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HCH and DDT Residues in Indian Soil: Atmospheric Input and Risk Assessment

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Chapter 3
HCH and DDT Residues in Indian Soil: Atmospheric Input and Risk Assessment

Paromita Chakraborty, Sanjenbam Nirmala Khuman, Bhupandar Kumar, and Bommanna Loganathan

3.1 Introduction

India is an agricultural country located in diverse climatic zones. Agriculture, with its allied sectors, is the largest source of livelihood in India, particularly in the vast rural areas contributing significantly to the Gross Domestic Product (GDP). Organochlorine pesticides (OCPs) such as dichlorodiphenyltricholoroethane (DDT) and hexachlorocyclohexane (HCH) have been extensively used in India for agricultural and public health purposes for more than five decades. DDT, HCH, and malathion (organophosphorous compound) constitute 70 % of the annual pesticide consumption (85,000 t) (Gupta 2004). OCPs were banned for agricultural practices in the late 1990s, but a substantial amount of these insecticides are still being used for exterminating insects that spread diseases such as malaria, kala-azar (black fever), etc. Due to widespread use, these pesticides continue to contaminate different environmental compartments because of their semi-volatile nature and long environmental lifetimes in soil and water (Kurt-Karakus et al. 2005).

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Owing to the hydrophobic and lipophilic properties and affinity towards particles, DDT and HCH accumulate in the organic matter of soil for longer period (Ockenden et al. 2003). Soil, therefore not only acts as the sink for these pollutants, but also acts as a secondary source by re-emitting these compounds into atmosphere (Harner et al. 2001; Wild and Jones 1995). Several studies (Babu et al. 2003; Rajendran and Subramanian 1999; Ramesh et al. 1991; Senthilkumar et al. 2001) have emphasized that HCHs and other OCPs with similar physicochemical properties are dissipated from soil under the tropical/subtropical conditions leading to their widespread distribution (Chakraborty et al. 2015; Agoramoorthy 2008; Rekha and Prasad 2006). In addition, our earlier studies have revealed the occurrence and distribution of persistent organic pollutants (POPs) in Indian atmosphere (Chakraborty and Zhang 2011). This chapter provides an overview of HCH and DDT residues in soil across northern, eastern, north-eastern, western, central, and southern part of India, their atmospheric input and the associated risk for human health.

3.2 Methodology

Concentrations of HCH isomers and DDT isomers and its metabolites in soil and air from different parts of India were compiled using recent literature. The data were used to elucidate the current status of contamination and human health risk estimation.

3.2.1 Sample Collection, Column Cleanup, and Instrumental Analysis

Surface soil samples were collected from the national capital city, New Delhi, and states, viz., Assam, Chattisgarh, Goa, Haryana, Karnataka, Maharashtra, Manipur, Tamilnadu, Tripura, Uttarpradesh, Uttarakhand, and West Bengal, and seven major Indian cities based on urban–suburban and rural transect. The details on the soil samples from different states have been given in Table 3.1. In most of the studies, Soxhlet was used for extraction of OCPs from the soil samples (Abhilash and Singh 2008; Chakraborty et al. 2015; Devi et al. 2013, 2015; Kumar et al. 2012; Singh et al. 2007). Some of the samples were processed by other extraction methods like shaker (Minh et al. 2006; Prakash et al. 2004; Ramesh et al. 1991), ultrasonication (Kumar et al. 2014) etc. After extraction, the sample extracts were subjected to column chromatography packed with alumina, silica gel, and sodium sulfate for cleanup. The sample extracts were further subjected to instrumental analysis in either Gas Chromatography (GC) equipped with electron capture detector (ECD) (Abhilash and Singh 2008; Agnihotri et al. 1996; Devi et al. 2013; IOG 2002; Jayashree and Vasudevan 2006; Kumar et al. 2012, 2014; Kumari et al. 2008; Mishra et al. 2003; Prakash et al. 2004; Ramesh et al. 1991; Singh et al. 2007) or Gas Chromatography interfaced with Mass Spectrometry techniques (Chakraborty et al. 2015).

Table 3.1 HCHs and DDTs residues in soil from various states of India

OCPs	Concentration (ng/g)	Soil type	Place	Year	References
HCHs	180–1586	Paddy fields	Dibrugarh	2009–2010	Mishra et al. (2003)
HCHs	345–1844	Paddy fields	Nagaon	2009–2010	
HCHs	75–2259	Tea gardens	Dibrugarh	2009–2010	
HCHs	223–1639	Tea gardens	Nagaon	2009–2010	
HCHs	178–1701	Others	Dibrugarh	2009–2010	
HCHs	98–1945	Others	Nagaon	2009–2010	
DDTs	75–2296	Paddy fields	Dibrugarh	2009–2010	
DDTs	166–2288	Paddy fields	Nagaon	2009–2010	
DDTs	218–2129	Tea gardens	Dibrugarh	2009–2010	
DDTs	351–1981	Tea gardens	Nagaon	2009–2010	
DDTs	172–1833	Others	Dibrugarh	2009–2010	
DDTs	181–1811	Others	Nagaon	2009–2010	
HCHs	122–638	Paddy fields	Dehradun	NA	Babu et al. (2003)
DDTs	13–238	Paddy fields	Dehradun	NA	
HCHs	5.83–85.083	Agricultural	Thiruvallur	2004–2005	Jayashree and Vasudevan (2006)
DDTs	1–10.5	Agricultural	Thiruvallur	2004–2005	
HCHs	89.40 (mean)	Agricultural	Aligarh	1998–1999	Nawab et al. (2003)
DDTs	34 (mean)	Agricultural	Aligarh	1998–1999	
Aldrin	1.46 (mean)	Agricultural	Aligarh	1998–1999	
HCHs	0.017–0.121	Forest	Assam	2006–2009	Devi et al. (2013)
DDTs	0.101–0.626	Forest	Assam	2006–2009	
Endos	0.161–0.463	Forest	Assam	2006–2009	
HCHs	0.015–0.097	Wildlife	Tripura	2006–2009	
DDTs	0.110–0.626	Wildlife	Tripura	2006–2009	
HCHs	0.018–0.149	Forest	Tripura	2006–2009	
DDTs	0.048–0.364	Forest	Tripura	2006–2009	
HCHs	0.006–0.140	Tea estate	Tripura	2006–2009	
DDTs	0.049–0.749	Tea estate	Tripura	2006–2009	
HCHs	0.029–0.234	Grassland	Tripura	2006–2009	
DDTs	0.096–0.549	Grassland	Tripura	2006–2009	
HCHs	0.029–0.234	Roadside	Manipur	2006–2009	
DDTs	0.096–0.549	Roadside	Manipur	2006–2009	
HCHs	0.080–2.950	Forest	Manipur	2006–2009	
DDTs	0.241–3.870	Forest	Manipur	2006–2009	
HCHs	0.079–1.642	Wetland	Manipur	2006–2009	
DDTs	0.328–5.208	Wetland	Manipur	2006–2009	
HCHs	7.1	Watershed	Vellar	1988–1989	Ramesh et al. (1991)
DDTs	1.5	Watershed	Vellar	1988–1989	
HCHs	0.003–0.33	Estuary	Hughli	1998–2000	Bhattacharya et al. (2003)
DDTs	0.003–0.119	Estuary	Hughli	1998–2000	

(continued)

Table 3.1 (continued)

OCPs	Concentration (ng/g)	Soil type	Place	Year	References
HCHs	6.4–212.2	Surface soil	New Delhi	2002	Prakash et al. (2004)
HCHs	0.2–212.2	Surface soil	Haryana	2002	
HCHs	6.91–637	Surface soil	Lucknow	2002	
HCHs	12.31–118.64	Sub surface soil	New Delhi	2002	
HCHs	10.97–382.97	Sub surface soil	Haryana	2002	
HCHs	0.08–7.25	Alluvial soil	Unnao, Gangetic plain	2003	Singh et al. (2007)
DDTs	BDL–74.06	Alluvial soil	Unnao, Gangetic plain	2003	
HCHs	53–99	Industrial	Lucknow	NA	Abhilash and Singh (2008)
HCHs	BDL-9	Dumpsite	Perungudi	1999–2001	Minh et al. (2006)
DDTs	BDL-63	Dumpsite	Perungudi	1999–2001	
HCHs	2–51	Agricultural	Haryana	NA	Kumari et al. (2008)
DDTs	1–66	Agricultural	Haryana	NA	
HCHs	14–158	Surface Alluvial soil	Farukabad	1991–1992	Agnihotri et al. (1996)
DDTs	27–337	Surface Alluvial soil	Farukabad	1991–1992	
HCHs	12–67	Subsurface Alluvial soil	Farukabad	1991–1992	
DDTs	28–295	Subsurface Alluvial soil	Farukabad	1991–1992	
HCHs	BDL-2.79	Surface soil	Itanagar, Guwahati, Tezpur, Dibrugarh	2012	Devi et al. (2015)
DDTs	0.28–2127	Surface soil	Itanagar, Guwahati, Tezpur, Dibrugarh	2012	
HCHs	0.24–59.8	Surface soil	Bangalore	2006–2007	Chakraborty et al. (2015)
DDTs	0.34–78	Surface soil	Bangalore	2006–2007	
HCHs	0.231–16.8	Surface soil	Chennai	2006–2007	
DDTs	0.35–10	Surface soil	Chennai	2006–2007	
HCHs	0.04–7.6	Surface soil	Mumbai	2006–2007	
DDTs	0.81–9.2	Surface soil	Mumbai	2006–2007	
HCHs	1.88–15.7	Surface soil	Goa	2006–2007	
DDTs	5.55–124.8	Surface soil	Goa	2006–2007	
HCHs	1.6–13.2	Surface soil	Agra	2006–2007	
DDTs	3.1–20.5	Surface soil	Agra	2006–2007	
HCHs	0.027–33.8	Surface soil	New Delhi	2006–2007	
DDTs	0.15–42	Surface soil	New Delhi	2006–2007	
HCHs	0.23–21.2	Surface soil	Kolkata	2006–2007	
DDTs	0.41–124	Surface soil	Kolkata	2006–2007	

3.2.2 *Risk Assessment*

Human exposure and consequent health risk to soil borne HCH and DDT residues in seven major Indian cities covering northern, eastern, western, and southern parts of India (Chakraborty et al. 2015) were estimated using the procedure described by the United States Environmental Protection Agency (USEPA). Incremental Lifetime Cancer Risk (ILCR) for each site was estimated (ATSDR 2005; USEPA 1989). ILCR for human was assessed from the estimated Lifetime Average Daily Dose (LADD) of DDTs and HCHs as per guidelines given by USEPA and Agency for Toxic Substances and Disease Registry (ATSDR) (ATSDR 2005; USEPA 1989). The equations used for estimating LADD and ILCR were as follows:

$$\mathrm{LADD}\left(\mathrm{mg\ kg^{-1}day^{-1}}\right) = (\mathrm{Cs} \times \mathrm{IR} \times \mathrm{F} \times \mathrm{EF} \times \mathrm{ED})/(\mathrm{BW} \times \mathrm{AT}), \tag{3.1}$$

where

Cs Pollutant concentration in soil (mg $\mathrm{kg^{-1}}$),
IR Soil ingestion rate (100 mg $\mathrm{day^{-1}}$ for adult and 200 mg $\mathrm{day^{-1}}$ for children),
F Unit conversion factor,
EF Exposure frequency (365 days $\mathrm{year^{-1}}$),
ED Lifetime exposure duration (adults—70 years; children—12 years),
BW Body weight (adults—70 kg; children—27 kg),
AT Averaging time for carcinogens (EF × ED).

$$\mathrm{ILCR} = \mathrm{LADD} \times \mathrm{CSF} \tag{3.2}$$

where CSF is cancer slope factor for a particular compound intake (mg $\mathrm{kg^{-1}}$ $\mathrm{day^{-1}}$).

3.3 Results and Discussion

3.3.1 *HCH and DDT: Production and Usage*

Unregulated use of synthetic pesticides started in India during 1948–1949 with the use of DDT for malaria control and HCH (also known as BHC) for locust control. Indian pesticides production started with the setting up of a BHC technical plant at Rishra near Kolkata in 1952. Hindustan Insecticides Ltd. set up two more units to manufacture DDT. Details of production and consumption of HCH and DDT in India have been given in Table 3.2.

India is the world's third largest consumer of technical HCH. The production, consumption, export, and import of HCH have been given in Table 3.2. About 10,43,000 t of HCH was produced between 1948 and 1997, and the total consumption of technical HCH in India during 1948–2000 was 10,57,000 t (IOG 2002; Wei

Table 3.2 Production, consumption, import, export, commencement, and ban of OCPs in India

				No. of plants				
Pesticide	Production (t)	Consumption (t)	Import/export (t)	Past	Now	Start year	Ban year	References
DDT	222721.82 1955–2009 5000 t/year 2011	85672 t (1988–2008)	Exp. 966 (1991–1999) Exp. 1.31 (2006–2010)	3	2	1955	1989	NIP (2011)
HCH	1042612 t (1948–1997)	1057000 t (1948–2000)	–	2	0	1954	1997	IOG (2002), Wei et al. (2007)
Lindane	6687 t (1990–2004)	2411 t (1995–2000)	Imp. 61 (2003–2005) Exp. 802.22 (1996–2008)	2	2	1995	2013	DGFT (2008); MCF (2001–2007), PPQS (2013))

et al. 2007). Cumulative consumption of the HCHs in India until 1985 was 5,75,000 t, and since then, about 45,000 t of HCH was used annually until it was banned in the year 1997.

During 2005-2010, DDT was used for malaria control in various states of India (Fig. 3.1) (NVBDCP 2010). It is very clear that DDT was extensively consumed by the north-eastern states of India particularly Assam. Since 1987, the production of DDT has decreased in India. DDT production was ceased in a plant at New Delhi in 1998. China is the largest consumer, importer, and exporter of DDT. The United States of America produced significant quantity of DDT, although production ceased in 1972. India and China are the only countries currently manufacturing and exporting DDT to other countries, where these insecticides are exclusively used for public health purposes. India is the only country where more than 1,00,000 t of DDT was applied since its inception, mainly in agricultural and vector control programs until it was banned for agricultural use in 1989. Among various states, Chhattisgarh has a major malaria problem and the state contributed about 13 % of the total malaria cases reported in the country. OCPs and synthetic pyrethroids have been used for national malaria control program in the past (Kumar et al. 2014).

Lindane is a gamma isomer of HCH, mainly used as insecticide. In India, lindane formulations are registered for usage in pharmaceutical products (Gupta 2004). In the year 2000, the production of lindane was 1107 t, and subsequently, India signed treaties against the usage of lindane. The production further declined in 2008 to 75 t (DGFT 2008; PPQS 2013). Being the largest exporter of lindane in the year 1998, India supplied 207 t to other countries. During 1990s with 1000 t of lindane production, India was reported to be the highest producer of lindane (DGFT 2008; MCF 2007; PPQS 2013). During 1990-2004, consumption of lindane in India was 6840 t, i.e., only 2 % of the global usage (Vijgen et al. 2006). Finally in 2013, Lindane has been banned in India.

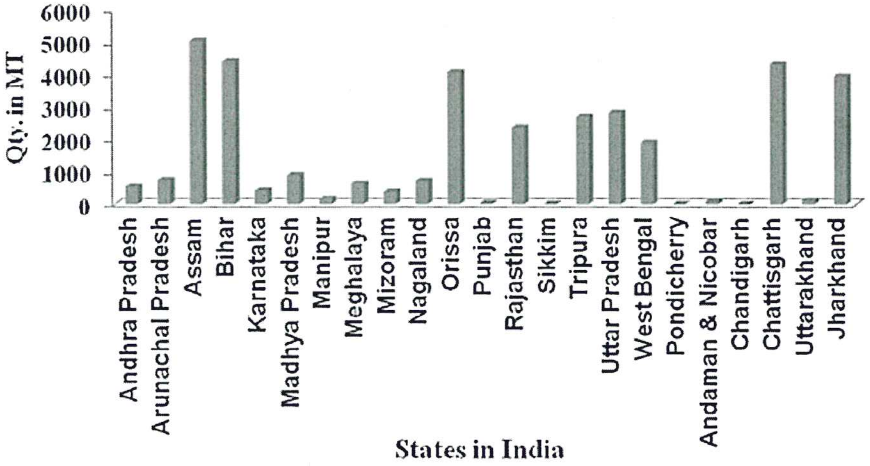

Fig. 3.1 DDT usage for malaria control in various states of India during 2005–2010 (NVBDCP 2010)

3.3.2 *Region-Specific Distribution and Atmospheric Input*

Maximum HCH and DDT residues observed in soil from different states of India between 2005-2015 have been given in Fig. 3.2.

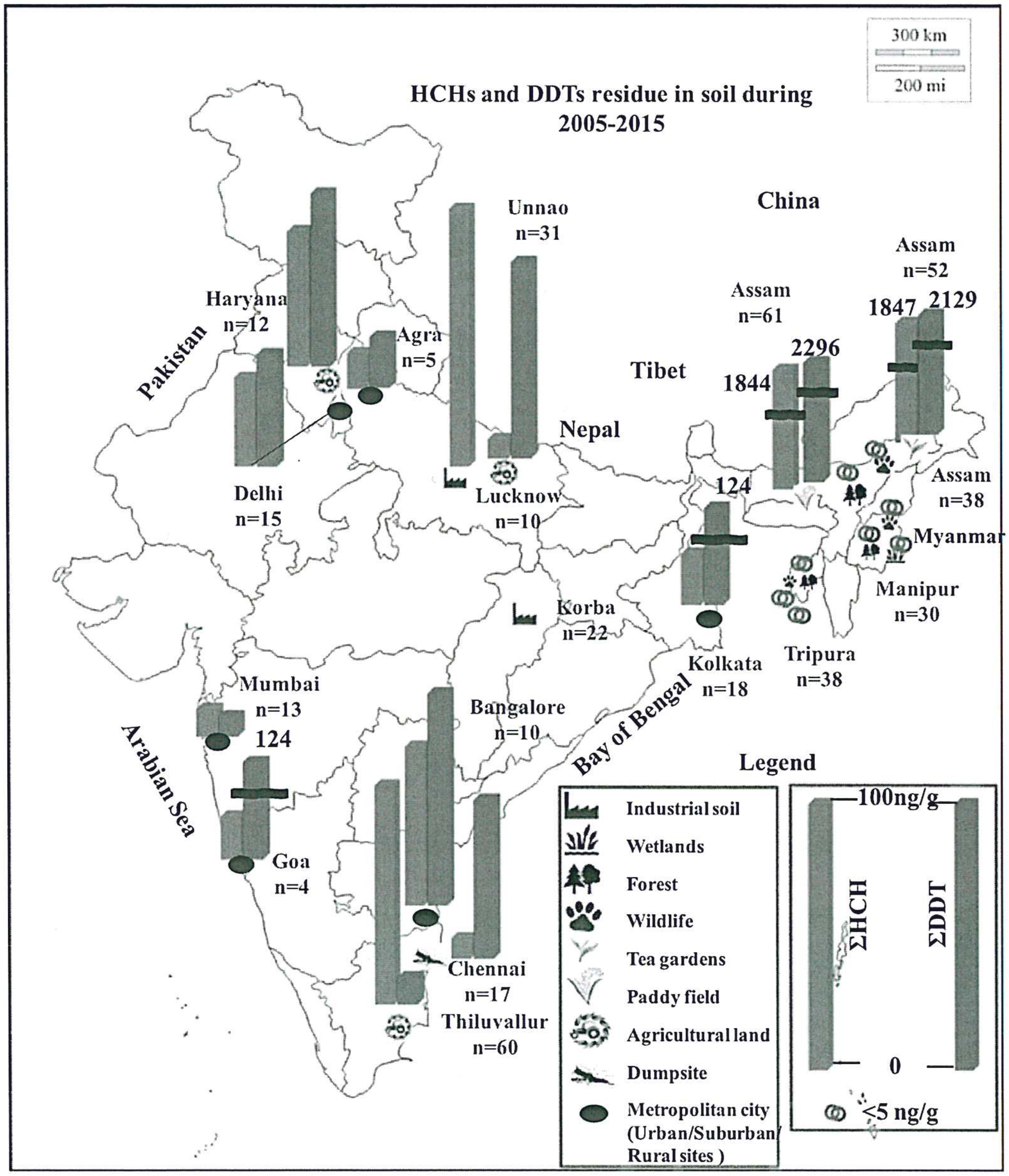

Fig. 3.2 Maximum HCH and DDT residues in surface soil from different states of India. Concentrations presented in this figure have been obtained from various studies in India (Abhilash and Singh 2008; Chakraborty et al. 2015; Devi et al. 2013; Jayashree and Vasudevan 2006; Kumar et al. 2014; Kumari et al. 1996; Mishra et al. 2003; Singh et al. 2007)

3.3.2.1 Northern India

HCH Elevated levels of HCH were observed in alluvial plains of Farukabad (Gupta 2004). In the capital city, New Delhi, and a nearby city, Agra, all the HCH isomers were prevalent in soil and ΣHCHs showed significant positive correlation with soil organic carbon (Chakraborty et al. 2015). Isomeric composition of soilborne HCH isomers in New Delhi ($\alpha = 4.4$ %, $\beta = 51.3$ %, $\gamma = 29.4$ %, $\delta = 15$ %) and Agra ($\alpha = 4.4$ %, $\beta = 51.3$ %, $\gamma = 29.4$ %, $\delta = 15$ %) showed elevated δ-HCH for sites in New Delhi close to Uttar Pradesh border as well as in all the sites of Agra located within Uttar Pradesh (Chakraborty et al. 2015). HCH isomers detected in the agricultural soil of New Delhi were attributed to the runoffs from the dump sites of the adjoining states (Prakash et al. 2004). In Haryana, a north Indian state in the western part of New Delhi, agricultural soil from paddy-wheat, cotton-wheat, and sugarcane fields were detected predominantly with γ-HCH (Kumari et al. 2008). Similarly at Kurukshetra in Haryana, all the HCH isomers, viz., α-HCH (33 %), β-HCH (35 %), γ-HCH (29 %), and δ-HCH (4 %), were observed (Kumar et al. 2012). Lindane usage was evident in all the studies. Chakraborty et al. (2010) reported that the wide range of fugacity fractions for δ-HCH showed deposition due to site-specific contamination especially in New Delhi and Agra possibly due to contamination from the nearby lindane manufacturing units (Prakash et al. 2004). Around Lindane producing factory, γ-HCH (lindane) was detected in all the soil samples, but a decreasing trend in the concentration of HCH was observed as the sampling sites extended from the center of lindane production to the outskirts of the industrial area in Lucknow (Abhilash and Singh 2008). Hence, the dumped waste from the HCH manufacturing unit in Haryana (Prakash et al. 2004) and the by-products released from lindane manufacturing unit in Uttar Pradesh (CAPE 2005) were attributed as the prime reasons for soilborne HCH isomers.

Higher atmospheric α-, β-, and δ-isomers of HCHs in New Delhi particularly from the suburban site, Gagan Vihar, and in the rural sites bordering the state of Uttar Pradesh have been evidenced in back trajectory analysis of air parcels during a passive air sampling (PAS) study (Chakraborty et al. 2010). Air parcels ending in New Delhi irrespective of the site of origin traversed across the lindane manufacturing unit located in Uttar Pradesh (Chakraborty et al. 2010). High atmospheric HCH isomers in Agra were also affected by a major cluster originating from north of Agra located more close to the lindane manufacturing unit (Chakraborty et al. 2010).

DDT Concentrations of DDDs and DDEs were on the higher side in soil from New Delhi, indicating historical usage of DDT. In addition, elevated *o,p′*-DDT levels and the highest *o,p′*-DDT/*p,p′*-DDT ratio were observed at Agra (average 7) followed by New Delhi (average 5), indicating ongoing usage of DDT (Chakraborty et al. 2010). The observed ratio of DDT/(DDD + DDE) for this study ranged between 0.09 and 2.39 with an average value of 0.75, indicating that DDT input in this area is both due to past and present usage (Chakraborty et al. 2015). In Kurukshetra city, *p,p′*-DDT, *o,p′*-DDT, *p,p′*-DDE, and *p,p′*-DDD occupied 12 %, 26 %, 30 %, and 33 % of total DDT respectively (Kumar et al. 2012). Among DDT analogues, *p, p′* DDE was found to be the dominant in Haryana, indicating past usage (Kumari et al. 2008). Mostly DDT isomers showed deposition at specified locations of New Delhi (Chakraborty et al. 2015). Very high concentration of DDT has been observed in Farukabad and somewhat lower concentration in Lucknow (Gupta 2004).

3.3.3 *Eastern and North-Eastern India*

HCH In most locations of Kolkata, a major metropolitan city in eastern India, α-HCH was found to be one- to two folds higher than γ-HCH, indicating ongoing use of technical HCH apart from Lindane (Chakraborty et al. 2015). Maximum soil-borne HCHs have been observed in Assam, a northeastern state of India with dominance of β-isomer particularly in paddy fields, and γ-HCH was dominant in tea garden (Mishra et al. 2003). Mean value of α/γ HCH in Dibrugarh (2.78) and Nagaon (2.51) suggests potential usage of technical HCH in Assam (Mishra et al. 2003). Soil from paddy fields contain substantially and significantly ($p < 0.05$) higher amount of HCHs compared to tea gardens, other agricultural fields, and fallow land (Mishra et al. 2003). More than 50 % of the soil samples taken from the forest cover of Manipur were detected with HCH with higher prevalence of β-HCH and δ-HCH concentration in Tripura (Devi et al. 2013). Therefore HCHs in the eastern and north-eastern states of India can be attributed to both lindane and technical HCH usage. High atmospheric HCH concentration was observed in Kolkata (Chakraborty et al. 2010). Most of the sampling sites demonstrated the usage and deposition of lindane in the background soil from the north-eastern states of India (Devi et al. 2013). Back trajectory analysis showed major air mass clusters originating from northern and eastern parts of India and traversed through Kolkata. Transboundary movement from Bangladesh before ending at Manipur, indicated potential long-range atmospheric transport of these pollutants from the source regions (Devi et al. 2011).

DDT An urban tilt of DDT has been observed in Kolkata. Apart from agricultural use (Guzzella et al. 2005), dicofol was used as an effective acaricide for tea cultivation in the north-eastern part of India (Saha et al. 2004). Soilborne DDTs from paddy fields showed substantially and significantly ($p < 0.05$) higher amount of DDT compared to tea gardens, other agricultural fields, and fallow land (Mishra et al. 2003). High average o,p'-DDT/p,p'-DDT ratios were observed in rural sites with highest DDT in soil from agricultural sites of Barasat, therefore suggesting the use of fresh DDT (Chakraborty et al. 2015). Dominance of p,p'-DDT has been observed in Assam with mean p,p'-DDT/((p,p'-DDE + p,p'-DDD) ratio of 1.25 and 1.82 in Dibrugarh and Nagaon respectively (Mishra et al. 2003). Higher p,p'-DDT for tea garden (having high organic carbon and high acidic soil) and more p,p'-DDE in paddy soil (having comparatively low organic carbon and high clay content) suggest more use of technical DDT to control malaria vectors or for intense paddy cultivation in the past and ongoing usage in tea plantation (Mishra et al. 2003). Low pesticide concentration was observed where there are less agricultural activities and vegetation cover. Higher concentration with the maximum load of soilborne DDT was found in wetland soil of Manipur (Devi et al. 2013). Wildlife sanctuary of Tripura contributed maximum amount of DDT load with dominance of metabolites, i.e., *p,p′*-DDE and *p,p′*-DDD (Devi et al. 2013). In Dibrugarh and Nagaon districts, 81,553 ha (24.09 %) and 160,035 ha (38.94 %) area is under paddy cultivation (summer, winter, and autumn paddy) with 66,309 ha (49.12 %) and 234,633 ha (61.25 %) total sown area (NIC 2004–2005) were subjected to high pesticide application, which may be a possible explanation for elevated pesticidal residues in these districts (Mishra et al. 2003).

3.3.4 Southern India

HCH Agricultural soil in Thiruvallur district of Tamil Nadu was highly contaminated with pesticidal residues particularly with higher concentration of γ-HCH, and the residual levels of α- and δ-HCH were lower than those of γ- and β-HCH (Jayashree and Vasudevan 2006). Tropical climate in southern India facilitates the post-application volatilization of 90 % HCHs from soil to atmosphere in the paddy fields (Takeoka et al. 1991), leading to the highest level of atmospheric gaseous phase HCHs in Chennai and very low HCH concentration in the particulate phase (average 0.55 %) (Chakraborty et al. 2010). Highest level of ΣHCHs was observed in Bangalore and γ-HCH was predominant, but a fair amount of α- and β-isomers were also present (Chakraborty et al. 2015). Elevated levels of γ-HCH in the urban sites are possibly due to its use in healthcare programs (Subramanian and Tanabe 2007).

DDT Over 60 % soilborne DDT in Chennai was comprised of DDT isomers (*o,p′*-DDT and *p,p′*-DDT), indicating ongoing technical DDT usage (Chakraborty et al. 2015). This reflects net volatilization of OCPs from soil to air particularly for the cities with higher ambient temperature under tropical climate (Chakraborty et al. 2015). In Bangalore, high DDT was found in a site which was a dumpsite in the past, whereas in other sites, the DDEs and DDDs were more prevalent (Chakraborty et al. 2015).

3.3.5 Central and Western India

HCH Residues of HCH isomers in soil of Korba, an industrial area in Chattisgarh, showed both technical and lindane usage (Kumar et al. 2014). Soilborne β-HCH dominated the concentrations of HCH isomers in Mumbai and Goa possibly due to the ongoing usage of technical HCH mainly for cotton cultivation practiced in the western and central parts of India (Chakraborty et al. 2015, 2010). Major contributions of atmospheric β-HCH were from Goa and Mumbai (Chakraborty et al. 2010). India was found to be a major source of global β-HCH emissions in 2000 (Li et al. 2003). Atmospheric models have shown that the contaminated air masses originating from the western and central parts of India were transported to Mumbai city via atmospheric transport (Chakraborty et al. 2010).

DDT DDT usage indicated past and ongoing application of technical DDT with higher concentration of soilborne DDE than parent isomers from Korba, attributed to the aerobic degradation of DDT coupled with the long-range atmospheric transport (LRAT) under tropical climatic conditions (Kumar et al. 2014). Soilborne DDT was high in Mumbai and Goa. Higher concentration of DDT in the urban centers particularly p,p'-DDT was found in coastal sites of Mumbai (Chakraborty et al. 2010) due to DDT application for vector control (Pandit et al. 2006) (Fig. 3.2).

3.4 Ecological Risk Assessment

Environmental risk assessment is expressed as the comparison of the estimated environmental concentration with guideline concentrations. Environmental quality guidelines such as soil quality guidelines (SQG) are usually based on toxicokinetics data of pollutants on plants and invertebrates from soil contact of different land uses. The soil quality guidelines for land uses are based on models designed to protect primary, secondary, and tertiary consumers from ingestion of contaminated soil and food. For all land uses, the soil contact values of contaminants are also called threshold effect concentration (TEC), above which adverse effects are not expected or rarely occur on the microorganisms and soils are considered to be clean or less polluted. Environmental guidelines for HCHs and DDTs in soil and sediment are not available in India. Therefore, recommended soil quality guidelines from National Oceanography and Atmospheric Administration (NOAA) USA and Canada government were applied in this study for the evaluation of ecotoxicological effects of HCHs and DDTs. The guideline concentration of 700 $\mu g\ kg^{-1}$ (agricultural and residential/parkland use) and 12,000 $\mu g\ kg^{-1}$ (commercial and industrial land use) for $\sum$DDT was established by Canadian Government. NOAA recommended 99.4–9940 $\mu g\ kg^{-1}$ for HCH in soil for mammals. Excluding the maximum values from the northeastern state, Assam, the concentrations of $\sum$DDT and $\sum$HCH in India were lower than the aforementioned guidelines.

Incremental Lifetime Cancer RiskAssessment in Indian Cities ILCRs for different exposure routes increased in the following order: inhalation < dermal contact < direct ingestion. The range and average of total calculated cancer risk for male child, female child, adult male, and adult female in all the seven Indian cities have been given in Tables 3.3, 3.4, 3.5, and 3.6.

Table 3.3 Male Child Cancer Risk (ILCR) for seven major Indian cities

	Bangalore	Chennai	Agra	New Delhi	Goa	Mumbai	Kolkata
Compound	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg±SD)	Range (Avg±SD)
α-HCH	3×10^{-10}–7×10^{-6} (1×10^{-6})	1×10^{-7}–5×10^{-6} (1×10^{-6})	4×10^{-7}–4×10^{-6} (2×10^{-6})	3×10^{-10}–3×10^{-6} (6×10^{-7})	1×10^{-7}–3×10^{-7} (2×10^{-7})	3×10^{-10}–2×10^{-6} (5×10^{-7})	4×10^{-10}–1×10^{-5} (2×10^{-6})
β-HCH	1×10^{-7}–1×10^{-5} (2×10^{-6})	1×10^{-7}–5×10^{-6} (1×10^{-6})	7×10^{-7}–8×10^{-6} (3×10^{-6})	4×10^{-11}–2×10^{-5} (2×10^{-6})	1×10^{-6}–1×10^{-5} (8×10^{-6})	1×10^{-10}–5×10^{-6} (2×10^{-6})	1×10^{-10}–1×10^{-5} (2×10^{-6})
γ-HCH	7×10^{-8}–4×10^{-5} (6×10^{-6})	1×10^{-7}–4×10^{-6} (1×10^{-6})	8×10^{-7}–3×10^{-6} (2×10^{-6})	2×10^{-11}–1×10^{-5} (1×10^{-6})	8×10^{-7}–1×10^{-6} (1×10^{-6})	1×10^{-10}–1×10^{-6} (7×10^{-7})	2×10^{-7}–8×10^{-6} (1×10^{-6})
δ-HCH	7×10^{-11}–2×10^{-6} (4×10^{-7})	1×10^{-10}–7×10^{-6} (7×10^{-7})	1×10^{-7}–1×10^{-6} (1×10^{-6})	BDL–4×10^{-6} (5×10^{-7})	6×10^{-8}–1×10^{-7} (1×10^{-7})	8×10^{-11}–4×10^{-7} (5×10^{-8})	7×10^{-11}–2×10^{-6} (4×10^{-7})
p,p'-DDE	4×10^{-9}–1×10^{-6} (2×10^{-7})	8×10^{-9}–6×10^{-8} (2×10^{-8})	4×10^{-8}–3×10^{-7} (2×10^{-7})	7×10^{-13}–6×10^{-7} (1×10^{-7})	1×10^{-7}–8×10^{-7} (4×10^{-7})	1×10^{-8}–1×10^{-7} (4×10^{-8})	4×10^{-9}–2×10^{-6} (2×10^{-7})
p,p'-DDD	1×10^{-9}–3×10^{-7} (5×10^{-8})	5×10^{-12}–8×10^{-9} (4×10^{-9})	1×10^{-8}–7×10^{-8} (4×10^{-8})	1×10^{-12}–8×10^{-8} (2×10^{-8})	4×10^{-11}–2×10^{-7} (9×10^{-8})	4×10^{-9}–4×10^{-8} (1×10^{-8})	3×10^{-9}–7×10^{-7} (8×10^{-8})
p,p'-DDT	1×10^{-8}–1×10^{-8} (1×10^{-8})	1×10^{-8}–9×10^{-7} (2×10^{-7})	9×10^{-11}–6×10^{-7} (2×10^{-7})	1×10^{-11}–4×10^{-7} (1×10^{-7})	4×10^{-10}–1×10^{-5} (3×10^{-6})	2×10^{-8}–2×10^{-7} (6×10^{-8})	2×10^{-11}–1×10^{-6} (2×10^{-7})

Table 3.4 Female Child Cancer Risk (ILCR) for seven major Indian cities

	Bangalore	Chennai	Agra	New Delhi	Goa	Mumbai	Kolkata
Compound	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg)
α-HCH	3×10^{-10}–7×10^{-6} (1×10^{-6})	1×10^{-7}–5×10^{-6} (1×10^{-6})	5×10^{-7}–4×10^{-6} (2×10^{-6})	3×10^{-10}–3×10^{-6} (6×10^{-7})	1×10^{-7}–3×10^{-7} (2×10^{-7})	4×10^{-10}–2×10^{-6} (5×10^{-7})	4×10^{-10}–1×10^{-5} (3×10^{-6})
β-HCH	1×10^{-7}–1×10^{-5} (2×10^{-6})	1×10^{-7}–5×10^{-6} (1×10^{-6})	7×10^{-7}–8×10^{-6} (3×10^{-6})	4×10^{-11}–2×10^{-5} (2×10^{-6})	1×10^{-6}–1×10^{-5} (8×10^{-6})	1×10^{-10}–5×10^{-5} (9×10^{-6})	1×10^{-10}–1×10^{-5} (2×10^{-6})
γ-HCH	5×10^{-8}–3×10^{-5} (4×10^{-6})	1×10^{-7}–3×10^{-6} (1×10^{-6})	6×10^{-7}–2×10^{-6} (2×10^{-6})	1×10^{-11}–9×10^{-6} (9×10^{-7})	6×10^{-7}–9×10^{-7} (7×10^{-7})	9×10^{-11}–1×10^{-6} (5×10^{-7})	1×10^{-7}–6×10^{-6} (1×10^{-6})
δ-HCH	7×10^{-11}–2×10^{-6} (4×10^{-7})	1×10^{-10}–8×10^{-6} (7×10^{-7})	2×10^{-7}–1×10^{-6} (1×10^{-6})	BDL–4×10^{-6} (6×10^{-7})	6×10^{-8}–1×10^{-7} (1×10^{-7})	9×10^{-11}–4×10^{-7} (5×10^{-8})	7×10^{-11}–2×10^{-6} (4×10^{-7})
p,p′-DDE	3×10^{-8}–1×10^{-5} (1×10^{-6})	6×10^{-8}–4×10^{-7} (1×10^{-7})	3×10^{-7}–2×10^{-6} (1×10^{-6})	5×10^{-12}–5×10^{-6} (1×10^{-6})	1×10^{-6}–6×10^{-6} (3×10^{-6})	8×10^{-8}–1×10^{-6} (2×10^{-7})	3×10^{-8}–1×10^{-5} (2×10^{-6})
p,p′-DDD	1×10^{-8}–2×10^{-6} (3×10^{-7})	3×10^{-11}–6×10^{-8} (3×10^{-8})	1×10^{-7}–5×10^{-7} (3×10^{-7})	1×10^{-11}–6×10^{-7} (1×10^{-7})	3×10^{-10}–1×10^{-6} (7×10^{-7})	3×10^{-8}–3×10^{-7} (8×10^{-8})	2×10^{-8}–5×10^{-6} (6×10^{-7})
p,p′-DDT	1×10^{-8}–9×10^{-7} (1×10^{-7})	1×10^{-8}–9×10^{-7} (2×10^{-7})	1×10^{-10}–7×10^{-7} (2×10^{-7})	1×10^{-11}–4×10^{-7} (1×10^{-7})	4×10^{-10}–1×10^{-5} (3×10^{-6})	3×10^{-8}–2×10^{-7} (6×10^{-8})	2×10^{-11}–1×10^{-6} (2×10^{-7})

Table 3.5 Male Adult Cancer Risk (ILCR) for seven major Indian cities

	Bangalore	Chennai	Agra	New Delhi	Goa	Mumbai	Kolkata
Compound	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg)
α-HCH	1×10^{-10}–2×10^{-6} (4×10^{-7})	5×10^{-8}–2×10^{-6} (5×10^{-7})	2×10^{-7}–1×10^{-6} (9×10^{-7})	1×10^{-10}–1×10^{-6} (2×10^{-7})	6×10^{-8}–1×10^{-7} (1×10^{-7})	1×10^{-10}–1×10^{-6} (2×10^{-7})	1×10^{-10}–5×10^{-6} (1×10^{-6})
β-HCH	4×10^{-8}–8×10^{-6} (1×10^{-6})	5×10^{-8}–2×10^{-6} (5×10^{-7})	3×10^{-7}–3×10^{-6} (1×10^{-6})	1×10^{-11}–8×10^{-6} (9×10^{-7})	5×10^{-7}–7×10^{-6} (3×10^{-6})	5×10^{-11}–2×10^{-5} (3×10^{-6})	5×10^{-11}–5×10^{-6} (1×10^{-6})
γ-HCH	2×10^{-8}–1×10^{-5} (2×10^{-6})	7×10^{-8}–1×10^{-6} (6×10^{-7})	3×10^{-7}–1×10^{-6} (1×10^{-6})	1×10^{-11}–5×10^{-6} (5×10^{-7})	3×10^{-7}–4×10^{-7} (4×10^{-7})	5×10^{-11}–7×10^{-7} (3×10^{-7})	8×10^{-8}–3×10^{-6} (6×10^{-7})
δ-HCH	3×10^{-11}–1×10^{-6} (1×10^{-7})	4×10^{-11}–3×10^{-6} (3×10^{-7})	8×10^{-8}–7×10^{-7} (4×10^{-7})	BDL–2×10^{-6} (2×10^{-7})	2×10^{-8}–7×10^{-8} (5×10^{-8})	3×10^{-11}–1×10^{-7} (2×10^{-8})	3×10^{-11}–9×10^{-7} (1×10^{-7})
p,p′-DDE	1×10^{-9}–6×10^{-7} (1×10^{-7})	3×10^{-9}–2×10^{-8} (1×10^{-8})	1×10^{-8}–1×10^{-7} (9×10^{-8})	5×10^{-12}–5×10^{-6} (1×10^{-6})	7×10^{-8}–3×10^{-7} (1×10^{-7})	6×10^{-9}–6×10^{-8} (1×10^{-8})	2×10^{-9}–1×10^{-6} (1×10^{-7})
p,p′-DDD	6×10^{-10}–1×10^{-7} (2×10^{-8})	2×10^{-12}–3×10^{-9} (1×10^{-9})	7×10^{-9}–2×10^{-8} (1×10^{-8})	1×10^{-11}–7×10^{-7} (1×10^{-7})	1×10^{-11}–8×10^{-8} (3×10^{-8})	1×10^{-9}–1×10^{-8} (4×10^{-9})	1×10^{-9}–2×10^{-7} (3×10^{-8})
p,p′-DDT	6×10^{-9}–6×10^{-9} (6×10^{-9})	5×10^{-9}–3×10^{-7} (8×10^{-8})	4×10^{-11}–2×10^{-7} (9×10^{-8})	1×10^{-10}–3×10^{-6} (1×10^{-6})	1×10^{-10}–4×10^{-6} (1×10^{-6})	9×10^{-9}–8×10^{-8} (2×10^{-8})	1×10^{-11}–5×10^{-7} (1×10^{-7})

Table 3.6 Female Adult Cancer Risk (ILCR) for seven major Indian cities

	Bangalore	Chennai	Agra	New Delhi	Goa	Mumbai	Kolkata
Compound	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg)	Range (Avg)
α-HCH	1×10^{-10}–3×10^{-6} (4×10^{-7})	6×10^{-8}–2×10^{-6} (6×10^{-7})	2×10^{-7}–2×10^{-6} (1×10^{-6})	1×10^{-10}–1×10^{-6} (3×10^{-7})	1×10^{-8}–3×10^{-7} (2×10^{-7})	1×10^{-10}–1×10^{-6} (2×10^{-7})	2×10^{-10}–6×10^{-6} (1×10^{-6})
β-HCH	5×10^{-8}–9×10^{-6} (1×10^{-6})	5×10^{-8}–2×10^{-6} (5×10^{-7})	3×10^{-7}–4×10^{-6} (1×10^{-6})	2×10^{-11}–9×10^{-6} (1×10^{-6})	6×10^{-7}–8×10^{-6} (3×10^{-6})	6×10^{-11}–2×10^{-5} (4×10^{-6})	6×10^{-9}–6×10^{-4} (1×10^{-5})
γ-HCH	3×10^{-8}–2×10^{-5} (2×10^{-6})	8×10^{-8}–2×10^{-6} (6×10^{-7})	3×10^{-7}–1×10^{-6} (1×10^{-6})	1×10^{-11}–5×10^{-6} (5×10^{-7})	3×10^{-7}–5×10^{-7} (4×10^{-7})	6×10^{-11}–8×10^{-7} (3×10^{-7})	9×10^{-8}–4×10^{-6} (7×10^{-7})
δ-HCH	3×10^{-11}–1×10^{-6} (1×10^{-7})	5×10^{-11}–3×10^{-6} (3×10^{-7})	9×10^{-8}–8×10^{-7} (5×10^{-7})	BDL–2×10^{-6} (2×10^{-7})	2×10^{-8}–8×10^{-8} (6×10^{-8})	3×10^{-11}–2×10^{-7} (2×10^{-8})	3×10^{-11}–1×10^{-6} (2×10^{-7})
p,p'-DDE	1×10^{-9}–7×10^{-7} (1×10^{-7})	3×10^{-9}–3×10^{-8} (1×10^{-8})	2×10^{-8}–1×10^{-7} (1×10^{-7})	3×10^{-13}–3×10^{-7} (7×10^{-8})	8×10^{-8}–3×10^{-7} (1×10^{-7})	5×10^{-9}–7×10^{-8} (1×10^{-8})	2×10^{-9}–1×10^{-6} (1×10^{-7})
p,p'-DDD	7×10^{-10}–1×10^{-7} (2×10^{-8})	2×10^{-12}–3×10^{-9} (1×10^{-9})	8×10^{-9}–3×10^{-8} (2×10^{-8})	8×10^{-13}–4×10^{-8} (1×10^{-8})	1×10^{-11}–9×10^{-8} (4×10^{-8})	1×10^{-9}–1×10^{-8} (5×10^{-8})	1×10^{-9}–3×10^{-7} (3×10^{-8})
p,p'-DDT	7×10^{-9}–4×10^{-7} (6×10^{-8})	6×10^{-9}–4×10^{-7} (9×10^{-8})	4×10^{-11}–3×10^{-7} (1×10^{-7})	8×10^{-12}–2×10^{-7} (7×10^{-8})	2×10^{-10}–5×10^{-6} (1×10^{-6})	1×10^{-8}–9×10^{-8} (2×10^{-8})	1×10^{-11}–6×10^{-7} (1×10^{-7})

ILCR varied only slightly in terms of gender differences. The highest and lowest average ILCR for male varied between 1×10^{-6} and 1×10^{-8} and female between 1×10^{-4} and 3×10^{-8}. Soil ingestion showed predominant risk for all the cities mostly ranging between 10^{-7} and 10^{-4}. Isomers of HCH have been found to be important source for ingestion risk in male and female children in Bangalore, Chennai, Agra, and Delhi (Tables 3.3 and 3.4). ILCR level of γ-HCH showed risk for all the Indian cities, predominantly Goa and Mumbai; this may be due to higher levels of γ-HCH found in their atmosphere (Chakraborty et al. 2010). Excluding Agra, the predominance of β-HCH showed high risk for all the Indian cities. In Mumbai, only one rural site was found at risk. However, β-HCH and γ-HCH showed predominant ILCR in all the cities due to their higher K_{OA} and relatively greater deposition in soil (Xiao et al. 2004) thereby leading to higher risk due to soil ingestion. Interestingly in Bangalore, only one rural site was found to have potential risk due to soilborne HCH. For children typically toddlers, soil ingestion pathway is higher in child leading to higher cancer risk due to exposure to soilborne pesticides from the infancy stage.

ILCR due to dermal contact exceeded 10^{-6} except for few sites in Goa, New Delhi, and Kolkata. Individual HCH isomers have an average ILCR range varying between 10^{-6} and 10^{-7} for all the age groups. ILCR for DDT in soil from seven major Indian cities were closely associated with the HCH residues. Lindane manufacturing units present in Agra, near New Delhi could be a reason for high risk in those areas. ILCRs caused by inhalation of soil particles were less among the three pathways ranging between 10^{-17} and 10^{-10}, indicating that the cancer risk caused by the inhalation of soil particles was negligible.

3.5 Conclusions

Indian climate varies from tropical region in the south to subtropical region in the north and temperate climate in the far north along the Himalayan Range. Such a diverse climatic variation can play a major role in the long-range atmospheric transport of POPs in India. Further monsoonal events can also cause significant impact on the movement of these compounds to the aquatic environment. Residues of HCH and DDT were due to extensive usage and large-scale production of DDT, technical HCH, and lindane for agricultural use in rural areas and vector (largely for mosquito) control in urban areas in last five decades. Soil is acting as sink for these compounds due to their tendency to bind with soil organic matter. Apart from local usage that is completely site specific in northern India, atmospheric transport can play an important role particularly in the movement of HCH isomers away from the source. Lower average ambient temperature during winter due to the subtropical climate in northern India was speculated to be the possible cause for deposition of HCH and DDT. All the cities showed potential risk due to exposure to soilborne HCH residues. We suggest that the soil ingestion pathway may be a potential cause for cancer risk due to chronic exposure from infant stage.

References

Abhilash P, Singh N (2008) Distribution of hexachlorocyclohexane isomers in soil samples from a small scale industrial area of Lucknow, North India, associated with lindane production. Chemosphere 73:1011–1015

Agnihotri N, Kulshrestha G, Gajbhiye V, Mohapatra S, Singh S (1996) Organochlorine insecticide residues in agricultural soils of the Indo-Gangetic plain. Environ Monit Assess 40:279–288

Agoramoorthy G (2008) Can India meet the increasing food demand by 2020? Futures 40:503–506

ATSDR (2005) Toxicology profile for polyaromatic hydrocarbons. ATSDR's toxicological profiles on CD-ROM. CRC Press, Boca Raton

Babu GS, Farooq M, Ray R, Joshi P, Viswanathan P, Hans R (2003) DDT and HCH residues in Basmati rice (*Oryza sativa*) cultivated in Dehradun (India). Water Air Soil Pollut 144:149–157

Bhattacharya B, Sarkar SK, Mukherjee N (2003) Organochlorine pesticide residues in sediments of a tropical mangrove estuary, India: implications for monitoring. Environ Int 29:587–592

CAPE (2005) Factsheet Lindane's Dirty Secret: Indian facilities dump toxic waste 2005 (Community Action for Pesticide Elimination)

Chakraborty P and Zhang G (2011) Organochlorine pesticides (OCPs), polychlorinated biphenyls (PCBs) and polybrominated diphenyl ethers (PBDEs) in the Indian atmosphere. In the chapter, contamination profiles of POPs in India in the book, global contamination trends of persistent organic chemicals. Taylor and Francis Books, Boca Raton

Chakraborty P, Zhang G, Li J, Xu Y, Liu X, Tanabe S et al (2010) Selected organochlorine pesticides in the atmosphere of major Indian cities: levels, regional versus local variations, and sources. Environ Sci Technol 44:8038–8043

Chakraborty P, Zhang G, Li J, Sivakumar A, Jones KC (2015) Occurrence and sources of selected organochlorine pesticides in the soil of seven major Indian cities: assessment of air–soil exchange. Environ Pollut 204:74–80

Devi NL, Qi S, Chakraborty P, Zhang G, Yadav IC (2011) Passive air sampling of organochlorine pesticides in a northeastern state of India, Manipur. J Environ Sci 23:808–815

Devi NL, Chakraborty P, Shihua Q, Zhang G (2013) Selected organochlorine pesticides (OCPs) in surface soils from three major states from the northeastern part of India. Environ Monit Assess 185:6667–6676

Devi NL, Yadav IC, Raha P, Shihua Q, Dan Y (2015) Spatial distribution, source apportionment and ecological risk assessment of residual organochlorine pesticides (OCPs) in the Himalayas. Environ Sci Pollu Res:1–13

DGFT (2008) Department of chemicals and petrochemicals, ministry of chemicals and director general of foreign trade, New Delhi. Government I. Eleventh five-year plan: 2008–2012, Planning Commision of India

Gupta PK (2004) Pesticide exposure—Indian scene. Toxicology 198:83–90

Guzzella L, Roscioli C, Vigano L, Saha M, Sarkar S, Bhattacharya A (2005) Evaluation of the concentration of HCH, DDT, HCB, PCB and PAH in the sediments along the lower stretch of Hugli estuary, West Bengal, northeast India. Environ Int 31:523–534

Harner T, Bidleman TF, Jantunen LMM, Mackay D (2001) Soil-air exchange model of persistent pesticides in the United States cotton belt. Environ Toxicol Chem 20:1612–1621

IOG (2002) Regionally based assessment of persistent toxic substances—Indian Ocean Regional Report global environment facility. United Nations Environmental Programme, Châtelaine

Jayashree R, Vasudevan N (2006) Residues of organochlorine pesticides in agricultural soils of Thiruvallur district, India. J Food Agric Environ 4:313–317

Kumar B, Mishra M, Verma VK, Kumar S, Sharma CS (2012) Distribution of dichlorodiphenyltrichloroethane and hexachlorocyclohexane in urban soils and risk assessment. J Xenobiot 3:1–9

Kumar B, Verma V, Mishra M, Gaur R, Kumar S, Sharma C (2014) DDT and HCH (organochlorine pesticides) in residential soils and health assessment for human populations in Korba, India. Hum Ecol Risk Assess 20:1538–1549

Kumari B, Singh R, Madan V, Kumar R, Kathpal T (1996) DDT and HCH compounds in soils, ponds, and drinking water of Haryana, India. Bull Environ Contam Toxicol 57:787–793

Kumari B, Madan V, Kathpal T (2008) Status of insecticide contamination of soil and water in Haryana, India. Environ Monitor Assess 136:239–244

Kurt-Karakus PB, Bidleman TF, Jones KC (2005) Chiral organochlorine pesticide signatures in global background soils. Environ Sci Technol 39:8671–8677

Li Y-F, Scholtz MT, Van Heyst BJ (2003) Global gridded emission inventories of β-hexachlorocyclohexane. Environ Sci Technol 37:3493–3498

MCF (2007) Performance of chemical & petrochemical industry at a glance Available via http://chemicals.nic.in/stat0107.pdf

Minh N, Minh T, Kajiwara N, Kunisue T, Subramanian A, Iwata H et al (2006) Contamination by persistent organic pollutants in dumping sites of Asian developing countries: implication of emerging pollution sources. Arch Environ Contam Toxicol 50:474–481

Mishra K, Sharma RC, Kumar S (2003) Contamination levels and spatial distribution of organochlorine pesticides in soils from India. Ecotoxicol Environ Saf 76:215–225

Nawab A, Aleem A, Malik A (2003) Determination of organochlorine pesticides in agricultural soil with special reference to γ-HCH degradation by Pseudomonas strains. Bioresour Technol 88:41–46

NIC (2004–2005) Agriculture contingency plan for district Nagaon Available via http://agricoop.nic.in/Admin_Agricoop/Uploaded_File/ASSAM16-NAGAON-26.7.2012.pdf

NIP (2011) National implementation plan, Stockholm convention on persistent organic Pollutants. Government of India, Pretoria

NVBDCP (2010) National vector borne disease control programme. National Rural Health Mission, Bhopal

Ockenden WA, Breivik K, Meijer SN, Steinnes E, Sweetman AJ, Jones KC (2003) The global re-cycling of persistent organic pollutants is strongly retarded by soils. Environ Pollut 121: 75–80

Pandit G, Sahu S, Sharma S, Puranik V (2006) Distribution and fate of persistent organochlorine pesticides in coastal marine environment of Mumbai. Environ Int 32:240–243

PPQS (2013) Pesticide and documentation unit, Directorate of Plant Protection, Quarantine & Storage. Available via http://ppqs.gov.in/IpmPesticides_Cont.htm

Prakash O, Suar M, Raina V, Dogra C, Pal R, Lal R (2004) Residues of hexachlorocyclohexane isomers in soil and water samples from Delhi and adjoining areas. Curr Sci 87:73–77

Rajendran RB, Subramanian A (1999) Chlorinated pesticide residues in surface sediments from the River Kaveri, South India. J Environ Sci Health Part B 34:269–288

Ramesh A, Tanabe S, Murase H, Subramanian A, Tatsukawa R (1991) Distribution and behaviour of persistent organochlorine insecticides in paddy soil and sediments in the tropical environment: a case study in South India. Environ Pollut 74:293–307

Rekha NSN, Prasad R (2006) Pesticide residue in organic and conventional food-risk analysis. J Chem Health Saf 13:12–19

Saha K, Saha T, Banerjee H, Bhattacharyya A, Chowdhury A, Somchoudhury A (2004) Persistence of dicofol residue on tea under North-East Indian climatic conditions. Bull Environ Contam Toxicol 73:347–350

Senthilkumar K, Kannan K, Subramanian A, Tanabe S (2001) Accumulation of organochlorine pesticides and polychlorinated biphenyls in sediments, aquatic organisms, birds, bird eggs and bat collected from south India. Environ Sci Pollut Res 8:35–47

Singh KP, Malik A, Sinha S (2007) Persistent organochlorine pesticide residues in soil and surface water of northern Indo-Gangetic alluvial plains. Environ Monitor Assess 125:147–155

Subramanian A, Tanabe S (2007) Persistent toxic substances in India. Dev Environ Sci 7:433–485

Takeoka H, Ramesh A, Iwata H, Tanabe S, Subramanian A, Mohan D et al (1991) Fate of the insecticide HCH in the tropical coastal area of South India. Marine Pollut Bull 22:290–297

USEPA (1989) Risk assessment guidance for superfund. Human health evaluation manual. volume I. EPA/540/1-89/002

Vijgen J, Yi LF, Forter M, Lal R, Weber R (2006) The legacy of lindane and technical HCH production. Organohalog Compd 68:899–904

Wei D, Kameya T, Urano K (2007) Environmental management of pesticidal POPs in China: past, present and future. Environ Int 33:894–902

Wild SR, Jones KC (1995) Polynuclear aromatic hydrocarbons in the United Kingdom environment: a preliminary source inventory and budget. Environ Pollut 88:91–108

Xiao H, Li N, Wania F (2004) Compilation, evaluation, and selection of physical-chemical property data for α-, β-, and γ-hexachlorocyclohexane. J Chem Eng Data 49:173–185