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Parental stress increases body mass index trajectory in pre-adolescents

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What is already known about this subject

- Rates of childhood obesity have increased since the mid-1970s.
- Research into behavioural determinants has focused on physical inactivity and unhealthy diets.
- Cross-sectional studies indicate an association between psychological stress experienced by parents and obesity in pre-adolescents.

What this study adds

- We provide evidence of a prospective association between parental psychological stress and increased weight gain in pre-adolescents.
- Family-level support for those experiencing chronic stress might help promote healthy diet and exercise behaviours in children.

Summary

Objective: We examined the impact of parental psychological stress on body mass index (BMI) in pre-adolescent children over 4 years of follow-up.

Methods: We included 4078 children aged 5–10 years (90% were between 5.5 and 7.5 years) at study entry (2002–2003) in the Children's Health Study, a prospective cohort study in southern California. A multi-level linear model simultaneously examined the effect of parental stress at study entry on the attained BMI at age 10 and the slope of change across annual measures of BMI during follow-up, controlled for the child's age and sex. BMI was calculated based on objective measurements of height and weight by trained technicians following a standardized procedure.

Results: A two standard deviation increase in parental stress at study entry was associated with an increase in predicted BMI attained by age 10 of 0.287 kg m^{-2} (95% confidence interval 0.016–0.558; a 2% increase at this age for a participant of average attained BMI). The same increase in parental stress was also associated with an increased trajectory of weight gain over follow-up, with the slope of change in BMI increased by 0.054 kg m^{-2} (95% confidence interval 0.007–0.100; a 7% increase in the slope of change for a participant of average BMI trajectory).

Conclusions: We prospectively demonstrated a small effect of parental stress on BMI at age 10 and weight gain earlier in life than reported previously. Interventions to address the burden of childhood obesity should address the role of parental stress in children.

Keywords: Obesity, parental psychological stress, pre-adolescent weight, prospective cohort.

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Introduction

Rates of childhood and adult obesity have increased dramatically in North America and elsewhere since the mid-1970s (1,2). Childhood is a critical period for determinants of energy balance, including biological programming and cognitive-social development that can influence the adoption of relevant health behaviours (3,4). Identifying factors that influence how obese phenotypes are determined during early life can facilitate prevention earlier in the life course. While research into behavioural determinants has focused on physical inactivity and unhealthy diets (5), psychological stress experienced by parents can also contribute to risk for obesity during childhood (6).

Although the role of psychological stress in parents in the aetiology of childhood overweight remains unclear, plausible pathways leading to positive energy balance in children include parenting changes that directly lead to positive energy balance in children (7,8) (e.g. preparation of convenient but unhealthy meals in an effort to help manage their own time (9)); the indication of parental obesity due to lifestyle and/or physiological dysfunction, which influences related attitudes and behaviours of children (10,11); and a role for stress experienced by children of stressed parents (12).

There has been little study of the relationship between parental stress and obesity outcomes in children (e.g. (6)), and apparently, no prospective assessment of the effects of parental stress on weight gain in children. We examined the relationship between perceived psychological stress in parents and attained body mass index (BMI) at age 10 and BMI trajectory over 4 years in pre-adolescent schoolchildren in the Children's Health Study, which provides data on a variety of factors that might confound associations between parental stress and obesity in children. Study participants included males and females aged 5–10 at study entry, and the majority of children were either of Hispanic ethnicity or non-Hispanic white race.

Methods

Study population

The Children's Health Study is a prospective cohort study of air pollution and respiratory health (13). Details on the study design are reported elsewhere (13). Enrolment was carried out in 45 participating schools located in 13 southern California communities in 2002 and 2003, as previously described (13). Written informed consent was obtained from parents or guardians at baseline, and the study was approved by the University of Southern California Institutional Review Board.

This secondary analysis includes data from baseline and annual follow-up visits over a 4-year period. Although all students in kindergarten and first grade were invited to participate, 5349 (65%) returned valid baseline questionnaires (14,15). This analysis was restricted to 4550 children followed up for at least 1 year (i.e. two or more measurements of height and weight). These participants had an average of 3.9 BMI measurements and there was complete follow-up (i.e. no missing measurements of BMI for any participants) before they were censored (i.e. 2.9 years of follow-up on average). Children were excluded from this analysis if they were missing data describing parental stress ($n = 472$). Therefore, the study population for this analysis included 4078 children (76% of all respondents).

Assessment of BMI, parental stress and covariates

BMI was calculated based on objective measurements of height and weight during annual follow-up visits (see Supporting Information Appendix S1 for details). Baseline and follow-up questionnaires were completed by parents (74% mothers), including information about demographic and family characteristics, respiratory health, parental stress at baseline, physical activity, characteristics of homes and other relevant covariates.

Perceived stress was assessed in the responding parent using the 4-item version of the Perceived Stress Scale (PSS), which is a widely used measure of the degree to which respondents believe their lives are unpredictable, uncontrollable or overwhelming (16). A single measure of this scale has been validated as a measure of manifestations including negative affective states and physical symptoms of stress (17,18). Scores ranged from 0 to 16, and a representative US sample found an overall mean and standard deviation of 4.49 and 2.96, respectively (16).

Other covariates from the baseline questionnaire and describing the built environment were identified as potential confounders of any of the pathways identified between parental stress and BMI trajectory of children based on prior evidence of a plausible relationship and a statistically significant unadjusted association with the slope of change in BMI in our cohort ($\alpha = 0.05$; see Supporting Information Appendix S1 for rationale). Questionnaire measures included age, sex, race or Hispanic ethnicity, use of

a Spanish language questionnaire, medical insurance coverage, parental educational attainment (an indicator of socioeconomic position), exercise classes, lessons or special programmes outside of school in which the child participated during the last 12 months (19), and presence of a daily smoker of cigarettes, cigars or pipes in the home (which may reflect exposure *in utero* (20)). Measures of the residential built environment at baseline included two walkability measures, including connectivity of the street network and household exposure to NO_X from traffic on local roads; proximity to food outlets; green cover based on the normalized difference vegetation index; and population density (population per square mile; see (14,21) for more detail on how these variables were compiled).

Statistical methods

Our mixed-effects modelling approach properly adjusts for age- and sex- specific effects on BMI growth in children, and provides a proper mechanism for assessing effects of risk factors on BMI growth. Letting *c*, *i* and *j* denote the study community, child and year of measurement, respectively, the following two-level linear model was used to examine the effect of parental stress at study baseline, S_{ci}, both on BMI, Y_{cij}:

$$Level1: Y_{cij} = A_{ci} + B_{ci}t_{cij} + e_{cij} \quad (1)$$

$$Level2a: A_{ci} = A_c + \beta_1 S_{ci} + \delta_1 Z_{ci1} + \ldots + \delta_q Z_{ciq} + e_{ci} \quad (2)$$

$$Level2b: B_{ci} = \beta_0 + \beta_2 S_{ci} + f_{ci} \quad (3)$$

where t_{cij} denotes age of participants at time of BMI measurements (centred at 10 years of age), A_c denotes town specific intercepts, and $Z_1, \ldots, Z_q$ denote adjustment factors such as sex, and race/ethnicity categories. Our results were obtained by combining equations (1–3) to fit the following unified mixed effects model:

$$Y_{cij} = A_c + \beta_0 t_{cij} + \beta_1 S_{ci} + \beta_2 S_{ci} \times t_{cij} + \delta_1 Z_{ci1} + \ldots + \delta_q Z_{ciq} + e_{ci} + f_{ci} t_{cij} + e_{cij} \quad (4)$$

In eqn (4), β_1 and β_2 correspond to the simultaneously estimated effects of parental stress on BMI level attained at age 10 (i.e. examining the main effect between individuals) and also the yearly slope of change in BMI during the follow-up period (22), respectively.

In our models, we required subjects to have at least two measurements of BMI to enable the conceptual subject-specific linear growth framework. BMI trajectory between measurements within the same individuals, and the effect of stress on BMI trajectory, were assumed to be linear over the 4-year average age change from 6–10 years.

The basic model included a set of design variables as fixed effects on BMI trajectory, namely, age, BMI at age 10, and community of residence at baseline, sex and racial/ethnic group. Additional confounders (identified as described above) were then considered in the final model using a manual forward stepwise approach. Potential confounders were entered into the model only if controlling for them individually changed the BMI trajectory coefficient of parental stress by at least 10%. This subset of potential confounders was then entered into a final model in order of greatest negative impact on the effect of parental stress on BMI trajectory. Indicators of missing data for confounders were also included in models adjusted for these covariates to allow all 4078 subjects into these models (23).

Interaction terms were added to the final model to test whether effects on attained BMI at age 10 or BMI trajectory were different by sex, Hispanic ethnicity and all other confounders entered into the final model ($\alpha = 0.05$), except for after-school exercise activity because the interaction model did not converge in that case. All analyses were conducted by using the SAS (SAS Institute, Cary, N.C., U.S.A) and R (R Foundation for Statistical Computing, Vienna, Austria) statistical software packages.

Results

The full age range at study entry was 5–10 years; however, 90% of participants were aged between 5.5 and 7.5 years with a mean of 6.7 years. The mean PSS among parents (i.e. parental stress) was 4.0 with a standard deviation of 2.9. Approximately, half of participants were male (50.5%) and the majority were of Hispanic ethnicity (54.1%). Around one-third of participants were non-Hispanic whites (32.2%), and the remainder was Asian (3.2%), African–American (2.7%) or another race/ethnicity (7.9%). One-quarter of parents used a Spanish-language questionnaire (24.9%). Only 12% of participants were not medically insured, while 21.8% lived with a parent without a high school diploma. Other characteristics of participants' families and their neighbourhood built environment are shown in Table 1.

After controlling for age and sex of participants, perceived stress in parents at baseline was positively

Table 1 Participant characteristics at baseline

Variables	*n* (%)*	Mean (SD): range
Dependent variable		
Parental stress (Perceived Stress Scale)		4.0 (2.9): 0–16
Design variables		
Age (years)		6.7 (0.7): 4.5–10.4
Male sex	2297 (50.5)	
Race/ethnicity		
Hispanic ethnicity	2462 (54.1)	
African–American race	122 (2.7)	
Asian race	145 (3.2)	
White non-Hispanic race	1464 (32.2)	
Other race	357 (7.9)	
Body mass index at baseline (kg m^{-2})		16.7 (2.7): 11.5–34.9
Gender- and age-specific body mass index percentile†		
Normal (BMI <85th pct)	3201 (70.4)	
Overweight (≥85th pct BMI <95th pct)	660 (14.5)	
Obese (≥95th pct BMI)	684 (15.1)	
Other covariates		
Number of after-school exercise activities in last 12 months?		
0	2913 (70.9)	
1	1000 (24.3)	
2+	198 (4.8)	
Parental education		
Did not finish high school	905 (21.8)	
High school diploma or some college	2364 (56.8)	
College diploma or greater	892 (21.4)	
Medical insurance coverage	3689 (88.3)	
Spanish language questionnaire	1133 (24.9)	
Any daily smoker inside home	309 (7.2)	
Any pets in home	3232 (76.1)	
Residential traffic-related pollution (ppm)		6.7 (6.1): 0–69
Residence in a food desert neighbourhood	2137 (51.9)	
Neighbourhood green space (NDVI)		0.09 (0.10): 0–0.50
Neighbourhood connectivity index		0.40 (0.06): 0–0.66
Neighbourhood population density (per km^2)		1703.9 (1426.7): 3.8–13978.3

*Numbers do not always add up to 4550 due to missing values.
†Body mass index percentile based on Centers for Disease Control and Prevention reference values for the year 2000.

Table 2 Associations of parental stress at baseline with attained BMI at age 10, slope of BMI change over a 4-year period

Effect	Model 1	Model 2
Attained BMI coefficient (95% CI)	0.443 (0.025–0.860)	0.287 (0.016–0.558)
Slope of BMI change coefficient (95% CI)	0.093 (0.078–0.108)	0.054 (0.007–0.100)

Model 1 includes adjustment for age, sex, racial/ethnic group and community of residence at baseline. Model 2 additionally adjusts for exposure to traffic-related pollution at the home, participant's level of extra-curricular exercise activity, residence in a 'food desert' and parental education. Effects of stress scaled across 2 SD of PSS (2.9 units).

associated with attained BMI at age 10 as well as BMI trajectory over a 4-year period (Table 2). In a model adjusted only for design variables, an increase in parental stress across two standard deviations of PSS was associated with 0.443 kg m^{-2} higher BMI at baseline (95% confidence interval 0.025–0.860). In this model, the slope of change in BMI over the study period was increased by 0.093 kg m^{-2} (95% confi

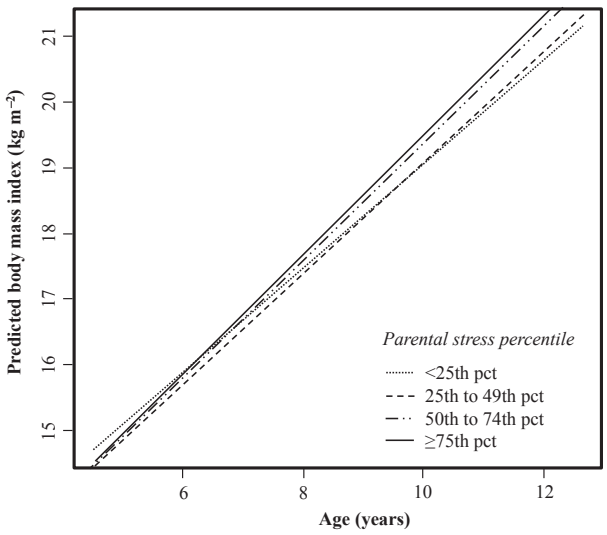

Figure 1 Predicted adjusted body mass index across increasing quartiles of parental stress.
Parental Stress Scale: 25th pct = 2, 50th pct = 4, 75th pct = 6. BMI trajectory is predicted by data from annual follow-up visits, applied to the age range available in the cohort from 5 to 13 years by parental stress quartile, based on the final model (model 2 in Table 2).

dence interval 0.078–0.108) across the same contrast (model 1 in Table 2).

Although adjusting for possible confounders led to large reductions in the size of effects on attained BMI and BMI trajectory, both effects remained statistically significant (model 2 in Table 2). After further adjusting for confounders including exposure to traffic-related pollution at the home, extra-curricular exercise activity, proximity to food outlets and parental education, we observed a 0.287 kg m^{-2} increase in attained BMI at age 10 across the interquartile range of parental stress (95% confidence interval 0.016–0.558). Across the same contrast, the slope of change in BMI was increased by 0.054 kg m^{-2} (95% confidence interval 0.007–0.100). These coefficients translate into roughly 2% and 7% increases in the attained BMI and BMI trajectory (respectively) for every two standard deviation increase in parental stress for participants of average attained BMI (16.7 kg m^{-2}) and average increase in attained BMI during follow-up (0.77 kg m^{-2}).

Figure 1 displays BMI trajectory across the age range available in the cohort (5 to 13 years) predicted by data from annual follow-up visits, which has been stratified by parental stress quartile, based on the final model (model 2 in Table 2). This figure indicates that there was a graded increase in the slope of change across increasing levels of parental stress, as well as a particularly large increase above (vs. below) the 50th percentile. Model results were also highlighted by considering the change in overweight and obesity rates at age 10 (120.5 months) based on gender-specific 85th and 95th percentiles of BMI from the Centers for Disease Control growth charts if all children in the sample were to have commensurate parental stress at the lower end of the 2 standard deviation range. Under this scenario, 2.7% of boys would move from overweight to normal, and approximately 1.2% would move from obese to overweight; for girls, the comparable shifts are 1.2% and 1.4%.

Effects of stress on attained BMI and the slope of change were stronger among children of Hispanic ethnicity (*P*-value for interaction < 0.05). Coefficients for both of these BMI outcomes were approximately 2.5 times larger in Hispanics than effects noted for the total population in Table 2; whereas these coefficients were diminished and not statistically significant among non-Hispanic children. Effects of parental stress did not differ for boys and girls in our cohort, nor by other covariates included in the final model that were tested.

Discussion

This is the first prospective study to demonstrate an association of parental stress with increased weight gain in pre-adolescents. This relationship was not fully explained by socioeconomic, lifestyle and environmental factors available for this analysis. Our results support prior studies indicating a cross-sectional positive relationship between parental stress with overweight and obesity in children (e.g. (6)).

Rates of obesity and other components of the metabolic syndrome have increased over the last 20 years, and it is possible that increasing stress in parents over this period contributed to weight gain in children (24). There are several plausible pathways, although we did not have data available to test their salience.

Parental stress may be associated with parenting behaviours causing positive energy balance in children, e.g. provision of an insecure food environment with under-nutritious food (7–9). Parental stress may also reflect obesity in parents, which could explain obesity in children due to shared genetic vulnerability (11), and the potential for lifestyle factors related to energy balance (i.e. diet and exercise) to be influenced by the conditions (e.g. nutrition environment; (10)) and social models (25) that parents offer children. Since parental stress was associated with other likely household-level stressors (e.g. low

income, residence in an apartment vs. house) in our cohort (26), it may act as a marker for psychosocial stress in children (12,27–30), which has been associated with weight gain (31,32) and metabolic syndrome (33,34) in adolescents.

Although the strength of the relationship of parental stress with both BMI outcomes was substantially reduced by controlling for confounders, several of these confounders reflect variables that may lie on the causal pathway, including level of exercise activity, as well as parental education, which could reflect a scarcity of resources to cope with stress effectively or residence in an environment with a scarcity of healthy, affordable food or that is activity unfriendly. Thus, the true stress effects may be larger than indicated by coefficients in the fully adjusted model in Table 2.

There was a wide age range among participants at baseline (5–10 years), although 90% of participants were between the ages of 5.5 and 7.5. A sensitivity analysis restricted to the narrower age group showed a similar pattern of effects of stress, so it is unlikely that the overall effect of stress on BMI is masking distinct effects of parental stress on participants of particularly old or young age at baseline. Pubertal stage, which can influence appetite control and weight gain through changes in levels of the hormone leptin, was not measured in this study (35). Therefore, if changes in the pubertal stage of children lead to changes in parental stress, then the association between parental stress and weight gain in children may be spurious. We only assessed parental stress at study baseline, and parents' experience of stress may have changed during follow-up. This would likely lead to non-differential misclassification and some attenuation of the size of the effect on BMI outcomes.

Beyond the pre-adolescent age range of our participants, the generalizability of these results is potentially limited based on the sociodemographic profile of the participants; namely, the unique racial and ethnic composition of our study population (mainly Hispanic and White, non-Hispanic schoolchildren) given that genetic and social factors help determine diet, weight gain and obesity. Given the importance of cultural, economic, political and other factors in determining parental and child stress and obesity, these results may be specific to US children.

The effects of stress on BMI were stronger among Hispanic children in our cohort. The average BMI at baseline of Hispanic children was 0.5 kg m^{-2} greater than children of other racial or ethnic backgrounds (data not shown), which is consistent with other studies that have observed a higher prevalence of overweight and obesity among Hispanic American children compared with other American children (e.g. (2)), and others that indicate that Hispanic children may be particularly vulnerable to parent stress for obesity (9). Some evidence indicates that obesity in Hispanic children may reflect genetic phenotypes characterized by behavioural (i.e. hyperphagia and sedentary lifestyle) and biological (i.e. promotion of adiposity) predispositions for positive energy balance (36,37). We cannot rule out the possibility that effects of stress reflect confounding by such genetic factors, or gene–stress interactions. Hispanic participants may also be more likely to experience stress related to immigration or acculturation. Hispanic children who used a Spanish language questionnaire in our survey also had higher stress (26), and acculturation stress has been associated with higher waist circumference in Mexican–American adults (38). However, the coefficient of stress on BMI among Hispanics in this analysis was not altered by limiting the sample to participants who used a Spanish-language questionnaire (data not shown), so stress related to a history of immigration or low acculturation does not explain the larger effect in Hispanic children. Future research should consider other reasons that Hispanic children are more susceptible to parental stress, including differences in how Hispanic parents respond to stress or how Hispanic children perceive stressors or cope with stress. Policies and other interventions (as described below) should be targeted in design to Hispanic communities.

Some participants were excluded from the analysis due to missing data on parental stress. These individuals were more likely to be of 'other' race/ethnicity (i.e. not Hispanic, African–American, Asian or White non-Hispanic; 28% vs. 5%), more likely to be of Hispanic ethnicity (55% vs. 50%) and slightly older (1.5 months); $P < 0.05$. Since the effect of parental stress on attained BMI and BMI trajectory was stronger for Hispanic participants, if these participants were more likely to have high parental stress, then effects reported in Table 2 may be biased towards the null, although there were only very weak positive associations between Hispanic ethnicity or use of a Spanish language questionnaire and parental stress, so this bias is unlikely to be very strong. Also, although we did not observe any effect of parental stress among children of 'other' race/ethnicity (data not shown) and thus, do not expect any bias associated with their exclusion, our study may have been under-powered to detect effects in this small subgroup to the small sample size. Finally, the age difference was very minor between participants with and without stress information; therefore,

bias in observed effects of stress due to these differences in excluded participants was unlikely.

This study links increased attained BMI at age 10 and increased BMI trajectory with parental stress in a population of pre-adolescents. Although the precise role of parental stress in children's energy balance remains unclear, emerging consistency in the relationship between chronic stress and BMI across a range of age groups suggests that interventions to reduce chronic household stress and stressful environmental conditions, and to increase resources for families to coping with stress, might help reduce obesity in children and its metabolic consequences (39). Family-level psychological, social, economic and educational support for those experiencing chronic stress might help promote healthy diet and exercise behaviours in children (e.g. (40)). Improved food security (e.g. (41)) and policies that provide a strong social safety net could lead to healthier eating habits and less obesity in families experiencing stressful conditions such as poverty or joblessness (42,43).

Conflict of interest statement

None of the authors have conflicts of interest to declare.

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Supporting information

Additional Supporting Information may be found in the online version of this article at the publisher's web-site:

Appendix S1. Methods.