Article

# Minor Physical Anomalies and Family Adversity as Risk Factors for Violent Delinquency in Adolescence

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**Objective:** Minor physical anomalies are considered indicators of disruption in fetal development. They have been found to predict behavioral problems and psychiatric disorders. This study examined the extent to which minor physical anomalies, family adversity, and their interaction predict violent and nonviolent delinquency in adolescence.

**Method:** Minor physical anomalies were assessed in a group of 170 adolescent boys from low socioeconomic status neighborhoods of Montréal. The boys had been enrolled in a longitudinal study since their kindergarten year, when an assessment of family adversity had been made on the basis of familial status and the parents' occupational prestige, age at the birth of the first child, and educational level. Adolescent delinquency was measured by using self-reported questionnaires and a search of official crime records.

**Results:** Results from logistic regression analyses indicated that both the total count of minor physical anomalies and the total count of minor physical anomalies of the mouth were significantly associated with an increased risk of violent delinquency in adolescence, beyond the effects of childhood physical aggression and family adversity. Similar findings were not found for nonviolent delinquency.

**Conclusions:** Children with a higher count of minor physical anomalies, and especially a higher count of anomalies of the mouth, could be more difficult to socialize for different and additive reasons: they may have neurological deficits, and they may have feeding problems in the first months after birth. Longitudinal studies of infants with minor physical anomalies of the mouth are needed to understand the process by which they fail to learn to inhibit physical aggression.

*(Am J Psychiatry 2000; 157:917–923)*

Serious violent delinquency is at its peak during late adolescence and early adulthood (1). However, violent behaviors do not appear suddenly during the teen years. Longitudinal studies have shown that childhood disruptive behaviors are among the best predictors of delinquency and adult antisocial personality (2, 3). These findings suggest that violent delinquency has precursors early in life (4). Therefore, etiological factors early in life should logically be studied. Precursors such as family context and perinatal complications are among the earliest factors that could influence behavioral development (5, 6).

In their elaboration of a theoretical framework for the study of violence, Raine et al. (7) suggested that biological factors predispose individuals to psychopathology specifically in adverse environments. Poor environmental conditions are expected to exacerbate physiological predispositions toward psychopathology, whereas favorable environmental conditions are expected to compensate for them. Few studies have simultaneously considered environmental conditions and perinatal risk factors. One of those rare studies (8) showed that individuals who had neuromotor deficits at age 1 year and grew up in unstable family environments were more at risk of becoming criminals than those with only one of these risk factors. Another study (9) showed that violent crimes were predicted by the interaction between maternal rejection at age 1 and birth complications. Taken together, these studies suggest that the development of violent behavior may be influenced by a biosocial process that takes place during the very first years of life.

Birth complications are not the earliest biological risk factors for behavioral disorder. During pregnancy, the fetus is exposed to various influences that could negatively affect its development. These influences are partly responsible for the development of minor physical anomalies, trivial aberrations that can be found on many parts of the body. Minor physical anomalies are considered indicators of fetal developmental disruption. The specific origin of these anomalies is not yet fully understood, but the interaction between environmental factors and genetic determinants is their most likely cause (10, 11). The central nervous system (CNS) may also be affected by factors responsible for minor physical anomalies because the development of the CNS is concurrent with the development of the organs that show the minor anomalies. Because neurological impairments are known to be associated

with behavioral problems (12), minor physical anomalies may well reflect CNS risk factors for the development of behavioral disorders.

Studies have shown that a higher count of minor physical anomalies is found in groups of individuals with psychiatric diagnoses such as schizophrenia (13), autism (14), and hyperactivity (15, 16) and among individuals with aggressive behaviors (17). Waldrop and Halverson (18) argued that it is the total number, and not any specific minor physical anomaly, that predicts behavior problems. An approach that associates the location of minor physical anomalies with behavioral disorders may also be informative. For example, a higher incidence of anomalies of the mouth has been linked with psychosis in three studies (19–21). Studies that have focused on the association between minor physical anomalies and aggressive behavior have not examined specific links with parts of the body where minor physical anomalies occur. Further, minor physical anomalies alone may not be sufficient to adequately predict violent delinquency. Current biosocial models of psychopathological development suggest that clear prediction of risk may be found when looking at minor physical anomalies in conjunction with environmental factors (7).

In support of this view, three studies have reported that deprived psychosocial environments influence the link between the total count of minor physical anomalies and behavioral disorders (22–24). In these studies of biosocial interactions, characteristics of the social environment were measured during adolescence or concurrently with outcome. In her developmental theory of antisocial behaviors, Moffitt (25) suggested that the interaction between early environmental characteristics and neuropsychological deficits engenders chronic violent antisocial behavior. Because antisocial behaviors tend to emerge from a childhood pattern of chronic disruptive behaviors, one would expect that the positive or negative impact of the environment on the development of violent behavior in individuals with CNS malformations could be traced back to childhood. Further, Moffitt suggested that this interaction could be a specific etiological mechanism for violent offenses as opposed to nonviolent offenses. Evidence supporting this assumption has been reported in empirical studies (9, 22).

The first aim of the study reported here was to test the hypothesis that the cumulative incidence of minor physical anomalies, family adversity during the preschool years as an index of family disadvantage, and their interaction predict violent delinquency at the end of adolescence in a study group of inner-city boys. The second aim was to identify the specific anomalies involved in violent delinquency. The third aim was to test the hypothesis that those predictions are specific to violent delinquency as opposed to nonviolent delinquency.

## Method

### *Sample*

Participants were involved in an ongoing longitudinal study of boys from lower socioeconomic status areas in Montréal (26). Fifty-three schools were selected because of the students' low score on a socioeconomic index that was based on family earnings, occupational prestige, and parents' schooling (27). Kindergarten teachers were asked to rate the behaviors of the boys in their classes. Only white, French-speaking boys whose mother and father were born in Canada were included in the sample (N=1,037) in order to have a culturally homogeneous group.

Data on minor physical anomalies were collected during a laboratory visit of a study group of 177 boys when they were 14 years of age. The boys were selected on the basis of teacher ratings of physical aggression and anxiety from age 6 to 12 (see reference 28) by using the Social Behavior Questionnaire (29). Physical aggression was measured with three items: fights with other children; kicks, bites, hits other children; and bullies other children. The anxiety scale comprised five items: fearful, distressed, worried, solitary, and cries. Each item was scored on a frequency scale ranging from 0 to 2. The Cronbach value for internal consistency for the anxiety scale was 0.76 when the boys were ages 6, 10, 11, and 12. For the physical aggression scale, the mean Cronbach value when the boys were between ages 6 and 12 was 0.84 and ranged from 0.78 to 0.87. The boys had to meet at least one of the following four overlapping criteria to be selected for the study group: 1) high aggressive or anxious behavioral pattern indicated by scores above the 70th percentile at age 6 and at least 2 other years, 2) low aggressive or anxious behavioral pattern indicated by scores below the 70th percentile at all assessments, 3) pattern of late-onset physical aggressiveness or anxiety indicated by scores above the 70th percentile only at age 12, and 4) prior visits to the laboratory. Compared to the rest of the sample (N=860), the study group was more aggressive, hyperactive, inattentive, and anxious in kindergarten, but was similar in socioeconomic characteristics and in prosociality. The behavioral differences were due to an overrepresentation of aggressive and anxious boys in the study group as a result of the selection criteria. Because all boys were recruited in regular schools, none of them had mental retardation, severe intellectual deficits, or significant physical handicaps. Data on minor physical anomalies and delinquent behaviors were available for 170 boys, 96% of the study group. Written informed consent was obtained from both the boys and their parents.

### *Assessment of Minor Physical Anomalies*

Minor physical anomalies were assessed as part of an extensive evaluation of body characteristics when the boys came to the laboratory. The 18 minor physical anomalies from the Waldrop scale were measured (30). A frequency scale was used in the analyses because it involved less judgment bias and was highly correlated with weighted scores for the anomalies (15, 18, 30). The anomalies were located in six different areas of the body: the mouth, ears, eyes, head, hands, and feet. The total count of these anomalies has been reported to be stable from birth to adolescence (31).

The frequency for one of the anomalies (a big gap between the first and the second toes) was exceptionally elevated (Table 1). The feet were the only anatomical region for which the cumulative count of minor physical anomalies was not correlated to the total count of anomalies and was negatively associated with anomalies of the eyes (pairwise r=–0.16, N=175) and with anomalies of the hands (pairwise r=–0.15, N=176). Our study group was drawn from a culturally homogeneous population that is known to have a relatively homogeneous gene pool (32). Thus, the toe gap may be characteristic of that population. We therefore decided to exclude this anomaly from the total count of minor phys-

**TABLE 1. Minor Physical Anomalies in 44 Violent Delinquent Adolescent Boys and 42 Nonviolent Delinquent Adolescent Boys**

| Location and Nature of Minor Physical Anomaly | Total Study Group (N=170)[a] | | Violent Delinquents (N=44)[b] | | Nonviolent Delinquents (N=42)[b] | |
|---|---|---|---|---|---|---|
| | N | % | N | % | N | % |
| Mouth | | | | | | |
| High steepled palate | 37 | 21.8 | 12 | 27.3 | 10 | 23.8 |
| Furrowed tongue | 31 | 18.2 | 10 | 22.7 | 7 | 16.7 |
| Smooth-rough spots | 67 | 39.4 | 23 | 52.3 | 20 | 47.6 |
| Ears | | | | | | |
| Malformed | 18 | 10.6 | 1 | 2.3 | 2 | 4.8 |
| Asymmetrical | 57 | 33.5 | 19 | 43.2 | 15 | 35.7 |
| Low seated | 47 | 27.6 | 10 | 22.7 | 9 | 21.4 |
| Soft and pliable | 57 | 33.5 | 17 | 38.6 | 13 | 31.0 |
| Adherent lobes | 64 | 37.6 | 15 | 34.1 | 15 | 35.7 |
| Eyes | | | | | | |
| Epicanthus | 80 | 47.3 | 21 | 47.7 | 23 | 54.8 |
| Hypertelorism | 15 | 8.8 | 6 | 13.6 | 4 | 9.5 |
| Head | | | | | | |
| Electric fine hair | 12 | 7.2 | 5 | 11.4 | 3 | 7.1 |
| Two or more hair whorls | 61 | 35.9 | 20 | 45.5 | 10 | 23.8 |
| Large circumference | 25 | 15.1 | 6 | 14.0 | 7 | 17.1 |
| Hands | | | | | | |
| Fifth finger curved | 43 | 25.3 | 14 | 31.8 | 12 | 28.6 |
| Single palmar crease | 7 | 4.1 | — | — | 1 | 2.4 |
| Feet | | | | | | |
| Third toe length | — | — | — | — | — | — |
| Partial syndactyly | 6 | 3.5 | 1 | 2.3 | — | — |
| Big gap between toes | 113 | 66.5 | 22 | 50.0 | 28 | 66.7 |

[a] Data missing on epicanthus for one participant, on electric fine hair for three participants, and on large circumference of the head for four participants.
[b] Data missing on large circumference of the head for one participant.

ical anomalies. A similar decision was made in a previous study when anomalies were found to be the norm rather than an exception (33). Because of this exclusion, we did not examine minor physical anomalies of the feet in analyses of separate anatomical regions.

### *Family Adversity*

Seven socioeconomic indices were used to create an index of family adversity (29). These indices were mother's and father's occupational prestige, mother's and father's age at birth of their first child, mother's and father's education level, and familial status. The accumulation of these different variables has been shown to increase the risk of behavioral disorders by creating stressful rearing conditions (34). Occupational prestige reflected a socioeconomic index of jobs in Canada (35), and familial status referred to whether both biological parents were living with the boy. Information on these indices was collected during a telephone interview with the mother at the end of the boy's kindergarten year. Because we hypothesized that environmental conditions have an impact on behaviors early in life, only the measure of adversity when the boy was age 6 was used, as it represented the earliest index of the socioeconomic conditions in which the boy grew up. Except for familial status, all indices were given a score of 1 if they were below the 30th percentile in the present sample and a score of 0 if they were above the 30th percentile. For familial status, a score of 1 was given if the boy was not living with his two biological parents during his kindergarten year. The maximum family adversity score was 7 for a boy living with one biological parent and a stepparent and was 4 for a boy living with one parent only. Therefore, the total family adversity score was divided by 7 if the boy was living with two parents and by 4 if he was living with one parent. The study group scores ranged from 0 to 1 (mean=0.34, SD=0.23), with higher scores representing more adversity. Study group members did not differ from the rest of the sample on each family adversity index. This composite measure of the degree of adversity in families when the boy was age 6 was shown to be predictive of a stable level of childhood physical aggression in the large sample and was highly correlated with family adversity scores when the boy was age 12 (r=0.85) (5). In addition, this index was related to verbal learning difficulties within this study group (28) and was associated with childhood externalizing disorders in a sample of more than 3,000 French-speaking children (36). The scores were standardized for easier interpretation of the results.

### *Delinquent Behavior*

At age 17, the boys were asked to respond to 27 items measuring delinquent behaviors that took place at home, at school, and with their friends (26). Four scales were created on the basis of items measuring physical aggression, theft, vandalism, and substance use. The Cronbach values for internal consistency of the four scales measured at age 17 were 0.78, 0.87, 0.73, and 0.82, respectively. Scores on the four scales at ages 16 and 17 were significantly correlated (r=0.62 [N=767], 0.67 [N=767], 0.45 [N=764], and 0.72 [N=766], respectively). Self-reported delinquency scales virtually identical to the one used in this study have been reported to have both concurrent and predictive validity (37). The score on the first scale (physical aggression) was considered a measure of self-reported violent delinquency, and the scores on the last three scales (theft, vandalism, and substance use) were summed to represent self-reported nonviolent delinquency. Data at age 16 were used for five participants whose data at age 17 were missing. Thirty-eight violent delinquents and 40 nonviolent delinquents were identified by using a 75th percentile cutoff point, given the skewed distributions of the two self-reported delinquency scales.

Criminal status was determined by a search of the criminal records for all boys in the sample as of age 19. Crimes were classified as violent or nonviolent according to the Canadian criminal code. Violent crimes such as illegal possession of a weapon, animal cruelty, and violent threats were found in 67 boys of the larger

**TABLE 2. Prediction of Violent and Nonviolent Deliquency in Adolescent Boys According to Logistic Regression Analyses of Physical Anomalies, Childhood Physical Aggression and Anxiety, and Familiy Adversity**

| Regression Analysis and Constituent Covariables | Violent Delinquency (N=44) | | | Nonviolent Delinquency (N=42) | | |
|---|---|---|---|---|---|---|
| | B | SE | Odds | B | SE | Odds |
| Regression 1: total anomalies | | | | | | |
| Childhood physical aggression | 0.28 | 0.15 | 1.3* | 0.05 | 0.15 | 1.1 |
| Childhood anxiety | –0.13 | 0.10 | 0.9 | –0.21 | 0.11 | 0.8* |
| Family adversity at age 6 | 0.36 | 0.19 | 1.4* | 0.03 | 0.19 | 1.0 |
| Total minor physical anomalies | 0.20 | 0.10 | 1.2* | –0.03 | 0.10 | 1.0 |
| Regression 2: anomalies of the mouth[a] | | | | | | |
| Family adversity at age 6 | 0.31 | 0.19 | 1.4 | 0.03 | 0.19 | 1.0 |
| Minor physical anomalies of the mouth | 0.52 | 0.24 | 1.7* | 0.23 | 0.24 | 1.3 |
| Regression 3: anomalies of the ears[a] | | | | | | |
| Family adversity at age 6 | 0.38 | 0.18 | 1.5* | 0.06 | 0.19 | 1.1 |
| Minor physical anomalies of the ears | 0.07 | 0.16 | 1.1 | –0.18 | 0.17 | 0.8 |
| Regression 4: anomalies of the eyes[a] | | | | | | |
| Family adversity at age 6 | 0.37 | 0.18 | 1.4* | 0.06 | 0.19 | 1.1 |
| Minor physical anomalies of the eyes | 0.15 | 0.31 | 1.2 | 0.22 | 0.30 | 1.2 |
| Regression 5: anomalies of the head[a] | | | | | | |
| Family adversity at age 6 | 0.37 | 0.19 | 1.4* | 0.03 | 0.19 | 1.0 |
| Minor physical anomalies of the head | 0.34 | 0.28 | 1.4 | –0.34 | 0.30 | 0.7 |
| Regression 6: anomalies of the hands[a] | | | | | | |
| Family adversity at age 6 | 0.38 | 0.18 | 1.5* | 0.08 | 0.19 | 1.1 |
| Minor physical anomalies of the hands | 0.19 | 0.37 | 1.2 | 0.21 | 0.37 | 1.2 |

[a] B, SE, and odds values for the covariables childhood physical aggression and anxiety were similar to those in regression 1.
* $p<0.05$.

sample and in 11 of the 170 boys in the study group (6.5%). Nonviolent crimes such as robbery, breaking and entering, and prostitution were found in 44 boys of the large sample and in five members of the study group (2.9%). Given the overlap between self-reports and official records of delinquency ($\chi^2=36.3$, df=1, $p<0.001$, for violent delinquency; and $\chi^2=19.4$, df=1, $p<0.001$, for nonviolent delinquency), we identified a violent delinquent group of 44 boys, or 25.9% of the study group, who had been arrested for a violent crime or had reported committing a violent offense, and a nonviolent delinquent group of 42 boys, or 24.7% of the study group, who had been arrested for a nonviolent crime or had reported committing a nonviolent offense. The violent and nonviolent groups were not mutually exclusive; 21 participants committed both violent and nonviolent offenses, 23 committed only violent offenses, and 21 committed only nonviolent offenses.

### *Statistical Analyses*

Logistic regression analyses were used to test the predictive value of minor physical anomalies and family adversity. To control for the selection criteria, mean scores for physical aggression and anxiety at ages 6, 10, 11, and 12 were used as covariates and entered in the first step of the analyses. Minor physical anomalies and family adversity were then forced in the second and third steps, and the interaction term was left free to enter in a forward stepwise selection in the last step of the analyses. These procedures were repeated for the total count of minor physical anomalies and separately for the minor physical anomaly score of each anatomical region. In the analyses, violent delinquents were compared to participants who did not commit violent offenses, and nonviolent delinquents were compared to participants who did not commit nonviolent offenses.

## Results

Table 1 shows the frequency of minor physical anomalies in the study group. High percentages of study group members had anomalies in the regions of the ears and the mouth. Inconsistent percentages were found for anomalies observed in other anatomical regions. Compared to the total study group, the violent delinquent boys had a higher percentage of anomalies of the mouth and, inconsistently, of some anomalies of other anatomical regions. After excluding the toe-gap anomaly, the mean total count of minor physical anomalies for 167 participants in the entire study group (data were missing for some anomalies) was 3.7 (SD=1.89, range=0–9). This mean is similar to means reported for other at-risk samples of preadolescents: mean=3.4 (SD=1.84, range=0–9) (17) and mean=3.58 (SD=2.4, range=0–10) (16). The total count of minor physical anomalies among the participants had the highest association with anomalies of the ears (r=0.70, N=167), followed by anomalies of the eyes (r=0.52, N=167) and anomalies of the mouth (r=0.51, N=167). Anomalies of the mouth were positively correlated with anomalies of the eyes (pairwise r=0.26, N=175).

We first tested to what extent the total count of minor physical anomalies, the family adversity score, and their interaction predicted violent delinquency during adolescence. The results depicted in Table 2 show a significant effect of the total count of minor physical anomalies, beyond significant effects of childhood physical aggression and family adversity. This result indicated that each increment of one anomaly augmented by a factor of 1.2 the risk of violent delinquency during adolescence, and each increment of one standard deviation on the family adversity index augmented that risk by 1.4. The effect of the interaction between the total minor physical anomaly count and family adversity in predicting violent delinquency did not reach statistical significance.

We then examined the relation of minor physical anomalies of each anatomical region and their interaction with family adversity at age 6 in predicting adolescent violent delinquency. Results of logistic regressions showed a sig-

nificant main effect for anomalies of the mouth, beyond the effect of childhood physical aggression (Table 2). Each increment of one anomaly in the area of the mouth augmented by a factor of 1.7 the risk of violent delinquency during adolescence. Family adversity no longer predicted violent delinquency, because of its significant association with minor physical anomalies of the mouth (r=0.16, N= 170). This result indicated that the cumulative count of minor physical anomalies of the mouth and family adversity shares a part of the variance that explained adolescent violent delinquency. The interaction between minor physical anomalies of the mouth and family adversity did not reach statistical significance and did not enter the model.

Further analyses were performed to examine whether the significant effect found for the total count of minor physical anomalies was attributable to anomalies of the mouth that were included in the scale. We repeated the analysis after having removed anomalies of the mouth from the total count of minor physical anomalies. The results indicated that the total count of anomalies no longer predicted adolescent violent delinquency, although family adversity still did. Finally, we examined whether the significant effects found were specific to violent delinquency, as suggested by Mednick and Kandel (22). Results presented in Table 2 support the hypothesis by showing no significant association between adolescent nonviolent delinquency and minor physical anomalies or between adolescent nonviolent delinquency and the interaction of minor physical anomalies and family adversity.

## Discussion

The goal of this study was to assess the contribution of minor physical anomalies, family adversity, and their interaction to the prediction of delinquency during adolescence. Minor physical anomalies of different anatomical regions were examined in an attempt to get a more precise idea of the organic structures and processes involved in the development of adolescent delinquency. Separate analyses were used to predict violent delinquency and nonviolent delinquency because the two types of delinquency could have different etiologies (25).

Adolescent boys with higher counts of anomalies, and especially with anomalies of the mouth, were found to be most at risk for violent delinquency. Previous studies of minor physical anomalies have shown that anomalies of the mouth are more frequent in children with psychoses and in adults with schizophrenia (19, 20). Anomalies of the mouth have been found in individuals with neurological deficits (38, 39) and could be associated with CNS dysfunctions that increase the risk for violent delinquency. The CNS develops in a sequential and hierarchical way (40), and each organ has a specific critical period of vulnerability to teratogens that may result in developmental disruption (10). For example, the critical period for the development of anomalies of the palate starts at the ninth week of gestation, whereas anomalies of the hands develop during the eighth week. It is then plausible that insults occurring at specific periods during gestation increase the risks for the development of violent delinquency as a result of atypical brain development. The identification of specific sites of minor physical anomalies that are related to specific behavior disorders should help clarify which part of the CNS may be affected and thus may be involved in a given behavioral problem. This clarification would be achieved by establishing the correspondence between the period of vulnerability for the affected organ and the CNS developmental phases.

Anomalies of the mouth could also impact children's behavior regulation in less direct ways. Orofacial structures are involved from birth onward in many functions such as communication, emotional expression, mastication, and deglutition, which may have a wide range of consequences on development (41). Anomalies of the mouth may affect sucking and other feeding behaviors during the first years of life and thus may affect physical development as well as the mother-child relationship (42). Infants who experience feeding problems such as failure to thrive often show neurological problems (43) and are reported to have more behavior problems during childhood than infants without feeding problems (44). Thus, children with minor physical anomalies of the mouth could be more difficult to socialize for different and additive reasons: they may have neurological deficits as well as feeding problems in the first months after birth.

Unlike Mednick and Kandel's study (22), the study reported here found that the interaction between minor physical anomalies and family adversity did not predict violent delinquency. Mednick and Kandel's use of a dichotomized score for minor physical anomalies may explain the differences in results compared to those reported here. It is possible that the significant interaction in their study reflected a nonlinear effect of minor physical anomalies, as their analyses did not control for quadratic effects.

The study reported here was limited to French-speaking, Caucasian males from low socioeconomic status areas in a large city, overrepresenting subjects with stable childhood physical aggression and anxiety. Replications in other populations will be needed to confirm the importance of anomalies of the mouth in the prediction of violent delinquency. Longitudinal studies of infants will also be needed to examine the process by which infants with these anomalies would fail to learn to inhibit physical aggression. Preventive interventions would be different depending on whether the risk for violent delinquency is through feeding problems, neurological deficits, or a mixture of these factors, or through other factors, such as parent-child interactions, that were not addressed in this study.

Received April 2, 1998; revisions received Dec. 3, 1998, and Nov. 12, 1999; accepted Nov. 18, 1999. From the Research Unit on Children's Psychosocial Maladjustment, University of Montréal; the Fernand Seguin Research Center, University of Montréal; the Institute of Psychiatry, University of London; and the Department of Psychiatry, University of Montréal. Address reprint requests to Dr. Tremblay, Research Unit on Children's Psychosocial Maladjustment, University of Montréal, 3050 blvd. Edouard-Montpetit, C.P. 6128 succ. centre-ville, Montréal, Québec, Canada H3C 3J7; gripret@ere.umontreal.ca (e-mail).

Supported by a fellowship from the Conseil Québécois pour la Recherche Sociale (Dr. Arseneault) and by grants from the Social Sciences and Humanities Research Council of Canada, the Conseil Québécois pour la Recherche Sociale, and Québec's Fonds pour la Formation des Chercheurs et l'Aide à la Recherche.

The authors thank the study group members.

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