Prospective associations between early long-term household tobacco smoke exposure and antisocial behaviour in later childhood

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ABSTRACT

Background Using a prospective birth cohort design, we estimate the relative contribution of long-term postnatal nicotine exposure from 17 to 86 months on children's subsequent antisocial behaviour by the end of fourth grade.

Method Parents reported the amount of household smoke exposure (at 17, 41, 65 and 86 months) for all 2055 children from the Quebec Longitudinal Study of Child Development. Main outcome measures include teacher- and child-reported antisocial and physically aggressive behaviour (at 121 months).

Results In terms of prevalence, 58% of parents reported that their children were never exposed to secondhand smoke in the home, while 34% and 8% of parents reported transient and continuous levels of secondhand smoke, respectively. When compared with never exposed children, children exposed to continuous secondhand smoke scored higher on self-reported aggressive behaviour and teacher-rated antisocial behaviour in fourth grade, B=0.336 (95% CI 0.155 to 0.517) and B=0.319 (95% CI 0.107 to 0.531), respectively. Similarly, children exposed to transient levels of secondhand smoke scored higher on aggressive and antisocial behaviour, B=0.714 (95% CI 0.456 to 0.972) and B=0.566 (95% CI 0.260 to 0.872), respectively.

Conclusions The observed prevalence is concordant with worldwide estimates of children's exposure to secondhand smoke. In comparison with their never exposed peers, children continuously and intermittently exposed to secondhand smoke in childhood showed an increased propensity toward physical aggression and antisocial behaviour by the end of fourth grade. We found no evidence of dose-dependence.

Environmental sources of tobacco smoke represent the most passive and preventable cause of disease and disability.[1] Secondhand smoke comprises 85% sidestream smoke emanated from a burning cigarette and 15% inhaled and then exhaled mainstream smoke. Because it contains a higher concentration of many dispersed respirable pollutants over a longer exposure period, sidestream smoke is considered more toxic than its mainstream counterpart.[2] Approximately 40% of children worldwide are exposed to toxic pollutants associated with secondhand smoke in their own homes.[3] Swelling current and future public health cost estimates is a putative link between environmental tobacco smoke and children's behaviour problems.[4]

Childhood antisocial behaviour, which violates major age-appropriate societal norms and the basic rights of others, is linked with social disruption in the school, neighbourhood and home.[5] It is relatively common among school-aged children and, if it persists across childhood, forecasts unhealthy lifestyle, mental health, and social and economic risks.[6] Long-term propensities are maximised when clinical symptoms persist by or before age 10.[7] As symptoms increase, so do the risks for adult substance use disorders, antisocial personality and high school dropout.[8] Aggression is a common clinical feature in childhood, followed by a peak in rule breaking in adolescence; both of which show developmental continuity.[5]

The National Health and Nutrition Examination Survey 2001–2004, a representative cross-sectional data set of American youth from ages 8 to 15, comprises assessments using the Diagnostic Interview Schedule for Children and serum metabolite measures of cotinine. These represent the most reliable measures of both conduct disorder and recent secondhand smoke exposure, respectively. With this data set, Braun *et al*[9] examined the cross-sectional relationship between serum cotinine levels and conduct disorder diagnosis. Secondhand smoke exposure predicted 9.15-fold greater odds of achieving DSM-IV criteria for conduct disorder. Using a dimensional perspective, Bandiera *et al*[10] found that increases in tobacco exposure predicted increases in the symptom count ratio for conduct disorder. Both studies included a number of significant control variables, including sociodemographic and racial factors, birth weight, and retrospectively reported prenatal tobacco use. Braun *et al*[9] also controlled for lead exposure. These findings were replicated with 8-year-old from the community-based, Scottish Health Survey, using both cotinine and child self-report measures of conduct disorder.[11] Clearly, these two data sets are advantaged by cotinine measures. However, they remain limited by their cross-sectional nature which precludes allusion to causality. Given that randomised experiments are not possible, carefully designed longitudinal research offers a natural experiment to investigate this link.

First generation birth cohort studies underscore the value of controlling for gestational tobacco smoke exposure and other family factors and suggest greater risks for externalising behaviour outcomes compared with internalising outcomes.[12–14] More than a decade later, using the German Infant Nutrition Intervention birth cohort, Rückinger *et al*[15] found that children exposed to both prenatal and postnatal tobacco smoke showed almost twice the estimated risk of conduct problems at age 10. This German study used parental

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(mostly mother) self-report follow-up data from the first to the sixth year and then retrospectively reported data at the age 10 follow-up (for ages 7–9). Children were considered exposed to tobacco smoke if parents reported 5 out of 9 years of smoking. The chief strength of this study was the age at which the outcome was measured. It also controlled for a number of significant factors associated with socioeconomic status, maternal age and screen time by children. Since mothers reported prenatal smoking, it was also possible to eliminate the confounding long-term influence of gestational exposure.

Past longitudinal birth cohort studies with prenatal and postnatal data are not without methodological challenges. First, birth cohort studies have generally asked mothers whether they smoked or not and how much at each follow-up, rather than asking whether someone smoked in the home where young children live and play. Second, none of the longitudinal studies have compensated for attrition bias, which can seriously challenge the interpretation of results even in the presence of sociodemographic and socioeconomic control variables. Participants who discontinue their participation in longitudinal studies tend to be comparably more socially disadvantaged or at-risk than those who continue as participants.[16] Third, most studies (with few exceptions[12 13]) have not controlled for parental dispositions toward violating social rules and the rights of others. It could be that parents with a history of antisocial behaviour may smoke around their children but that the antisocial parental characteristics account for the observed relation between secondhand smoke and child deviance. Two compelling investigations find that the link between gestational exposure and later antisocial behaviour is likely accounted for by a confluence of family background and genetic factors.[16 17] Accordingly, this finding underscores the potential value of prenatal smoking as a control variable because it might also act as a surrogate for other co-occurring family background and genetic factors in postnatal secondhand smoke investigations. Based on experimental animal research[18 19] and non-experimental prospective longitudinal research in humans,[13 14] it is also plausible that above and beyond whether parents are antisocial or not, being exposed to tobacco smoke during early childhood may induce risk for more developmental neurotoxicity. Consequently, because of the distinct importance of early childhood brain development in humans,[20] earlier exposure to secondhand smoke might be more noxious than later exposure.[21] Few studies have considered a prolonged period of secondhand smoke exposure in childhood with fewer studies projecting its estimates to several years later. Studies of developmental psychopathology would benefit from examining the influence of exposure during the critical periods in early childhood brain development. For these reasons, further prospective investigation is warranted.

Hence, the objective of this study is to estimate a prospective association between long-term household nicotine exposure from infancy to the end of first grade, and children's subsequent teacher-reported antisocial behaviour and child-reported aggressive behaviour at the end of fourth grade. We expect to find a modest dose–response relationship once adjustments for possible confounders and corrections for attrition bias are implemented.

METHODS

Participants

Analyses were conducted using data from the Québec Longitudinal Study of Child Development. This sample originates from a randomly selected stratified sample of 2837 infants born between 1997 and 1998 in Quebec, Canada. At the inception of the study, 93 children were deemed ineligible and 172 were untraceable due to incorrect coordinates. Of the 2572 remaining children, 14 were unreachable and 438 refused participation. Beginning at 5 months post partum, 2055 infants were followed up annually for the early childhood phase, representing 82% of the eligible target population. Of these, 39% were firstborn. Baseline measures were taken when children were 5 months old. Follow-up occurred at age 10, in the spring of fourth grade. For each follow-up, informed consent was obtained from parents. During the school-aged phase, teachers and children also gave consent. Participants in this IRB approved study were included in this study if they had complete data on maternal reports of environmental smoke exposure from the baseline measures at 5 months post partum (n=2055).

Predictor variable (average child age 17, 41, 65 and 86 months)

Household exposure to tobacco smoke

Following parental consent at each follow-up, mothers were asked, 'Does one or other of the parents or another person smoke in the house?' Based on their responses, children were categorised into one of three environmental tobacco smoke groups: never exposed; transient environmental smoke (exposure at one to three time points); and continuous environmental smoke (exposure at all four time points).

Outcome measures (average child age 121 months)

Antisocial behaviour

Teachers completed the antisocial behaviour factor of the Social Behaviour Questionnaire[22 23] (six items: Bragged about accomplishments and antisocial behavior; Used or conned others; Did not seem to feel guilty after misbehaving; Engaged in risky or dangerous behaviours; Was unconcerned about the feelings of others; and Did not keep promises, $\alpha=0.80$). The items were scored as follows: never or not true (0), sometimes or somewhat true (1), or often or very true (2). Scores were rescaled from 0 to 10.

Physical aggression

To corroborate the teacher measures, children were asked about their own physically aggressive behaviour toward others using the age 10 child self-report version of Social Behaviour Questionnaire. Items asked children whether they: fight often; physically attack other; and hit, bite or kick other children ($\alpha=0.78$).[22–24] The items were scored as follows: never or not true (0), sometimes or somewhat true (1), or often or very true (2). Scores were rescaled from 0 to 10.

Gestational and early childhood control variables

Weight for gestational age was derived from birth records and standardised by gender and months of pregnancy using Canadian norms. Children were coded as either 0 (normal weight) or 1 (below the 10th percentile). When children were 5 months old, mothers self-reported their smoking behaviour during pregnancy. Mothers responded to two questions 'Did you smoke during pregnancy?' and 'How many cigarettes did you smoke while pregnant?' Responses were then coded as (smoked=1 or never smoked=0). Approximately 25% of mothers reported smoking during pregnancy, which is consistent with prevalence rates found in other studies and is also correlated with physiological measures.[24 25] Mothers also self-reported gestational consumption of alcohol (coded as 0=no alcohol consumption or 1=alcohol consumption) and illicit drugs (coded as 0=no drug consumption or 1=drug consumption).

At the 5-month assessment, parents reported income using an 8-point scale (from 1=less than $10 000 to 9=over $C80 000), the number of siblings present to account for birth order, maternal age at child birth and maternal education (finishing high school=1 and not=0). Maternal depressive symptoms over the previous week were assessed using a 12-item abbreviated version of the Center for Epidemiological Studies Depression Scale, with responses ranging on a 4-point scale (α=0.78).[26] Parental and family characteristics were also measured in early childhood. To assess parental history of antisocial behaviour, both parents completed a questionnaire. The items assessed the extent to which parents had engaged in antisocial behaviour during adolescence and adulthood and were derived from the NIMH-Diagnostic Interview Schedule. Items were scored as (0=no, 1=yes) and were summed to create a parental score. Adolescent items included: Starting fights; Theft, involvement with youth protection or police; Expulsion or suspension from school; Truancy; and Running away from home. Adult items included: Arrests; Being fired from a job; Trouble at work, with family, or with the police due to drug or alcohol abuse; Starting fights (fathers only); and Hitting or throwing things at the spouse or partner (mothers only). Family configuration (two parent = 1 and not = 0) was measured at 5 months and family functioning was measured at 17 months (based on 12 items designed to measure family communication, problem solving, control of disruptive behavior, and demonstration of affection, α = .98).[26 27] Maternal hostile parenting was also observed. At the 17-month assessment, trained examiners indicated the extent to which mother: Screamed at child; Seemed disturbed by the child; Hit the child; and Scolded or put down the child. Items were rated on a 5-point scale where higher scores reflect more hostile parenting (α=0.75).[23] At 41 months, mothers also reported child exposure to domestic aggression (by answering the following question: How often does your child see adults or teenagers in your house physically fighting, hitting or otherwise trying to hurt others?').[23]

Data analysis

We aimed to examine the relationship between child exposure to environmental smoke from 17 to 86 months ($ENSM_i$) and two measures of child antisocial behaviour in fourth grade ($ANTI_{i4thgrade}$). In order to reduce the possibility of competing explanations and minimise the possibility of omitted variable bias, our intent was to account for variables that are likely to be statistically correlated with either secondhand smoke or childhood antisocial behaviour and thus represent potential candidates as control variables. These include: (1) Individual child factors ($CHILD_i$) available in the data set that may act as potential confounders (including: sex; weight per gestational age; gestational exposure to tobacco; illicit drugs and alcohol; exposure to aggression; and age at grade four assessment) and (2) Family factors (FAM_i) available in the data set that may act as potential confounders (including: parent antisocial behaviour; presence of domestic violence; number of siblings; maternal depressive symptoms, age, hostility and level of education; and family income, configuration and functioning). Our position is that gestational smoking, as a candidate control variable, may afford a better estimate of the unique contribution of postnatal household smoke by accounting not only for its own long-term influence but as a proxy for other confounding variables. Our initial intent bears upon an adjusted model, where: $ANTI_{i4thgrade}$ represents child deviance in terms of teacher-reported antisocial and self-reported aggressive behaviour; $ENSM_i$ represents early childhood exposure to environmental smoke; and FAM_i and $CHILD_i$ represent family and child control variables for each individual $child_i$. Finally, a_1 and e_i represent the constant and the stochastic error term, respectively.

$$ANTI_{i4thgrade} = a1 + \beta 1\, ENSM_i + \gamma 1\, CHILD_i + \gamma 2\, FAM_i + e_i$$

The analyses compared children who were not exposed to environmental smoke with children exposed to transient and consistent exposure. Maximum likelihood regression estimation (in Mplus) was implemented according to the above equation. Missing data on outcome measures and covariates were adjusted in the regression analyses using full information maximum likelihood methods in Mplus.[28] In the final analyses, a more parsimonious model that omits non-significant covariates was estimated and is reported in the Results section.

RESULTS

The descriptive statistics for independent, dependent and control variables are reported in table 1. In the present sample, 58% of parents reported that their children were never exposed to secondhand smoke in the home, while 34% and 8% of parents reported transient and continuous levels of secondhand smoke, respectively.

Table 2 reports the unadjusted coefficients for the relationship between transient and continuous secondhand smoke exposure and later antisocial and aggressive behaviour. Compared with never exposed children, those who were exposed to continuous and transient secondhand smoke during childhood both scored 11% of an SD higher than never exposed children on self-reported physical aggression. Children exposed to transient and continuous amounts of secondhand smoke in turn scored 11%

Table 1 Descriptive statistics for household exposure to tobacco smoke, antisocial behaviour outcomes and control variables

Predictor			
Never exposed (N=1672)	1199	(58%)	
Transient exposure (N=360)	689	(34%)	
Continuous exposure (N=23)	167	(8%)	
Gestational smoke (yes=1)	25%	0	1
Sex (boys=1)	51% boys	0	1
	Mean (SD)	**Min**	**Max**
Outcome variable			
Antisocial behaviour	1.08 (1.78)	0	10
Aggressive behaviour	1.09 (1.80)	0	10
Control variables			
Weight for gestational age	0.11 (413)	0	2
Gestational alcohol	0.36 (0.48)	0	1
Gestational illicit drugs	0.01 (0.11)	0	1
Maternal depression (5 months)	1.39 (1.33)	0	9.23
Parent antisocial (5 months)	0.47 (0.84)	0	6
Number of siblings (5 months)	0.83 (0.91)	0	4
Family functioning (5 months)	1.69 (1.45)	0	10
Maternal age (5 months)	29.32 (5.21)	16.40	44.50
Maternal education (5 months)	0.80 (0.40)	0	1
Family configuration (5 months)	0.81 (0.39)	0	1
Family income (5 months)	5.82 (2.28)	1	9
Exposure to fighting (41 months)	0.06 (0.24)	0	1
Hostile parenting (17 months)	1.05 (1.20)	0	8.83
Exact age in months at follow-up	121.78 (3.10)	116	128

Table 2 Unadjusted and adjusted regression coefficients (unstandardised) for the prospective association between early childhood secondhand smoke exposure and teacher-rated antisocial and self-reported aggressive behaviour by the end of fourth grade

	Physical aggression (unadjusted)		Physical aggression (adjusted)	
	B	(95% CI)	B	(95% CI)
Continuous exposure	0.423	(0.241 to 0.604)***	0.336	(0.155 to 0.517)**
Transient exposure	0.794	(0.531 to 1.108)***	0.714	(0.456 to 0.972)***
R square	0.021			0.104
	Antisocial behaviour (unadjusted)		**Antisocial behaviour (adjusted)**	
	B	(95% CI)	B	(95% CI)
Continuous exposure	0.406	(0.193 to 0.619)**	0.319	(0.107 to 0.531)*
Transient exposure	0.693	(0.386 to 1.00)***	0.566	(0.260 to 0.872)**
R square	0.018			0.076

Omitted group=Never exposed to household tobacco smoke. The regression for antisocial behaviour is adjusted for sex, parental antisocial behaviour, number of siblings and maternal age. The regression for physical aggression is adjusted for sex, maternal education and exposure to physical fighting in the home. *p≤0.05, **p≤0.01 and ***p≤0.001.

and 12% of an SD higher than never exposed children on teacher-reported antisocial behaviour. The explained variance in both models is quite modest, suggesting that factors, exogenous to our specified model, play a role in the prospective associations observed.

The adjusted coefficients for the child and family control variables are also included in table 2 for relative comparisons of effect sizes. Children exposed to continuous secondhand smoke scored 11% and 9% of an SD higher on aggressive behaviour and antisocial behaviour, respectively, when compared with never exposed children. Similarly, children exposed to transient levels of secondhand smoke scored 8% and 9% of an SD higher on physically aggressive and antisocial behaviour, respectively, in the fourth grade. The explained variance is this model remained small.

Comment

In 2006, the US Surgeon General published a comprehensive piece which provides a clear health advisory to actively avoid contexts with cigarette smoke.[1] Because secondhand tobacco smoke exposure contains a higher concentration of many dispersed respirable pollutants over a longer exposure period, it is considered more toxic than its mainstream exhaled counterpart.[2] Child exposure to tobacco smoke in domestic settings has become an international concern.[3]

Approximately 42% of children in this population-based birth cohort lived with at least one person who smoked in the house at some point during the six early childhood years that span the study reported here. Of these, the majority was intermittently exposed to secondhand smoke and a much smaller percentage was continuously exposed to secondhand smoke. In general, the results suggest that living with one or more smokers during early childhood poses detectable risks for later antisocial behaviour. This behavioural propensity ultimately has repercussions for the life course trajectories of individuals and their ability to make a productive contribution to society.

Secondhand household smoke exposure made a unique contribution in predicting teacher-rated antisocial behaviour and self-reported physical aggression at the end of fourth grade. Both intermittent and continuous exposure levels showed links with both forms of deviant behaviour. The most remarkable finding is that even low doses, indicating intermittent exposure at one to three time points between 17 and 86 months, yielded significant prospective associations with antisocial and aggressive behaviour. Interestingly, a dose–response relationship was not linked with either outcome. This study does support the US Surgeon General's position that there is no safe dosage level.[1 4]

Household smoke exposure remained influential despite the stringent intent to statistically control for a range of substantively important confounding variables. Gestational exposure to maternal smoking was not significantly related to the antisocial outcomes examined in this study. The inclusion of other statistically important sociodemographic and psychosocial confounding variables likely reduced its influence as a control variable.[16 17]

Our measure extended from infancy to age seven. The first 5 years of life are characterised by many critical moments in brain development.[29] These periods culminate in a sensitive period of growth and development of key cognitive, sensorimotor and socio-emotional skills between ages five and seven.[21] Such skills, which are based in the frontal lobe, eventually play a role in eventual academic and personal success.[20]

Both antisocial and aggressive behaviours are rooted in common neurobiological processes which, although not directly assessed here, remain relevant to our findings.[8] They offer an explanatory framework which suggests developmental mechanisms in the frontal lobe likely operate in the link between smoke exposure and child antisocial behaviour. First, randomised experiments using rhesus monkeys[18] and rodents[29] conclude that gestational and environmental exposure to nicotine evokes reduced cellular density and increased cell size in the frontal cortex and midbrain. In fact, these studies find that the effects of prenatal and postnatal exposure are not significantly different in regional selectivity, direction and magnitude of neurotoxic effects.[29] The chief strength of these findings is that they emanate from randomised animal experiments which preclude social confounders such as parental antisocial behaviour and low socioeconomic status. Second, there is enough evidence, from both human and animal models, which suggests that even low doses of nicotine disrupt cellular communication in the frontal lobe by extended activation of neuronal nicotinic acetylcholine receptors (nAChRs).[19] These are present in the developing human brain from the third trimester onward, and are meant to regulate important aspects of brain maturation during critical developmental periods such as gestation, early childhood and adolescence.[19 29] Extended activation of nAChRs, most often via environmental tobacco smoke (even at low doses),

may induce dysfunctional coordination between the thalamus and the sensory cortex which consequently dysregulates sensory processing over the long term.[19 30] Consequently, poor corticothalamic–thalamocortical coordination could negatively impact the modulation of neurotransmitters such as dopamine and serotonin.[30–32] Third, structural brain deficits might also play a role in sensory processing dysregulation.[33] In humans, prenatal tobacco smoke exposure predicts long-term decrements in regional volumes of cortical grey matter by adolescence[31] and white-matter integrity by young adulthood.[34] It also interferes with the development of the orbitofrontal cortex,[34] which is recruited for the processing of cues associated with reward, punishment and for the control of impulsive behaviour.[35] Postnatal smoke exposure predicts similar structural disturbances in the orbitofrontal regions recruited for social and emotional regulation in both adults and children.[36–38] These can influence misconstruals of reward-related information and lead to antisocial behaviours.[39] In addition to often misperceiving the intents of others as hostile, individuals who engage in antisocial behaviour are more likely to misconstrue rewards from the environment and often incorrectly process the absence of a reward, thus increasing the likelihood of aggression toward others.[38 40] A confluence of the above micro and macro processes likely reinforces defective sensory processing which, in turn, could influence negative perceptions of others and their intentions as hostile, leading to the habitual antisocial behaviour responses reported by both teachers and children's own self-ratings. Although these clinical interpretations are speculative, they are consistent with our epidemiological findings and integrate them within an established neuroscientific framework.

Indeed, the frontal lobe undergoes important growth and development from birth onward, until middle childhood, which covers the time window of secondhand smoke exposure examined in this study. Nevertheless, non-experimental studies like this one preclude definitive statements about causal mechanisms, especially when they involve neural mechanisms in living humans. As such, the non-experimental nature of this study represents a primary limitation of this study. Although we used diverse data sources and aimed to control influential confounders with reliable measures, the explained variance was low, indicating that our models suffered from omitted variable bias. A second important limitation is that we did not use a biomarker such as cotinine to accurately measure the amount of secondhand smoke more precisely. Our approach to measurement of secondhand smoke might not have been sensitive enough to detect a dose-dependent association. As it stands, our study suggests that even low levels of secondhand smoke influence later neurobehavioural development. The role of other contextual and individual variables providing competing explanations merits further investigation. A third limitation of this study is that it does not consider smoking in cars as another source of domestic pollution. Secondhand smoke in cars is 23 times more toxic than in a house; making it much more harmful for children given their higher relative ventilation needs compared with adults.[41–43]

Notwithstanding such limitations, we found a modest yet robust prospective association between secondhand smoke exposure and child antisocial behaviour even after correcting for attrition bias. Although prenatal smoking does have an influence on later child behaviour, the results of this study suggest that the postnatal period might be as important for the prevention of impaired neurobehavioural development and the promotion of an unpolluted domestic environment for children.[1 3 19]

What is already known on this subject?

Infants and young children account for the largest global disease burden associated with postnatal secondhand household smoke exposure, probably due to underdeveloped neurological, immune and respiro-circulatory systems. There is an increasingly robust association between tobacco smoke exposure and developmental psychopathology in children, adding to current and future disease burden estimates in public health. This longitudinal birth cohort study overcomes crucial methodological challenges such as attrition bias and confounding influences due to gestational smoke exposure, parental history of conduct disorder and family adversity variables.

What this study adds?

Postnatal household exposure to tobacco smoke during critical periods in childhood development presents important neurobiological risks that increase the chances of developmental psychopathology in youth, above and beyond important confounders. These may influence eventual impairments in social/occupational functioning which are costly to the individual, the entourage and society.

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