Eur J Pediatr (2011) 170:923–929
DOI 10.1007/s00431-010-1379-0

ORIGINAL PAPER

Attention-deficit hyperactivity disorder in children chronically exposed to high level of vehicular pollution

Shabana Siddique · Madhuchanda Banerjee · Manas Ranjan Ray · Twisha Lahiri

Received: 2 November 2010 / Accepted: 7 December 2010 / Published online: 30 December 2010

Abstract The purpose of this study is to explore whether sustained exposure to vehicular air pollution affects the behavior and activities of children. The prevalence of attention-deficit hyperactivity disorder (ADHD) was assessed in two childhood populations. In a cross-sectional study 969 school-going children (9–17 years) and 850 age- and sex-matched children from rural areas were assessed, following the criteria of Diagnostic and Statistical Manual of conduct disorders (DSM-IV) of American Pediatric Association. Data of ambient particulate matter with a diameter of less than 10 μm (PM_{10}) were obtained from Central Pollution Control Board and aerosol monitor. ADHD was found in 11.0% of urban children in contrast to 2.7% of the control group ($p<0.001$). Major risk factors were male gender, lower socioeconomic status, 12–14 year age group, and PM_{10} level in breathing air. ADHD was more prevalent among boys both in urban and rural areas. It was prevalent among 18.0% of the boys enrolled in Delhi against 4.0% of the girls, giving a male/female ratio of 4.5:1. Inattentive type of ADHD was predominant followed by hyperactive–impulsive type and combined type of ADHD. Controlling potential confounder, ambient PM_{10} level was positively correlated with ADHD (OR= 2.07; 95% CI, 1.08–3.99). *Conclusion*: The results of this study point to a possible association between air pollution and behavioral problems in children. Though gender, socioeconomic status, and age play a very important factor in ADHD prevalence, the association is highest and strongest between particulate pollution and prevalence of ADHD.

Keywords ADHD · Air pollution · Children · Urban · PM_{10}

Introduction

Vehicular pollution is often associated with respiratory and cardiovascular diseases in children, but few studies have explored its neurodegenerative effects in a developing country like India. Attention-deficit/hyperactivity disorder (ADHD) is one of the most common behavioral changes diagnosed in children and adolescents. It is characterized by inattentiveness, overactivity, and impulsiveness. Children with ADHD are forgetful, restless, prone to fail, unable to follow tasks, unpredictable, and moody. The problem starts in early childhood and persists in adulthood in the majority of cases [5].The disorder is characterized by marked inattention, hyperactivity, and impulsiveness. In most cases, symptoms can be treated by catecholamine-releasing drugs [33]. Children with ADHD are at a greater risk of substance abuse, conduct, and mood disorders. Studies in laboratory animals have shown that air pollution may cause brain damage [10]. Investigations in humans have illustrated brain lesions and impairment of olfactory function among

S. Siddique (✉)
Health Canada, Environmental Health Center (EHC, HECS),
Room # B-36, 50, Columbine Driveway,
Tunneys Pasture, AL: 0800C,
Ottawa K1A 0K9 ON, Canada
e-mail: shim23@gmail.com

M. Banerjee
Indian Institute of Technology, (IIT),
Guwahati, India

M. R. Ray
Chittaranjan National Cancer Institute,
Kolkata, India

T. Lahiri
Nature, Environment and Wildlife Society (NEWS),
Kolkata, India

residents of highly polluted Mexico City [15]. These reports indicate that chronic exposure to high level of urban air pollution may cause changes in the brain activity that may manifest themselves in behavioral alterations. Long-term concentration of black carbon particles from mobile sources is also associated with decreases in cognitive test scores; decreases in cognitive functioning are seen in verbal and nonverbal intelligence constructs as well as memory constructs [28].

In a situation-by-situation analysis, hyperactive children were most consistently and significantly more active than the controls during structured school activities [24]. Interestingly, a study conducted in Ontario has revealed that children with ADHD also had mathematical learning and reading disorders [11].

Certain factors like lower breathing zone, more time spent outdoors, immature immunity, and developing organs make a child more susceptible to the ill effects of air pollutants, but it's not only the physical health which is at stake, there is evidence which shows that air pollutants also affect the mental health of a child. In India, very little work has been done so far on mental health of a child in relation to air pollution.

Air pollution is viewed as a serious problem in Delhi. The city has the dubious distinction of one of the most polluted cities in the world. In 2005, the annual average concentration of respirable suspended particulate matter, i.e., PM_{10}, was 2.7 times higher than the Indian standards for residential areas. The major sources of the city's air pollution are road traffic (72%), industrial emissions (20%), and emissions from household activities (8%) [22]. Motor vehicles are responsible for a substantial part of Delhi's air pollution. The motor vehicle fleet of Delhi presently stands at 4.2 million, which is more than Mumbai, Kolkata, and Chennai put together [2].

The goal of this study was to explore whether sustained exposure to high level of urban air pollution has any impact on the behavior and activities of the children.

Materials and methods

Participants

A total number of 969 school children of Delhi, 490 boys and 479 girls, from different areas of National Capital Region of Delhi were enrolled for ADHD screening. The children were selected randomly from schools located within 3 km radius of the Central Pollution Control Board monitoring stations. The children were selected from houses which were in close proximity to the school. Care was taken so that the children represented every section of the society with comparable representations from low, medium, and high socioeconomic status. The children were aged between 9 and 17 years (median age, 14 years). Another group of 850 age-matched children, 442 boys and 408 girls, from rural areas of Uttaranchal and West Bengal where ambient air pollution level was much less due to lesser number of automobiles and air-polluting industries, was enrolled as control. The protocol of this study conducted during 2003–2005 was approved by the Institutional Ethics Committee of Chittaranjan National Cancer Institute.

Inclusion and exclusion criteria

Apparently healthy children were included in this study. Children staying away from home such as residing in school hostel and those with history of neurological problems like epilepsy, autism, mental retardation, sensory deficits, and severe learning disabilities (more than 2 years in the same class) were excluded from our study. Mothers of children recruited for the study were asked about their smoking status and the smoking habits of the members of the household. Children having a family member who smokes (to exclude the influence of Environmental Tobacco Smoke (ETS)) and coming from households with alcohol abuse were also excluded from our study. Only children from household using liquid petroleum gas (LPG) as the cooking fuel were selected from the rural areas. Care was taken to select the children who resided in the current address for the past 6 years.

Air pollution level

Air quality data with respect to particulate matter with a diameter of less than 10 μm (PM_{10}), oxides of sulfur (SOx) and nitrogen in ambient air of study areas were collected from Central and State Pollution Control Boards from their fixed site monitoring stations. In addition, the level of PM_{10} in indoor air of the households of participating children was measured by portable, battery-operated laser photometer (DustTrak™ Aerosol monitor, model 8520, TSI Inc., MN, USA). Monitoring was done in 60 randomly selected urban and 60 rural households, 20 in each season—winter, summer, and monsoon. The monitor was placed in the living room 4 ft above the floor. Monitoring was done for three consecutive days for 8 h (8 A.M. to 6 P.M.) in home (living room) and school (classroom).

Diagnosis of ADHD

ADHD was screened following the criteria prescribed in the Diagnostic and Statistical Manual of conduct disorders (DSM-IV) of American Pediatric Association [1]. DSM-IV manual emphasizes that presence of six or more signs and symptoms of either category #1 *(inattention)* or of category #2 (*hyperactivity–impulsivity)* confirms the diagnosis of

ADHD. Children showing symptoms of both category #1 and category #2 are diagnosed with *combined type* (category #3) or mixed type of ADHD.

Validity and reliability of questionnaire

In evaluating ADHD symptomatology, it is important to obtain independent reports about the child's behavior at school from the teacher and about the child's behavior at home from the parents [13]. Accordingly, we have obtained reports from the parent as well as the class teacher of the school.

Determination of socioeconomic status

Socioeconomic status (SES) of the child's family was ascertained following the procedure of [30].

Assessment of BMI

Body mass index (BMI) of a child was calculated by dividing the body weight in kilogram by the square of the standing height in meter. The body weight was categorized as "underweight", "healthy", "at risk of overweight", or "overweight" following the procedure of BMI; for age, separate growth charts for girls or boys formulated by the Center for Disease Control, USA were used.

Statistical analysis

The data were statistically analyzed by Student's *t* test and Chi-square test. Univariate and multivariate analyses of the gathered database were performed. Altogether, we analyzed all the potential risk factors (like gender, socioeconomic status, ETS, age, PM_{10}, BMI, and outdoor exposure) to identify significant influence on the prevalence of ADHD. Univariate conditional logistic regression was done first to determine which variables were potential risk factors. Multivariate conditional logistic regression analysis was then used to determine which factors were statistically significant. Univariate and multivariate logistic regression analyses of data were done by EPI info 6 and SPSS statistical packages (SPSS, USA). A descendent stepwise, conditional logistic regression adjusted over potential confounding variables was also done with the help of SPSS statistical package.

Results

Demography

In Delhi, a total of 1,000 questionnaires were distributed among children; of these, 31 were not accepted due to incomplete filling. Likewise, in the rural control areas, 1,000 questionnaires were distributed; of these, 150 were not accepted due to incomplete filling. The characteristics of both urban (Delhi) and rural (control) children are presented in Table 1. It is evident that the urban and rural children were comparable ($p>0.05$) with respect to age, gender, SES, and BMI.

Air pollution level

The annual average concentrations of PM_{10} were 161.3±4.9 μg/m^3. In contrast, the concentrations of these pollutants were significantly lower in control areas, 74.6±3.3 μg/m^3. Mean concentrations of sulfur dioxide and nitrogen dioxide in Delhi's air during this period were 9.6±1.0 and 50.1±7.1 μg/m^3, respectively, and it was within the permissible limit. In the control areas, the concentrations were 5.6±2.2 and 30.3±5.2 μg/m^3, respectively (Table 2).

Prevalence of ADHD

ADHD was diagnosed in 107 children (11.0%) of Delhi, in contrast to 2.7% (23/850) of control children. Thus, ADHD was 4.1 times more prevalent among school children of Delhi when compared with their rural counterparts ($p<0.001$; Table 3).

Difference between boys and girls

ADHD was more prevalent among boys both in urban and rural areas. It was prevalent among 18.0% of the boys enrolled in Delhi against 4.0% of the girls, giving a male/female ratio of 4.5:1. In the control group, 4.3% of the boys and 1.0% of the girls had the disorder, giving a male/female ratio of 4.3:1. When a comparison was made among boys and the girls of urban and control areas, urban boys and girls had 4.2 and 4.0 times greater prevalence of ADHD than their rural counterparts, respectively (Table 3).

Type of ADHD

Inattentive type of ADHD was predominant in Delhi (5.9%) as well as in control group (2.0%; $p<0.01$). It was followed by hyperactive–impulsive type (4.2% vs. 0.4%, $p<0.001$) and combined type (1.1% vs. 0.2%; $p<0.05$) of ADHD (Table 4).

Prevalence of ADHD in different age groups

Prevalence of ADHD in Delhi was found to be highest (14.2%) in children belonging to the age group of 12–14 years old, followed by (12.0%) 15–17 years age group. The prevalence was lowest (5.9%) in the age group of 9–11 years

Table 1 Demographic characteristics of the children

Characteristics	Control (n=850)	Delhi (n=969)	p value
Age wise distribution (%)			
9–11 years	28.5 %	29.7 %	0.56*
12–14 years	40.9 %	39.3 %	0.50*
15–17 years	30.6 %	30.9 %	0.86*
Boys/girls	1.08	1.02	
Median height in cm	132	141.5	
Median body weight in kg	40	49.5	
Median BMI in kg/m^2	15	17.5	
SES (%)			
Low	41.0 %	41.7 %	0.78*
Medium	40.3 %	40.5 %	0.96*
High	18.6 %	17.9 %	0.68*

*Statistically not significant. P values were obtained from chi-square tests for categorical variables

old. Even in the control group, prevalence of ADHD was highest in the age group of 12–14 years (3.6%), followed by 15–17 years (2.1%) and 9–11 years (1.5%). Children belonging to the age group of 12 to 14 years old were the most affected, and when compared with children belonging to age group of 9–11 years old, the odds was 2.631 at 95% confidence interval (95% CI; 1.490–4.64; Table 5).

ADHD and socioeconomic status

The prevalence of ADHD in Delhi was more in children from low (13.6%) and medium (10.2%) SES compared with high SES (6.9%). An inverse relationship existed between prevalence of ADHD and socioeconomic status; the higher the socioeconomic status, the lower is the prevalence (Table 5).

Body mass index and ADHD prevalence

The prevalence of ADHD was 11.4% in children of Delhi with normal BMI, and more or less similar prevalence was seen in underweight, at risk of getting overweight and obese children; however, no relation was found to exist between BMI and ADHD prevalence (Table 5).

Air pollution and ADHD prevalence

Compared with the control group, where the ambient air quality standard was better than ambient air quality standard of Delhi, the prevalence of ADHD was found to be much higher in children of Delhi (odds ratio (OR)=4.47; 95% CI, 2.77–7.29). Children of Delhi residing at areas where the level of particulate pollution was very high displayed a higher prevalence of ADHD than children residing in areas of comparatively low level of particulate pollution.

Multivariate logistic regression analysis showed that the prevalence of ADHD was related to PM_{10} (OR=2.066; 95% CI, 1.079–3.958), age (OR=1.568; 95%CI, 1.044–2.354), the most vulnerable being 12–14-year age group, and socioeconomic status (OR=1.232; 95%CI, 1.027–2.085; Table 6). After controlling potential confounders like age, gender, socioeconomic status, and parental smoking, PM_{10} was found to be positively and strongly associated with ADHD prevalence (Table 6). Taking the prevalence of ADHD during low pollution level (PM_{10} <120 μg/m^3) as constant (OR=1) in conditional logistic regression analysis, prevalence of ADHD was found to increase when particulate pollution was elevated (Table 5)

Discussion

The study was conducted to examine whether chronic exposure to high level of air pollution affects the behavior

Table 2 Air quality data

Ambient air (annual average)	Control	Delhi
PM_{10} (μg/m^3)	74.6±3.3	161.3±4.9*
SOx (μg/m^3)	5.6±2.2	9.6±1.0*
NOx (μg/m^3)	30.3±5.2	50.1±7.1*

*p<0.05, compared with control in chi-square tests

Table 3 Comparison of the prevalence (%) of ADHD in control and exposed group

	Control (n=850)	Delhi (n=969)
Boys (%)	4.3	18.0*
Girls (%)	1.0	4.0**
Total (%)	2.7	11.0***

*p<0.01; **p<0.005;***p<0.001 compared with control in chi-square test

Table 4 Prevalence (%) of subtype of ADHD in control and exposed group

ADHD category	Control (n=850)	Delhi (n=969)
Inattentive (%)	2.0	5.9*
Hyperactive–impulsive (%)	0.4	4.2**
Combined (%)	0.2	1.1***

*p<0.01; **p<0.001;***p<0.05 compared with control in chi-square test

of the children. We found a fourfold rise in the prevalence of ADHD among children residing in Delhi compared to children residing in less polluting rural areas of the country. About half of the world's children reside in the South and Southeast Asia. These regions are rapidly undergoing industrialization. In the process, threats to children's health, both physical and mental, are believed to be increasing [29]. In two previous reports in Delhi in the 1990s, 5% and 11.2% of children brought to pediatric outpatients' clinics by their parents for some behavioral problems were diagnosed with ADHD [3, 27]. In contrast to these studies on referred cases, our study was population-based cross-sectional, and we found ADHD prevalence as high as 11.0%. It implies that the problem has probably been intensified in the intervening years with meteoric rise in vehicular population and worsening air quality. The problem was more common in boys than in girls of comparable age, for reasons not known.

Table 5 Conditional logistic regression analysis for association between prevalence of ADHD and different confounding factors

Confounders	Odds ratio
Age	
9–11 years old	1
12–14 years old	2.631 (1.49–4.64)
15–17 years old	2.173 (1.191–3.963)
Socioeconomic status	
High SES	1
Medium SES	0.999 (0.558–1.790)
Low SES	1.173 (1.006–2.085)
BMI	
Normal	1
Underweight	0.93 (0.59–1.46)
At risk	0.92 (0.40–2.11)
Overweight	0.93 (0.27–3.18)
PM_{10}	
<120 $\mu g/m^3$	1
120–139 $\mu g/m^3$	1.824 (1.070–3.629)
140–200 $\mu g/m^3$	2.201 (1.162–5.032)
>200 $\mu g/m^3$	2.770 (1.381–5.555)

The results are expressed as odds ratio with 95% CI in parentheses

Table 6 Multivariate logistic regression analysis showing strong association between PM_{10} and ADHD prevalence

Confounding factors	OR
Gender	1.517 (1.005–2.288)*
Age	1.568 (1.044–2.354)*
PM_{10}	2.066 (1.079–3.958)**
SES	1.232 (1.027–2.085)
BMI	0.988 (0.732–1.708)

The results are expressed as odds ratio with 95% CI in parentheses
*p<0.05; **p<0.001

Children with neurological problems like epilepsy, autism, mental retardation, sensory deficits, and severe learning disabilities could influence our study. Thus, for this reason, such children were excluded from the sampling population. It was also important to exclude children exposed to high level of indoor air pollution. Therefore, children from non-smoking homes were selected. The problem of indoor air pollution is massive in villages where the women use biomass fuel for cooking purpose, so, it was important to select children from households using LPG as the cooking fuel.

At a given level of harmful environmental exposure, socioeconomic factors may modify the health effects by influencing the susceptibility characteristics of children [17]. Indeed, our study revealed that children from the low socioeconomic status were the worst sufferers; the prevalence of ADHD was highest in this group. But, after negating this confounding factor, the prevalence of ADHD was positively and strongly correlated with the PM_{10} level of the city (OR=2.066; 95% CI, 1.079–3.958).

Children suffering from ADHD show worse social adaptability [18]. Our study reflected the same picture; children diagnosed with ADHD were found to be the disturbing element in the class and were reprimanded by the teacher often. A cross-sectional analysis of children and adolescents revealed that children and adolescents with attention-deficit disorder/attention-deficit/hyperactivity disorder were at the odds of being overweight [31]. A systematic review of the literature suggests that obese individuals may present with higher than expected prevalence of ADHD [12]. Our data, however, suggest no positive relation between BMI and prevalence of ADHD.

Human and animal studies have confirmed that the brain is a target organ for several environmental pollutants. A developing brain is particularly vulnerable to toxic chemical insults, and such insults may have long-lasting or even irreversible developmental consequences [32].

The respiratory and olfactory epithelia are particularly vulnerable to toxicological insults of inhaled pollutants. It may be said that neurodegenerative disorders may begin early in life, with air pollutants playing a crucial role [8]. Experimental studies on rodents have shown that the CNS can be targeted by airborne solid ultrafine particles and that the most likely mechanism is from deposits on the olfactory mucosa of the nasopharyngeal region of the respiratory tract and subsequent translocation via the olfactory nerve [23].

Damage to the endothelial blood–brain barriers by the free radical activity carried on the PM particle's surface would disrupt the tight junctions and facilitate particle translocation. Respiratory tract inflammation may play a role by chronically increasing the levels of circulating cytokines that can cross the blood–brain barrier and evoke an inflammatory response [25]. Increased concentrations of pre- and anti-inflammatory serum cytokines are seen in Mexico City children, along with evidence of significant respiratory tract damage and breakdown of their nasal epithelial barriers. Although the children are chronically exposed to a complex mixture of air pollutants, a variety of evidences suggest that particulate matter may play a role in the development of neurodegenerative diseases, including ADHD [7, 9].

It has been hypothesized that ADHD is due to non-cortical dysfunction that manifests early in ontogeny [14], but the precise etiology of ADHD is currently unknown. Stress could be a risk factor because increased pressure for school performance is an important contributing factor for ADHD [26]. Maternal smoking is another independent risk factor for ADHD in offspring [6, 19, 20].This confounding factor could not have affected our study design as children were selected from household of non-smokers (present and past).

ADHD has a significant impact on multiple domains of health-related quality of life in children and adolescents. They have impaired learning ability, decreased self-esteem, social problems, family difficulty, and potential long-term effects [4]. In addition, the problems of children with ADHD have a significant impact on parents' emotional health and parents' time to meet their own needs, and they interfered with family activities and family cohesion [16]. ADHD increases the risk for developing antisocial and substance use disorders in adolescence, which, in turn, increases the risk for criminal behavior in adolescence and adulthood [21].

Our study clearly showed that the prevalence of ADHD increased when particulate pollution was elevated, using conditional logistic regression analysis. This strengthened our aim to establish a positive relation between particulate pollution and ADHD prevalence. Thus, we can conclude that though gender, socioeconomic status, and age play a very important factor in ADHD prevalence, the association is highest and strongest between particulate pollution and prevalence of ADHD.

There is a need to improve research in the developing countries like India on environment and children's health, the neurobehavioral aspect in particular. Assessment of behavioral abnormality is very difficult particularly in children. The causes for the development of ADHD are many, exposure to air pollution is just one of them. Because of the complexity, integrated approaches and a combination of different intervention measures and policies are necessary to reduce environmental exposure and adverse health effects in children. Researchers and pediatricians may contribute to the improvement of children's environmental health by risk communication and health advocacy at community and government levels.

Acknowledgement I would like to thank Central Pollution Control Board (CPCB), Delhi for providing the fund to carry out the study.

References

1. American Pediatric Association (APA) (2000) Diagnostic and statistical manual of mental disorders, 4th edition (DSM-IV), text revision. American Pediatric Association, Washington, DC
2. Badami MG (2005) Transport and urban air pollution in India. Environ Manage 36:195–204
3. Bhatia MS, Nigam VR, Bohra N, Malik SC (1991) Attention deficit disorder with hyperactivity among pediatric outpatients. J Child Psychol Psychiatry 32:297–306
4. Brook V, Boaz M (2006) Adolescents with attention-deficit hyperactivity disorder/learning disability and their proneness to accidents. Indian J Pediatr 73:299–303
5. Brookes KJ, Xu X, Chen W, Zhou K, Neale B, Lowe N (2006) The analysis of 51 genes in DSM-IV combined type attention deficit hyperactivity disorder: association signals in DRD4, DAT1 and 16 other genes. Mol Psychiatry 11:934–953
6. Button TM, Thapar A, McGuffin P (2005) Relationship between antisocial behavior, attention-deficit hyperactivity disorder and maternal prenatal smoking. Br J Psychiatry 187:155–160
7. Calderon-Garciduenas L, Valencia-Salazar G, Rodriguez-Alcaraz A, Gambling TM, Garcia R, Osnaya N (2001) Ultrastructural nasal pathology in children chronically and sequentially exposed to air pollutants. Am J Respir Cell Mol Biol 24:132–138
8. Calderon-Garciduenas L, Azzarelli B, Acuna H, Garcia R, Gambling TM, Osnava N (2002) Air pollution and brain damage. Toxicol Pathol 30:373–389
9. Calderon-Garciduenas L, Maronpot RR, Torres-Jardon R, Henriquez-Roldan C, Schoonhoven R, Acuna-Ayala H (2003) DNA damage in nasal and brain tissues of canines exposed to air pollutants is associated with evidence of chronic brain inflammation and neurodegeneration. Toxicol Pathol 31:524–538
10. Calderon-Garciduenas L, Reed W, Maronpot RR, Henriquez-Roldan C, Delgado-Chavez R, Calderon-Garciduenas A (2004) Brain inflammation and Alzheimer's-like pathology in individuals exposed to severe air pollution. Toxicol Pathol 32:650–658
11. Capano L, Minden D, Chen SX, Schacher RJ, Lckowicz A (2008) Mathematical learning disorder in school-age children with attention-deficit hyperactivity disorder. Can J Psychiatry 53:392–399
12. Cortese S, Angriman M, Maffeis C, Isnard P, Konofal E, Lecendreux M (2008) Attention-deficit/hyperactivity disorder (ADHD) and obesity: a systematic review of the literature. Crit Rev Food Sci Nutr 48:524–37

13. de Nijs PF, Ferdinand RF, de Bruin EI, Dekker MC, van Duijn CM, Verhulst DC (2004) Attention-deficit /hyperactivity disorder (ADHD): parents' judgment about school, teachers' judgment about home. Eur Child Adolesc Psychiatry 13:315–320
14. Halperin JM, Schulz KP (2006) Revisiting the role of prefrontal cortex in the pathophysiology of attention-deficit hyperactivity disorder. Psychol Bull 132:560–581
15. Hudson R, Arriola A, Martinez-Gomez M, Distel H (2006) Effect of air pollution on olfactory function in resident of Mexico City. Chem Senses 31:79–85
16. Klassen AF, Miller A, Fine S (2004) Health-related quality of life in children and adolescents who have a diagnosis of attention-deficit/hyperactivity disorder. Pediatrics 114:541–547
17. Kohlhuber M, Heinrich J, van Den Hazel P, Zuurbier M, Louise Bistrup M, Koppe JG, Bolte G (2006) Children's environmental health: why should social disparities be considered? Acta Paediatr Suppl 95:26–30
18. Kukla L, Hruba D, Tyrlik M, Matejova H (2008) Conduct disorders in seven-year-old children—results of ELSPAC study 1. Co-morbidity. Cas Lek Ces 147:269–277
19. Laucht M, Schmidt MH (2004) Maternal smoking during pregnancy: risk factor for ADHD in the offspring? Z Kinder Jugendpsychiatr Psychother 32:177–185
20. Linnet KM, Dalsgaard OC, Wisborg K, Henriken TB, Rodriguez A, Kotimaa A (2003) Maternal lifestyle factors in pregnancy risk of attention-deficit hyperactivity disorder and associated behavior: review of the current evidence. Am J Psychiatry 160:1028–1040
21. Mannuzza S, Klein RG, Moulton JL 3rd (2008) Lifetime criminality among boys with attention deficit hyperactivity disorder: a prospective follow-up study into adulthood using official arrest records. Psychiatry Res 160:237–246
22. Mo EF (1997) Ministry of Environment and Forest, Government of India. White paper on pollution in Delhi with an action plan. Ministry of Environment and Forest, Government of India, New Delhi
23. Oberdorster G, Sharp Z, Atudorei V, Elder A, Gelein R, Kreyling W, Cox C (2004) Translocation of inhaled ultrafine particles to the brain. Inhal Toxicol 16:437–445
24. Porrino LJ, Rapoport JL, Behar D, Sceery W, Ismond DR, Bunney WE Jr (1983) A naturalistic assessment of the motor activity of hyperactive boys. I. Comparison with normal controls. Arch Gen Psychiatry 40:681–687
25. Rivest S (2001) How circulating cytokines trigger the neural circuits that control the hypothalamic–pituitary–adrenal axis. Psychoneuroendocrinology 26:761–788
26. Schneider H, Eisenberg D (2006) Who receives a diagnosis of attention-deficit/hyperactivity disorder in the United States elementary school population? Pediatrics 117:601–609
27. Sidana A, Bhatia MS, Chaudhary S (1998) Prevalence of and pattern of psychiatric morbidity in children. Indian J Med Sci 52:556–558
28. Suglia SF, Gryparis A, Wright RO, Schwartz J, Wright RJ (2008) Association of black carbon with cognition among children in a prospective birth cohort study. Am J Epidemiol 167:280–286
29. Suk WA, Ruchirawat KM, Balakrishnan K, Berger M, Carpenter D, Damstra T, de Garbino JP, Koh D, Landrigan PJ, Makalinano I, Sly PD, Xu Y, Zheng BS (2003) Environmental threats to children's health in Southeast Asia and the Western Pacific. Environ Health Perspect 111:1340–1347
30. Tiwari SC, Kumar A, Kumar A (2005) Development and standardization of a scale to measure socio-economic status in urban and rural communities in India. Indian J Med Res 122:309–314
31. Waring ME, Lapane KL (2008) Overweight in children and adolescents in relation to attention-deficit/hyperactivity disorder: results from a national sample. Pediatrics 122:e1–6
32. Winneke G, Walkowiak J, Lilienthal H (2002) PCB-induced neuro developmental toxicity in human infants and its potential mediation by endocrine dysfunction. Toxic Rev 181:161–165
33. Wohl M, Purper-Ouakil D, Mouren MC, Ades J, Gorwood P (2005) Meta-analysis of candidate genes in attention-dificit hyperactivity disorder. Encephale 31:437–447