrichloroethane (DDT) and hexachlorocyclohexane (HCH) were determined in 313 human omental fat samples collected from subjects from all five zones of India during 1977–1980. The median concentration of 2,2-bis-(p-chlorophenyl)-1,1-dichloroethylene (p,p'-DDE), total DDT, beta HCH, and total HCH were 3.4, 6.0, 1.3, and 1.9 mg/kg, respectively. The calculated national mean levels for DDT and HCH were 11.1 and 3.5 mg/kg, respectively. Although the values of DDT and HCH were not as high as those reported earlier, there is still a need for close monitoring of the bioaccumulation of these chemical residues in the Indian population.

Key terms: human fat, organochlorine compounds, pesticides.

The residues of organochlorine pesticides have received worldwide attention in the last few decades since studies have shown an accumulation of these chemicals in the environment, and in human tissues as well (9, 11, 13, 27). There are numerous reports concerning acute (12, 19, 25) and chronic (8, 17, 30) toxic effects of pesticides in man. Higher residues of these chemicals have been reported in patients suffering from diseases such as cirrhosis, atherosclerosis, hypertension, idiopathic amyloidosis, and certain forms of cancer (6, 22, 26).

The biological monitoring of dichlorodiphenyltrichloroethane (DDT) and hexachlorocyclohexane (HCH) is very important, especially in underdeveloped and developing countries like India, where public health programs are still dependent upon these organochlorine chemicals as pesticides. Earlier reports from India (2, 4, 15, 23, 24) have indicated varying levels of DDT and HCH in human fat samples (table 1). All these reports were confined, however, to the north zone of India.

The present paper reports the results of the first nationwide monitoring program for DDT and HCH residues in human adipose tissue.

Samples and methods

The solvents were glass-distilled and free of interfering residues. Organochlorine pesticide standards were obtained from the Environmental Protection Agency,

Research Triangle Park, North Carolina (USA), with certified purities of 97–100 %.

Human omental fat samples (N = 313) were collected during medicolegal autopsies during routine post-mortem. The subjects were between the ages of 10 and 60 years (202 men and 111 women) and were without any history of occupational exposure to organochlorine pesticides.

Figure 1 illustrates the sample collection stations of all five zones of India (North, South, East, West, and Central). The samples were freed of nonfatty material and dried with cellulose paper. The insecticides were extracted through the process of weighing a quantity of dried fat in hexane and heating the mixture for 1 h in a water bath. The undissolved tissue was isolated and extracted in a small portion of fresh hexane. All the hexane extracts were combined and dried for the determination of fat contents. The fat was dissolved in 50 ml of hexane, and 25 ml of the hexane extract was subjected to liquid : liquid extraction (7) with the

Table 1. Mean dichlorodiphenyltrichloroethane (DDT) and hexachlorocyclohexane (HCH) residues in human fat.

Country of origin	Total DDT	Total HCH	Reference
India			
North zone	26.0	.	Dale et al (4)
North zone	21.8	.	Ramachandran et al (24)
North zone	2.2	.	Bhaskaran et al (2)
North zone	1.8	2.3	Kaphalia & Seth (15)
North zone	22.3	16.9	Ramachandran et al (23)
North zone	9.1	4.3	Kalra & Chawla (14)
National mean	11.1	3.5	Present study
Turkey	7.1	1.7	Karakaya & Ozalp (16)
Canada	2.6	0.07	Mes et al (20)
	2.1	0.16	Mes et al (21)
United Kingdom	2.5	0.29	Abbott et al (1)
South Africa	6.4	6.2	Wiese (31)
Australia	9.3	0.7	Wassermann et al (28)
Japan	2.47	1.54	Curley et al (3)

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Figure 1. City locations used for sample collections from different zones of India. (North zone: 1 = Agra, 2 = Chandigarh; south zone: 3 = Bangalore; east zone: 4 = Calcutta; west zone: 5 = Ahmedabad, 6 = Bombay; and central zone: 7 = Bhopal).

Table 2. Minimum detection limit and percentage recovery of organochlorine pesticides and their metabolites. (HCH = hexachlorocyclohexane, p,p'-DDE = 2,2-bis-(p-chlorophenyl)-1,1-dichloroethylene, DDT = dichlorodiphenyltrichloroethane)

Compound	Minimum detection limit (µg/kg)	Recovery ^a (%)
Alpha HCH	0.1	92–96
Gamma HCH	0.04	96–101
Beta HCH	0.2	86–94
p,p'-DDE	2.5	95–98
o,p'-DDT	0.5	94–96
p,p'-DDT	0.5	96–99

^a Five sets.

use of dimethylformamide (9 : 1, volume per volume). After partition from dimethylformamide the hexane extract was concentrated to 1 ml for alumina column cleanup. Two grams of 5 % deactivated alumina was wet-packed in a column, and the sample was charged. The column was eluted with 50 ml of hexane, and the eluent was concentrated to 1 ml for analysis by gas-liquid chromatography.

Table 2 contains the average recovery and detectability data from the duplicate analysis of two 500-mg samples of human adipose tissue. In the recovery experiment 0.1 ml of the reference mixture was added to one of the 500-mg human fat samples, and the spiked and unspiked samples were put through the entire procedure. Appropriate corrections were made from the results of the spiked sample analysis, and,

depending upon the results of the unspiked sample, the recovery was calculated.

The final analysis was done by gas-liquid chromatography with a nickel-63 (Ni^{63}) electron capture detector and a glass column (1.8 m in length with an outer diameter of 0.6 cm) packed with 3 % trifluoropropyl, methyl silicone (DC QF-1) on chromosorb W (80/100). The confirmation of isomers was done on a 5 % OV-17 (50 % phenyl, methyl silicone) column.

The height and area of the unknown concentrations were compared with that of the standards of known concentrations.

Results

The mean levels of the DDT and HCH isomers in the samples are given in tables 3 and 4, respectively, according to five zones of India. Table 5 presents summation of the results for all the samples combined. The frequency distribution for the DDT and HCH residues is shown in figures 2 and 3, respectively. Figure 4 shows the distribution of the DDT and HCH residues in the men and women separately and in the different age groups of the subjects from whom the samples were taken.

Discussion

Table 3 shows that the total DDT was highest (mean 17.2 mg/kg) in the samples from the west and lowest (6.5 mg/kg) in those from the east. For total HCH, shown in table 4, the maximum (5.1 mg/kg) and minimum (1.1 mg/kg) levels were observed in the south and central zones, respectively. Such a wide variation in the bioaccumulation of DDT and HCH in different zones of India might be due to differences in the intake of these chemicals because of varying residues or due to differences in eating habits or due to a combination of these. Racial and geographic variation in the residues of these chemicals in human body fat have also been reported earlier (5, 18).

The calculated national mean levels for DDT, HCH, and their major isomers p,p'-DDE [2,2-bis-(p-chlorophenyl)-1,1-dichloroethylene] and beta HCH were 11.1, 3.5, 7.4, and 2.5 mg/kg, respectively. However, the most recurrent values for DDT, HCH, p,p'-DDE, and beta HCH were only 4.0, 1.1, 2.0, and 0.7 mg/kg, respectively. Furthermore, it was observed that p,p'-DDE and beta HCH contributed about 60–70 % of the total DDT and HCH in the fat tissues.

The frequency distribution curves for DDT and HCH residues were found to be highly skewed. Half of the total samples (50 %) showed DDT residues below the level of 6.0 mg/kg and HCH residues below 1.8 mg/kg.

In contradiction to an earlier report (29), no significant correlation was found between the bioaccumulation of DDT or HCH with age. This finding might be

Table 3. Mean and range of dichlorodiphenyltrichloroethane (DDT) residues (mg/kg) in human fat samples from inhabitants of various zones of India.

Zone	Number of samples	p,p'-DDE		o,p'-DDT		p,p'-DDT		Total DDT	
		Mean	Range	Mean	Range	Mean	Range	Mean	Range
East	45	4.2	0.3–16.4	0.01	0.2–2.4	1.9	0.4–11.8	6.5	1.4–37.1
West	114	12.0	0.2–128.1	0.7	0.4–23.0	3.2	0.1–25.0	17.2	1.3–176.1
North	24	5.9	0.4–51.3	0.3	0.3–5.0	8.5	0.9–70.6	15.4	1.9–131.6
South	116	4.3	0.2–64.4	0.9	0.4–20.7	2.2	0.03–12.9	7.8	0.2–80.7
Central	14	6.4	0.3–29.9	0.2	0.9–12.8	1.9	0.1–24.1	9.2	1.0–37.1

Table 4. Mean and range of hexachlorocyclohexane (HCH) residues (mg/kg) in human fat samples from inhabitants of various zones of India.

Zone	Number of samples	Alpha HCH		Gamma HCH		Beta HCH		Total HCH	
		Mean	Range	Mean	Range	Mean	Range	Mean	Range
East	45	0.3	0.06–0.6	0.3	0.01–0.8	1.05	0.06–3.9	1.6	0.1–4.84
West	114	0.4	0.01–4.6	0.2	0.02–1.7	2.6	0.10–17.5	3.2	0.2–20.6
North	24	0.5	0.01–5.5	0.2	0.02–1.3	1.5	0.07–9.0	2.2	0.2–11.0
South	116	1.0	0.01–21.0	1.2	0.01–28.0	2.9	0.01–15.7	5.1	0.02–94.5
Central	14	0.2	0.05–0.5	0.04	0.01–0.08	0.8	0.3–1.5	1.1	0.25–1.9

Figure 2. Frequency distribution curve of dichlorodiphenyltrichloroethane (DDT) residues in human fat samples. (No = number)

Figure 3. Frequency distribution curve of hexachlorocyclohexane (HCH) residues in human fat samples. (No = number)

Table 5. Average dichlorodiphenyltrichloroethane (DDT), hexachlorocyclohexane (HCH), and their isomer residue levels in human fat samples (N = 313). (p,p'-DDE = 2,2-bis-(p-chlorophenyl)-1,1-dichloroethylene)

Compound	Mean (mg/kg)	Median (mg/kg)	Mode (mg/kg)	Range (mg/kg)	Range covering central 90 % observations (mg/kg)
Total DDT	11.1	6.0	4.0	0.2–176.1	0.7–32.7
p,p'-DDE	7.4	3.4	2.0	0.2–128.1	0.3–27.3
Total HCH	3.5	1.9	1.1	0.03–94.6	0.2–10.5
Beta HCH	2.5	1.3	0.7	0.01–45.7	0.12–7.9

Figure 4. Distribution of dichlorodiphenyltrichloroethane (DDT) and hexachlorocyclohexane (HCH) residues according to the age and sex of the person from which the samples were taken.

due to a relatively high daily dietary intake of these chemicals as residues in food materials from infancy (10, 13, 24). Furthermore, the residues show somewhat higher levels for men than for women (figure 4).

Although the residue buildup of DDT and HCH in the general population of India was not found to be as high as reported earlier (4, 23, 24), there is still need for close monitoring of the bioaccumulation of these chemical residues in Indians.

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Inhaled dust and disease. By Paul F Holt. John Wiley & Sons, Chichester—New York—Brisbane—Toronto—Singapore 1987. 325 p. ISBN 0-471-91665-2. (Price: GBP 35.50)

Professor PF Holt is excellently qualified to write an overview on inhaled dusts and their related diseases. He started to research industrial lung diseases 50 years ago and since 1975 has been Professor Emeritus at the Reading University in the United Kingdom.

The causal relationship between the regular inhalation of various kinds of dust and disease is well known. The dusts discussed in this book cover a wide range of inorganic and organic types, eg, asbestos, coal, quartz, china clay, talc, cotton, etc. The primary emphasis is placed, however, on fibrous silicates and, especially, asbestos. Asbestos-related questions are discussed in 10 of the 22 chapters of the book.

The fact that mesothelioma has been discussed in detail is not surprising in light of the author's report of a doubling in incidence in Great Britain in 1967—1976 and again in 1976—1984. Mesothelioma is considered a malignant tumor. Benign types are not treated. Mesothelioma is nowadays regarded as a disease that is nearly always caused by asbestos exposure. Children are at a greater risk than adults to incur mesothelioma if exposed to asbestos dust. Sometimes an environmental exposure to asbestos in childhood has resulted in death from mesothelioma many years later. In a few young children however the occurrence of mesothelioma is probably not related to exposure to asbestos. The most disconcerting aspect of the relationship between malignant mesothelioma and asbestos is the documented association of mesothelioma with apparently low levels of exposure for relatively

brief periods in the past. This phenomenon implies a lack of a dose-response relationship.

The association between lung cancer and asbestos exposure is also discussed in the book. Professor Holt states that the risk of developing bronchial carcinoma appears to be directly related to the intensity of exposure to asbestos. A linear relationship between accumulated asbestos exposure and lung cancer has been widely accepted on the basis of the results of studies on asbestos textile and factory workers and ladders. Other types of dose-response relationships are also discussed, as are also the major findings of experimental research.

The health hazards of man-made mineral fibers are not so clear. Large epidemiologic studies of populations exposed to these fibers have shown no excess occurrence of mesotheliomas, whereas an excess of lung cancer has been reported for workers with 30 years or more since their first exposure. The early environmental conditions of rock-wool and slag-wool production might have helped to increase the incidence of lung cancer in this group of workers.

On the whole the book is very well written and easy to read. It covers the main epidemiologic evidence of the various exposures and diseases, and it illustrates the size of the problem. This book is valuable for occupational health and safety experts who are interested in various exposures which may cause occupational lung diseases. The diseases themselves have not been described in detail however.

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