ORIGINAL RESEARCH

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Two Lifestyle Risks Intertwined: Parental Smoking Predicts Child Gambling Behavior at Age 12 Years

Abstract: Background. *Parental smoking can create a toxic environment for child development. A parental smoking lifestyle can predispose children to executive deficits, influencing precocious risk activities. Using a prospective birth cohort design, we examine the association between 2 lifestyle factors by estimating the relative contribution of long-term parental household smoking in predicting subsequent precocious child gambling behavior.* Method. *Parents reported on the amount of household smoke exposure from ages 1.5 to 7.5 years for children from the Quebec Longitudinal Study of Child Development. The main outcome measure was children's self-report of gambling behavior (at age 12 years).* Results. *Sixty percent of parents reported that their children were never exposed to secondhand smoke in the home, while 27% and 13% reported transient and continuous levels of secondhand smoke, respectively. Overall, 16% of children reported gambling participation. When compared with never-exposed children, children exposed to secondhand smoke had 18% more chances of having participated in gambling at age 12 years (odds ratio = 1.18; 95% CI = 1.080-1.293). These results are adjusted for competing explanations and possible individual and family confounders.* Conclusions. *Higher levels of early childhood household smoke exposure are associated with greater odds of reporting gambling participation at age 12 years, which is more than several years before it is normative youthful behavior. By connecting the neurotoxic influence of one lifestyle factor on another, we show a nontrivial link between 2 public health issues (smoke exposure and precocious gambling) associated with considerable individual and societal costs that are amenable to community information campaigns.*

Keywords: child behavior; developmental neurotoxicity; executive function; secondhand smoke; youth gambling

Although the prevalence of cigarette smoking as a lifestyle risk is on the decline, there remain a significant number of smokers.[1] Tobacco smoke has a major role in mortality and morbidity estimates of both smokers and their entourage.[2,3] Young children with parents who smoke in the home have little control over such parental lifestyle choices, and thus their secondhand smoke exposure. The American Academy

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of Pediatrics has summarized astounding medical costs attributed to children's secondhand smoke inhalation at more than $260 million per day in the United States alone.[4]

Secondhand smoke comprises 85% sidestream smoke (from the cigarette itself) and 15% inhaled-exhaled smoke into the environment.[5] Being an important source of nicotine, secondhand smoke also disperses more than 250 physiologically toxic chemicals into the breathing environment.[5-7] When children are exposed to smoke where they live and play, they are at risk for a number of short- and long-term health problems.[8] There are costs other than medical, in that children's cardiorespiratory systems are more vulnerable to environmental pollutants, which could also impede brain development.[3]

Secondhand smoke can be especially neurotoxic during early childhood brain development, when cellular communication and synaptogenesis are exuberant.[9,10] From birth to about age 5 years, multiple neurological systems consolidate in order to fulfill their vital biopsychosocial potential.[11] The anoxia from cardiorespiratory distress,[2] coupled with the harmful characteristics of secondhand smoke, predicts disruptions in cellular communication, structural development of brain tissue, and epigenetic mutations.[12] Neurotoxicity during critical periods in brain development could ultimately compromise the brain's executive system.[12] For example, experiments with animal models have shown that, during sensitive periods of brain growth, exposure to secondhand smoke has effects on structural and cellular development in the prefrontal cortex executive system, which manages working memory, impulse control, and mental flexibility skills.[13,14] These help to simultaneously focus and filter, monitor, and make and revise decisions during information processing.[15] Developmental consolidation of these will affect reward processing.

Pre- and postnatal exposure to secondhand smoke independently increase risks of childhood impulsivity, strongly associated with attention deficit/hyperactivity disorder.[12,16] Interestingly, early impulsivity is strongly linked with gambling behavior in later childhood.[17] Normative youth gambling participation is at age 17 years.[17,18] Precocious youth gambling behavior predicts disordered gambling in adulthood and the notion of developmental impulsivity in problematic and pathological gambling in adolescents and adults is firmly established in neuroimaging research.[18] Heightened impulsivity is accompanied by diminished prefrontal and anterior cingulate cortex functioning.[19] A persuasive test of developmental neurotoxicity would be to examine whether secondhand smoke exposure predicts behavior that is associated with impulsivity, such as early youth gambling behavior.

The existing longitudinal birth cohort studies with prenatal and postnatal secondhand smoke data are not without their own methodological challenges. First, smoking during pregnancy is not only a potential confound but also serves as a proxy for other stable family background and genetic characteristics which often go unmeasured in postnatal studies of secondhand smoke. This would especially apply in light of the predictive relationship between gestational smoking and later attention deficits in childhood.[20] Second, birth cohort studies typically have mother self-reported smoking status at each follow-up. As such, we do not know whether mothers or others smoked in the indoor living environment where children live and play. Third, birth cohort studies have had difficulty compensating for attrition bias at follow-up. This can further confound interpretations even when sociodemographic and socioeconomic confounds have been controlled. Fourth, it is plausible that parents with lifetime history of antisocial behavior may become smokers around their children and it is equally plausible that such antisocial parental characteristics ultimately account for any association found between household smoke and children's neurodevelopmental outcome factors.[21,22] Most secondhand smoke studies do not consider life-course antisocial predispositions in parents (exceptions, see Pagani and Fitzpatrick,[20] Maughan et al,[22] and Hermann et al[23]). Last, being exposed to secondhand smoke when brain development is exponential could induce its own independent risks for deregulation in the corticothalamic relay processes and deficits in structural growth and development. These may directly and indirectly influence the brain's executive system.[12] Few studies have examined the influence of household secondhand smoke exposure over a prolonged critical childhood period, and such exceptions have established links with antisocial behavior.[20,24] Antisocial behavior is strongly tied to impulsivity in later childhood,[25] just as gambling is tied to impulsivity in adulthood.[26] None have followed up several years later with precocious child gambling behavior as a practical indicator of neurodevelopmental disruption.

For the above reasons, further investigation of prospective links between early childhood exposure to a long-term neurotoxic environment, due to parental lifestyle choices, and a correlate of later child impulsivity is warranted. Using a longitudinal birth cohort design, the purpose of this study was to estimate the prospective association between household smoke exposure, from infancy to the end of first grade, and subsequent self-reported child gambling participation at the end of sixth grade, reflecting early involvement in a gateway behavior toward more gambling activity across development. It was expected that higher levels of early childhood household smoke exposure from ages 1.5 to 7.5 years would predict child gambling behavior at age 12 years.

Methods

Participants

This institutional review board–approved study was conducted using data from the Quebec Longitudinal Study

of Child Development (coordinated by the Institut de la Statistique du Québec), a birth cohort of 2837 newborns from the Canadian province of Quebec between 1997 and 1998. The sample was randomly selected and stratified by provincial region. Eighteen percent of the eligible participants were not retained in the sample due to different reasons: First Nation status made 93 of the children ineligible, 186 were untraceable, and at the 5-month baseline assessment participation was refused by parents for 438 of the children in the study. For the 2055 children left (82% of the original eligibility list) informed written parental consent was obtained for the annual follow-ups taking place during early childhood, and informed consent was obtained systematically from parents, teachers, and children for each biennial school age follow-up. Secondary analyses included all participants with complete data on tobacco smoke exposure from ages 1.5 to 7.5 years, gambling participation at age 12 years, and controls (n = 548).

Predictor: Childhood Household Smoke Exposure (Ages 1.5, 2.5, 3.5, 4.5, 5.5, 6.5, and 7.5 Years)

Mothers were asked, annually, "Does one or other of the parents or another person smoke in the house?," with 2 possible response choices (yes = 1, no = 0). Based on the answers obtained for each year, a continuous index, ranging from 0 to 7, was created and used in the analyses (mean = 1.84, SD = 2.66). Parent-reported household smoke exposure has reliably been used in other studies,[24,27,28] with one study using both parent-reports and serum cotinine showing consistent results in terms of effect sizes.[29]

Outcome: Early Gambling Participation (Age 12 Years)

In a semistructured interview in the spring of sixth grade, children were asked "In your lifetime, have you ever gambled, played games for money (eg, lotteries, scratch tickets, video poker, casino, cards, dice, bingo, betting on sports events)?" Based on the 2 possible response choices (yes = 1, no = 0), a dichotomous variable was produced and retained in its categorical form in the analyses. Self-reported measures of gambling behavior in children have been used in previous studies.[17]

Individual and Family Control Variables (From 5 Months to Age 10 Years)

Individual. Given that sex is a known risk factor for gambling problems, this variable, which was directly obtained from birth records, was included as a covariate (girls = 0, boys = 1). Weight for gestational age was also obtained at birth and dichotomized using Canadian norms (above the 10th percentile = 0, below the 10th percentile = 1). Given that inattention has often been associated to impulsivity and gambling,[17,30] it was included as a control variable in this study. At age 6 years, teachers were asked "Would you say that this child . . . (a) was easily distracted, had trouble sticking to any activity; (b) was unable to concentrate, could not pay attention for long; and (c) was inattentive?" The response choices for each item were never (=0), sometimes (=1), frequently (=2). A sum of these 3 items was computed to create a continuous score of inattention ranging from 0 to 6. Using the median (2.00) as the cutoff point, the sample was then divided into 2 subgroups (0 = below the 50th percentile, 1 = above the 50th percentile).

Family

At 5 months, mothers reported on their level of education (completed high school = 0, no high school diploma = 1), household income in 1997-1998 Canadian dollars ($30 000 or more = 0, $29 999 or less = 1), family configuration (two parents = 0, single parent = 1). Mothers were also asked whether they had smoked during pregnancy to create a variable for gestational smoke (not exposed = 0, exposed = 1). Parental antisocial behavior during adolescence and adulthood was assessed using the National Institute of Mental Health–Diagnostic Interview Schedule.[31] A composite score for both parents was computed. Maternal depressive symptoms were assessed with the Center for Epidemiological Studies Depression Scale–Short Form (Cronbach's α = .78).[32] Both parental mental health variables were assessed when the child was 5 months old. Family functioning was measured using a 12-item scale completed by mothers at 17 months (α = .98).[33] Several studies have shown that gambling behavior is often associated with parental gambling.[30,34] At the age 10 assessment, parents were asked whether they had participated in 11 different types of gambling activities over the past 12 months: lottery tickets; casino; bingo; cards for money; horse races, dogs, or other animals; played the stock or commodities markets; slot, poker, or other gambling machines; bowling, pool, golf or other games of skill for money; dice games for money; sports betting; or played another game for money. For each gambling activity, each parent was given a 1 for having participated or a 0 for not having participated. These response codes were summed to compute an index ranging from 0 to 11 for each parent. Then, both scores (mother and father) were averaged to create a parental gambling variable, which was used in its continuous form (mean = 1.37, SD = 0.99). In cases of missing data on one parent, the sole parent average was used.

Data Analytic Strategy

Using logistic regression, we aimed to examine the relationship between child exposure to environmental smoke from 17 to 86 months ($ENSM_i$) and child gambling behavior at the end of sixth grade ($CGMB_{iage12)}$. In order to reduce the possibility of competing explanations and minimize 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 youth gambling behavior and thus represent potential candidates as control variables. Potential confounders included (*a*) individual

child factors ($CHILD_i$) such as child sex, weight for gestational age, gestational exposure to tobacco, inattention and (*b*) family factors (FAM_i) such as family income, configuration, and functioning; maternal level of education and depressive symptoms; parental antisocial behavior and parental gambling. 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 $CGMB_{iage12}$ represents self-reported gambling behavior; $ENSM_i$ represents early childhood exposure to environmental smoke; and FAM_i and $CHILD_i$ represent family and child control variables, respectively, for each individual $child_i$. Finally, a_1 and e_i represent the constant and the stochastic error term, respectively.

$$CGMB_{iage12} = a_1 + \beta_1 ENSM_i + \gamma_1 CHILD_i + \gamma_2 FAM_i + e_i$$

A logistic regression analysis was conducted using SPSS software, in order to predict the probability for the individual to fall into one of the two categories of the dichotomous dependent variable (gambler vs nongambler) based on the independent variable (level of exposure to secondhand smoke) while controlling for the other variables included in the model.

Results

The data in this longitudinal study were obtained from a population-based sample, using multiple sources. Only 25% of the original cases (n = 2223) had total complete data on all 13 variables in this study (n = 548). The reported results are corrected for attrition bias. We compensated for incomplete data, missing at random, using SPSS multiple imputation, which generates probable values, thus creating several "complete" data sets which are then used to produce individual outputs that are then combined. The resulting pooled output estimates probable results had the original dataset been complete.[35]

Descriptive statistics of predictor, outcome, and control variables are reported in Table 1. More than half of children (60%) were never exposed to early childhood household smoke (score of 0), 27% were transiently exposed (score of 1 to 6), and 13% were continuously exposed (score of 7) from ages 1.5 to 7.5 years. As for the outcome variable, 84% of children had never gambled, as opposed to 16% reporting gambling participation at age 12 years. As reported in the Supplemental Web Appendix (Table 3), Preliminary analyses of bivariate correlations revealed significant associations between early childhood household smoke exposure and the gambling outcome. Individual and family control variables were also significantly associated with early childhood household smoke exposure.

Table 2 reports the association between early childhood household smoke exposure and gambling behavior at age 12 years. Unit increases in early childhood household smoke exposure corresponded to 18% increased chances of gambling participation, 95% confidence interval (CI): 1.080-1.293. In the adjusted model, 4 variables were significantly associated with child gambling: early childhood household smoke exposure, child sex, parental antisocial behavior, and parental gambling. Specifically, boys had 1.56 more chances of self-reporting gambling behavior than girls (95% CI: 1.186-2.051). Unit increases in parental antisocial behavior predicted 1.61 greater odds of gambling participation (95% CI: 1.000-2.582). Finally, for every unit increase in parental gambling, the probability of having participated on gambling by age 12 years was 1.21 times higher (95% CI: 1.022-1.422). The model explains 13.3% of the variance on gambling behavior at age 12 years (Nagelkerke's $R^2 = 0.133$).

Discussion

Given that protecting brains and prioritizing early child development have become social and economic policy issues, exposure to household tobacco smoke has become critical to public health as a preventable lifestyle risk factor.[12,36] Moreover, the consequences associated with excessive gambling, which is rooted in childhood, also translate into considerable individual and societal costs.[37] Although most individuals gamble in a responsible manner, others overindulge, explaining why present trends show young adults engaging in at-risk behaviors, which emerge early in development, that can lead to addictions.[38]

There are several mechanisms that could explain how one parental lifestyle risk begets early child gambling behavior. First, processing of rewards might become dysfunctional in children exposed to neurotoxic environments, especially secondhand smoke.[12] In rats, nicotine exposure dysfunctionally decreases dopaminergic-2 receptors and increases dopaminergic-1 receptors in the nucleus accumbens, the brain's reward processing system.[39] In humans, similar reward processing dysfunction has been associated with impulsive and antisocial decision making.[40] While incorrectly anticipating potential rewards, impulsive and antisocial people release excessive dopamine in the nucleus accumbens, leading to an overactivation of their reward system. Compared with normative samples, substance dependent individuals simultaneously suffer from neural hypo- and hyperresponsiveness, coined as reward deficiency syndrome.[18] This is also common in individuals with attention deficit/hyperactivity disorder.[41] This makes them pursue more potent rewards, like powerful drugs, to compensate for this deficiency and to obtain a sense of contentment.[42] Gamblers who risk becoming pathological experience a greater subjective value representation of rewards than nonpathological gamblers. Pathological gamblers show unusually high activity during anticipation of larger rewards compared with smaller rewards.[43] Second, sustained attentional effort in more conventional reward systems offered in the labor market may be too difficult for some people, and so they choose to gamble. Mice exposed to

Table 1.

Descriptive Statistics for the Early Childhood Household Smoke Exposure Predictor, Gambling Outcome, and Individual and Family Control Variables, Quebec Longitudinal Study of Child Development (1997-1998 Birth Cohort).

		Mean	(SD)	Min-Max
Independent variable				
Early childhood household smoke exposure[a]		1.84	(2.66)	0-7
(ages 1.5-7.5 years)	0 = Never exposed 1 = Transiently exposed 2 = Continuously exposed	59.9% 27.3% 12.8%		
Control variables				
a. Inattention (age 6 years)	0 = Below 50th percentile 1 = Above 50th percentile	53.0% 47.0%		
b. Sex	0 = Girls 1 = Boys	48.8% 51.2%		
c. Gestational smoke (5 months)	0 = Not exposed 1 = Exposed	74.9% 25.1%		
d. Weight for gestational age	0 = Above 10th percentile 1 = Below 10th percentile	91.6% 8.4%		
e. Family configuration (5 months)	0 = Two parents 1 = Single parent	92.0% 8.0%		
f. Family functioning (age 1.5 years)	0 = Normal 1 = Problematic	83.5% 16.5%		
g. Family income (5 months)	0 = $30 000 or more 1 = $29 999 or less	70.5% 29.5%		
h. Maternal education (5 months)	0 = Completed high school 1 = No high school diploma	83.9% 16.1%		
i. Maternal depression (5 months)	0 = No 1 = Yes	84.9% 15.1%		
j. Parental antisocial behavior (5 months)	0 = No 1 = Yes	86.5% 13.5%		
k. Parental gambling[a] (age 10 years)		1.37	(0.99)	0-8
Dependent variable				
Gambling lifetime participation				
(age 12 years)	0 = Never participated 1 = Has participated	84.3% 15.7%		

[a]Applies when treated as a continuous variable.

Table 2.

Association Between Early Childhood Household Smoke Exposure and Gambling Participation at Age 12 Years, Quebec Longitudinal Study of Child Development (1997-1998 Birth Cohort).

Variable	Odds Ratio	95% CI
Early childhood household smoke exposure	1.182**	1.080-1.293
Inattention	0.951	0.203-4.445
Sex	1.560**	1.186-2.051
Gestational smoke	0.651	0.368-1.150
Weight for gestational age	1.060	0.498-2.253
Family configuration	0.924	0.305-2.805
Family functioning	1.145	0.639-2.055
Family income	0.800	0.519-1.233
Maternal education	1.117	0.601-2.077
Maternal depression	0.980	0.601-1.598
Parental antisocial behavior	1.607*	1.000-2.582
Parental gambling	1.205*	1.022-1.422

*$P < .05$. **$P < .01$.

secondhand smoke at a developmental period compatible with early human childhood show prefrontal abnormalities when compared to other brain regions.[44] This might negatively influence the capacity to sustain goal-directed effortful behavior required for motivational and social functioning and the ability to unlearn antisocial behavior.[45,46] Finally, human exposure to tobacco smoke during gestation has been associated with premature thinning of the corpus callosum, having a catastrophic influence on interhemispheric connectivity.[47] Because erratic connectivity has been identified in youth with conduct disorder,[48] this chronic smoke exposure could play a role in the early development of self-regulation risks.

Although we judge our interpretations as conservative, there are several limitations associated with data mining in population-based, noncausal longitudinal studies for secondary analyses. First, our measure of early gambling behavior at age 12 years is very inclusive and includes low-impact activities such as lottery and scratch tickets. This measure naturally showed a lot of variance. Second, this study underestimates the total amount of possible exposure by omitting other various domestic environments where children could be exposed to secondhand smoke, especially when transported in cars. Third, the nonexperimental nature of this study precludes any definitive statements about causal mechanisms, especially given that our household exposure measure may not have been ideal. More specifically, we did not objectively measure the amount of smoke present in every house or the amount of cotinine in bodily fluids, which limited our ability to establish a smoking gun. Nevertheless, we believe the measure used was robust enough to estimate the unique contribution of our less than ideal measure of smoke exposure in association with gambling in a middle class (lower risk) sample. This suggests not only that our results are robust but also that effect size is conservatively interpreted from our birth cohort born between spring 1997 and spring 1998.

Notwithstanding such limitations, the strength of this birth cohort study is that it sheds light on secondhand smoke risks at a window of time when the frontal lobe undergoes important growth and development from birth onward, until middle childhood. Prospective associations between household smoke and attention deficit/hyperactivity disorder and antisocial behavior have been established.[12,20] The use of a prospective longitudinal design from birth onward represents a major strength because it does not depend on retrospective recall and allows the tracking of the environment and human health and development without researcher bias or interference. Even low levels of poorly measured household smoke have been significantly linked with later neurobehavioral development.[20,24] The net risk estimate becomes more than modest when taking a population health perspective, especially when the quantity and quality of the preexisting and concurrent individual and family confounders are given consideration: child sex, weight for gestational age, gestational exposure, inattention, socioeconomic adversity (family income, configuration, and dysfunction), maternal factors (depressive symptoms and level of education), parental antisocial behavior, and parental gambling. These attest to our efforts to eliminate competing explanations for the observed link. Thus, although prenatal smoking does have a nontrivial influence on later child behavior, the results of this study suggest that childhood environment is as important for the prevention of impaired neurobehavioral development and therefore supports the promotion of an unpolluted domestic environment for children.[4,49]

Health care professionals have an obligation to counsel parents to quit smoking, guide children never to become smokers, and educate families

about the ills associated with secondhand smoke. Although most nonsmokers report an indoor smoking ban, smokers have a much lower rate of prohibiting household smoking. Today's family environment, which includes the car and all other living spaces, remains the primary source of secondhand smoke in families with parents who smoke.[50] Fewer parents ban smoking in the car than in the home.[4] Legislation efforts are not typically meant for personal living spaces like homes and cars. Nevertheless, because smoking is an addictive behavior that affects children's development, strict or legal nonsmoking policies and sanctions may be optimal interventions because their connotation as dangerous may influence smokers' behavior. In fact, the burdens to youngsters and society might be reduced more significantly if smoke-free information campaigns become more rigorously encouraged in both cars and homes through social media.[50,51] Legislation- and media-induced reductions of secondary smoke in homes and vehicles are likely to have a promising effect on population health than more general public education campaigns.[5]

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Ethical Approval

This study was approved by the institutional review board of the University of Montreal (Canada).

Informed Consent

Informed written parental consent was obtained for the annual follow-ups taking place during early childhood, and informed consent was obtained systematically from parents, teachers, and children for each biennial school age follow-up.

Trial Registration

Not applicable, because this article does not contain any clinical trials.

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Supplemental Material

Supplemental material for this article is available online. AJLM

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