Psychiatry Research 225 (2015) 164–168

Contents lists available at ScienceDirect

Psychiatry Research

journal homepage: www.elsevier.com/locate/psychres

The effects of prenatal exposure to alcohol and environmental tobacco smoke on risk for ADHD: A large population-based study

Ji-Youn Han [a], Ho-Jang Kwon [b,c,*], Mina Ha [b,c], Ki-Chung Paik [c,d], Myung-Ho Lim [c,d], Sang Gyu Lee [e], Seung-Jin Yoo [c], Eun-Jung Kim [c]

[a] Department of Dental Hygiene, Baekseok Culture University, Cheonan, South Korea
[b] Department of Preventive Medicine, College of Medicine, Dankook University, Cheonan, South Korea
[c] The Environmental Health Center (Neurodevelopment), Dankook University Medical Center, Cheonan, South Korea
[d] Department of Psychiatry, College of Medicine, Dankook University, Cheonan, South Korea
[e] Graduate School of Public Health, Yonsei University, Seoul, South Korea

ARTICLE INFO

Article history:
Received 29 August 2013
Received in revised form
5 October 2014
Accepted 4 November 2014
Available online 13 November 2014

Keywords:
ADHD
Environmental tobacco smoke
Alcohol

ABSTRACT

Attention deficit hyperactivity disorder (ADHD) is caused by the interaction of genetic and environmental factors. The objective of this study was to examine the effects of prenatal exposure to alcohol and environmental tobacco smoke (ETS). Among the 30,552 parents who responded to a survey, the answers of 19,940 who replied to questions on prenatal exposure to ETS, alcohol consumption, and completed the DuPaul Rating Scale were analyzed. Results revealed that risk of ADHD significantly increased as a result of exposure to alcohol by 1.55 times (95% CI 1.33–1.82), maternal smoking during pregnancy by 2.64 times (95% CI 1.45–4.80), and paternal smoking during pregnancy by 1.17 times (95% CI 1.98–1.39). When the subjects whose mothers did not smoke during pregnancy were divided into 4 groups, the prevalence was 1.16 times higher (95% CI 1.02–1.33) in the group exposed to ETS but not alcohol, 1.19 times higher (95% CI 0.91–1.57) in the group exposed to alcohol but not ETS, and 1.58 times higher (95% CI 1.31–1.91) in the group exposed to ETS and alcohol. The differences between the groups were statistically significantly ($P < 0.0001$). This result shows that simultaneous exposure to ETS and alcohol during pregnancy increases the risk of ADHD.

1. Introduction

Attention-deficit hyperactivity disorder (ADHD) is one of the most commonly observed disorders in preschool and school age children, and it is characterized by increased hyperactivity and impulsiveness and poorer attention. (American Psychiatric Association, 1994). Due to these core symptoms of ADHD, children with this disorder are more likely to have behavioral problems, lower academic achievement, and difficulty in establishing social relations (Barkley, 1990). The ADHD prevalence rates vary across countries from 7.2% to 11.4% (Baumgaertel et al., 1995; Wolraich et al., 1996; Montiel-Nava et al., 2002). The prevalence rates reported in South Korean elementary school children were 7.6% (Cho and Shin, 1994), and 6.1% (Pyo et al., 2001).

Many studies have examined the causes of ADHD, identifying various risk factors that may contribute to the development of ADHD. Environmental factors include complications from pregnancy and delivery (Sprich-Buckminster et al., 1993) and perinatal factors. Other factors, such stress during pregnancy (Linnet et al., 2003) and exposure to heavy metals such as lead (Needleman, 1982), mercury (Anderson et al., 1981), and manganese (Collipp et al., 1983) are also reported to increase the risk of ADHD.

Alcohol consumption and smoking during pregnancy have been extensively studied. Consumption of alcohol during pregnancy is reported to increase the risk of ADHD (Streissguth et al., 1994; Mick et al., 2002; Knopik et al., 2005). Studies on understanding this association have produced consistent results; however, there are some contradictory evidences. For example, Boyd et al. (1991) and Leech et al. (1999) reported that the use of alcohol was not considered a risk factor for ADHD. These negative results may be due to the absence of a sufficiently large number of heavy drinking mothers in those samples. The association between exposure to smoking during pregnancy and ADHD has been demonstrated by many studies. However, only a few studies have demonstrated the association between ADHD and maternal exposure to ETS among those who did not smoke. Smoking during pregnancy increases the risk for ADHD by 2.7 times (Milberger et al., 1998). Linnet et al. (2005) reported that smoking in pregnancy increased the prevalence of hyperkinetic disorder by 3 times, and Lindblad and Hjern (2010) reported that smoking increased the risk for ADHD by 2.86 times.

* Corresponding author at: Department of Preventive Medicine, College of Medicine, Dankook University, 29 Anseo-Dong, Dongnam-Gu, Cheonan, Chungnam, South Korea. Tel.: +82 415503879; fax: +82 415566461.
E-mail address: hojangkwon@gmail.com (H.-J. Kwon).

http://dx.doi.org/10.1016/j.psychres.2014.11.009
0165-1781/

Alcohol consumption and smoking often coexist. According to a study by Grant et al. (2004), the prevalence of nicotine dependence among people with alcohol dependence is more than three times higher than in the general population. The prevalence of alcohol dependence is about four times higher among people with nicotine dependence than in the general population. The degree of alcohol dependency is known to have some correlation with the degree of nicotine dependency (Batel et al., 1995). It has been proposed that the concurrent use of alcohol and nicotine gives synergy to the toxicity of each. For example, the process of genotype mutation caused by nicotine is accelerated by alcohol (Henning et al., 1999).

There is much research on prenatal exposure to tobacco smoke and alcohol consumption in pregnancy, but this may well be the first study that simultaneously examines prenatal exposure to alcohol, maternal smoking, and ETS in relation to ADHD in such a large sample. This study was conducted in order to assess the effects of exposure to ETS and the consumption of alcohol during pregnancy on the incidence of ADHD. In our study, we hypothesized that children from mothers exposed to ETS and alcohol during pregnancy showed higher ADHD prevalence than those from mothers without such an exposure.

2. Method

2.1. Participants

This study surveyed the parents of elementary students in Cheonan, Korea for two years in 2007 and 2008. We distributed questionnaires with an official cover letter to 53 schools in Cheonan, and asked parents to complete the questionnaires with the help of class and health teachers. Among 30,552 parents who responded to the survey, the responses of the 19,940 who answered the questions on prenatal exposure to alcohol and ETS, and completed the ADHD DuPaul Rating Scale, were analyzed. This study was approved by the Institutional Review Board of Dankook University Hospital, and written consent of the participants was obtained.

2.2. Materials

The Korean version of the DuPaul Rating Scale (K-ARS; Kim et al., 2003) was utilized and completed by the parents. The K-ARS consists of 18 questions, nine items about attention and nine items about hyperactivity, scored on a 4-point scale (0–3). We classified children as having ADHD if the total inattentive and hyperactivity scores were ≥ 19. The K-ARS classifies children as "inattentive-type ADHD" if the total score is 19 or higher, and the score of attention deficit items is 10 or higher; "hyperactivity-type ADHD" if the total score is 19 or higher, and the score of hyperactivity items is 10 or higher; or "mixed-type ADHD" if both conditions are satisfied.

2.3. Variables

The variables measuring exposure used in this study were the mother's current alcohol consumption patterns, the mother's prenatal alcohol consumption, current parental smoking habits, and prenatal smoking habits. For maternal exposure to ETS during pregnancy, we selected paternal smoking as the source of ETS exposure. The outcome variable was the score of ADHD measured by the K-ARS.

The confounding variables were gender, age, father's education, mother's age at childbirth, marriage status, delivery complications, history of child vaccinations, and family history of ADHD. The association between ADHD and demographic variables including patient's gender and age, father's education, mother's age at childbirth, marital status, delivery complications, history of child vaccinations, and family history of ADHD were analyzed by χ^2 tests. In addition, the subjects were divided into four groups according to prenatal exposure to ETS and alcohol to perform interaction analysis. The exposure variables and the outcome variable were also analyzed through multiple logistic regression. We used Statistical Package for the Social Sciences (SPSS), Version 18.0, for the analyses.

Table 1
ADHD prevalences by Socio-demographics and prenatal exposure to alcohol and environmental tobacco smoke.

Characteristics		ADHD			
		All(*N*)	Yes (*N*)	%	*p**
All		19,940	1769	(8.9)	
Sex	Male	9855	1195	(12.1)	< 0.0001
	Female	10,085	574	(5.7)	
Age	< 9	10,252	926	(9.0)	0.422
	10 ~ 11	6024	536	(8.9)	
	≥ 12	2734	228	(8.3)	
	Unknown	930	79	(8.5)	
Father's education	< 12	435	89	(20.5)	< 0.0001
	12	8092	808	(10.0)	
	≥ 13	11,122	831	(7.5)	
	Unknown	291	41	(14.1)	
Marriage status	Single	2419	283	(11.7)	< 0.0001
	Couple	16,774	1422	(8.5)	
	Unknown	747	64	(8.6)	
ADHD family history	No	18,443	1528	(8.3)	< 0.0001
	Yes	183	26	(14.2)	
	Unknown	1314	215	(16.4)	
Mother's age at childbirth	≤ 20	120	25	(20.8)	< 0.0001
	21 ~ 29	11,285	957	(8.5)	
	30 ~ 34	6523	541	(8.3)	
	35 ~ 39	1246	135	(10.8)	
	≥ 40	217	30	(13.8)	
	Unknown	549	81	(14.8)	
History of vaccination	Not completely	3999	484	(12.1)	< 0.0001
	Completely done	15,664	1240	(7.9)	
	Unknown	277	45	(16.2)	
Delivery complication	No	8283	663	(8.0)	< 0.0001
	Yes	226	30	(13.3)	
	Unknown	11,431	1076	(9.4)	
Prenatal ETS	No	5447	424	(7.8)	0.002
	Yes	14,493	1345	(9.3)	
Prenatal alcohol use	No	17,524	1478	(8.4)	< 0.0001
	Yes	2416	291	(12.0)	

*χ^2 test,excluding missing values.

3. Results

Of the 19,940 respondents, 1769 (8.9%) indicated that their child met K-ARS criteria for ADHD. In addition, 14,493 children (72.7%) were prenatally exposed to ETS, and 2416 (12.1%), to alcohol (Table 1).

Maternal alcohol consumption during pregnancy was significantly associated with ADHD. The risk of ADHD was 1.55 times higher (95% CI 1.33–1.82) in children of mothers who used alcohol during pregnancy than in those whose mothers did not consume alcohol. The risk of ADHD was 2.47 times higher (95% CI 1.66–3.68) in the group of mothers who currently smoked than in the group who did not smoke. If the mother smoked during pregnancy, the risk of ADHD was approximately 2.64 times higher (95% CI 1.45–4.80). With regard to paternal smoking, the risk of ADHD was 1.17 times higher (95% CI 1.98–1.39) if the father smoked during the mothers' pregnancy than in the group with fathers who did not smoke (Table 2).

The subjects with mothers who did not smoke during pregnancy were divided into four groups according to prenatal exposure to ETS and alcohol. The prevalence rate of ADHD was significantly higher in the group with prenatal exposure to ETS and alcohol (12.8%) than in the group without exposure to either (7.5%) [$P < 0.0001$]. In addition, compared to the group without exposure to ETS and alcohol, the prevalence and risk was 1.16 times higher (95% CI 1.02–1.33) in the group exposed to ETS but not exposed to alcohol, 1.19 times higher (95% CI 0.91–1.57) in the group not exposed to ETS but exposed to alcohol, and 1.58 times higher (95% CI 1.31–1.91) in the group exposed to both ETS and alcohol. The differences were statistically significantly (interaction, $P < 0.0001$) (Table 3).

We had analyzed the association between ADHD subtype and prenatal exposure to ETS and alcohol. Across all subtype of ADHD, there were no significant differences in the magnitude of effect estimates. (data not shown).

4. Discussion

ADHD is a disorder characterized by inattention, impulsiveness and hyperactivity, and its prevalence, although varying across countries and studies, has been reported to be 2.2–17.8% (Skounti et al., 2007). In our study, the prevalence of ADHD was 8.9% in elementary school children. This is similar to the prevalence of ADHD diagnosed through structured interviews based on the DSM-IV in previous studies.

In our study, 72.7% of children were prenatally exposed to ETS which reflected paternal smoking during pregnancy. According to the National Health and Nutrition Examination Survey by Korea Centers for Disease Control and Prevention (2013), the lifetime prevalence rate of smoking in men aged 30–39 years was 75%, and current smoking rate was 54.8%, while 55.3% of women aged 19–29 years were exposed to ETS. Lee et al. (2010) reported 16.4% maternal alcohol consumption during pregnancy in a sample of 1000 people. In our study also, 12.1% of pregnancies had maternal alcohol consumption.

In our study, alcohol consumption in pregnancy increased the prevalence of ADHD by 1.55 times, and maternal smoking in pregnancy increased its prevalence by 2.64 times. Moreover, the interaction between exposure to environmental tobacco smoking and alcohol in pregnancy increased the prevalence of ADHD by 1.58 times in the group with mothers who did not smoke during pregnancy, but consumed alcohol and were exposed to tobacco smoke. This result shows that simultaneous exposure to environmental tobacco smoke and alcohol during pregnancy increases the risk of ADHD.

The harmful effect of a pregnant woman's alcohol consumption and smoking on the fetus has been studied thoroughly, and smoking during pregnancy is known to cause behavioral and mental disorders in the child (Andres and Day, 2000). The association between exposure to smoking during pregnancy and ADHD has been demonstrated by many studies. Some animal experiments have shown that exposure to nicotine during pregnancy results in hyperactivity in the child (Slotkin et al., 1993; Eriksson et al., 2000), and the possible mechanisms for this increase may be the number of nicotine receptors and an abnormality in the dopamine system (Marks et al., 1993). Smoking may reduce the amount of blood sent to the fetus, hinder the supply of nutrition and oxygen (Suzuki et al., 1980), and have direct impact on the brain of the fetus through exposure to the tar and carbon monoxide in cigarettes (Ernst et al., 2001). Many studies with human subjects report that exposure to nicotine during pregnancy increases the risk of ADHD (Herrmann et al., 2008; Roza et al., 2009). Thapar et al. (2003) and Obel et al. (2009) reported that smoking during pregnancy increases the risk of hyperactivity–inattention, and Eskenazi and Castorina (1999) found an association between smoking during pregnancy, neurobehavioral development, and behavioral problems. Further, Rauh et al. (2004) reported that

Table 2
Association between ADHD and maternal alcohol use and parental smoking.

Total no.	Total no.	ADHD(+)		Unadjusted		Adjusted†	
		N	%	OR	(95% CI)	OR	(95% CI)
Maternal alcohol intake	19,940	1769	(8.9)				
Never	6742	500	(7.4)	1	Referent	1	Referent
Alcohol used except pregnancy period	10,782	978	(9.1)	1.25	1.11,1.39	1.31	1.17,1.48
Alcohol used during pregnancy	2416	291	(12.0)	1.71	1.47,1.99	1.55	1.33,1.82
Maternal smoking	19,940	1769	(8.9)				
Never smoked	14,339	1188	(8.3)	1	Referent	1	Referent
Smoking except pregnancy	155	36	(23.2)	3.35	2.30,4.89	2.47	1.66,3.68
Smoking during pregnancy	64	16	(25.0)	3.69	2.09,6.52	2.64	1.45,4.80
Missing	5382	529	(9.8)				
Paternal smoking	19,940	1769	(8.9)				
Never smoked	2130	161	(7.6)	1	Referent	1	Referent
Smoking except pregnancy	2121	160	(7.5)	1.00	0.80,1.25	0.95	0.76,1.20
Smoking during pregnancy	13,900	1277	(9.2)	1.24	1.04,1.47	1.17	1.98,1.39
Missing	1789	171	(9.6)				

†Odds ratio and 95% confidence intervals estimated using multiple logistic regression model adjusted for gender, age, father's education, mother's age at childbirth, marriage status, delivery complication, history of child vaccinations, family history of ADHD.

Table 3
Association between ADHD and prenatal exposure to factors among mothers who did not smoke during pregnancy.

	Group	Total no.	ADHD		Unadjusted		Adjusted[†]	
		19,876	*N*	(%)	OR	(95% CI)	OR	(95% CI)
Group	ETS(*x*), alcohol use (*x*)	4728	353	(7.5)	1	Referent	1	Referent
	ETS(0), alcohol use(*x*)	12,753	1115	(8.7)	1.19	1.05,1.35	1.16	1.02,1.33
	ETS(*x*), alcohol use (0)	719	71	(9.9)	1.36	1.04,1.78	1.19	0.91,1.57
	ETS(0), alcohol use (0)	1676	214	(12.8)	1.81	1.52,2.17	1.58	1.31,1.91
	P-interaction					< 0.0001		

[†]Odds ratio and 95% confidence intervals estimated using multiple logistic regression model adjusted for gender, age, father's education, mother's age at childbirth, marriage status, delivery complication, history of child vaccinations, family history of ADHD.

exposure to environmental tobacco smoke during pregnancy caused a delay of neurobehavioral development, resulting in growth that was as much as two times slower than in embryos not exposed to smoke. Our result, that smoking and exposure to environmental tobacco smoke during pregnancy increased the prevalence of ADHD, is consistent with the results of these earlier studies.

Alcohol consumption during pregnancy can cause fetal alcohol spectrum disorders, which consist of neurobehavioral disorders, psychomotor retardation, other problems, as well as various internal anomalies that may have no morphological manifestations (Mattson et al., 2011). Prenatal exposure to alcohol in humans causes brain abnormality (Sowell et al., 1996; Kuehn et al., 2012). Exposure to alcohol during the third trimester, when the brain undergoes dramatic development, results in microcephaly and the loss of Purkinje cells (Goodlett et al., 1991). Sulik and Johnston (1983) have reported that facial and brain alterations were associated with first trimester exposure in an animal study. Experiments on animal development have reported that heavy alcohol consumption during pregnancy is a neurotoxin (Olney et al., 2000). Streissguth et al. (1989) and Mattson et al. (2013) reported that heavy alcohol consumption during pregnancy is associated with neurobehavioral disorders. Streissguth et al. (1990) also reported that moderate levels of prenatal alcohol exposure can have long lasting effects on IQ and learning problems in young school aged children. These results are consistent with our report that alcohol consumption during pregnancy increased the prevalence of ADHD. The results are consistent with those from previous studies (Brown et al., 1991; Delaney-Black et al., 2000).

Several studies reported that the concurrent exposure to tobacco and alcohol increases toxicity; for example, Henning et al. (1999) reported the process of genotype mutation caused by nicotine is accelerated by alcohol. Moreover, it has been reported that concurrent smoking and alcohol consumption can affect the development of tumors by damaging the antioxidant defense mechanism (Gao et al., 2002). The incidence and severity of various diseases are also different in those who neither smoke nor drink from those who engage in both (Vaillant et al., 1991). Additionally, leukemia in children has been associated with alcohol consumption and smoking in parents (MacArthur et al., 2008). Previous neurobehavioral studies have focused primarily on the association of the mother's smoking, alcohol consumption, or both, during pregnancy and the child's risk for ADHD. Some animal studies have shown that exposure to nicotine during pregnancy results in an increased number of nicotine receptors and abnormalities in the dopamine system (Marks et al., 1993). A few studies also suggest that ethanol stimulates the release of both dopamine in the ventral tegmental area (Yoshimoto et al., 1992). There are few epidemiologic studies on the interaction of exposure to ETS and alcohol in connection to the risk or prevalence of ADHD. Thus, the results of the present study are notable.

Nevertheless, this study has certain limitations. The first pertains to the generalizability of the results. The subjects were from Cheonan, which has urban, rural, and industrial areas, but there may be some limitations in applying the results of this study to all children in South Korea. Second, the questionnaire survey was based on the parental recall, which may have an information bias, and this raises questions about the accuracy of the results. Another limitation is that there were no professional assessments or diagnoses. It would not be possible to obtain formal diagnostic data for such a large sample since few children sampled from a general population would be clinically diagnosed with ADHD. Additionally, the cultural climate of South Korea, which views women's smoking more negatively than in other countries, may have caused an information bias. Subjects who did not reply to the questionnaire on mother's alcohol consumption and smoking during pregnancy were excluded from the study, which may represent a different segment of the population, however, the excluded subjects did not differ from the included group in any general characteristics. Another important limitation of this study is that the levels of alcohol consumption and frequency of smoking during pregnancy are unknown. A large majority of these women may have consumed alcohol in measures that were probably too low to cause adverse effects. Therefore, the magnitude of the effects of heavy drinking may be underestimated in our study.

Despite these limitations, to the best of our knowledge, this is the first study that considered a non-smoking mother's exposure to environmental tobacco smoke and alcohol consumption during pregnancy and this study is meaningful because it identified an increase in the prevalence of ADHD under those conditions. Future studies could explore this result on the role of environmental factors in increasing risk for ADHD through follow-up studies and the inclusion of professional assessments.

Acknowledgements

Project funding was provided by the Ministry of Environment, the Republic of Korea.

References

American Psychiatric Association, 1994. Diagnostic and Statistical Manual Of Mental Disorders, 4th ed. American Psychiatric Association, Washington, D.C.
Anderson, R.J., Hart, G.R., Crumpler, C.P., Lerman, M.J., 1981. Clonidine overdose: report of six cases and review of the literature. Annals of Emergency Medicine 10, 107–112.
Andres, R.L., Day, M.C., 2000. Perinatal complications associated with material tobacco use. Seminars in Neonatology 5, 231–241.
Barkley, R.A., 1990. Attention Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment. Guilford Press, New York.
Batel, P., Pessione, F., Maître, C., Rueff, B., 1995. Relationship between alcohol and tobacco dependencies among alcoholics who smoke. Addiction 90, 977–980.
Baumgaertel, A., Wolraich, M.L., Dietrich, M., 1995. Comparison of diagnostic criteria for attention deficit disorders in a German elementary school sample. Journal of the American Academy of Child and Adolescent Psychiatry 34, 629–638.

Boyd, T.A., Ernhart, C.B., Greene, T.H., Sokol, R.J., Martier, S., 1991. Prenatal alcohol exposure and sustained attention in the preschool years. Neurotoxicology Teratology 13, 49–55.
Brown, R.T., Coles, C.D., Smith, I.E., Platzman, K.A., Silverstein, J., Erickson, S., Falek, A., 1991. Effects of prenatal alcohol exposure at school age, II: attention and behavior. Neurotoxicology Teratology 13, 369–376.
Cho, S.C., Shin, Y.O., 1994. Prevalence of disruptive behavior disorders. Journal of the Korean Academy of Child and Adolescent Psychiatry 5, 141–149.
Collipp, P.J., Chen, S.Y., Maitinsky, S., 1983. Manganese in infant formulas and learning disability. Annals of Nutrition and Metabolism 27, 488–494.
Delaney-Black, V., Covington, C., Templin, T., Ager, J., Nordstrom-Klee, B., Martier, S., Leddick, L., Czerwinski, R.H., Sokol, R.J., 2000. Teacher-assessed behavior of children prenatally exposed to cocaine. Pediatrics 106, 782–791.
Eriksson, P., Ankarberg, E., Fredriksson, A., 2000. Exposure to nicotine during a defined period in neonatal life induces permanent changes in brain nicotinic receptors and in behaviour of adult mice. Brain Research 853, 41–48.
Ernst, M., Moolchan, E.T., Robinson, M.L., 2001. Behavioral and neural consequences of prenatal exposure to nicotine. Journal of the American Academy of Child and Adolescent Psychiatry 40, 630–641.
Eskenazi, B., Castorina, R., 1999. Association of prenatal maternal or postnatal child environmental tobacco smoke exposure and neurodevelopmental and behavioral problems in children. Environmental Health Perspectives 107, 991–1000.
Gao, C.M., Takezaki, T., Wu, J.Z., Liu, Y.T., Li, S.P., Ding, J.H., Su, P., Hu, X., Xu, T.L., Sugimura, H., Tajima, K., 2002. Glutathione-S-transferases M1 (GSTM1) and GSTT1 genotype, smoking, consumption of alcohol and tea and risk of esophageal and stomach cancers: a case-control study of a high-incidence area in Jiangsu Province, China. Cancer Letters 188, 95–102.
Goodlett, C.R., Thomas, J.D., West, J.R., 1991. Long-term deficits in cerebellar growth and rotarod performance of rats following "binge-like" alcohol exposure during the neonatal brain growth spurt. Neurotoxicology and Teratology 13, 69–74.
Grant, B.F., Hasin, D.S., Chou, S.P., Stinson, F.S., Dawson, D.A., 2004. Nicotine dependence and psychiatric disorders in the United States: results from the national epidemiologic survey on alcohol and related conditions. Archives of General Psychiatry 61, 1107–1115.
Henning, S., Cascorbi, I., Münchow, B., Jahnke, V., Roots, I., 1999. Association of arylamine N-acetyltransferases NAT1 and NAT2 genotypes to laryngeal cancer risk. Pharmacogenetics 9, 103–111.
Herrmann, M., King, K., Weitzman, M., 2008. Prenatal tobacco smoke and postnatal secondhand smoke exposure and child neurodevelopment. Current Opinion in Pediatrics 20, 184–190.
Kim, Y.S., So, Y.K., Noh, J.S., Choi, N.K., Kim, S.J., Koh, Y.J., 2003. Normative data on the Korean ADHD rating scales (K-ARS) for parents and teachers. Journal of the Korean Neuropsychiatry Association 42, 352–359.
Knopik, V.S., Sparrow, E.P., Madden, P.A., Bucholz, K.K., Hudziak, J.J., Reich, W., Slutske, W.S., Grant, J.D., McLaughlin, T.L., Todorov, A., Todd, R.D., Heath, A.C., 2005. Contributions of parental alcoholism, prenatal substance exposure, and genetic transmission to child ADHD risk: a female twin study. Psychological Medicine 35, 625–635.
Korea Centers for Disease Control and Prevention, 2013. Korea Health Statistics 2012: Korea National Health and Nutrition Examination Survey (KNHANES, V-3).
Kuehn, D., Aros, S., Cassorla, F., Avaria, M., Unanue, N., Henriquez, C., Kleinsteuber, K., Conca, B., Avila, A., Carter, T.C., Conley, M.R., Troendle, J., Mills, J.L., 2012. A prospective cohort study of the prevalence of growth, facial, and central nervous system abnormalities in children with heavy prenatal alcohol exposure. Alcoholism Clinical and Experimental Research 36, 1811–1819.
Leech, S.L., Richardson, G.A., Goldschmidt, L., Day, N.L., 1999. Prenatal substance exposure: effects on attention and impulsivity of 6-year-olds. Neurotoxicology and Teratology 21, 109–118.
Lee, S.H., Shin, S.J., Won, S.D., Kim, E.J., Oh, D.Y., 2010. Alcohol use during pregnancy and related risk factors in Korea. Psychiatry Investigation 7, 86–92.
Lindblad, F., Hjern, A., 2010. ADHD after fetal exposure to maternal smoking. Nicotine and Tobacco Research 12, 408–415.
Linnet, K.M., Dalsgaard, S., Obel, C., Wisborg, K., Henirksen, T.B., Rodriguez, A., Kotimaa, W., Moilanen, I., Thomsen, P.H., Olsen, J., Jarvelin, M.R., 2003. Maternal lifestyle factors in pregnancy risk of attention deficit hyperactivity disorder and associated behaviors: review of the current evidence. American Journal of Psychiatry 160, 1028–1040.
Linnet, K.M., Wisborg, K., Obel, C., Secher, N.J., Thomsen, P.H., Agerbo, E., Henriksen, T.B., 2005. Smoking during pregnancy and the risk for hyperkinetic disorder in offspring. Pediatrics 116, 462–467.
MacArthur, A.C., McBride, M.L., Spinelli, J.J., Tamaro, S., Gallagher, R.P., Theriault, G., 2008. Risk of childhood leukemia associated with parental smoking and alcohol consumption prior to conception and during pregnancy: the cross-Canada childhood leukemia study. Cancer Causes Control 19, 283–295.
Marks, M.J., Grady, S.R., Collins, A.C., 1993. Downregulation of nicotinic receptor function after chronic nicotine infusion. Journal of Pharmacology and Experimental Therapeutics 266, 1268–1276.
Mattson, S.N., Crocker, N., Nguyen, T.T., 2011. Fetal alcohol spectrum disorders: neuropsychological and behavioral features. Neuropsychology Review 21, 81–101.
Mattson, S.N., Roesch, S.C., Glass, L., Deweese, B.N., Coles, C.D., Kable, J.A., May, P.A., Kalberg, W.O., Sowell, E.R., Adnams, C.M., Jones, K.L., Riley, E.P., 2013. Further development of a neurobehavioral profile of fetal alcohol spectrum disorders. Alcoholism Clinical and Experimental Research 37, 517–528.
Mick, E., Biederman, J., Faraone, S.V., Sayer, J., Kleinman, S., 2002. Case-control study of attention-deficit hyperactivity d isorder and maternal smoking, alcohol use, and drug use during pregnancy. Journal of the American Academy of Child and Adolescent Psychiatry 41, 378–385.
Milberger, S., Biederman, J., Faraone, S.V., Jones, J., 1998. Further evidence of an association between maternal smoking during pregnancy and attention deficit hyperactivity disorder: findings from a high-risk sample of siblings. Journal of Clinical Child Psychology 27, 352–358.
Montiel-Nava, C., Peña, J.A., López, M., Salas, M., Zurga, J.R., Montiel-Barbero, I., Pirela, D., Cardozo, J.J., 2002. Estimations of the prevalence of attention deficit hyperactivity disorder in Marabino children. Nature Reviews Neurology 35, 1019–1024.
Needleman, H.L., 1982. Lead and impaired abilities. Developmental Medicine and Child Neurology 24, 196–198.
Obel, C., Linnet, K.M., Henriksen, T.B., Rodriguez, A., Jarvelin, M.R., Kotimaa, A., Moilanen, I., Ebeling, H., Bilenberg, N., Taanila, A., Ye, G., Olsen, J., 2009. Smoking during pregnancy and hyperactivity–inattention in the offspring-comparing results from three Nordic cohorts. International Journal of Epidemiology 38, 698–705.
Olney, J.W., Ishimaru, M.J., Bittigau, P., Ikonomiduo, C., 2000. Ethanol-induced apoptotic neurodegeneration in the developing brain. Apoptosis 5, 515–521.
Pyo, K.S., Park, S.H., Kim, S.H., Cho, Y.R., Kim, H.R., Moon, K.R., 2001. The Prevalence of Attention Deficit Hyperactivity Disorder in Urban Elementary School Children. Department of Psychiatry, Chosun University, Gwangju.
Rauh, V.A., Whyatt, R.M., Garfinkel, R., Andrews, H., Hoepner, L., Reyes, A., Diaz, D., Camann, D., Perera, F.P., 2004. Developmental effects of exposure to environmental tobacco smoke and material hardship among inner-city children. Neurotoxicology and Teratology 26, 373–385.
Roza, S.J., Verhulst, F.C., Jaddoe, V.W., Steegers, E.A., Mackenbach, J.P., Hofman, A., Tiemeier, H., 2009. Maternal smoking during pregnancy and child behaviour problems: the Generation R Study. International Journal of Epidemiology 38, 680–689.
Skounti, M., Philalithis, A., Galanakis, E., 2007. Variations in prevalence of attention deficit hyperactivity disorder worldwide. European Journal of Pediatrics 166, 117–123.
Slotkin, T.A., Lappi, S.E., Seidler, F.J., 1993. Impact of fetal nicotine exposure on development of rat brain regions: critical sensitive periods or effects of withdrawal? Brain Research Bulletin 31, 319–328.
Sowell, E.R., Jernigan, T.L., Mattson, S.N., Riley, E.P., Sobel, D.F., Jones, K.L., 1996. Abnormal development of the cerebellar vermis in children prenatally exposed to alcohol: size reduction in lobules I–V. Alcoholism: Clinical and Experimental Research 20, 31–34.
Sprich-Buckminster, S., Biederman, J., Milberger, S., Faraone, S.V., Lehman, B.K., 1993. Are perinatal complications relevant to the manifestation of ADD? Issues of comorbidity and familiarity. Journal of the American Academy of Child and Adolescent Psychiatry 32, 1032–1037.
Streissguth, A.P., Bookstein, F.L., Sampson, P.D., Barr, H.M., 1989. Neurobehavioral effects of prenatal alcohol: part III. PLS analyses of neuropsychologic tests. Neurotoxicology and Teratology 11, 493–507.
Streissguth, A.P., Barr, H.M., Sampson, P.D., 1990. Moderate prenatal alcohol exposure: effects on child IQ and learning problems at age 7 1/2 years. Alcoholism: Clinical and Experimental Research 14, 662–669.
Streissguth, A.P., Barr, H.M., Sampson, P.D., Bookstein, F.L., 1994. Prenatal alcohol and offspring development: the first fourteen years. Drug Alcohol Dependence 36, 89–99.
Sulik, K.K., Johnston, M.C., 1983. Sequence of developmental alterations following acute ethanol exposure in mice: craniofacial features of the fetal alcohol syndrome. The American Journal of Anatomy 166, 257–269.
Suzuki, K., Minei, L.J., Johnson, E.E., 1980. Effect of nicotine upon uterine blood flow in the pregnant rhesus monkey. American Journal of Obstetrics and Gynecology 136, 1009–1013.
Thapar, A., Fowler, T., Rice, F., Scourfield, J., van den Bree, M., Thomas, H., Harold, G., Hay, D., 2003. Maternal smoking during pregnancy and attention deficit hyperactivity disorder symptoms in offspring. American Journal of Psychiatry 160, 1985–1989.
Vaillant, G.E., Schnurr, P.P., Baron, J.A., Geber, P.D., 1991. A prospective study of the effects of cigarette smoking and alcohol abuse on mortality. Journal of General Internal Medicine 6, 299–304.
Wolraich, M.L., Hannah, J.N., Pinnock, T.Y., Baumgaertel, A., Brown, J., 1996. Comparison of diagnostic criteria for attention-deficit hyperactivity disorder in a county-wide sample. Journal of the American Academy of Child and Adolescent Psychiatry 35, 319–324.
Yoshimoto, K., McBride, W.J., Lumeng, L., Li, T.K., 1992. Alcohol stimulates the release of dopamine and serotonin in the nucleus accumbens. Alcohol 9, 17–22.