Childhood predictors of smoking in adolescence: a follow-up study of Montréal schoolchildren

Margaret R. Becklake, Heberto Ghezzo, Pierre Ernst

Abstract

**Background:** The factors that cause children to become smokers in adolescence remain unclear. Although parental smoking and peer pressure may play a role, physiological factors such as lung volume have also been identified.

**Methods:** To investigate these and other possible childhood predictors of teenage smoking, we gathered follow-up data on 191 Montréal schoolchildren, aged 5–12 years (average 9.2 yr) when first examined. At an average age of 13.0 years, they answered further questions on their health and smoking behaviour and provided a second set of spirometric measurements.

**Results:** At the second survey, 80% of the children had entered high school and 44% had become smokers. Reaching puberty between the surveys was the most significant determinant of becoming a smoker: 56.4% of the 124 children postpubertal at the second survey had taken up smoking, versus 17.9% of the 67 who were still prepubertal (p = 0.001). We found salivary cotinine level, a measure of uptake of environmental tobacco smoke, to be an independent predictor of becoming a teenage smoker; even after adjustment for sex, socioeconomic status of parents, a crowding index, and the numbers at home of siblings, adult smokers and cigarettes smoked, it remained significant for both groups: postpubertal (odds ratio [OR] 1.2, 95% confidence interval [CI] 1.2–3.0) and prepubertal (OR 2.1, 95% CI 1.0–4.5). The influence of forced vital capacity was marginally significant only in the postpubertal group (OR 5.0, 95% CI 0.88–28.3).

**Interpretation:** The proportion of nicotine absorbed from that available in environmental tobacco smoke during childhood is associated with subsequent smoking in adolescence. The more efficient absorption of nicotine seen in some children may be related to physiological factors such as lung capacity.

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13.0 years. None of the grade 5 children reported being a smoker, although 14 children (7.3%) did not answer the question. Four children at the first survey had concentrations of salivary cotinine that were markedly elevated, but in only 1 child did the level approach 20 µg/L, the usual cutoff for defining a smoker. At the time of the second survey, 84 children (44.0%) had become smokers; of these, 10 boys and 10 girls smoked more than 10 cigarettes per week. Attaining puberty between surveys was the most significant determinant of becoming a teenage smoker (56.4% of postpubertal children compared with 17.9% of those still prepubertal when the follow-up survey was administered, p = 0.001). Subsequent analyses were therefore stratified by the children’s pubertal status at the second survey (Table 2).

The only factor associated with becoming a teenage smoker in both pre- and postpubertal children was salivary cotinine concentration (adjusted in the analysis for sex, number of siblings, socioeconomic status of the parents, crowding index, number of smokers at home and total number of cigarettes smoked in the home). In prepubertal children, a marginally significant childhood factor was female sex; in postpubertal children, forced vital (lung) capacity and the presence of adult smokers in the home, whether single (i.e., 1 smoker) or multiple. The specific high school currently attended, which was recorded at the second survey, also showed marginal significance (p = 0.09; data not shown).

**Interpretation**

Our study population was small, and multiple comparisons were made; our results must therefore be interpreted with caution. Moreover, salivary cotinine levels may reflect current smoking by the child as well as environmental tobacco smoke; given the low rates in the first survey both of salivary cotinine and of reported smoking, however, this effect is likely to have been small.

An unexpected finding in the multivariate analysis was the lack of any significant association between becoming a teenage smoker and the number of smokers in the home in childhood (p = 0.99). Also unexpected was the association between a decrease in the number of adult smokers in the home between surveys (thought to indicate a positive parental attitude toward smoking cessation) and a decrease in the number of adolescents becoming smokers (the reduction in risk is shown in Table 2). This could have been caused not only by parental disapproval but alternatively by nonsmoking teenagers pressuring their parents to quit. It nonetheless shows the importance of a dynamic attitude at home against smoking.

As for the anatomic and physiologic findings, the most important observation was that salivary cotinine in childhood (a measurement of the nicotine actually entering the bloodstream) was an independent predictor of adolescent smoking, after adjustment in the analysis for amount smoked at home and other relevant factors (see final footnote, Table 1). Given also the association (admittedly not strong) between forced vital capacity and adolescent smoking, we suggest that lung size (or some associated characteristic) increases the uptake of environmental tobacco smoke, maximizes the influence of passive smoking in childhood and induces smoking in adolescence. In this context, the recently reported reduced density of dopamine D1 receptors in the ventral striatum of the brains of adult smo-
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Correspondence to: Dr. Margaret R. Becklake, Respiratory Epidemiology and Clinical Research Unit, Montreal Chest Institute, Rm. K1.33, 3650 St Urbain Street, Montréal QC H2X 2P4; margaret.becklake@mcgill.ca