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Never-smokers with a positive family smoking history are more likely to be overweight or obese than never-smokers with a negative family smoking history

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Abstract

To shed light on the complex relationship between smoking and body weight, we used never-smokers stratified on family smoking history to model the effects of a diathesis for smoking on body weight without the potential confound of metabolic changes or decreased physical activity caused by chronic tobacco smoke exposure. Participants were 100 family history negative never-smokers (FH−; 2 never-smoking parents) and 71 family history positive never-smokers (FH+; 2 ever-smoking parents). Controlling for significant group differences in race and age, BMI was significantly higher in FH+ (26.7±.6) than in FH− (24.5±.4; $F=10.351$ $p<.01$). Further analysis using logistic regression showed that FH+ were 2.7 times as likely to be overweight/obese ($BMI \geq 25$; 95% C.I. 1.398–5.351; $p<.01$). FH+ scored significantly higher on the Dieting and Bingeing Severity Scale than FH− and were significantly more likely to score in the severe or at-risk range. FH+ drank significantly more alcohol than FH−; they scored significantly higher on the CAGE and on the Michigan Alcohol Screening Test. Our analyses provide support for the role of inherited and/or environmentally-driven tendencies towards disinhibited eating and/or risky behaviors in general in the observed differences in BMI. No group differences in BMI or likelihood of being overweight/obese emerged based on prenatal exposure to nicotine in FH+ smokers, although our sample was too small to rule out an association. Further research in larger samples, using more complex statistical models, will be needed to disentangle these issues and identify causal pathways.

Keywords

BMI; disordered eating; family history; never-smokers; nicotine; prenatal exposure; weight

1. Introduction

Smoking suppresses appetite and body weight. Adolescents who take up smoking gain less weight than those who do not (Stice & Martinez, 2005), and smokers on the whole weigh less than never-smokers (Williamson, Madans, Anda, Kleinman, Giovino, & Byers, 1991). Paradoxically, however, people who smoke heavily may actually weigh more than never-smokers (Chiolerio, Jacot-Sadowski, Faeh, Paccaud, & Cornuz, 2007), possibly due to a clustering of risky behaviors (or behaviors promoting weight gain) such as overuse of alcohol,

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poor nutritional practices, and lack of exercise in addition to smoking (Chiolero, Wietlisbach, Ruffieux, Paccaud, & Cornuz, 2006). Upon quitting, most smokers gain weight, reaching, on average, a weight that approximates what they would have weighed had they never smoked (Williamson et al., 1991). Substantial individual differences in postcessation weight gain exist (Williamson et al., 1991), however, with large weight gain often being associated with patterns of disordered or disinhibited eating (Hudmon, Gritz, Clayton, & Nisenbaum, 1999).

Since concerns about weight have been shown to promote smoking initiation and hinder quitting, especially among girls and women (Saules, Tate, & Pomerleau, in press), better knowledge of the complex relationship between smoking and body weight could contribute to the development of targeted strategies for both prevention and treatment. Unfortunately, our understanding of the effects of a diathesis for smoking upon body weight in smokers is complicated by metabolic changes or decreased physical activity caused by chronic tobacco smoke exposure. To avoid this potential confound, we used never-smokers stratified on family smoking history as a model for comparing individuals with high versus low susceptibility to reinforcement by nicotine, and specifically to initiation and maintenance of smoking because of nicotine's weight- and appetite-suppressing effects. (See Pomerleau, Pomerleau, Snedecor, et al., under review, for a more detailed exposition of the rationale for this approach and its application in the field of alcohol dependence.)

In the current study, history positive never-smokers (two biological parents with a history of at least 3 years of regular smoking) were compared with history negative never-smokers (two biological parents who had smoked fewer than 100 lifetime cigarettes) on Body Mass Index, an estimate of body composition used extensively as an indicator of obesity, as well as on several measures of disordered eating and alcohol use. We hypothesized that history-positive never-smokers would have a higher Body Mass Index than history-negative never-smokers, and that they would show greater evidence of disinhibited eating and alcohol abuse, factors that could help to explain differences in Body Mass Index. In addition, to study the possible influence of prenatal exposure to nicotine (Winzer-Serhan, 2008), history-positive never-smokers whose mothers had smoked during their gestational period were compared with history-positive never-smokers whose mothers had abstained from smoking during pregnancy.

2. Method

2.1 Participants

Participants were 171 never-smokers, ages 21–55, recruited from the local community via newspaper, radio, and television advertisements to participate in a laboratory study of reactivity to nicotine administration. To ensure that they had actively rejected smoking, they had to have tried at least 1 cigarette, but not more than 100 cigarettes, in their lifetime. Participants had to be either family history positive (FH+; $n=71$) or family history negative (FH–; $n=100$) for smoking. To be considered FH+, both biological parents were required to have been regular daily smokers for at least three years at some time in their lives; for FH–, both biological parents are/were never-smokers (<100 lifetime cigarettes) and never regular users of other tobacco products. All participants endorsed either “fairly certain” or “very certain” as their level of confidence about parental smoking status. Nonsmoking status of participants was verified via exhaled carbon monoxide levels (Vitalograph BreathCO monitor), and a test of nicotine metabolites in urine (Quickscreen™ Cotinine (COT) Test; Craig Medical Distributors Inc.). See Pomerleau et al. (under review) for further information on inclusion/exclusion criteria and screening procedures.

2.2. Study instruments

A **General History** developed in the Nicotine Research Laboratory to assess age, race, sex, weight, height, and other person variables was used to collect demographic data. **Body Mass Index** (BMI; also known as the Quetelet Index) was calculated by dividing mass (in kilograms) by height (in meters) squared.

The following instruments were used to assess personal and family history of tobacco use/exposure: 1) **A Smoking History** developed in the Nicotine Research Laboratory to assess current and lifetime smoking status, as well as early experimentation with tobacco, use of other tobacco or nicotine-containing products, degree of nicotine dependence, and other smoking-related variables, was used to document lifetime non-smoking status. 2) **The Non-Smokers Survey** (NSS; Pomerleau, Pomerleau, Snedecor, & Mehringer, 2002), an instrument developed to assess personal and family history of smoking, includes an assessment of prenatal exposure to nicotine (i.e., maternal smoking during pregnancy, as reported by participant, including “don’t know” as an option to avoid forcing a choice in the face of participant uncertainty).

Measures of eating and body image were assessed using the following instruments: 1) The **Dieting and Bingeing Severity Scale** (DBSS; Krahn, Kurth, Demitrack, & Drewnowski, 1992; Kurth, Krahn, Nairn, & Drewnowski, 1993) is a self-report assessment of the severity of dieting and bingeing behavior adapted from an instrument developed by Krahn and colleagues. 2) The **Three Factor Eating Questionnaire** (TFEQ; Stunkard & Messick, 1985) is a self-report assessment of eating behavior with Restrained Eating, Disinhibited Eating, and Hunger subscales.

As a proxy for a general proclivity to substance self-administration, the following measures of alcohol use and abuse were administered: 1) A Substance Intake History queries alcohol use; it assesses drinks per week and also includes the **CAGE** (Mayfield, McLeod, & Hall, 1974), a 4-question screening test for alcohol dependence (dichotomized as 0–1 vs. 2–4). 2) **The Michigan Alcohol Screening Test** (MAST; Moore, 1972) is a widely used 24-item questionnaire for detecting alcoholism.

2.3 Procedure

Questionnaires used in this study were part of a baseline assessment battery for a laboratory study of response to nicotine administration based on differences in family history of smoking (Pomerleau et al., under review). The protocol and all consent forms were approved by the University of Michigan Medical School Institutional Review Board. The current study included all individuals who qualified for enrollment in the study, whether or not they completed the laboratory protocol, and had no missing questionnaire data on essential assessments.

2.4 Data analysis

Group differences based on family history of smoking were analyzed in SPSS, using independent t-tests, ANCOVA, Chi-Square tests, and Logistic Regression techniques as appropriate. Differences based on prenatal exposure to nicotine were analyzed using independent t-tests.

3. Results

3.1 Sample characteristics

FH+ participants were significantly older (35.7 ± 1.3) than FH- participants (31.9 ± 1.0 , $t = -2.419$, $p < .05$). They were more likely to be White (FH+: 90.1%; FH-: 69.0%; $\chi^2 = 10.737$; $p < .01$). Women comprised 47.9% of FH+ and 49.0% of FH- (NS). Because they differed

significantly between groups, race (White vs. non-White) and age were controlled for in all subsequent analyses.

3.2 Differences in Body Mass Index and overweight status

All participants had a BMI of at least 18.5 (that is, above the cutpoint for being underweight). BMI was significantly higher ($26.7 \pm .6$) in FH+ than in FH- ($24.5 \pm .4$; $F=10.351$, $p<.01$). The analysis using Logistic Regression showed that FH+ were 2.7 times as likely as FH- to be overweight/obese ($BMI \geq 25$; 95% C.I. 1.398–5.351; $p<.01$).

3.3 Differences in measures of disordered eating

FH+ scored significantly higher ($2.8 \pm .1$) on the Dieting and Bingeing Severity Scale than FH- ($2.5 \pm .1$; $F=5.066$, $p<.05$) and were 2.6 times as likely (95% C.I. 1.118–5.990; $p<.05$) to score in the severe or at-risk categories. Because of the possibility that being overweight could influence or be influenced by eating behaviors, the ANCOVA and Logistic Regression analyses were rerun with weight category added to the model; differences for both remained marginally significant ($p<.10$).

On the TFEQ, marginally significant differences were observed for Disinhibited Eating (FH+: $4.9 \pm .4$; FH-: $4.0 \pm .3$; $F=3.014$; $p<.10$), which disappeared after controlling for weight category. No significant differences were detected for either Cognitive Restraint or Hunger.

3.4 Differences in measures of alcohol use/abuse

FH+ consumed significantly more drinks per week than FH- (FH+: $2.9 \pm .6$; FH-: $1.5 \pm .4$; $F=4.463$; $p<.05$). On the CAGE, 18.3% of FH+ scored in the high range (2–4), as opposed to 5.1% of FH-, yielding an odds ratio of 4.8 (95% C.I. 1.499–15.176; $p<.01$) with age and race included in the Logistic Regression analysis. FH+ scored significantly higher on the MAST (FH+: $3.6 \pm .7$; FH-: $2.0 \pm .6$; $F=4.444$; $p<.05$).

3.5 Differences in prenatal exposure to nicotine in FH+ never-smokers

No group differences in BMI or likelihood of being overweight/obese emerged between FH+ subsamples who knew their mother had ($n=24$) or had not ($n=25$) smoked while pregnant.

4. Discussion

As hypothesized, BMI and likelihood of overweight/obesity were elevated in never-smokers with a positive family smoking history. Possible explanations for this difference—i.e., inherited and/or environmentally-driven tendencies towards disinhibited eating and/or risky behaviors in general—also received support, suggesting that a negative family smoking history may confer protection against excessive weight indirectly, via reducing the likelihood of unhealthful behaviors. No differences based on prenatal exposure emerged in never-smokers with a positive family smoking history.

The following limitations of our study should be noted: 1) Family history was categorized on the basis of parental smoking. Using more rigorous selection criteria (e.g., smoking in additional first-degree relatives; minimum smoking requirements for parents) might have allowed for clearer differentiation between Family History groups. 2) The subset of participants with a positive family smoking history who indicated knowledge of whether they were prenatally exposed to nicotine via maternal smoking was too small to rule out the possible impact of this variable.

In addition to the factors explored in our study, possible contributors to the observed differences in BMI include childhood environmental influences (e.g., less emphasis on physical activity)

and/or inherited metabolic and other constitutional factors that might have led to weight-control smoking in the ever-smoking parents. Further research in larger samples, in the context of longitudinal studies permitting validation of parental smoking status and prenatal nicotine exposure via direct assessment of the parents, using more complex statistical models and a broader range of environmental factors, will be helpful in disentangling these issues and identifying causal pathways.

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