Tabac, grossesse et maladies de l’esprit

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Research article

Smoking and mental illness: results from population surveys in Australia and the United States
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Conclusion: Mental illness is associated with both higher rates of smoking and higher levels of smoking among smokers. Further, a significant proportion of smokers have mental illness. Strategies that address smoking in mental illness, and mental illness among smokers would seem to be important directions for tobacco control. As the majority of smokers with mental illness are not in contact with mental health services for their condition, strategies to address mental illness should be included as part of population health-based mental health and tobacco control efforts.
Chronic exposure to cigarette smoke during gestation results in altered cholinesterase enzyme activity and behavioral deficits in adult rat offspring: Potential relevance to schizophrenia


induced increase of locomotor activity. Additionally, it was observed increase of acetylcholinesterase and butyrylcholinesterase activity in the brain and serum, respectively. We demonstrated that animals exposed to cigarettes in the prenatal period had increased the risk for psychotic symptoms in adulthood. This also occurs in a dose-dependent manner. These changes provoke molecular events that are not
Development/Plasticity/Repair

Prenatal Nicotine Exposure Mouse Model Showing Hyperactivity, Reduced Cingulate Cortex Volume, Reduced Dopamine Turnover, and Responsiveness to Oral Methylphenidate Treatment

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Cigarette smoking, nicotine replacement therapy, and smokeless tobacco use during pregnancy are associated with cognitive disabilities later in life in children exposed prenatally to nicotine. The disabilities include attention deficit hyperactivity disorder (ADHD) and conduct disorder. However, the structural and neurochemical bases of these cognitive deficits remain unclear. Using a mouse model we show that prenatal nicotine exposure produces hyperactivity, selective decreases in cingulate cortical volume, and radial thickness, as well as decreased dopamine turnover in the frontal cortex. The hyperactivity occurs in both male and female offspring and peaks during the “active” or dark phase of the light/dark cycle. These features of the mouse model closely parallel the human ADHD phenotype, whether or not the ADHD is associated with prenatal nicotine exposure. A single oral, but not intraperitoneal, administration of a therapeutic equivalent dose (0.75 mg/kg) of methylphenidate decreases the hyperactivity and increases the dopamine turnover in the frontal cortex of the prenatally nicotine exposed mice, once again paralleling the therapeutic effects of this compound in ADHD subjects. Collectively, our data suggest that the prenatal nicotine exposure mouse model has striking parallels to the ADHD phenotype not only in behavioral, neuroanatomical, and neurochemical features, but also with respect to responsiveness of the behavioral phenotype to methylphenidate treatment. The behavioral, neurochemical, and anatomical biomarkers in the mouse model could be valuable for evaluating new therapies for ADHD and mechanistic investigations into its etiology.
Abstract
Many epidemiological studies support a relationship between maternal smoking during pregnancy and adverse neurobehavioral effects later in life. Prenatal exposure to tobacco seems to increase the risks for cognitive deficits, attention deficit/hyperactivity disorder, conduct disorder, criminality in adulthood and a predisposition in the offspring to start smoking and alcohol abuse. Nicotine
Does Prenatal Nicotine Exposure Sensitize the Brain to Nicotine-Induced Neurotoxicity in Adolescence?

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Offspring of women who smoke during pregnancy are themselves more likely to take up smoking in adolescence. We evaluated neurotoxicant effects of prenatal and adolescent nicotine exposure in developing rats to evaluate whether these contribute to a biological basis for this relationship. Rats were given nicotine or vehicle throughout pregnancy and the offspring then again received nicotine or vehicle during adolescence (postnatal days PND30–47.5); this regimen reproduces the plasma nicotine levels found in smokers. Indices of neural cell number (DNA concentration and content), cell size (protein/DNA ratio), and cell membrane surface area (membrane/total protein) were then evaluated in brain regions during adolescent nicotine administration (PND45) and up to 1 month post-treatment. By itself, prenatal nicotine administration produced cellular alterations that persisted into adolescence, characterized by net cell losses in the midbrain and to a lesser extent, in the cerebral cortex, with corresponding elevations in the membrane/total protein ratio. The hippocampus showed a unique response, with increased DNA content and regional enlargement. Adolescent nicotine treatment alone had similar, albeit smaller effects, but also showed sex-dependence, with effects on protein biomarkers preferential to females. When animals exposed to nicotine prenatally were then given nicotine in adolescence, the net outcome was worsened, largely representing summation of the two individual effects. Our results indicate that prenatal nicotine exposure alters parameters of cell development leading into adolescence, where the effects add to those elicited directly by adolescent nicotine; neurotoxicant actions may thus contribute to the association between maternal smoking and subsequent smoking in the offspring.

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Tabac, Grossesse, troubles de l’esprit


Secondhand Smoke Exposure and Depressive Symptoms

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Abstract

Objective—To evaluate the association between secondhand smoke (SHS) exposure and depression. Tobacco smoking and depression are strongly associated, but the possible effects of SHS have not been evaluated.

Methods—The 2005 to 2006 National Health and Nutrition Examination Survey (NHANES) is a cross-sectional sample of the noninstitutionalized civilian U.S. population. SHS exposure was measured in adults aged ≥20 years by serum cotinine and depressive symptoms by the Patient Health Questionnaire. Zero-inflated Poisson regression analyses were completed with adjustment for survey design and potential confounders.

Results—Serum cotinine-documented SHS exposure was positively associated with depressive symptoms in never-smokers, even after adjustment for age, race/ethnicity, gender, education, alcohol consumption, and medical comorbidities. The association between SHS exposure and depressive symptoms did not vary by gender, nor was there an association between SHS smoke exposure and depressive symptoms in former smokers.

Conclusions—Findings from the present study suggest that SHS exposure is positively associated with depressive symptoms in never-smokers and highlight the need for further research to establish the mechanisms of association.
**Conclusion:** Evidence from longitudinal studies suggests that the association between smoking and depression is bidirectional. To better estimate these effects, future research should consider the potential utility of: (a) shorter intervals between surveys with longer follow-up time, (b) more accurate measurement of depression, and (c) adequate control of confounding.
Prenatal tobacco exposure: is it a risk factor for early tobacco experimentation?

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Few studies have considered the etiological role of the fetal environment on the offspring's substance use. This prospective study examines the relations between the mother's prenatal and current smoking and the offspring's smoking experimentation. A low SES birth cohort of 589 10-year-olds, who have been followed since their gestation, completed a self-report questionnaire about their substance use. Half were female, and 52% were African-American. Detailed data on exposure to tobacco and other substances in the prenatal and postnatal periods were collected from the mothers. During pregnancy, 52.6% of the mothers were smokers; 59.7% were smokers when their children were 10. Six percent of the children (37/589) reported ever smoking cigarettes, 3% had had one full alcoholic drink, and none had started to use other drugs. Maternal smoking during pregnancy was significantly associated with an increased risk of the child's tobacco experimentation. Offspring exposed to more than ½ pack per day during gestation had a 5.5-fold increased risk for early experimentation. Structural equation modeling showed that prenatal tobacco exposure had a direct and significant effect on the child's smoking and that maternal current smoking was not significant. Prenatal tobacco exposure also predicted child anxiety/depression and externalizing behaviors, and these outcomes affected child smoking through the mediating effect of peer tobacco use.
Association of Tobacco and Lead Exposures With Attention-Deficit/Hyperactivity Disorder

**WHAT'S KNOWN ON THIS SUBJECT:** Prenatal tobacco and childhood lead exposures have been associated with hyperactivity and inattentive symptoms, but little is known about their independent and potential combined effects on attention-deficit/hyperactivity disorder, as defined with current diagnostic criteria.

**WHAT THIS STUDY ADDS:** This study is the first to determine the independent effects of tobacco and lead exposures on ADHD in a nationally representative sample of US children using DSM-IV criteria for outcome assessment, and provides the first estimate of the joint effects of these common toxicants on ADHD.

**CONCLUSIONS:** Prenatal tobacco and childhood lead exposures are associated with ADHD in US children, especially among those with both exposures. Reduction of these common toxicant exposures may be an important avenue for ADHD prevention. *Pediatrics* 2009;124:e1054–e1063

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**KEY WORDS**

attention deficit/hyperactivity disorder, lead exposure, tobacco exposure, toxicant interactions, joint effects
Prenatal Smoking Exposure and the Risk of Psychiatric Morbidity Into Young Adulthood

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Context: Prenatal smoking exposure modulates brain development, which may deviate mental development of the offspring.

Objective: To study the effects of prenatal smoking exposure on psychiatric morbidity and mortality among Finnish young adults by means of population-based longitudinal register data.

Design: Information on maternal smoking as reported by the mothers (0, <10, or >10 cigarettes a day) and other background factors (maternal age and parity and child’s sex, gestational age, birth weight, and 5-minute Apgar score) was derived from the Finnish Medical Birth Register. Information on children’s psychiatric diagnoses related to outpatient visits (1998-2007), children’s inpatient care (1987-2007), and mothers’ psychiatric inpatient care (1969-1989) was obtained from the Finnish Hospital Discharge Register. Information on deaths and their causes for the children (1987-2007) was received from the Cause-of-Death Register.

Setting: Population-based study of all singletons born in Finland from 1987 to 1989 with information on prenatal smoking exposure.

Patients: The source population included all singleton births in Finland from January 1, 1987, through December 31, 1989 (n = 175,869), excluding children with major congenital anomalies (3.1%) and children who died during the first week of life (0.3%).

Main Outcome Measures: Psychiatric morbidity and mortality.

Results: The prevalence of maternal smoking was 15.3%. The risk of psychiatric morbidity was significantly higher in the exposed children than in the unexposed children. Among the offspring of mothers who smoked fewer than 10 cigarettes a day, 21.0% had any psychiatric diagnoses (adjusted odds ratio [OR], 1.53 [95% confidence interval (CI), 1.47-1.60]) compared with 24.7% among those of mothers who smoked more than 10 cigarettes a day (1.83 [1.74-1.96]) and 13.7% in the unexposed children (the reference group). The risk was significantly increased for most of the psychiatric diagnoses. The strongest effects were in psychiatric disorders due to psychoactive substance use and in behavioral and emotional disorders. The risk of mortality was significantly higher in children exposed to more than 10 cigarettes a day (OR, 1.69 [95% CI, 1.31-2.19]) compared with unexposed children.

Conclusion: Prenatal smoking exposure is associated with an increased risk of psychiatric morbidity, whereas prenatal exposure to more than 10 cigarettes a day increases the risk of mortality in childhood, adolescence, and young adulthood.

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Figure 2
Effect sizes and 95% confidence intervals reported in six longitudinal studies on smoking predicting depression in adolescents.
Figure 3
Effect sizes and 95% confidence intervals reported in 12 longitudinal studies on depression predicting smoking in adolescents.
• Conclusions
  – Importance du repérage et de la prise en charge du tabagisme chez femme enceinte
  – Repérage des troubles psy chez la femme enceinte