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Associations prospectives entre l'exposition à la fumée secondaire résidentielle à l'enfance et les difficultés psychosociales à la préadolescence

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Mémoire présenté à la Faculté des études supérieures et postdoctorales
en vue de l'optention du grade de Maîtrise ès science (M. Sc.)
en psychoéducation, profil mémoire et stage

Août, 2015

Résumé

Introduction

La fumée secondaire est considérée comme un agent neurotoxique. À ce jour, la littérature traitant des liens entre l'exposition à la fumée secondaire à l'enfance et les comportements antisociaux repose majoritairement sur des devis transversaux ou porte sur la période développementale de l'enfance.

Objectif

D'une part, nous souhaitons repousser l'âge auquel les comportements antisociaux sont mesurés en le portant à la préadolescence. De plus, nous souhaitons utiliser des données autorapportées afin d'utiliser une mesure possiblement plus valide des comportements antisociaux. Finalement, nous souhaitons confirmer les résultats longitudinaux précédemment rapportés par les parents ou les enseignants à l'âge de 10 ans.

Méthode

Les parents de 1035 enfants ayant participé à l'Étude longitudinale des enfants du Québec ont rapporté si au moins un individu fumait au domicile (entre l'âge d'un an et demi et sept ans). Les variables dépendantes autorévélées (mesurées à douze ans) comprenaient les problèmes de conduite, l'agressivité proactive, l'agressivité réactive, l'indiscipline scolaire et le risque de décrochage.

Résultats

Après l'inclusion des variables de contrôle, nous avons observé que l'exposition à la fumée secondaire est prospectivement associée aux problèmes de conduite, l'agressivité proactive, l'agressivité réactive, l'indiscipline scolaire et le risque de décrochage autorévélés.

Discussion

Les jeunes exposés à la fumée secondaire à l'enfance rapportent avoir plus de comportements antisociaux. Nos résultats corroborent les recommandations du *American Surgeon's General* indiquant qu'aucune exposition à la fumée secondaire ne peut être considérée sécuritaire à l'enfance.

Mots-clés

Agression, problèmes de conduite, risque de décrochage, comportements antisociaux, exposition à la fumée tabagique

Abstract

Introduction

Secondhand smoke is considered a developmental neurotoxicant. Up to this day, research on secondhand smoke and later antisocial behavior has remained cross-sectional or in the developmental period of childhood.

Objective

We sough to extend previous research by extending to age 12 our prospective associational model. Also, we use self-reported data for validity reasons and to cross-match previous research using parent and teacher-rated data at age 10.

Method

Parents reported the amount of household smoke exposure (between ages 1.5 and 7) for 1035 children from the Quebec Longitudinal Study of Child Development. Main outcome measures include self-reported conduct problems, proactive aggression, reactive aggression, dropout risk, and school indiscipline at age 12.

Results

After adjusting for potential confounders, we observed that secondhand smoke is prospectively associated to conduct problems, proactive and reactive aggression, school indiscipline, and dropout risk.

Discussion

Children exposed to household smoke reported increased risks of reporting antisocial behavior at age 12. Our findings corroborate the recommendation of the American Surgeon's General that no smoke exposure can be considered safe.

Key words

Aggression, conduct problems, dropout risk, antisocial behavior, dropout risk, tobacco smoke exposure

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INTRODUCTION GÉNÉRALE

Position du problème

À ce jour, environ 20 % de la population adulte en Amérique du Nord consomme des produits dérivés du tabac (Agaku, King, & Dube, 2014). L'exposition aux composés résultant de la combustion ou de toute autre forme de consommation du tabac s'avère nocive pour l'être humain, et ce de la gestation à l'âge adulte (US Department of Health and Human Services, 2014). La nicotine, l'ingrédient actif et addictif du tabac, de même que les composés carcinogènes dégagés par la combustion du tabac sont associés à diverses problématiques de santé physique et mentale (DiFranza, Aligne & Weitzman, 2004). Néanmoins, les risques associés à la consommation de tabac ne s'appliquent pas seulement aux fumeurs. Les individus se trouvant dans des environnements clos où il est permis de fumer s'exposent à divers risques découlant de la présence de fumée secondaire et tertiaire (Matt, 2011). Ces deux types de fumées pourraient augmenter les risques de développer des problèmes de nature comportementale et neuropsychologique (Pagani, 2014). Une récente étude menée par Pagani et Fitzpatrick (2013) a observé qu'environ 40% des participants ont été exposés, périodiquement ou continuellement, à la fumée secondaire résidentielle entre l'âge de 1 an et demi et 7 ans. Étant donné cette proportion, le nombre d'individus potentiellement touché s'avère significatif. À ce jour, plusieurs recherches transversales et longitudinales portent sur l'association entre l'exposition à la fumée secondaire à l'enfance et la santé mentale. Plus spécifiquement, les individus exposés à la petite enfance ou à l'enfance sont plus à risque de développer des problèmes neurodéveloppementaux (Braun, Kahn, Froehlich, Auinger & Lanphear, 2006) ; Kabir, Connolly & Alpert, 2011), tel que les troubles de l'apprentissage, ou comportementaux (Bandiera, Richardson, Lee, He & Merikangas, 2011). Cependant, d'autres

recherches utilisant des devis longitudinaux sont nécessaires afin de déterminer si ces associations se maintiennent jusqu'à la préadolescence.

Relevé de littérature

Les comportements antisociaux et le risque de décrochage

Les comportements antisociaux se définissent par des actions qui brisent sciemment les règles sociales tout en causant un tort à un individu, un groupuscule ou, plus largement, à la société (Zuddas, 2014). Ces comportements peuvent relever de la criminalité, de la consommation abusive de substances et du refus de se conformer à une figure d'autorité (Wakefield, Pottick & Kirk, 2014). À l'enfance, lorsqu'ils atteignent un certain niveau de sévérité ces comportements peuvent mener à des problématiques ultérieures telles que la criminalité et la consommation de substances illicites (Miech, Caspi, Moffitt, Wright & Silva, 1999).

Les comportements antisociaux sont un problème de santé publique vu leur fréquence relativement élevée et les coûts qu'ils entraînent pour les individus et la société (Foster & Jones, 2005). Par ailleurs, les corolaires des comportements antisociaux, tels que le décrochage scolaire, ont des coûts qui s'additionnent à ceux des comportements antisociaux (Cohen & Piquero, 2009). Plusieurs approches en sciences humaines ont tenté par le passé d'expliquer l'émergence, le maintien ou l'exacerbation de ces comportements. À l'inverse, la littérature étaye une multitude de facteurs de protection influençant la trajectoire développementale qui mène au maintien ou à l'exacerbation des comportements antisociaux. À la petite enfance, les comportements antisociaux suivent une trajectoire normative où l'agressivité, une des premières manifestations antisociales, augmente pour atteindre un pic vers l'âge de deux ans (Baillargeon et al., 2007). Elle peut être de nature proactive lorsque le but est de dominer ou d'obtenir quelque chose ou réactive lorsqu'elle sert de réponse à une situation suscitant une émotion négative (Fite, Raine, Stouthamer-Loeber, Loeber & Pardini,

2009). Chez une population normative et neurotypique, ces comportements diminuent avec les années alors que l'enfant entre à la maternelle et que de nouvelles attentes sociales lui sont formulées (Baillargeon et al., 2007). Cependant, chez une minorité d'individus ces comportements diminuent nettement moins rapidement, stagnent ou augmentent malgré la maturation (Tremblay et al., 2004). Ces comportements deviennent donc de plus en plus problématiques et mènent à d'autres problèmes tel que la diminution de la performance académique et le décrochage scolaire (Kokko, Tremblay, Lacourse, Nagin & Vitaro, 2006).

À l'approche de l'adolescence, les comportements antisociaux tendent à augmenter dans la population générale (Moffitt, 1993). Néanmoins, l'étude des diverses trajectoires des comportements antisociaux indique que, malgré la normalité relative de ces comportements dans cette période développementale, certains marqueurs indiquent qu'ils persévèreront passé la fin de l'adolescence (Miech et al., 1999). Plus spécifiquement, un début précoce et d'intensité élevée à l'enfance est un indicateur de la persévération des comportements antisociaux passé l'adolescence (Hyde, Burt, Shaw, Donnellan & Forbes, 2015).

Les comportements antisociaux ont une base génétique. Plusieurs estimés d'héritabilité génétique effectués sur les comportements antisociaux établissent la contribution de la génétique à 50% (Vitaro et al., 2015). Malgré cette proportion élevée, cela indique que 50% de la variance peut être attribuée à des facteurs environnementaux. Des facteurs sociaux tels que la maltraitance (MacMillan et al., 2014), l'exposition à la violence fraternelle (Kendler, Morris, Lönn, Sundquist & Sundquist, 2014) ou conjugale (Sousa et al., 2011) sont associés au développement de différents types de comportements antisociaux, malgré le fait qu'ils

pourraient aussi relever d'un facteur génétique commun. Le statut socioéconomique, souvent calculé comme un index intégrant l'éducation parentale et le revenu, est connu pour sa capacité prédictive du développement des comportements antisociaux (Miech et al, 1999). Parallèlement, d'autres facteurs environnementaux biologiques tels que l'exposition à des drogues illicites, l'alcool ou les composés du tabac durant la grossesse sont des facteurs de risque connus en ce qui a trait au développement des comportements antisociaux sur lesquels il est possible d'intervenir (Liebschutz et al., 2015). De plus, d'autres facteurs de risque de nature biologique peuvent émerger après la grossesse. Un exemple serait l'exposition à la fumée secondaire qui, selon la littérature récente, met l'enfant à risque autant au plan de la santé physique que mentale (Pagani, 2014).

Les connaissances actuelles identifient des relations transactionnelles entre les comportements antisociaux à l'enfance et les difficultés scolaires, le tout étant associé fortement au décrochage scolaire (Wang & Fredricks, 2014). Dans l'optique de cerner l'ampleur de la problématique des comportements antisociaux, l'ajout de paramètres estimant le fonctionnement scolaire s'avère primordial. Ce type de mesure devrait comprendre la performance académique, le redoublement scolaire et l'engagement scolaire (Archambault & Janosz, 2009).

L'exposition à la fumée secondaire

Le tabac, lorsqu'il est fumé, produit deux types de fumées résiduelles. La fumée secondaire est un mélange composé de la fumée qui est inhalée, subissant ainsi des modifications biochimiques lorsqu'elle entre dans le système respiratoire, et qui exhalée par le

fumeur (Schick & Glantz, 2005). Elle comprend aussi la fumée qui émane de la cigarette qui brûle. La fumée tertiaire est formée des résidus de la fumée secondaire qui s'accumule dans les environnements clos où il y a présence de fumée secondaire (Matt, 2013). La fumée secondaire s'avère plus nocive pour l'être humain que la fumée primaire (inhalée par le fumeur) vu sa plus haute teneur en nicotine et en composés carcinogènes (Schick & Glantz, 2005).

À la petite enfance, deux hypothèses visent à expliquer les associations observées entre l'exposition à la fumée secondaire résidentielle et les relations précédemment observées avec les difficultés psychosociales telles que les comportements antisociaux et les difficultés scolaires (Kabir, Connolly & Alpert, 2011; Pagani & Fitzpatrick, 2014). Premièrement, la fumée secondaire s'avère être un irritant des voies respiratoires qui peut causer une détresse circulatoire (difficulté à acheminer l'oxygène, à évacuer le dioxyde de carbone et à faire circuler le sang) et l'apnée du sommeil (Barnoya & Glantz, 2005; Beebe, Raush, Byars, Lanphear & Yolton, 2012; Jara, Benke, Lin & Ishman, 2015). Cela pourrait avoir un impact sur le développement du cerveau des enfants puisque le manque d'irrigation sanguine pourrait altérer le fonctionnement et le développement de ce dernier (Kheirandish-Gozal, Yoder, Kulkarni, Dozal & Decety, 2014). Deuxièmement, les modèles expérimentaux utilisant des animaux démontrent que l'exposition à la nicotine ou à l'ensemble des produits présents dans la fumée secondaire est neurotoxique (Slotkin, Pinkerton & Seidler, 2006). La neurotoxicité des dérivés du tabac fumé peut opérer de trois façons distinctes : tout d'abord, elle peut altérer la transmission de l'information entre les cellules nerveuses et donc causer des problèmes de communication cellulaire – par exemple, les rats exposés à la fumée secondaire tendent à

sécréter des quantités excessives de dopamine dans les régions associées au circuit de récompense (Pinheiro et al., 2015); ensuite, elle peut modifier la cytoarchitecture cérébrale, engendrant ainsi des altérations dans le tissu cérébral. Par exemple les individus fumant la cigarette ont une plus grande myélinisation des fibres nerveuses dans le corps calleux, lorsque comparés à des non-fumeurs, possiblement au détriment de la matière grise (Van Ewijk et al., 2015) – ceci peut hypothétiquement augmenter l'impulsivité vu la plus grande vitesse de transmission de l'information conjuguée avec une capacité moindre de traitement de l'information ; enfin, l'exposition à la fumée secondaire pourrait avoir des effets épigénétiques. L'épigénétique est un processus par lequel des conditions environnementales causent des changements au niveau génomique après la conception – par exemple, dans une étude portant sur des singes rhésus, l'exposition aux composés du tabac durant la grossesse mettait à risque la deuxième génération de singes et non la première, indiquant des changements génétiques apparaissant post-conception (Holloway, Cuu, Morrison, Gerstein & Tarnopolsky, 2007).

Ces résultats imputés aux effets toxiques et neurotoxiques de la fumée du tabac ont mené à plusieurs incitatives de décideurs publics et de professionnels de la santé mentale. Ces initiatives se reflètent par de nouvelles lois votées, de programmes de prévention et d'intervention et des stratégies gouvernementales éducatives (Picketts, Schober, Brody, Curtin & Giovino, 2006). Cependant, plusieurs autres initiatives sont à développer et à mettre en place.

La nécessité de poursuivre la recherche sur la fumée secondaire

Poursuivre la recherche sur les risques que pose l'exposition à la fumée secondaire à l'enfance est une nécessité en santé publique pour plusieurs raisons. Premièrement, nombre d'études populationnelles ont omis de contrôler statistiquement l'exposition aux composés du tabac durant la gestation. Donc, elles n'ont pas été en mesure d'isoler la contribution unique de l'exposition à la fumée secondaire sur la santé mentale (Eskenazi & Castorina, 1999; DiFranza, Aligne, & Weitzman, 2004; Herrmann, King, & Weitzman, 2008). Deuxièmement, certaines des meilleures études ont utilisé des modèles transversaux, ce qui limite les hypothèses quant à la directionalité et le maintien dans le temps de leurs résultats (Braun et al., 2006; Bandiera et al., 2011). Plus spécifiquement au niveau des comportements antisociaux, la majorité des études longitudinales disponibles ont été conduites des sujets âgés au maximum de dix ans (voir Rückinger et al., 2010; Pagani & Fitzpatrick, 2013) et mesurent les comportements antisociaux à l'aide de données rapportées par les parents ou les enseignants. Ces associations doivent être répliquées à des périodes développementales ultérieures afin de déterminer si ces risques perdurent dans le temps ou s'estompent avec la maturation. Parallèlement, l'utilisation de mesures rapportées par d'autres personnes que l'enfant pose problème vu la nature cachée que les comportements antisociaux acquièrent plus les individus approchent de l'adolescence (Loeber, Burke, & Pardini, 2009).

Bref, vu les difficultés potentielles pouvant être attribuées à l'exposition à la fumée secondaire et l'état de la littérature scientifique, de nouvelles études s'avèrent nécessaires afin de valider les associations observées entre l'exposition à la fumée secondaire et les comportements antisociaux.

ARTICLE

Lévesque-Seck, F., Archambault, I., Janosz, M., & Pagani, L. (in preparation for The Journal of Epidemiology and Community Health). Early childhood household smoke exposure and psychosocial impairment in middle school: Relationships with antisocial behavior and dropout risk

Abstract

Background

Secondhand smoke is considered a developmental neurotoxicant**.** Using data from a birth cohort design, we investigate the prospective association between early childhood tobacco smoke exposure from age 1.5 to 7 and later antisocial behavior.

Method

Parents reported the amount of household smoke exposure (between ages 1.5 and 7) for 1035 children from the Quebec Longitudinal Study of Child Development. Main outcome measures include self-reported conduct problems, proactive aggression, reactive aggression, dropout risk, and school indiscipline at age 12.

Results

After adjusting for potential confounders, increases of one standard deviation of exposure to household tobacco smoke forecasted a 19% standard deviation unit increase in conduct problems (unstandardized $\beta = 0.07$; 95% confidence interval [CI] from 0.04 to 0.09), a 11% standard deviation unit increase in proactive aggression (unstandardized $\beta = 0.04$; 95% CI from 0.01 to 0.07), a 13% standard deviation unit increase in reactive aggression (unstandardized $\beta = 0.07$; 95% CI from 0.03 to 0.12), a 13% standard deviation unit increase in school indiscipline (unstandardized $\beta = 0.13$; 95% CI from 0.05 to 0.20), and a 10% standard deviation unit increase in dropout risk (unstandardized $\beta = 0.07$; 95% CI from 0.01 to 0.12).

Conclusion

Children exposed to household smoke reported increases in conduct problems, proactive and reactive aggression, classroom indiscipline, and dropout risk at age 12. These findings warrant

fostering parental awareness by policy makers and health practitioners about the psychosocial cost of early exposure to secondhand smoke. Our results support tobacco control regulation and prevention initiatives.

EARLY CHILDHOOD HOUSEHOLD SMOKE EXPOSURE AND PSYCHOSOCIAL IMPAIRMENT IN MIDDLE SCHOOL: RELATIONSHIPS WITH ANTISOCIAL BEHAVIOR AND DROPOUT RISK

Although on the decline, people are still smoking at a significant prevalence (Agaku, King, & Dube, 2014). Children and adults exposed to secondhand smoke risk a vast number of concurrent and long-lasting health problems (Öberg, Jaakkola, Woodward, Peruga, & Prüss-Ustün, 2011). These illnesses, caused by tobacco smoking and its byproducts, have been attributed a major role in mortality and morbidity rates in the general population (US Department of Health and Human Services, 2014). Secondhand smoke, resulting from cigarette consumption, releases a cocktail of pollutants comprising 85% side-stream smoke (from the cigarette itself) and 15% of exhaled smoke into the environment (Schick & Glantz, 2005). Apart from being a major source of nicotine, secondhand smoke contains more than 250 toxic chemicals such as carbon monoxide, ammonia, and multiple heavy metals (Schick & Glantz, 2005). Beyond physical health, these chemicals present in secondhand smoke are hypothesized as hampering brain development in childhood (Pagani, 2014). Thus, leading to impulsive and dysfunctional behaviors such as impulsivity and antisocial behavior (Bandiera, Richardson, Lee, He, & Merikangas, 2011; Kabir, Connolly, & Alpert, 2011; Pagani & Fitzpatrick, 2013).

Indeed, the physiological mechanisms of firsthand and secondhand tobacco smoke have been studied extensively in animals and humans and multiple causal effects have been demonstrated on physical health and behavior (Leberl, Kratzer, & Taraseviciene-Stewart, 2013; Fricker, Deane, & Hansbro, 2014). In relation to brain development, causal experiments

with animals have demonstrated that secondhand smoke is neurotoxic and affects the developing brain (Cohen & George, 2013; Slotkin et al., 2015). When considering the developmental changes in the brain circuitry of children from gestation to the end of childhood, childhood exposure to neurotoxic compounds contained in household tobacco smoke becomes worthy of research attention in public health (Jacobs et al., 2013).

Young children living in a household with parents who smoke exert virtually no control over their exposure to secondhand smoke. Between birth and age 5, the brain consolidates multiple neurological systems that will later be vital for academic and occupational functioning (Shonkoff, 2011). Experiments with rodents have demonstrated that exposure to secondhand smoke has effects on development and consolidation of key areas of the executive system during sensitive periods of brain growth (Counotte et al., 2009). In humans, exposure to secondhand smoke has been associated in humans with sleep apnea and cardiorespiratory distress (Barnoya & Glantz, 2005). This state of anoxia, coupled with the neurotoxic characteristics of secondhand smoke, have been associated with disruptions in cellular communication, structural development of brain tissue, and epigenetic mutations (Pagani, 2014). Disruptions in the executive system would then explain the relationship between childhood household smoke exposure and developmental psychopathology (Pagani & Fitzpatrick, 2013).

Childhood antisocial behavior is characterized by proactive intentions to harm others and unremorseful violation of societal norms (Zuddas, 2014). These represent a multitude of behaviors that include aggression, criminal offenses, theft, refusal to comply with authority,

and destruction of property (Wakefield, Pottick, & Kirk, 2014). Childhood antisocial behavior is associated with poor adjustment in the majority of social environments children evolve into (Kim, Kochanska, Boldt, Nordling, & O'Bleness, 2014). Functional impairment associated with antisocial behavior can persist across an individual development, evolving in intensity, form, and frequency (Moffit, 1993). Although aggression is relatively common in its physical form in early childhood, it is expected to diminish significantly when children start kindergarten (Wildeboer et al., 2015). From school age onwards, this type of antisocial behavior can manifest itself as reactive or intentional (Fites, Rubens, Preddy, Raines, & Pardini, 2014). Consistently high levels of aggression, whether proactive or reactive, before or at the age of 10 places an individual at risk for a number of long-lasting mental health problems (Moffit, 1993).

A true indication of individual dysfunction in childhood and adolescence is characterized by psychosocial and academic impairment (Foster & Jones, 2005). For instance, measures of student engagement, behavior problems, and academic performance are considered to be robust predictors of dropout risk (Wang & Fredericks, 2014). Grade retention early in elementary school has been associated with persistent worsening of behavioral and academic adjustment by the middle school transition (Pagani, Tremblay, Vitaro, Boulerice, & McDuff, 2001). When combined with other family risk factors during this transition, grade retention predicts later dropout from high school by age 21 (Pagani et al., 2008). Student engagement during the middle school transition forecasts subsequent dropout from high school (Archambault, Janosz, Fallu, & Pagani, 2009). In these analyses, school indiscipline and inattendance made the most powerful contribution in predicting high school dropout.

Studies are increasingly finding links between exposure to secondhand smoke and child maladjustment (Jacobs et al., 2013), yet these have been mostly cross-sectional (Eskenazi & Castorina, 1999; DiFranza, Aligne, & Weitzman, 2004; Herrmann, King, & Weitzman, 2008). For example, exposure to environmental tobacco smoke has been associated with conduct disorder (Bandiera, Richardson, Lee, He, & Merikangas, 2011). Associations have also been observed between household tobacco smoke exposure and higher prevalence of learning disabilities (Kabir, Connolly, & Alpert, 2011) and ADHD (Braun, Kahn, Froelich, Auinger, & Lanphear, 2006). Exposure to household tobacco smoke has also been associated with a greater average of days missed at school and higher childhood illness during school age years (Levy, Winickoff, & Rigotti, 2011). In a vast sample of Chinese non-smoking adolescents, exposure to household tobacco smoke between 1 and 7 days per week was associated with a 14% to 28% increased risk of self-reported poor academic performance (Ho, Lai, Wand, & Lam, 2010). Moreover, these risks increased with the number of people smoking per household.

The strengths of longitudinal analyses have recently been applied to examine exposure to secondhand smoke and it later correlates. In adults, associations have been found between adult exposure to secondhand smoke and later increased risks for psychiatric hospitalization (Hamer, Stamatakis, & Batty, 2010). Rückinger et al. (2010) also observed increased risks for parent-reported conduct problem, hyperactivity, emotional problems, and peer difficulties in children exposed to secondhand smoke prenatally and postnatally at age 10. Among the most compelling studies that have implemented prospective population-based designs, we find several limitations. Using data from a birth cohort, Pagani and Fitzpatrick (2013) found early

exposure to household secondhand smoke to be prospectively associated with higher levels of teacher-reported antisocial behavior at age 10. Antisocial behavior becomes increasingly covert as children move into adolescence (Loeber, Burke, & Pardini, 2009), indicating that this relationship warrants verification with self-reported data. Second, this replication with self-report needs to extend beyond age 10, which represents an important moment as it the age of the transition to middle school. This academic transition harbors many markers for later school dropout (Kieffer, Marinelle, & Neugebauer, 2014).

Hence, the purpose of this study is to prospectively examine the association between household tobacco smoke exposure from ages 1.5 to 7 and later antisocial behavior at age 12 using self-reported data. It is expected that higher levels of early childhood household smoke exposure will predict more self-reported antisocial behavior.

Methods

Participants

The original birth cohort sample for this study, randomly selected and stratified by region, comprised 2837 children from the birth registry in Quebec (Canada) between 1997 and 1998. Between participant enrollment and the 5 months baseline, 93 children were ineligible, 186 were untraceable, and 438 refused to participate to this study. Leaving 2055 children (82% of eligible participants) that were followed up in the early childhood data collection. School age follow-ups occurred biennially and informed consent was obtained systematically from parents, teachers, and children. All children (N = 1035) with complete data on mother-reported exposure to household tobacco smoke exposure from ages 1.5 to 7 and outcome data

emanating from the Social Behavior Questionnaire (Pagani, Fitzpatrick, Barnett, & Dubow, 2010) or the MASPAQ (Brière, Fallu, Morizot, & Janosz, 2014) were included in these analyses.

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

Between ages 1.5 and 7, mothers were asked annually 'Does one or other of the parents or another person smoke in the house?'. These answers produced a index ranging from 0 to 7 (Pagani & Fitzpatrick, 2014; Pagani, Nguyen, & Fitzpatrick, 2015). Parental self-reports have been recognize a good reliability in assessing childhood exposure to secondhand smoke and have been successfully cross-matched with plasma, urine, and hair cotinine to determine their validity (Avila-Tang et al., 2013).

Outcomes: Antisocial behavior (age 12)

Conduct Problems. Children were asked about their truant behavior and conduct problems (Brière, Fallu, Morizot, & Janosz, 2014). Items asked if they had (in the last 12 months): Stayed out later than their parent said they could; Stayed out all night without permission; Skipped a day of school without permission; Been questioned by policemen about something; Ran away from home; Threatened to hit someone in order to force them to do something they didn't want to do; Broke down a door or a window in order to get into a place and take something; Threatened or roughed up other people to get something; and Were part of a group that did bad things ($\alpha = 0.53$). The items were scored as follows: never (0); once or twice (1); often (2); or very often (3). Scores were rescaled from 0 to 10.

Proactive Aggression. Children were asked about their proactive aggression behavior (Tremblay et al., 1991; Vitaro, Gendreau, Tremblay, & Oligny, 1998; Dickson et al., 2015). Items asked if they had (in the past 6 months): Threatened to hit people; Encouraged other children to pick on a particular child; Tried to dominate other children; and Scared other children to get what they want ($\alpha = 0.51$). 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.

Reactive Aggression. Children were asked about their reactive aggression behavior (Tremblay et al., 1991; Vitaro, Gendreau, Tremblay, & Oligny, 1998; Dickson et al., 2015). Items asked if they had (in the past 6 months): Reacted with anger and fighting to an accident; Reacted in an aggressive manner when something was taken away from them; Reacted in an aggressive manner when teased; and Reacted in an aggressive manner when contradicted ($\alpha = 0.71$). All items comprised hitting, pushing, or slapping as examples of reacting in an aggressive manner. 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).

School Indiscipline. Children were asked about their behaviors related to classroom indiscipline and failing subjects at school (Archambault, Janosz, Fallu, & Pagani, 2009). Items asked if they had (in the past school year): Disrupted the class on purpose; Been rude to their teacher; Used hidden notes or other means not allowed in order to cheat on a test; Missed school without a valid reason; and Ever failed one or more subjects on their report card ($\alpha =$

0.51). The items were scored as follows: never (0); once or twice (1); often (2); or very often (3). Scores were rescaled from 0 to 10.

Dropout Risk. Children were asked about their school engagement, grade retention history, and academic performance (Archambault & Janosz, 2009). Items, forming a dropout risk index, asked children about: Their performance in Language Arts; Their performance in mathematics; Grade retention history; Liking school; How they would rate their grades when compared with other students of their age at school; How important is it for them to get good marks; and How far do they plan to go in school, based on their own wishes ($\alpha = 0.56$). 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 (Ages: 5 to 29 months)

At 5 months, mothers were asked 'Did you smoke during pregnancy?'. Responses were coded as smoked (= 1) or did not smoke (= 0). Mother-reported gestational illicit drug consumption (0 = no consumption or 1 = consumed illicit drugs) was also included in the analyses. Weight for gestational age was calculated directly from birth records using Canadian norms. Children were coded as 10^{th} percentile and higher (=0) and below 10^{th} percentile (=1). Parental antisocial behavior in adolescence and adulthood was also assessed with several items from the NIMH-Diagnostic Interview Schedule (Shaffer et al., 1996). All items were scored as no (= 0) and yes (= 1) and then rescaled to produce a parental score ranging from 0 to 1. Adolescent items included: Starting fights; Theft; Involvement with youth protection or policemen; Expulsion or suspension from school (father only); Running away from home

(mother only); and Skipping school (mother only). Adult items included: Arrests; Being fired from a job; Having trouble at work, with their family, or with the police due to drug or alcohol abuse; Starting fights (fathers only); and Hitting or throwing things at a spouse or partner (mothers only) (Pagani & Fitzpatrick, 2013). Mother-reported number of siblings of the child at birth and maternal age at childbirth were also included in the analyses. Mothers reported their level of education coded as no high school diploma (= 0) and high school diploma obtained (= 1). Mothers reported household income at 5 months (29999k or less = 1 or 30000K and more = 0). Family configuration was assessed at 5 months (single parent family = 1 or two parents = 0). Maternal depressive symptoms at 5 month was assessed through 12 questions from the Center for Epidemiological Studies Depression Scale (α=0.78) (Radloff, 1977; Pagani & Fitzpatrick, 2013). Items assessed various depressive symptoms covering vegetative (e.g.: 'had poor appetite'), cognitive (e.g.: 'had trouble keeping their mind on what they were doing'), and behavioral (e.g.: 'had crying spells'). Trained examiner observed maternal hostile parenting during interviews at age 1.5 (items: Screamed at the child; Seemed disturbed by the child; Hit the child; and Scolded or put down the child) (α=0.75) (Caldwell & Bradley, 1994; Fitzpatrick, Pagani, & Barnett, 2012). At 17 months mother reported family functioning by answering questions from the McMaster family assessment tool (α=0.98) (Epstein, Baldwin, & Bishop, 1983). This scale reflects various aspects of family life in terms of communication, problem solving, and general household climate. Child temperament was derived by meaning mother-reported perception of the difficulty and unpredictability of the child at 17 month (Pagani, Fitzpatrick, Barnett, & Dubow, 2010). Early cognitive skills were measured using the Imitation Sorting Task. This standardized test assesses attention and working memory at 29 months (Alp, 1994). At age 4, mothers reported, on one single 4-point

Likert Scale, child exposure to fighting with the following question: 'How often does your child see adults or teenagers in your house physically fighting, hitting or otherwise trying to hurt others?' (Fitzpatrick, Pagani, & Barnett, 2012). Sex was also implemented as a control variable (Boys =1).

Data analysis

These secondary analyses using a series of multiple linear regressions aims to model the linear relationship between early childhood household smoke exposure and later antisocial behavior and dropout risk. Only linear relationships were investigated. To exclude other potential explanatory variables, we selected multiple individual and family factors to be integrated as control variables since they may be potentially related to early childhood household smoke exposure or antisocial behavior. More specifically, this model also includes prenatal exposure to tobacco smoke in an effort to isolate the unique contribution of early childhood household smoke exposure. In the adjusted model: $ANTI_{i12years}$ represents self-reported antisocial behavior, $DR_{i12years}$ represents self-reported dropout risk, $ENSM_i$ represents early childhood household smoke exposure, and $CHILD_i$ and FAM_i represents individual, gestational, and family control variables for every individual $child_i$. Finally, a1 represents the constant and e_i the stochastic error term.

$$ANTI_{i12years} = a1 + \beta\ 1ENSM_i + \gamma 1\ CHILD_i + \gamma 2FAMi + e_i$$
$$DRi_{12years} = a1 + \beta\ 1ENSM_i + \gamma 1\ CHILD_i + \gamma 2FAMi + e_i$$

Results

As the retained sample (n = 1035) represented 47% of the original cases (n = 2223), analyses comparing retained cases to those from the original sample were required to identify

a possible attrition bias. The retained sample had more mothers that finished high school (89% vs 80%; $t_{2218} = 5.769$; $p < 0.001$), had less mothers reporting smoking during pregnancy (22% vs 27%; $t_{2210} = -2.676$; $p < 0.01$), and had less boys (47% vs 55%; $t_{2177.77} = -3.481$; $p = 0.001$). To compensate for attrition bias, we imputed all missing data using NORM multiple imputation software. Using an iterative method, NORM uses the valid observations for each case to impute missing data with an expectation-maximization algorithm (Shaffer, 1999). Data was imputed for cases with data on early childhood household smoke exposure and missing on outcomes. This was implemented to reduce to 20% the number of cases with imputed data (vs. 53%).

Table 1 reports descriptive statistics of predictor, outcome, and control variables. Boys comprised 47.2% boys of the sample. Continuous household smoke exposure in early childhood between the ages of 1.5 and 7 was of 12.8%, transient exposure comprised 27.3% of the sample, and never exposed children represented the remaining 59.9%.

Table 2 reports on the relationship between household smoke exposure in early childhood and pre-existing individual and family factors implemented as control variables. The model indicates that parental antisocial behavior, number of siblings at birth, family functioning, maternal age at childbirth, and maternal education, and maternal gestational smoking significantly predict early childhood household smoke exposure. Individual variables were non-significant with the exception of child early cognitive skills. Table 3 reports the correlations between predictor, outcome, and control variables.

Table 4 reports unadjusted and adjusted regression coefficients which reflect the linear relationship between early childhood household smoke exposure and antisocial behavior at age 12. In the adjusted models, early childhood household smoke exposure predicted increases in self-reported conduct problems, proactive and reactive aggression, school indiscipline, and dropout risk at age 12. More specifically, in the adjusted model, for every standard deviation increase in household smoke exposure was prospectively associated with a 19% standard deviation unit increase in conduct problems (unstandardized $\beta = 0.07$; 95% confidence interval [CI] from 0.04 to 0.09), a 11% standard deviation unit increase in proactive aggression (unstandardized $\beta = 0.04$; 95% CI from 0.01 to 0.07), a 13% standard deviation unit increase in reactive aggression (unstandardized $\beta = 0.07$; 95% CI from 0.03 to 0.12), a 14% standard deviation unit increase in school indiscipline (unstandardized $\beta = 0.13$; 95% CI from 0.05 to 0.20), and a 10% standard deviation unit increase in dropout risk (unstandardized $\beta = 0.07$; 95% CI from 0.01 to 0.12). Sex was the only control variable significantly predictive of these outcomes. The explained variance by these models ranged from small (proactive aggression, $R^2 = 3\%$; reactive aggression, $R^2 = 7\%$; conduct problems, $R^2 = 6\%$) to moderate (dropout risk, $R^2 = 15\%$; school indiscipline, $R^2 = 13\%$). This could in turn indicate either a possible poor fit of the model or the absence of other potential explanatory variables. Table 5 reports non-imputed regression coefficient for comparison purposes.

Discussion

This study sought to replicate and extend previous research examining the relationship between early childhood exposure to secondhand smoke and later social and academic impairment. Our results support the Surgeon's General recommendations that no exposure to

household secondhand smoke in childhood can be considered safe (US Department of Health and Human Services, 2014). Past longitudinal research has suggested that secondhand smoke is associated with antisocial behavior. However, even when using prospective designs such studies did not rely on self-reports. Several past reviews, mostly based on cross-sectional data, described associations between secondhand smoke exposure in childhood and different indicators of academic achievement, performance, and functioning (Eskenazi & Castorina, 1999; DiFranza, Aligne, & Weitzman, 2004; Herrmann, King, & Weitzman, 2008). Our results suggest that living in a home with smokers prospectively influences later child antisocial behavior at age 12, above and beyond potential confounders.

In this study, children exposed to household secondhand smoke between ages 1.5 and 7 were more likely to instigate aggression toward other children, threaten others with violence, and induce fear to access property of others. Increased risks were also found for using reactive aggression to problem solve in situations related to sharing, teasing, and discussing opinion divergences. Also, increases in our measure of early household smoke exposure were associated with a proportional increase in risks of engaging in early delinquent behaviors such as skipping school, fighting, breaking and entry, being questioned by police officers, and being affiliated with a gang. Academically, children exposed to early household secondhand smoke showed increased risks for poor academic performance, failing subjects at school, and grade retention. There was also a link with not liking school as much as their non-exposed peers. Lastly, increased exposure to household smoke also forecasted more overt classroom misbehavior such as disruptiveness, insolence, and cheating.

Multiple mechanisms in early childhood could explain these observed associations. From birth onward, vital systems in children undergo massive structural and functional changes (US Department of Health and Human Services, 2006). The underdevelopment of cardiorespiratory systems leaves them more fragile to environmental pollutants, which could potentially hamper brain development (Pagani, 2014). The developing brain is known to be sensitive to snoring-induced anoxia and circulatory distress, mainly instigated by carbon monoxide present in secondhand smoke (Barnoya & Glantz, 2005; Beebe, Raush, Byars, Lanphear, & Yolton, 2012; Jara, Benke, Lin, & Ishman, 2015). Hence, when added to the presence of airborne neurotoxic particles induced by secondhand and thirdhand smoke, this may create a situation of jeopardizing brain development (Grandjean & Landrigan, 2014).

Ethical constraints in research involving humans create the necessity to draw inferences from animal models. Despite the identification of causal effects from secondhand smoke exposure in animal models, inferring that the same mechanisms affect humans similarly is hazardous. Interspecies differences conjugated with the limits of experimental designs hampers the internal and external validity of animal research when conclusions are to be drawn on human phenomenon (Reagan-Shaw, Nihal & Ahmad, 2008; Van der Worp et al., 2010). Despite being speculative, these studies offer some hypotheses about the explanatory mechanisms between early childhood tobacco smoke exposure and later functional impairment. First, exposure to secondhand smoke affects cortico-thalamic connectivity in experiments with rodents, through the hyperactivation of nicotinic acetylcholine receptors (Heath, King, Gotti, Marks, Picciotto 2010). The cortico-thalamic relay system has a role in reward expectation and task difficulty evaluation. Individuals showing alterations in these

pathways are at risk for inaccurate reward processing, punishment anticipation, and impairment in weighting the risks of their behavioral responses (Krebs, Boehler, Roberts, Song, Woldorff, 2012). Second, rats exposed to nicotine during lactation showed a decreased number of dopaminergic-2 receptors and a higher number of dopaminergic-1 receptors in the nucleus accubens, suggesting dopaminergic system alterations leading to excessive or even insatiable need for reward (Pinheiro et al., 2015). The authors attribute their results to nicotine-induced reward system dysfunctions that are hypothesized to function similarly in humans. Similar alterations in humans of dopaminergic systems of the nucleus accubens are associated with impulsive and antisocial traits (Buckholtz et al., 2010). When confronted with rewards, impulsive and antisocial individuals release more dopamine in the nucleus accubens than other individuals, leading to an excessive activation of the reward system. This could, in turn, explain why some individuals engage in antisocial and impulsive behavior (Buckholtz et al., 2010) and substance abuse (Ernst & Luciana, 2015). Third, experiments found that mice exposed to secondhand smoke in infancy showed increased oxidative activity in multiple cortical regions, with the prefrontal cortex exerting less antioxidant protection when compared to other brain regions (Torres et al., 2012). In another experiment targeting oxidation in the prefrontal cortex, mice exposed to secondhand smoke did not show altered basic learning and reward processing (Johnson et al., 2013). However, exposure to secondhand smoke decreased reversal learning, motivational functioning, and social behaviors. Thus, impacting negatively their capacity to unlearn negative behaviors and to apply goal-directed effortful actions. In human, these types of prefrontal abnormalities are associated with core-characteristics of antisocial behavior and impulsivity such as poor social conduct, impairments in decision-making and emotional-processing, difficulties to self-regulate, demonstrating cognitive

rigidity, and decreases in goal-directed behavior (Yang & Raines, 2009). As these features are key aspects of learning and socialization processes, these abnormalities could lead to poor academic and behavioral functioning in school as well as increases of the likelihood to impulsively engage in aggression (Yang & Raines, 2009). Fourth, exposure to tobacco smoke during gestation has been associated with corpus callosum thinning possibly leading to lower inter-hemispheric connectivity (Paus et al., 2008). Observations on current smokers also show excessive myelination of white matter in the corpus callosum (Van Ewijk et al., 2015). Similar alterations in the corpus callosum have been identified among individuals with conduct disorder indicating that erratic or exacerbated connectivity in this region could play a role in antisocial behavior (Zhang et al., 2014). Accordingly, these dysfunctions could translate in aggression and antisocial behavior due to increased impulsivity and impaired decisions-making skills. Hypotheses for these observed associations come from the fact that the corpus callosum is the leading brain route bridging higher cognitive abilities, located in frontal cortexes, with motor functions situated in parietal regions (Catani & De Schotten, 2008). As white matter and grey matter evolve in an opposite trends, this brain tissue ratio could potentially lead to impaired processing capacities, impulsivity, and decision-making impairments, despite higher information transferring speed. (Zhang et al., 2014). Which could in turn increase risks for antisocial behavior, aggression, and exhibit lower academic adjustment. However, these explanatory mechanisms are speculative. Future studies on the direct neurotoxic effect of secondhand smoke will evaluate the degree to which animal research can inform on human problems.

Nevertheless, this study bears limitations to be mentioned. First, our measure of

household household smoke does not objectively measure the amount of smoke present in every house or the actual amount of cotinine in bodily fluids. Second, it underestimates the total amount of possible exposure by omitting various other household environments where children could be exposed to secondhand smoke, especially when transported in cars. Recent research indicates that second and thirdhand smoke from car interiors is associated with similar health problems (Kabir et al., 2009). When considering these limitations, the interpretation of our results might be seen as conservative. Third, by nature of their designs, longitudinal models rely on natural experiments. Although we sought to generate results as free as possible from confounding explanations, our results cannot be interpreted as causal. Ethical constraints precluding randomized experiments in humans call for more control in associational models.

In terms of strengths, this study using self-reports may better estimate antisocial behavior. When children approach adolescence, antisocial behavior becomes increasingly covert, making adults less apt to observe and report these behaviors accurately (Loeber et al., 2009). Also, this study somewhat captures the potential noxious presence of thirdhand smoke, a tobacco compound resulting from the residuals deposits of secondhand smoke over time (Matt, 2013). These prospective associations, similar to those found previous studies, supports the links found between early childhood household smoke exposure and antisocial behavior. When taking into account the observed relationships with academic adjustment, our results suggests further investigations on secondhand smoke exposure in childhood to identify the developmental course leading to psychosocial dysfunction in adulthood.

Household smoke is an early modifiable risk factor that requires simple behavior redirection to vanish. This study offers insights on the later behavioral challenges children exposed to secondhand smoke might encounter. Moreover, the continuity of these associations in time as well as the presence of relationships despite controlling for many potential confounders identifies that early childhood exposure to secondhand smoke might be a factor that clinicians should look at when assessing patients. Also, these results become relevant when taking into account the later societal consequences of antisocial behavior such as its consolidation as well as lifestyle risks in adulthood (Cohen & Piquero, 2009). These observed associations with later dropout indicators at the middle school transition suggest later risks for criminality, dropout, occupational impairment, poverty, and higher mortality (Montez, Hummer, & Hayward, 2012). These findings warrant fostering parental awareness by policy makers and public health practitioners about the long-term psychosocial cost of early exposure to secondhand smoke and the later neurodevelopmental impairment (Foster & Jones, 2005; Pelham, Foster, & Robb, 2007; Max, Sung, & Shi, 2014), which adds to the global medical burden of secondhand smoke (Max, Sung, & Shi, 2015). Therefore, our results support tobacco control regulation, educational campaigns for the general public, prevention initiatives by public health practitioners, and smoke-free environments for young children.

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Table 1. *Descriptive statistics for the predictor, outcomes, and pre-existing individual and family control variables.*

Independent Variables			
Early childhood household smoke exposure in early childhood (ages 1.5 to 7)			
1. Never exposed	59.9%		
2. Transiently exposed	27.3%		
3. Continuously exposed	12.8%		
Control Variables	Mean	Standard Deviation	Min - Max
1. Temperament (age 1.5)	0.14	0.35	0-1
2. Early cognitive skills (age 2)	0.27	0.44	0-1
3. Sex (Boys = 1)	0.47	0.50	0-1
4. Child exposure to fighting (age 3)	0.07	0.26	0-1
5. Weight for gestational age	0.07	0.26	0-1
6. Gestational smoke exposure (5 mo)	0.23	0.42	0-1
7. Gestational illicit drug exposure (5 mo)	0.01	0.10	0-1
8. Number of siblings at birth (5 mo)	0.16	0.37	0-1
9. Family income (5 mo)	0.22	0.42	0-1
10. Family configuration (5 mo)	0.04	0.20	0-1
11. Family functioning (age 1.5)	0.13	0.34	0-1
12. Maternal hostile parenting (age 1.5)	0.05	0.21	0-1
13. Maternal depression (5 mo)	0.12	0.32	0-1
14. Parental antisocial behavior (5 mo)	0.09	0.28	0-1
15. Maternal education (5 mo)	0.89	0.32	0-1

16. Maternal age at childbirth †	29.54	5.03	16.5-44.40
Dependant Variables			
1. Conduct problems (age 12)†	0.72	0.93	0-10
2. School indiscipline (age 12)†	2.95	2.39	0-10
3. Dropout risk (age 12)†	1.67	1.89	0-10
4. Proactive agression (age 12)†	0.49	0.97	0-10
5. Reactive agression (age 12)†	1.01	1.48	0-10

†Applies when treated as a continuous variable.

Table 2. *Adjusted unstandardized regression coefficients reflecting the relationship between early household tobacco smoke exposure from ages 1.5 to 7 and pre-existing individual and family characteristics.*

Independent Variables	*Dependent variable* β(SE)
	Early childhood household smoke exposure in early childhood (ages 1.5 to 7)
1. Temperament (age 1.5)	-0.19(0.17)
2. Early cognitive skills (age 2)	-0.32(0.13)*
3. Sex (Boys = 1)	0.10(0.12)
4. Child exposure to fighting (age 3)	0.09(0.24)
5. Weight for gestational age	0.13(0.23)
6. Gestational smoke exposure (5 mo)	4.00(0.15)***
7. Gestational illicit drug exposure (5 mo)	-0.05(0.60)
8. Number of siblings at birth (5 mo)	0.36(0.17)*
9. Family income (5 mo)	0.15(0.16)
10. Family configuration (5 mo)	0.0.17(0.15)
11. Family functioning (age 1.5)	0.37(0.19)*
12. Maternal hostile parenting (age 1.5)	0.00(0.29)
13. Maternal depression (5 mo)	-0.22(0.19)
14. Parental antisocial behavior (5 mo)	0.48(0.22)*
15. Maternal education (5 mo)	-0.71(0.20)**
16. Maternal age at childbirth	-0.06(0.01)***

Adjusted *R* Square	0.49

Standard Errors are presented in parentheses. Asterisk reflect associated probability: *** $p < .001$, ** $p < .01$, and * $p \leq .05$.

Table 3a. *Correlation matrix reflecting the relationships between predictor variable outcome variables.*

Variables	1.	2.	3.	4.	5.	6.
1. Early childhood household smoke exposure in early childhood (ages 1.5 to 7)	1	_	_	_	_	_
2. Conduct problems (age 12)	0.16***	1	_	_	_	_
3. Proactive aggression (age 12)	0.10**	0.43***	1	_	_	_
4. Reactive aggression (age 12)	0.12***	0.30***	0.44***	1	_	_
5. School indiscipline (age 12)	0.18***	0.36***	0.32***	0.33***	1	_
6. Dropout risk (age 12)	0.19***	0.13***	0.13***	0.19***	0.40***	1

Standard Errors are presented in parentheses. Asterisk reflect associated probability: *** $p < .001$, ** $p < .01$, and * $p \leq .05$.

Table 3b. *Correlation matrix reflecting the relationships between predictor variable and preexisting individual and gestational characteristic.*

Variables	1.	2.	3.	4.	5.	6.	7.	8.
1. Early childhood household smoke exposure in early childhood (ages 1.5 to 7)	1	_	_	_	_	_	_	_
2. Temperament (age 1.5)	0.02	1	_	_	_	_	_	_
3. Early cognitive skills (age 1.5)	-0.09**	-0.07*	1	_	_	_	_	_
4. Sex (boys = 1)	0.01	0.06	0.02	1	_	_	_	_
5. Child exposure to fighting (age 3)	0.10**	0.03	0.00	-0.01	1	_	_	_
6. Weight for gestational age	0.07**	-0.03	0.05	0.02	0.01	1	_	_
7. Gestation smoke exposure	0.68***	-0.04	-0.04	-0.02	0.09**	0.09**	1	_
8. Gestational illicit drug exposure	0.10**	-0.02	-0.02	-0.02	0.04	0.01	0.13***	1

Standard Errors are presented in parentheses. Asterisk reflect associated probability: *** $p < .001$, ** $p < .01$, and * $p \leq .05$.

Table 3c. *Correlation matrix reflecting the relationships between predictor variable and preexisting family characteristics.*

Variables	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Early childhood household smoke exposure in early childhood (ages 1.5 to 7)	1	_	_	_	_	_	_	_	_	_
2. Number of siblings (5 months)	0.01	1	_	_	_	_	_	_	_	_
3. Family income (5 months)	0.19***	-0.01	1	_	_	_	_	_	_	_
4. Family configuration (5months)	0.09**	-0.02	0.30***	1	_	_	_	_	_	_
5. Family functioning (5months)	0.10**	0.06	0.12***	0.09**	1	_	_	_	_	_
6. Maternal hostile parenting (age 1.5)	0.03	0.01	0.11**	0.00	0.04	1	_	_	_	_
7. Maternal depression (5 months)	0.02	0.01	0.12***	0.13***	0.28***	0.06*	1	_	_	_
8. Parental antisocial behavior (5 months)	0.21***	0.04	0.11***	-0.04	0.04	0.03	0.07*	1	_	_
9. Maternal education (5months)	-0.25***	-0.06	-0.15***	-0.15***	-0.10**	-0.11***	-0.08**	-0.07*	1	_
10. Maternal age at childbirth	-0.23***	0.26***	-0.32***	-0.13***	0.02	-0.14***	-0.08*	-0.07*	0.23***	1

Standard Errors are presented in parentheses. Asterisk reflect associated probability: *** $p < .001$, ** $p < .01$, and * $p \leq .05$.

Table 3d. *Correlation matrix reflecting the relationships between outcome variables and preexisting individual and gestational characteristic.*

Variables	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. Conduct problems (age 12)	1	_	_	_	_	_	_	_	_	_	_	_
2. Proactive aggression (age 12)	0.41***	1	_	_	_	_	_	_	_	_	_	_
3. Reactive aggression (age 12)	0.30***	0.44***	1	_	_	_	_	_	_	_	_	_
4. School indiscipline (age 12)	0.36***	0.32***	0.33***	1	_	_	_	_	_	_	_	_
5. Dropout risk (age 12)	0.13***	0.13***	0.19***	0.40***	1	_	_	_	_	_	_	_
6. Temperament (age 1.5)	0.02	0.04	0.03	-0.01	0.03	1	_	_	_	_	_	_
7. Early cognitive skills (age 1.5)	-0.05	-0.04	-0.05	-0.10**	-0.12***	-0.07*	1	_	_	_	_	_
8. Sex (boys = 1)	0.14***	0.12***	0.23***	0.24***	0.16***	0.01	0.02	1	_	_	_	_
9. Child exposure to fighting (age 3)	0.06*	0.04	0.05	0.05	0.09**	0.03	0.00	-0.01	1	_	_	_
10. Weight for gestational age	-0.05	-0.01	0.02	0.00	0.00	-0.03	0.05	0.02	0.01	1	_	_
11. Gestation smoke exposure	0.07*	0.03	0.04	0.10**	0.12***	0.02	-0.04	-0.02	0,09**	0.09**	1	_
12. Gestational illicit drug exposure	0.04	0.00	0.02	0.01	0.01	0.04	-0.02	-0.02	0.04	0.01	0.13***	1

Standard Errors are presented in parentheses. Asterisk reflect associated probability: *** $p < .001$, ** $p < .01$, and * $p \leq .05$.

Table 3e. *Correlation matrix reflecting the relationships between outcome variables and preexisting family characteristics.*

Variables	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
1. Conduct problems (age 12)	1	_	_	_	_	_	_	_	_	_	_	_	_	_
2. Proactive aggression (age 12)	0.41***	1	_	_	_	_	_	_	_	_	_	_	_	_
3. Reactive aggression (age 12)	0.30***	0.44**	1	_	_	_	_	_	_	_	_	_	_	_
4. School indiscipline (age 12)	0.36***	0.32***	0.33***	1	_	_	_	_	_	_	_	_	_	_
5. Dropout risk (age 12)	0.13***	0.13***	0.19**	0.40***	1	_	_	_	_	_	_	_	_	_
6. Number of siblings (5 months)	0.08*	0.09**	0.01	0.10***	0.01	1	_	_	_	_	_	_	_	_
7. Family income (5 months)	0.08**	0.07*	0.07*	0.12***	0.18***	0.00	1	_	_	_	_	_	_	_
8. Family configuration (5 months)	0.06*	-0.02	0.06	0.14***	0.11***	-0.02	0.23***	1	_	_	_	_	_	_
9. Family functioning (5 months)	0.09**	0.03	0.05	0.02	0.02	0.06	0.13***	-0.09	1	_	_	_	_	_
10. Maternal hostile parenting (age 1.5)	0.04	0.06	0.03	0.07*	0.09**	0.01	0.10***	0.00	0.04	1	_	_	_	_
11. Maternal depression (5 months)	0.05	0.01	0.05	0.04	0.08**	-0.01	0.12***	0.13***	0.28***	0.06*	1	_	_	_
12. Parental antisocial behavior (5 months)	0.09**	0.08**	0.09**	0.13***	0.07*	0.04	0.11***	-0.04	0.04	0.03	0.07*	1	_	_

13. Maternal education (5 months)	-0.01	-0.09**	-0.08**	-0.14***	-0.31**	-0.06	-0.29***	-0.15***	-0.10**	-0.11***	-0.08**	-0.07*	1	_
14. Maternal age at childbirth	-0.05	-0.07*	-0.09**	-0.14***	-0.17***	0.26***	-0.32***	-0.13	0.02	-0.14***	-0.08**	-0.07*	0.23***	1

Standard Errors are presented in parentheses. Asterisk reflect associated probability: *** $p < .001$, ** $p < .01$, and * $p \leq .05$.

Table 3f. *Correlation matrix reflecting the relationships between control variables (preexisting individual, gestational, and family characteristics).*

Variables	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
1. Temperament (age 1.5)	1	_	_	_	_	_	_	_	_	_	_	_	_	_	_	_
2. Early cognitive skills (age 1.5)	-0.07**	1	_	_	_	_										
3. Sex (boys = 1)	0.01	0.02	1	_	_	_	_	_	_	_	_	_	_	_	_	_
4. Child exposure to fighting (age 3)	0.03	0.00	-0.01	1	_	_	_	_	_	_	_	_	_	_	_	_
5. Weight for gestational age	-0.03	0.05	0.02	0.01	1		_	_	_	_	_	_	_	_	_	_
6. Gestation smoke exposure	0.02	-0.04	-0.02	0.09**	0.09**	1	_	_	_	_	_	_	_	_	_	_
7. Gestational illicit drug exposure	0.04	-0.02	-0.02	0.04	0.01	0.13***	1	_	_	_	_	_	_	_	_	_
8. Number of siblings (5 months)	0.04	-0.03	-0.02	0.22***	-0.07*	-0.03	-0.02	1	_	_	_	_	_	_	_	_

9. Family income (5 months)	0.01	-0.08*	-0,02	0.10**	-0.04	0.15***	0.13***	0.00	1	_	_	_	_	_	_	_
10. Family configuration (5 months)	-0.03	-0.01	0.04	0.01	-0.02	0.08**	0.21***	-0.02	0.30***	1	_	_	_	_	_	_
11. Family functioning (5 months)	0.05	0.02	0.02	0.12***	0.01	0.07*	0.07*	0.06	0.13***	0.09**	1	_	_	_	_	_
12. Maternal hostile parenting (age 1.5)	0.02	-0.02	0.06*	-0.01	0.04	-0.01	0.02	0.01	0.11***	0.00	0.04	1	_	_	_	_
13. Maternal depression (5 months)	0.08*	0.00	0.03	0.04	0.01	0.02	0.05	-0.01	0.12***	0.13***	0.28***	0.06*	1	_	_	_
14. Parental antisocial behavior (5 months)	-0.01	0.02	0.02	0.09**	0.01	0.21***	0.16***	0.04	0.11***	-0.04	0.04	0.03	0.07*	1	_	_
15. Maternal education (5 months)	-0.05	0.11**	0.01	-0.14***	-0.03	-0.19***	-0.05	-0.06	-0.29***	-0.15***-	-0.10**	-0.11***	-0.08**	-0.07*	1	_
16. Maternal age at childbirth	-0.02	0.03	-0.04	0.02	-0.03	-0.12***	-0.06	0.26***	-0.32***	0.13***	0.02	-0.14***	-0.08*	-0.07*	023***	1

Standard Errors are presented in parentheses. Asterisk reflect associated probability: *** $p < .001$, ** $p < .01$, and * $p \leq .05$.

Table 4. *Adjusted and unadjusted imputed unstandardized regression coefficients reflecting the relationship between early household tobacco smoke exposure from ages 1.5 to 7 and self-reported conduct problems, school indiscipline, dropout risk, proactive aggression and reactive aggression at age 12.*

	Conduct problems (unadjusted)		Conduct problems (adjusted)	
	β(SE)	(95% CI)	β(SE)	(95% CI)
Early childhood household smoke exposure in early childhood (ages 1.5 to 7)	0.06(0.01)	(0.04 to 0.08)***	0.07(0.02)	(0.04 to 0.09)***
R square	0.03		0.06	
	Proactive aggression (unadjusted)		Proactive aggression (adjusted)	
	β(SE)	(95% CI)	β(SE)	(95% CI)
Early childhood household smoke exposure in early childhood (ages 1.5 to 7)	0.04(0.01)	(0.02 to 0.06)**	0.04(0.02)	(0.01 to 0.07)*
R square	0.01		0.03	
	Reactive aggression (unadjusted)		Reactive aggression (adjusted)	
	β(SE)	(95% CI)	β(SE)	(95% CI)
Early childhood household smoke exposure in early childhood (ages 1.5 to 7)	0.07(0.02)	(0.03 to 0.10)***	0.07(0.02)	(0.03 to 0.12)**
R square	0.01		0.07	
	School indiscipline (unadjusted)		School indiscipline (adjusted)	
	β(SE)	(95% CI)	β(SE)	(95% CI)

Environmental tobacco smoke exposure in early childhood	0.16(0.03)	(0.11 to 0.21)***	0.13(0.04)	(0.05 to 0.20)**
R square	0.01		0.13	
	Dropout risk (unadjusted)		Dropout risk (adjusted)	
	β(SE)	(95% CI)	β(SE)	(95% CI)
Environmental tobacco smoke exposure in early childhood	0.13(0.02)	(0.09 to 0.18)***	0.07(0.03)	(0.01 to 0.12)*
R square	0.03		0.15	

Adjusted model includes temperament, sex, early cognitive skills, child exposure to violence, weight for gestational age, gestational smoke exposure, gestational exposure to illicit drugs, number of siblings, family income, family configuration, family functioning, maternal hostile parenting, maternal depression, parental antisocial behaviour, maternal education, and maternal age at childbirth. Standard Errors are presented in parentheses. Asterisk reflect associated probability: *** $p < .001$, ** $p < .01$, and * $p \leq .05$

Table 5. *Adjusted and unadjusted non-imputed unstandardized regression coefficients reflecting the relationship between early household tobacco smoke exposure from ages 1.5 to 7 and self-reported conduct problems, school indiscipline, dropout risk, proactive aggression and reactive aggression at age 12.*

	Conduct problems (unadjusted)		Conduct problems (adjusted)	
	β(SE)	(95% CI)	β(SE)	(95% CI)
Environmental tobacco smoke exposure in early childhood	0.06(0.01)	(0.04 to 0.08)***	0.08(0.02)	(0.04 to 0.11)***
R square	0.03		0.05	
	Proactive aggression (unadjusted)		Proactive aggression (adjusted)	
	β(SE)	(95% CI)	β(SE)	(95% CI)
Environmental tobacco smoke exposure in early childhood	0.04(0.01)	(0.02 to 0.06)**	0.07(0.02)	(0.02 to 0.16)**
R square	0.01		0.07	
	Reactive aggression (unadjusted)		Reactive aggression (adjusted)	
	β(SE)	(95% CI)	β(SE)	(95% CI)
Environmental tobacco smoke exposure in early childhood	0.07(0.02)	(0.03 to 0.10)***	0.10(0.03)	(0.04 to 0.16)**
R square	0.01		0.07	
	School indiscipline (unadjusted)		School indiscipline (adjusted)	
	β(SE)	(95% CI)	β(SE)	(95% CI)
Environmental tobacco smoke exposure in early childhood	0.16(0.03)	(0.11 to 0.21)***	0.12(0.05)	(0.02 to 0.22)*
R square	0.01		0.09	

	Dropout risk (unadjusted)		Dropout risk (adjusted)	
	β(SE)	(95% CI)	β(SE)	(95% CI)
Environmental tobacco smoke exposure in early childhood	0.13(0.02)	(0.09 to 0.18)***	0.05(0.04)	(-0.02 to 0.13)^{NS}
R square	0.03		0.11	

Adjusted model includes temperament, sex, early cognitive skills, child exposure to violence, weight for gestational age, gestational smoke exposure, gestational exposure to illicit drugs, number of siblings, family income, family configuration, family functioning, maternal hostile parenting, maternal depression, parental antisocial behaviour, maternal education, and maternal age at childbirth. Standard Errors are presented in parentheses. Asterisk reflect associated probability**:** *** $p < .001$, ** $p < .01$, * $p \leq .05$, and ^{NS} $p > .05$.

CONCLUSION GÉNÉRALE

Plusieurs risques liés à l'exposition à la fumée secondaire sont désormais connus et de nombreuses études étayent la morbidité physique et psychologique associée à cette exposition. La fumée secondaire contient de nombreux composés pouvant être carcinogènes, altérer la santé cardiovasculaire et agir comme composé neurotoxique (Barnoya & Glantz, 2005; Grandjean & Landrigan, 2014; Pagani, 2014). Malgré des progrès significatifs en ce qui a trait la réduction des espaces où les individus peuvent être exposés à la fumée secondaire, ce sujet est toujours d'actualité et constitue une préoccupation de plusieurs décideurs publics. À ce jour, les études sur l'exposition à la fumée secondaire à l'enfance l'ont associée à des problématiques neurodéveloppementales (Braun et al., 2006) de même qu'à des difficultés comportementales (Bandiera et al. 2011). Ces problématiques peuvent s'avérer sévèrement handicapantes pour un individu en limitant sa participation sociale et son inclusion dans la société, justifiant ainsi l'intérêt que les professionnels de la santé publique doivent accorder à l'exposition à la fumée secondaire (Cohen& Piquero, 2009).

Puisque l'éthique en recherche et les droits de l'Homme restreignent toute expérimentation qui aurait trait à la fumée secondaire sur les humains, les chercheurs se trouvent dans l'obligation d'investiguer les corolaires d'une exposition à l'enfance par l'étude d'expériences naturelles grâce à des modèles associatifs. Ces modèles obligent à prendre en compte la notion de contrôle statistique. Le but de cette étude était donc de déterminer si l'exposition à la fumée secondaire à l'enfance était associée à des difficultés comportementales, sociales et occupationnelles à la préadolescence, lorsque l'exposition est isolée à l'aide de multiples variables de contrôle. Parallèlement, notre étude avait pour objectif de répliquer de précédentes études sur l'exposition à la fumée secondaire à l'enfance et les

comportements antisociaux ultérieurs tout en intégrant des mesures autorapportées afin d'éviter les biais possibles qui découlent de mesures rapportées par de tierces personnes (Loeber et al., 2009). Parallèlement, nous avons tenté de porter à douze ans l'âge auquel ces associations prospectives sont observées. Globalement, les individus exposés entre l'âge d'un an et demi et sept ans étaient plus susceptibles de commettre des actes antisociaux. De plus, ils rapportaient davantage de difficultés fonctionnelles dans la sphère scolaire.

La prise de décision et la socialisation sont des tâches qui requièrent des processus mentaux sophistiqués et un haut niveau de coordination entre différents systèmes neurologiques (Barkley, 2012). Par exemple, une situation de résolution de problème implique qu'un individu doit solliciter son système moteur pour établir un contact visuel (Grant & Spivey, 2003) et ses fonctions exécutives dans le but de déterminer les comportements qui seront les plus efficaces dans la résolution du problème (Wiley & Jarosz, 2012). Par ailleurs, la socialisation et la prise de décision requièrent une flexibilité cognitive qui permet à un individu d'adopter différents comportements lorsqu'il est confronté à une situation où son répertoire comportemental est insuffisant (Armbruster, Ueltzhöffer, Basten & Fiebach, 2012). Cependant, cette fonction exécutive s'avère déficitaire chez les individus antisociaux (Villa-Ballo et al., 2015). Le contrôle de l'impulsivité joue aussi un rôle dans la socialisation et la prise de décision. Ce contrôle permet aux individus de traiter l'information provenant de l'environnement adéquatement avant d'émettre une réponse comportementale, permettant ainsi de réagir de façon appropriée lorsque les situations sociales s'avèrent complexes (Franken, van Strien, Nijs & Muris, 2008). En tirant des conclusions des modèles utilisant des animaux, il est possible d'émettre l'hypothèse selon laquelle les individus exposés à la fumée

secondaire pourraient présenter des altérations neurologiques dans les systèmes gérant la transmission de l'information entre divers systèmes cognitifs, des différences au sein des tissus cérébraux associés au fonctionnement exécutif ou dans les circuits responsables de l'apprentissage (Paus et al., 2008; Torres et al., 2012; Pinheiro et al., 2015). Ces différences anatomiques et physiologiques pourraient expliquer la plus grande propension des participants de notre étude exposés à la fumée secondaire à rapporter l'usage de comportements moins fonctionnels au plan social et occupationnel (Yang & Raines, 2009; Buckholtz et al., 2010.; Zhang et al., 2014). Cela se reflèterait par des comportements transgressant les règles sociales, une plus grande utilisation de comportements agressifs et des difficultés dans des environnements où les attentes sociales sont élevées, tel que l'école (Zuddas, 2014).

Malgré les limites inhérentes des modèles associatifs, qui obligent à tirer des conclusions sur la base de modèles expérimentaux basés sur des animaux, nos résultats suggèrent que les décideurs publics et les professionnels de la santé mettent en place des stratégies permettant de diffuser ce constat : l'exposition à la fumée secondaire à l'enfance est associée à des difficultés comportementales ultérieures et, à ce jour, aucune exposition ne peut être qualifiée de sécuritaire.

L'impact attribué aux politiques sociales

Plusieurs outils législatifs ont été mis en place afin de diminuer l'exposition à la fumée secondaire dans la population générale. Une des actions mises massivement de l'avant au cours des dernières années est l'utilisation de la loi pour bannir la fumée du tabac des endroits publics et privés (Pickett et al, 2006). Cependant, évaluer l'effet direct des lois à large spectre

sur l'exposition à la fumée secondaire s'avère difficile au niveau empirique vu les nombreuses contraintes méthodologiques (notamment la diffusion des connaissances scientifiques, les programmes d'intervention visant l'exposition à la fumée secondaire implantés parallèlement aux lois ou les effets des programmes visant la cessation de la consommation du tabac) (Callinan, Clarke, Doherty & kelleher, 2010). Cependant, de nombreuses études populationnelles ont tenté d'évaluer l'effet attribué aux efforts législatifs dirigés vers la diminution de l'exposition à la fumée secondaire. Entre 1988 et 2002, une diminution de 70 % de la quantité totale de la cotinine sanguine, un dérivé de la nicotine indiquant une exposition au tabac, a été observée au sein d'un échantillon représentatif de 29 849 Américains non-fumeurs (Pirkle, Bernet, Caudill, Sosnoff & Pechacek, 2006). Ce changement coïncide avec la publication, aux États-Unis, des premiers rapports institutionnels décrivant les risques associés à l'exposition à la fumée secondaire de même qu'au début des initiatives parlementaires visant à réduire l'exposition à la fumée secondaire par le biais de la loi (Pirkle et al., 2006). Par ailleurs, la portée des lois est associée à l'ampleur de l'exposition. Dans un échantillon de plus de 5 000 individus provenant de diverses régions des États-Unis, les individus vivant dans des comtés avec des lois à grande portée (lois votées au niveau de l'État) avaient des niveaux de cotinine sanguine significativement plus faible que ceux des comtés avec des lois limitées ou sans loi (Pickett et al., 2006). Par ailleurs, les individus provenant d'états avec des lois limitées (lois votées au niveau municipal) avaient des niveaux de cotinine moins élevés que ceux habitant dans des états sans loi sur la présence de fumée secondaire dans les espaces publics. À un niveau plus local, des chercheurs ont comparé les différences entre des individus fréquentant différents collèges avec des politiques différentes envers la présence de fumée secondaire (types de règlements par ordre croissant : interdiction de fumer dans le collège

seulement, permission dans des zones désignées à l'extérieur seulement, fumée du tabac complètement bannie sur les terrains du collège, et produits du tabac complètement bannis sur les terrains du collège) (Fallin, Roditis & Glantz, 2015). Leurs résultats suggèrent que des politiques plus restrictives quant aux produits du tabac sont associées à une réduction de l'exposition à la fumée secondaire chez les collégiens. Par ailleurs, des lois prohibant la fumée du tabac dans les voitures, un espace privé, tendent à démontrer une certaine réduction du nombre d'enfants exposés à la fumée secondaire dans les voitures (Elton-Marshal, Leatherdale, Driezen, Azagba & Burkhalter, 2015). Cependant, leurs résultats jettent un doute quant à la capacité à faire respecter ces lois puisque certains territoires, malgré des lois qui prévoient des conséquences telles que des amendes, ne voient pas le nombre d'enfants exposés à la fumée secondaire dans un véhicule diminuer.

Ces diminutions, attribuées à la mise en place de dispositifs législatifs, sont aussi associées à certains bénéfices en termes de santé physique. La mise en place de lois prohibant la consommation de cigarettes dans les lieux de travail et les espaces publics est associée à une baisse du nombre d'infarctus et d'hospitalisation pour des problèmes cardiovasculaires (Lightwood & Glantz, 2009). De plus, les bénéfices estimés de ces lois augmenteraient en fonction du nombre d'années écoulées depuis la mise en place de ces lois (Lightwood & Glantz, 2009). Cependant, d'autres études seront nécessaires pour estimer si ces lois sont ou seront associées à une réduction de la morbidité psychiatrique à l'enfance et la préadolescence.

Les programmes d'intervention ciblant l'exposition à la fumée secondaire à l'enfance

Les efforts déployés par les décideurs publics et les professionnels de la santé publique ne se bornent pas seulement à des outils législatifs. En ce sens, plusieurs initiatives et programmes éducatifs ont été évalués afin de déterminer leur capacité à réduire l'exposition des enfants à la fumée secondaire, et ce dans diverses situations d'intervention. Chi et al. (2015) ont développé un programme s'adressant aux femmes enceintes non-fumeuses afin de leur fournir du contenu éducatif et de leur enseigner des techniques pour confronter les individus fumant au domicile. Les résultats de l'évaluation de ce programme indiquent une grande diminution du nombre de foyers où il y a présence de fumée secondaire dans le groupe expérimental. Par ailleurs, les futures mères ont rapporté se sentir plus autonomisées, avoir plus de connaissances sur les effets de la fumée secondaire, avoir un meilleur sentiment d'auto-efficacité et utiliser plus de comportements visant à se protéger de la fumée secondaire. Eakin et al. (2014) ont tenté d'évaluer l'efficacité de l'ajout de quatre courtes entrevues motivationnelles sur la fumée secondaire au programme Head Start afin de réduire l'exposition à la fumée secondaire au domicile. Les participants faisant partie du groupe Head Start + entrevues ont vu la quantité totale de nicotine aérienne diminuer au domicile lorsque comparés au groupe Head Start seulement. Par ailleurs, la proportion de parents rapportant avoir cessé de fumer et avoir appliqué une interdiction totale de fumer au domicile augmenta significativement par rapport au groupe Head Start seulement. Utilisant un modèle d'intervention systémique, Blanch et al. (2013) ont tenté d'évaluer les effets d'un programme avec une composante adolescent, parents et école ayant pour but de diminuer l'exposition des enfants à la fumée secondaire. Leurs résultats suggèrent que le programme a entraîné une diminution de l'exposition à l'école, au

domicile et dans les transports contrairement au groupe contrôle où seule une diminution au domicile a été observée.

Des interventions ont aussi été mises en place pour des populations plus vulnérables. Un programme d'intervention destiné aux mères ayant un enfant en voie de recevoir son congé des soins intensifs néonataux, consistant en une entrevue de dix minutes sur l'asthme et les risques associés à la fumée secondaire, démontre une diminution de l'exposition à la nicotine huit mois après avoir obtenu congé de l'hôpital (Blaakman et al., 2015). Par ailleurs, ces familles, lorsque comparées au groupe contrôle, avaient plus de chances de vivre dans un domicile sans fumée et les enfants avaient moins de contacts directs avec des fumeurs lorsque ces derniers fumaient. Finalement, un programme, destiné aux enfants atteints d'un cancer, démontre une augmentation du nombre de familles vivant dans un domicile sans fumée suite à une intervention comprenant trois séances d'éducation sur la fumée secondaire et deux rappels téléphoniques (Peck, Tyc, Huang & Zhang, 2015).

Un nouveau de paradigme en intervention ?

Plusieurs modèles et programmes d'intervention curative ont été développés afin de diminuer l'incidence ou la gravité des comportements antisociaux et ses conséquences. Ces différents programmes démontrent des niveaux d'efficacité appréciables, mais sans toutefois parvenir à assurer un fonctionnement normatif à l'ensemble des individus présentant des traits antisociaux (Hoffman, Asnaani, Vonk, Sawyer & Fang, 2012; Schwalbe, Gearing, MacKenzie, Brewer & Ibrahim, 2012).

Ce constat mena au développement des programmes de prévention qui ciblent les enfants présentant des marqueurs précoces les prédisposant aux comportements antisociaux et à ses multiples corolaires. Plusieurs initiatives, telles que Head Start (Webster-Stratton, 1998), ces Années incroyables (Webster-Stratton, Jamila-Reid & Stoolmiller, 2008) ou Tools of the Mind (Barnett et al., 2008), visent à aider les enfants ou leurs parents à acquérir les outils de base afin de combler des déficits présents en bas âge qui sont associés à la réussite scolaire et qui sont reconnus comme des facteurs protecteurs des comportements antisociaux. Ces programmes visent donc à compenser pour un manque de stimulation en bas âge pouvant mener à des problèmes d'adaptation ultérieurs.

Plus largement, la majorité des programmes d'intervention en prévention se basent sur le paradigme de la stimulation. Les programmes d'intervention comportementale intensive basés sur l'analyse appliquée du comportement visent à stimuler en bas âge les enfants ayant un trouble du spectre de l'autisme ou une déficience intellectuelle afin de favoriser l'apprentissage et l'autonomie fonctionnelle (Lovaas, 1987). Les programmes basés sur une approche cognitive comportementale cherchent quant à eux à outiller les enfants ou les adolescents avec des techniques leur permettant de faire face à leurs difficultés (Turgeon & Parent, 2012). Les interventions basées sur les fonctions exécutives, telles que Tools of the Mind, tentent d'améliorer l'attention, l'impulsivité et les capacités mnésiques des enfants à une période de grande plasticité cérébrale (Barnett et al., 2008). Cependant, ces interventions ne ciblent qu'une partie de la problématique.

Les interventions préventives devraient intégrer la notion de « protection » du cerveau lorsqu'elles visent la stimulation cognitive (Shonkoff, 2011). Plusieurs situations, telles que l'exposition à des composés neurotoxiques comme la fumée secondaire ou le stress chronique, posent un stress sur le développement neurologique et sont associées à plusieurs problématiques de santé mentale émergeant à l'adolescence ou à l'âge adulte (Greandjean & Landrigan 2014; Shonkoff, 2011 respectivement). L'idée n'est pas de limiter ou d'arrêter d'investir dans des programmes de stimulation. Cependant, l'objectif d'un programme de stimulation est d'enseigner ou de consolider des stratégies permettant un meilleur fonctionnement. Ces deux processus, pour fonctionner de façon optimale, devraient inclure des stratégies qui visent à protéger le cerveau des éléments environnementaux neurotoxiques afin de s'assurer qu'il soit en mesure d'intégrer et utiliser les stratégies enseignées (Shonkoff, 2011).

Forces et limites de la présente étude

L'objectif de la présente étude était d'observer les associations prospectives entre l'exposition à la fumée secondaire à l'enfance et les difficultés psychosociales à la préadolescence. Ces résultats s'ajoutent à un corps de littérature observant nombre d'associations au sein de divers échantillons populationnels entre l'exposition à la fumée secondaire et les comportements antisociaux. Cette étude s'avère novatrice pour plusieurs raisons. Premièrement, ce modèle longitudinal prospectif permet d'éviter la question de la directionalité et d'observer des associations à long terme. Deuxièmement, l'intégration de plusieurs composés tératogènes neurotoxiques dans les analyses permet d'isoler statistiquement la contribution de l'exposition à la fumée secondaire. Plus spécifiquement, contrôler pour l'exposition aux composés du tabac

durant la gestation permet d'observer la contribution unique de l'exposition postnatale aux composés de la fumée secondaire. Troisièmement, l'utilisation de données autorapportées pour mesurer les comportements antisociaux permet d'obtenir une meilleure fiabilité quant aux résultats comparativement à des données rapportées par des parents ou des enseignants (Loeber et al., 2009). Parallèlement, ces résultats autorapportés concordent avec les associations précédemment observées avec des données rapportées par les enseignants (voir Pagani & Fitzpatrick, 2013) et par les parents (voir Rükinger et al. 2010) à l'âge de dix ans, renforçant ainsi les résultats de cette étude ainsi que ceux précédemment rapportés vu la stabilité des associations malgré le passage du temps.

Les résultats de cette étude suggèrent que l'exposition à la fumée secondaire est associée aux comportements antisociaux, à l'agressivité et au fonctionnement académique. Cependant, plusieurs limites quant à l'interprétation de ces résultats sont à mentionner. Premièrement, la mesure d'exposition ne permet pas de quantifier l'exposition réelle et donc de discriminer les enfants hautement exposés des enfants faiblement exposés. De plus, cette dernière est unidimensionnelle. Elle ne permet de mesurer qu'une seule source d'exposition, soit l'exposition au domicile. Par ailleurs, plusieurs mesures, étant autorapportées, sont possiblement teintées par un biais de désirabilité sociale pouvant affecter leur exactitude (King & Bruner, 2000). Ce type de biais pourrait expliquer les faibles fréquences observées chez les variables dépendantes, malgré le fait qu'elles soient similaires aux fréquences observées précédemment dans cet échantillon (Pagani & Fitzpatrick, 2014) ou d'autres échantillons (Kokko, Tremblay, Lacourse, Nagin & Vitaro, 2006; Vitaro, Brendgen, Girard, Boivin, Dionne & Tremblay, 2015). Par ailleurs, contrairement aux mesures autorapportées

d'exposition à la fumée secondaire (Avila-Tang et al., 2013), les mesures autorapportées d'exposition prénatale aux composants du tabac sont considérées comme controversées, vu leur faible validité (Florescu, Ferrence, Einarson, Selby, Soldin & Koren, 2009). Parallèlement, plusieurs mesures incluses dans les variables de contrôle sont rétrospectives, donc basées sur la mémoire des participants. Un autre facteur limitant la portée des résultats est l'attrition (environ 53 % de l'échantillon) relativement élevée. Cependant, des méthodes d'imputation multiple ont été utilisées afin de réduire l'impact sur la validité des données (Schaffer, 1999). De plus, la faible cohérence interne des instruments de mesure des comportements antisociaux et du risque de décrochage peut occasionner des problèmes de fidélité et, possiblement, de validité. De faibles coefficients de cohérence interne, étant des indicateurs de l'homogénéité des items des échelles, pourraient indiquer que certains items d'une même échelle mesurent différents construits (Streiner, 2003). Néanmoins, la cohérence interne est une mesure sensible au nombre d'items dans une échelle. Vu le faible nombre d'items dans certaines échelles (p.ex. : L'agressivité proactive comprend 4 items), ces résultats pourraient être attribuables à la taille des échelles plutôt qu'un réel problème de cohérence interne (Streiner, 2003). Finalement, le type de devis utilisé ne permet pas de valider les hypothèses imputant un rôle neurotoxique à l'exposition à la fumée secondaire résidentielle à la petite enfance, et ce malgré l'apport des études portant sur des animaux (Van Ewijk et al., 2015). Ce type d'études, bien qu'informatives, ne se généralise pas automatiquement à l'Homme vu les différences interespèces quant à la métabolisation des composés toxiques (Reagan-Shaw, Nihal & Ahmad, 2008). De plus, leur nature expérimentale limite leur validité externe, limitant d'autant plus la comparaison à une expérience « naturelle » telle que celle utilisée dans la présente étude. Ces biais découlent de facteurs tels que l'homogénéité des

individus dans les modèles expérimentaux, l'induction d'une exposition à la fumée secondaire différente par rapport à celle vécue chez une population humaine de même que l'adéquation entre l'administration de la variable indépendante et de la mesure des variables dépendantes (p.ex. : les altérations neurophysiologiques) pouvant difficilement correspondre au développement humain (Van der Worp et al., 2010).

D'autres études seront nécessaires afin de confirmer les associations observées. Plusieurs ajouts et modifications méthodologiques pourraient s'avérer intéressants. La mise à l'épreuve de ce modèle par des méthodes plus intégratives, par exemple une modélisation par équations structurelles, pourrait s'avérer intéressante. Ce type d'analyse permettrait de prendre en compte les interrelations entre les différentes variables et leur représentativité au sein d'un même construit latent (Tomarken & Waller, 2004). Ce type d'analyse permet aussi de réduire l'erreur de type I attribuée à l'utilisation de plusieurs régressions linéaires multiples (Tomarken & Waller, 2004). Néanmoins, ce type d'erreur peut être estimé et corrigé sans l'utilisation d'équations structurelles, par l'entremise de la Correction de Bonferroni par exemple (Simes, 1986). L'utilisation de marqueurs biologiques de l'exposition à la fumée secondaire pourrait améliorer la validité des données et permettre de quantifier l'exposition totale des individus. De plus, d'autres études pourraient se pencher sur ces associations durant ou après la puberté afin de vérifier si ces associations se maintiennent ultérieurement. En définitive, d'autres recherches utilisant des devis méthodologiques plus robustes ou l'avancement des technologies d'imagerie cérébrales pourront trancher quant au caractère neurotoxique de la fumée secondaire. Dans l'attente des résultats de ces futures investigations empiriques, ces résultats soutiennent qu'aucune exposition à la fumée secondaire ne peut être

considérée comme sans risque à l'enfance. Ils soutiennent la mise en place de lois visant à réduire l'exposition à la fumée secondaire chez les jeunes enfants. Ils suggèrent aussi la mise en place de programmes d'intervention visant à réduire l'exposition à la fumée secondaire de même que des stratégies éducatives nationales visant à inciter les fumeurs à ne pas fumer dans des espaces clos fréquentés par de jeunes enfants.

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