

ARTICLE

Pesticide Pollution and Child Growth

Gurbinder Singh and S.P. Singh*

ABSTRACT

The modern technology brought in pesticides and herbicides which were used senselessly to obtain more produce resulting in very detrimental health outcomes. Environmental contamination is wide spread and exposure occurs both at workplace and in residential areas. Accordingly, this paper is focused on to understand the effect of pesticide pollution on the growth and development of growing children of the Jat Sikh boys of age group 6–18 years living in the cotton belt of Bathinda in terms of height, weight and Intelligence Quotient (IQ). It was found that children living in the polluted area were shorter in height and lighter in body weight than control group and they also performed lower in the I.Q. score.

Keywords: Pesticide pollution, Growth and development, Jat Sikh boys, Bathinda

Introduction

Modern technology ushered in Green revolution, White revolution and Blue revolution during the latter half of twentieth century in north India. People have reaped rich in the initial years but slowly and steadily the gains from them are fizzling out and ultimately are resulting in nightmare for the people because of their harmful fallouts. These revolutions have engraved a bad destiny on the foreheads of our present generation, which is full of tragedy and sufferings. The strides taken forward to obtain prosperity are also leaving long trails of sorrows behind which could be easily read from the eyes of the victims. The industrialisation and modernisation are heavily polluting our environment and this pollution is emerging as a major threat for the wellbeing of living organisms. Introduction of toxicity in the physical environment causes challenges to survival, reproduction, growth and well being of humans and other organisms. Physiological pathways may be distributed by many materials that are now common place pollutants and these pathways in turn may affect growth and development. Epidemiological studies on the effect of pollutants on human health are very challenging because it is impossible to expose a human being to toxic substances and therefore there is a greater difficulty to recognise the subjects who got exposed to the toxic substances for a long time and compare them with their counterparts who are living without being exposed to the toxic substances. A child's physical growth is very

sensitive to the toxic substances; therefore, it can be used as an important issue of health to ascertain the effect of pollutants.

Environmental contamination is wide spread and exposure occurs both at workplace and in residential areas. Compared with adults, the children are more vulnerable to the effect of exposure to environmental toxicity [Landrigan, 1990]. Children breathe proportionately more air, drink more water and eat more food than adults. The rapidly developing organ systems of children are highly susceptible to the effect of toxins. Children's ability to detoxify and excrete toxic compounds is lower than that of the adults, their immune system is underdeveloped, their play activities involve close contact with the environment and their hand to mouth activity contributes to their greater vulnerability. The foetus is especially at greater risk since many contaminations pass through the placenta during the sensitive period of development. Finally, because children have more years of life than adults they are more susceptible to chronic multistage diseases that may be triggered by early exposure. The various types of pollutants impart detrimental effect on child health, growth and development. According to some studies the growth is highly eco-sensitive [Johnston, 1993]. The prenatal exposure to toxicants results in lower birth weight and shorter gestation. The decrease in birth weight ranges from 220 to 500 g [Baibergenova *et al.*, 2003; Karamus and Zhu, 2004; Rylander *et al.*, 2000]. Exposure to air pollution and

Mr. Gurbinder Singh, Science Teacher, Govt. High School Gurthari, Bathinda-151001, Punjab, and Dr. S.P. Singh, Professor, Department of Human Biology, Punjabi University, Patiala-147002. Email: spsingh@humanbiologyjournal.com

*Corresponding author

pesticides result in smaller head circumference and smaller skull in the newborn [Boback *et al.*, 2001; Berkowitz *et al.*, 2004]. The children of polluted areas have been found to be affected in neuro-developmental attributes where general intelligence and I.Q. are reduced along with retardation in cognitive functioning [Eskenazi and Castornia 1999; Litverk *et al.*, 1999; Weiss 2004; Sarkar and Mohiuddin, 2002; Ayotte *et al.*, 2003; Garcia *et al.*, 2003; Wakefield, 2003]. A study by Rosenlund *et al.* [2009] showed that there was a strong association between estimated NO₂ exposure per 10 µg/m³ and lung function, especially expiratory flows, in linear regression models adjusted for age, gender, height and weight. Heart rate, its variability, and blood pressure have been shown to be susceptible to the effects of air pollution [Magari *et al.*, 2002]. Reduction in forced expiratory volume was reported in the children, either born to the mothers exposed to air pollution or in the children exposed to air pollution in early childhood [Turnovska *et al.* 2009]. Pollutants like Polycyclic Aromatic Hydrocarbons (PAH), Environmental Tobacco Smoke (ETS) and ambient carbon monoxide along with particulate matter exerts many detrimental effects, which result in low birth weights of infants in many regions of the world [Misra and Nguyen, 1999; Han *et al.*, 2000; Yang, 2000; Boback *et al.*, 2001]. Exposure to arsenic (As) in drinking water has been associated with a decline in intellectual function in children. This association has been established recently on the basis of a cross-sectional study of 201 10-year-old children in Bangladesh [Wasserman *et al.*, 2004]. Mexican study by Calderon *et al.* [2001] and a Taiwanese study by Tsai [2003] have established tentative adverse association between arsenic exposure and children's intellectual function.

The effect of synthetic pesticides on the growth and development is being documented in various parts of the world. Berkowitz *et al.* [2004] studied the combined effect of in-utero pesticide exposure and, maternal paraoxonase activity on birth weight, length, head circumference and gestation age among 404 births between May 1998 and May 2002. The results showed that maternal level of chlorpyrifos above the defined limits coupled with low maternal paraoxonase activity were associated with a significant reduction in head circumference and head size. Pesticides exposure is responsible for birth defects, genetic disorders or mutations, reproductive system deformities, neurological damage, mild cognitive dysfunction and other health effects [Jeyaratnam, 1990; Hemminki *et al.*, 1980; Faustman *et al.*, 2000; Bosma *et al.*, 2000]. Early exposure

to pesticides was found to hinder pre-schoolers hand eye coordination, recall and ability to draw pictures [Faustman *et al.*, 2000]. Woman exposed to pesticides through agricultural or floricultural work have been documented to have significantly higher risk of children born with musculoskeletal defects, growth retardation and learning behaviour disorders [Faustman *et al.*, 2000; Hemminki *et al.*, 1980].

Besides the toxic pollutants like synthetic pollutants, the heavy metals also possess great detrimental effects on the growth and development of children along with other health hazards. Significant impairments of height, body weight, brain and intelligence were reported in the children poisoned by milk powder containing arsenic in Japan when compared with that of control group of the same age of 16 years after the poisoning event [Wang and Haung, 1994]. Although selenium is thought to be essential for various immune responses, the excess supplementations have adverse effect on certain immunological functions [Hitoshi, 2008].

Neonates with cord serum having the traces of Polychlorinated Biphenyls (PCB's) had decreased birth weight of approximately 160–190 g and smaller head circumferences compared to less exposed children [Fein *et al.*, 1984]. A study in the Netherlands found that lower birth weight and delayed growth rate were correlated with higher PCB levels measured in cord plasma and mother's blood [Patandin *et al.*, 1998]. A recent study of Great Lakes fish eaters on the effect of toxicants on birth weight found a significant decrease (500 g) in birth weight of infants born to mothers with higher level of PCB in blood [Karmus and Zhu, 2004]. The PCB's exposure delays the timing of menarche in girls [Denham *et al.*, 2005]. Away from PCB's, pesticides and heavy metals like lead, mercury and selenium also decrease the rate of growth in children. Noise stress also acts as a potent inhibitor of normal growth rate in children. Birth weight has been shown in several studies to be reduced in relation to noise exposure from airports and other sources [Knipschild *et al.*, 1981]. Maternal exposure to methyl-mercury has been associated with decrements in cognitive function in the child [Budtz *et al.*, 2007; Jedrychowski *et al.*, 2006; Oken *et al.*, 2005]. In a study of women in Tagum, the Philippines, a fish eating community using metallic mercury in gold mining and processing, cord blood mercury was associated with developmental and language deficits at 2 years of age [Ramirez *et al.*, 2003].

The Punjab Scenario

Malwa region of Punjab, which is also the area under study in this paper, is highly contaminated with excess and indiscriminate use of pesticides and poor quality of underground drinking water. Therefore, in order to study the effect of pesticide pollution on the growth and development of growing children, a study was designed in which height, weight and intelligence quotient (IQ) of the Jat Sikh boys of age group 6–18 years living in the cotton belt of Bathinda were measured. It is important to mention that the cotton crop is generally sprayed with pesticides more than 30 times by the farmers of this region. In order to find out the effect of pesticide pollution, a control area was selected, which was taken from around the canal irrigation areas having plenty of water and mainly farming in paddy, which needs very little pesticide spray. Thus the two areas presented a contrast picture of very high pesticide polluted area vis-à-vis a very less pesticide polluted area. This control area is having good quality of drinking water, same socio-economic status, geographical conditions and the subjects belong to the same endogenous group.

The Punjab state is the second highest consumer of pesticides in India (6972 MT) after Uttar Pradesh. Leading pesticides which are used include monocrotophos, acephate, endosulphan and chloropyrifos [Pesticide inf., 2002]. About 54% of total pesticides used in Indian agriculture are consumed on cotton alone though it accounts for only 5% of the total cultivated area [Puri *et al.*, 1999]. Cotton contributes about 55% of total share in the consumption of pesticide while it is only 17% in case of rice. When the area under these two crops is compared, the share of cropped area under rice is 24% as compared to 5% for cotton. Thus it results in a manifold concentration of pesticide in area under cotton crops [Agnihotri, 1999]. In drinking water, pesticide like heptachlor (0.006 ppm) and malathion (0.269 ppm) were found much above the permissible limits of 0.00003 ppm for heptachlor and 0.0005 ppm for malathion in the affected area under study. Besides pesticides, the drinking water also contained heavy metals like selenium, arsenic, and mercury above the permissible limit. Traces of pesticides like heptachlor, endosulfan, aldrin, ethion and chloropyrifos were detected in the vegetables and fruits of the affected area (PGMIER Report 2006). Punjab Pollution Control Board Punjab reported traces of Dichloro Diphenyl Trichloroethane (DDT) and

Benzene Hexachloride (BHC) from the canal water of the affected area. The abnormal number of cancer cases (107) and deaths due to cancer (489) in last 10 years (PGIMER, Chandigarh Report 2006), which were abnormal by any standards, puts a stamp on the extent of pollution in the environment in which people of affected areas were living. Pollution imparts a negative effect on the health and wellbeing of every human but the thinner skin of women makes them more susceptible to absorption of toxic substances than men under similar levels of exposure. As persistent toxic substances like pesticides are stored in the fatty tissue, women having higher percentage of body fat than men store more toxic substances in their bodies. Toxic substances are also eliminated from the body through urine, a women's renal function is slightly less efficient than man's especially during pregnancy thus making her much more vulnerable. Exposure to the toxic pollutants possess a variety of reproductive health impacts in women and they have increased spontaneous abortions, miscarriages, delayed pregnancies, infertility, children born with birth defects, still births and pregnancy problems. Women of affected area under study are facing the above said problems at a higher rate than the control area. A house-to-house data collection was carried out regarding the number of pregnancy-related problems faced by the women in the past 7 years, which are represented in the Table 1.

The children who live in the cotton belt of Malwa region of Punjab in India with high level of pollution have been described as affected and those living in the non-cotton belt area of Malwa region with very low-level of pollution are described as the control.

Table 1: Pregnancy-related problems in polluted and control areas

Abnormalities	Affected area total 191		Control area total 185	
	n	%	n	%
Still births	6	3.14	2	1.08
Birth defects	7	3.66	2	1.08
Spontaneous abortions	9	4.71	3	1.62
Premature births	5	2.62	2	1.08
Pregnancy problems	15	7.85	8	4.32

Results

1. Pollution and Physical Growth of Children

Height: In the boys of the affected group, height increases from 110.19 cm at 6 years to 169.70 cm at the age of 18 years, whereas in control group it increases from 116.15 cm to 178.47 cm, respectively, in the age groups of 6–18 years as shown in the Table 2 and illustrated in Figure 1.

There was a gain of 59.51cm in height in the case of the affected group and this gain was 62.32 cm in the control group boys starting from 6 years to 18 years. The statistically significant difference between the two groups was observed in all the age groups. Interestingly, a backlog of about 6 cm in height of affected children was noticed till the age of 6 years. It indicated that a major portion of the delay occurred during the first few years of life. In order to

Table 2: Comparison of height of boys (cm) of affected area and control area

Age (yrs)	Affected area (Jajjal)			Control area			t-score
	Mean	S.D	SEM	Mean	S.D	SEM	
5.5-6.499	110.19	4.18	0.76	116.15	3.20	0.58	6.21***
6.5-7.499	115.88	4.48	0.82	122.60	3.10	0.57	6.72***
7.5-8.499	119.65	4.53	0.83	125.60	3.45	0.63	5.67***
8.5-9.499	127.10	6.54	1.40	132.20	4.68	0.85	3.11**
9.5-10.499	131.57	5.96	1.10	137.61	5.34	0.97	4.11***
10.5-11.499	139.17	5.94	1.08	145.75	6.00	1.09	4.27***
11.5-12.499	143.01	7.70	1.40	151.42	5.54	1.05	4.80***
12.5-13.499	151.20	8.28	1.50	156.20	6.06	1.17	2.63*
13.5-14.499	153.10	9.50	1.70	162.65	6.70	1.22	4.57***
14.5-15.499	161.43	8.70	1.60	167.30	4.20	0.77	3.29**
15.5-16.499	164.10	9.20	1.70	173.55	5.25	0.94	4.87***
16.5-17.499	167.75	4.50	0.80	176.62	3.10	0.57	9.05***
17.5-18.499	169.70	3.90	0.71	178.47	3.34	0.62	9.33***

***P<0.001; **P<0.001; *P<0.05

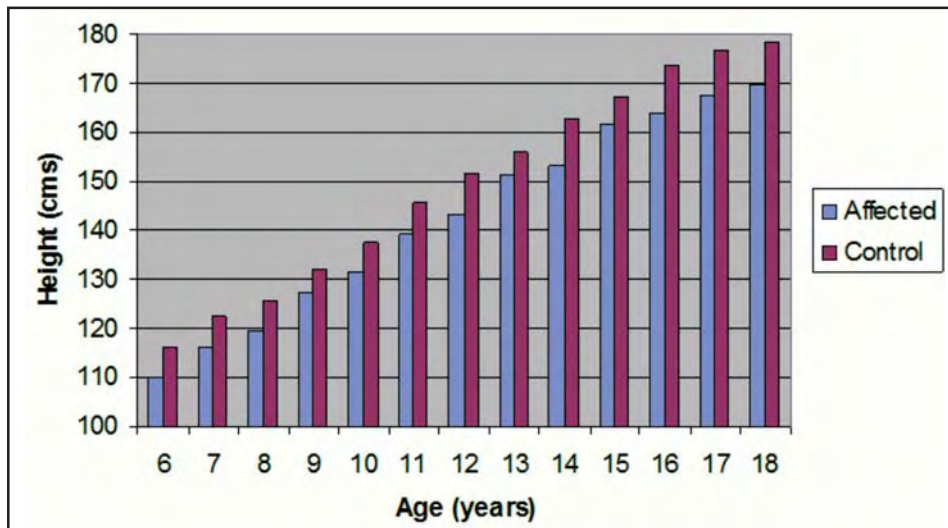


Figure 1: Comparison of height of affected area boys vs control area boys

obtain an average figure of difference per year between the two groups the sum of difference at all age groups were divided by the number of age groups. It was found that on an average the affected group was lagging behind their counterparts by 7.10 cm at each age group. The backlog in the affected group of children from the control group ones has been expressed in the percentage of height,

which was obtained from the difference between the control and the affected group divided by the height of the control group and multiplied by 100. The affected group of boys had a backlog of 4.77% in height as compared to their control counterparts.

Weight:- The affected group of boys had gained weight from 15.95 kg at 6 years to 50.53 kg at 18 years with a net

Table 3: Comparison of weight of boys (kg) of affected area and control area

Age (yrs)	Affected area (Jajjal)			Control area			t-score
	Mean	S.D	SEM	Mean	S.D	SEM	
5.5-6.499	15.95	1.58	0.29	17.58	1.53	0.28	4.10***
6.5-7.499	17.38	2.60	0.48	19.17	1.48	0.27	3.25**
7.5-8.499	19.47	1.80	0.32	21.20	3.50	0.64	2.40*
8.5-9.499	21.80	3.10	0.56	23.10	2.20	0.41	1.88
9.5-10.499	23.75	3.60	0.65	25.00	1.81	0.33	1.71
10.5-11.499	27.40	5.90	1.10	28.80	3.00	0.60	1.12
11.5-12.499	29.45	5.30	0.96	32.20	2.80	0.54	2.50*
12.5-13.499	34.75	8.50	1.50	37.50	5.15	0.96	1.54
13.5-14.499	35.60	6.05	1.10	41.25	6.10	1.10	3.50**
14.5-15.499	42.95	8.08	1.47	44.00	6.13	1.12	0.57
15.5-16.499	44.03	7.90	1.44	51.30	7.07	1.30	3.75***
16.5-17.499	47.40	4.10	0.74	52.80	2.60	0.48	6.10***
17.5-18.499	50.53	8.80	1.61	53.43	2.40	0.44	1.75

***P<0.001; **P<0.001; *P<0.05

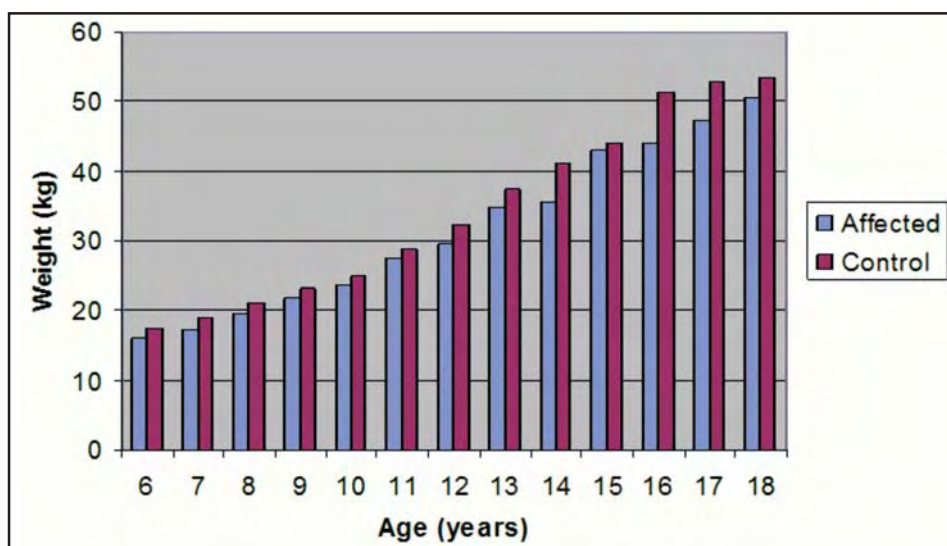


Figure 2: Comparison of weight of boys (kg) of affected area and control area

gain of 34.58 kg; whereas in the case of control group weight had increased from 17.58 kg at 6 years to 53.43 kg at 18 years of age and the net gain is 35.85 kg as shown in Table 3 and illustrated in Figure 2.

2. Pollution and Intelligence Quotient (IQ) of Children

The intelligence score of children was measured with

Ravan's coloured and standard progressive matrices [Ravan *et al.*, 2000]. The score increases with age in boys of both groups as shown in Table 4 and illustrated in Figure 3.

The boys of the affected group had lesser intelligence quotient score than the control and the difference is statistically significant in 12 age groups out of the total 13. The boys of age 6 years of control group perform better

Table 3: Comparison of IQ score of boys of affected area and control area

Age (yrs)	Affected area (Jajjal)			Control area			t-score
	Mean	S.D	SEM	Mean	S.D	SEM	
5.5-6.499	10.97	3.84	0.70	17.73	4.88	0.89	5.98***
6.5-7.499	13.77	3.45	0.63	19.73	4.75	0.87	5.50***
7.5-8.499	12.83	4.99	0.91	20.70	4.39	0.80	6.50***
8.5-9.499	14.00	5.60	1.03	21.57	5.74	1.05	5.10***
9.5-10.499	15.37	5.01	0.91	21.16	5.75	1.05	4.20***
10.5-11.499	17.10	6.48	1.18	28.97	8.51	1.55	6.10***
11.5-12.499	22.50	9.98	1.80	29.39	11.30	2.10	2.47*
12.5-13.499	22.80	12.19	2.23	32.27	11.10	2.06	3.12**
13.5-14.499	32.30	12.56	2.29	34.50	10.85	1.98	0.73
14.5-15.499	31.30	12.20	2.23	38.07	9.18	1.67	2.40*
15.5-16.499	30.43	10.96	2.00	40.65	10.25	1.84	3.76***
16.5-17.499	28.76	10.37	1.89	40.07	10.50	1.90	4.20***
17.5-18.499	28.17	11.45	2.09	40.69	9.69	1.78	4.50***

***P<0.001; **P<0.001; *P<0.05

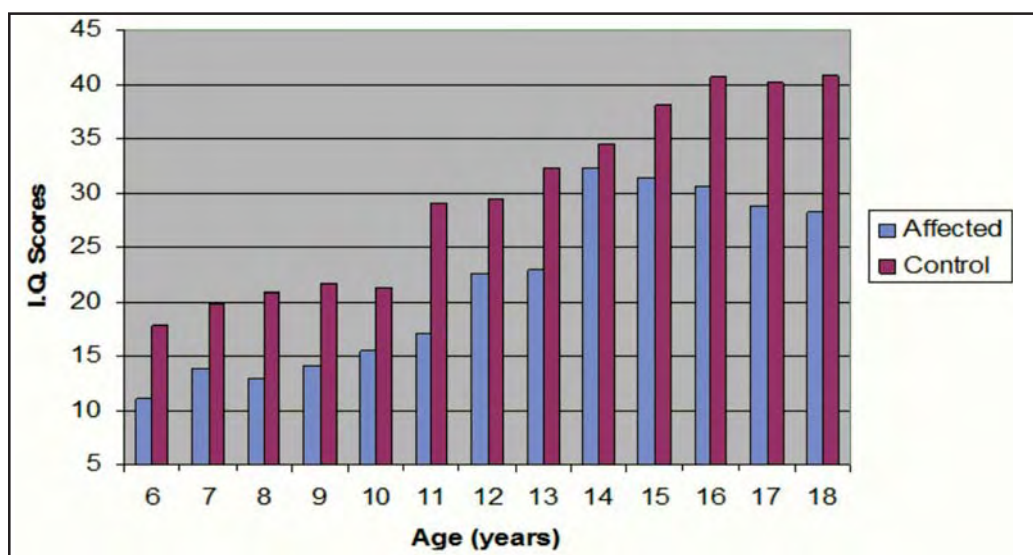


Figure 3: Comparison of IQ score of boys of affected area and control area

than the boys of 11 years of affected group. The affected group had a backlog of 27.3% than their counter parts.

Summing up the findings it was found the children living in the polluted area were shorter in height and lighter in body weight than the control group and they also performed lower in the I.Q. score. It may be concluded that children exposed to the pollutants suffer from growth retardation and perform lower on IQ scores.

References

- Agnihotri, N.P. 1999. Pesticide Safety Evaluation and Monitoring. *All India Research project on Pesticide Residue*. Indian Agricultural Research Institute, New Delhi. pp. 22-23.
- Ayotte, P., Muckle, G., Jacobson, J.L., Jacobson, S.W. and Dewailly, E. 2003. Assessment of pre and postnatal exposure to polychlorinated biphenyls: Lesson from the cohort study. *Environmental Health Perspective*, 111: 1253-1258.
- Baibergenova, A., Kudiyakov, R., Zdeb, M. and Carpenter, D.O. 2003. Low birth weight and residential proximity to PCB contamination waste sites. *Environmental Health Perspective*, 111: 1352-1357.
- Berkowitz, G.S., Wetmur, J.G., Deych, E.B., Obel, J., Lapinski, R.H., Godbold, J.H., Holzman, I.R. and Wolf, M.S. 2004. *In vitro* pesticide exposure maternal paraoxnase activity and head circumferences. *Environmental Health Perspective*, 112: 358-391.
- Boback, M., Richard, M. and Wodsworth, M. 2001. Air pollution and birth weight in Britain in 1946. *Epidemiology*, 16: 358-359.
- Bosma, H. *et al.* 2000. Long term pesticide exposure linked to mild cognitive dysfunction. *The Lancet*, 356(9233): 912-913.
- Budtz, J.E., Keiding, N., Grandjean, P. and Weihe, E. 2007. Confounder selection in environmental epidemiology: assessment of health effects of pre-natal mercury exposure. *Epidemiology*, 17: 27-35.
- Calderon, J., Navarro, M.E., Jimenez, C.M.E., Santos, D., Golden, A. and Rodriguez, L.I. 2001. Exposure to arsenic and lead and neuropsychological development in Mexican Children. *Environmental Research*, 85(2): 69-76.
- Denham, M., Schell, L.M., Deane, G., Gallo, M.V., Ravenscroft, J. and DeCarpio, A.T. 2005. The Relationship of lead, mercury, mirex, dichlorodiphenyldichlorodiethyle, hexachlorobenzene, and polychlorinated biphenyls to timing of menarche among akwesasne Mohawk girls. *Pediatrics*, 115: 127-134.
- Eskenazi, B. and Castornia, R. 1999. Association of prenatal and postnatal child environmental tobacco smoke exposure and neuro-developmental and behavioral problem in children *Environmental. Health Perspective*, 107: 991-999.
- Faustman, M., Silbernagel, S.M. and Ponce, R.A. 2000. Mechanisms Underlying Children's Susceptibility to Environmental Toxicants. *Environmental Health Perspectives*, 108(1): 13-21.
- Fein, G.G., Jacobson, J.L., Jacobson, S.W., Schwartz, P.M. and Dowler, J.K. 1984. Prenatal exposure to polychlorinated biphenyls: effect on birth size and gestational age. *Journal of Pediatrics*, 105: 315-320.
- Garcia, S.J., Seidler, F.J. and Sloykhain, T.S. 2003. Developmental neurotoxicity elicited by pre or postnatal chlorpyrifox exposure: Effect of neurospecific protein indicator changing vulnerabilities. *Environmental Health Perspective*, 111: 297-303.
- Han, S., Pfizenmaier, D.H., Garcia, E., Equez, M.L., Kemp, F.W. and Bogden, J.D. 2000. Effect of lead exposure before pregnancy and dietary calcium during on foetal development and lead accumulation. *Environmental Health Perspective*, 108: 527-531.
- Hemminki, K., Saloniemi, I., Lvoma, K., Salonen, J. and Partunen, T. 1980. Congenital Malformation by the Parental Occupation in Finland. *Int. Arch. Occup. Environ. Health*, 46: 93-98.
- Hitoshi, U. 2008. Affects of selenium status and supplementary seleno-chemical sources on mouse T-cell mitogenesis. *Journal of Trace Elements in Medicine and Biology*, 22(1): 9-16.
- Jedrychowski, W., Jankowski, J., Flak, E., Skarpura, A., Mroz, E. and Sochacka, T.E. 2006. Effect of pre natal exposure to mercury on cognitive and psychomotor function in one -year old infants: epidemiological cohort study in Poland. *Ann. Epidemiology*, 16: 439-447.
- Jeyaratnam. 1990. Acute Pesticide Poisoning: A major global health problem. *World Health Statistics Quarterly*: 43.
- Johnston, F.E. 1993. The enduring effects of environmental deprivation on growth and development. *J. Hum. Ecol.*, 4:117-134.
- Karamus, W. and Zhu, X. 2004. Maternal concentration of polychlorinated biphenyls and dichlorodiphenyl, dichloroethylene and birth weight in Michigan fish eaters: A cohort study. *Environmental Health*, 3:1-5.
- Knipschild, P., Meijer, H. and Salle, H. 1981. Aircraft noise and birth weight. *International Archives of Occupational and Environmental Health*, 48: 131-136.
- Landrigan, P.J. 1990. Health effects of environmental toxins in deficient housing. *Bull NY Acad. Med.*, 66: 491-499.
- Litverk, P.F., Wasserman, G., Kline, J.K. and Graziano, J. 1999. The Yugoslavia study of environmental lead exposure. *Environmental Health Perspective*, 107: 9-14.
- Magari, S.R., Schwartz, J. and Williams, P.L. 2002. The association of particulate air concentration with heart

- rate variability. *Environmental Health Perspective*, 110:875-880.
- Misra, D.P. and Nguyen, R.H.N. 1999. Environmental tobacco smoke and low birth weight: A hazard in the workplace. *Environmental Health Perspective*, 108: 803-813.
- Oken, E., Wright, R.O., Klienman, K.P., Bellinber, D., Amarasiriwardena, C.J. and Hu, H. 2005. Maternal fish consumption, hair mercury, and infant cognition in a U. S. cohort. *Environmental Health Perspective*, 113: 1376-1380.
- Patandin, S., Koopman, E.C., De-Ridder M.A.J., Weisglaskupers, N. and Sauer, P.J.J. 1998. Effect of environmental exposure to polychlorinated biphenyls and dioxins on birth size and growth in Dutch children. *Pediatrics Research*, 44: 447-450.
- Pesticide Information. 2002 Pesticide in India-Environment and Health Source Book. New Delhi: *Toxilink*. 24(4):9-21.
- Post Graduate Institute of Medical Education and Research, Chandigarh. 2006. An epidemiological study of cancer cases reported from villages of Talwandi Sabo Block, District Bathinda, *Punjab Report*.
- Puri, S.N., Murthy, K.S. and Sharma, O.P. 1999. IPM for sustainable crop production. Management in Sundaran, V. *et al.*, *Handbook of cotton in India*, ISCI, Bombay.
- Ramirez, G.B., Pagulayan, O., Akagi, H., Francisco, R.A., Lee, L.V. and Berroya, A. 2003. Tagum Study II: follow up study at two years of age after pre-natal exposure to mercury. *Pediatrics*, 111: 289-295.
- Ravan, J., Ravan, C.J. and Court, H.J. 2000. Standard Progressive, Matrices Including the Parallel and plus versions, Set A, B, C, D and E. *Oxford Psychologists Press*, Oxford.
- Rosenlund, M., Forastiere, F., Porta, D., Sario, M., Badaloni, C. and Perucci, C.A. 2009. Traffic-related air pollution in relation to respiratory symptoms, allergic sensitization and lung function in schoolchildren. *Thorax*, 64: 573-580.
- Rylander, L., Stromberg, U. and Hagmar, L. 2000. Lowered birth weight among infants born to women among infants born to women with intake of fish contaminated with persistent organochlorine compounds. *Chemosphere*, 40: 1255-1262.
- Sarkar, P.C. and Mohiuddin, M.D. 2002. Arsenic poisoning and its impact on the socioeconomic and cultural life of people of Bangladesh. *South Asian Anthropologist*, 2: 97-102.
- Tsai, S.Y. 2003. The Effects of chronic arsenic exposure from drinking water on the neurobehavioral development in adolescence. *Neuro. Toxicology*, 24(4-5):747-753.
- Turnovska, T.H. and Marinov, B.I. 2009. The influence of air pollution during intrauterine development and early childhood on respiratory functions at later age. *International Journal of Hygiene and Environmental Health*, 212(5):519-532.
- Wakefield, J. 2003. Size in the city: New York pollution may lower birth weight. *Environmental Health Perspectives*, 108: 373-400.
- Wang, L. and Huang, J. 1994. Chronic arsenic from drinking water in some areas of Xinjiang China. In: Arsenic in the Environment. Part II: *Human Health and Ecosystem Effects*. New York: John Wiley & Sons, 159-172.
- Wasserman, G.A., Liu, X., Parvej, F., Ahsan, H., Factor L.P, Van, G.A., *et al.* 2004. Water arsenic exposure and children's intellectual function in Araihasar, Bangladesh. *Environmental Health Perspectives*, 108: 765-768.
- Weiss, B. 2004. Pesticides. *Pediatrics*, 113(4): 1030-1036.
- Yang, C.Y. 2000. Association between chlorination of drinking water and adverse pregnancy outcome in Taiwan. *Environmental Health Perspectives*, 108: 765-768.