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Gender, Stress in Childhood and Adulthood, and Trajectories of Change in Body Mass

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Abstract

Despite substantial evidence of the linkage between stress and weight change, previous studies have not considered how stress trajectories that begin in childhood and fluctuate throughout adulthood may work together to have long-term consequences for weight change. Working from a stress and life course perspective, we investigate the linkages between childhood stress, adulthood stress and trajectories of change in body mass (i.e., Body Mass Index, BMI) over time, with attention to possible gender variation in these processes. Data are drawn from a national longitudinal survey of the Americans' Changing Lives ($N=3,617$). Results from growth curve analyses suggest that both women and men who experienced higher levels of childhood stress also report higher levels of stress in adulthood. At the beginning of the study period, higher levels of adulthood stress are related to greater BMI for women but not men. Moreover, women who experienced higher levels of childhood stress gained weight more rapidly throughout the 15-year study period than did women who experienced less childhood stress, but neither childhood nor adulthood stress significantly modified men's BMI trajectories. These findings add to our understanding of how childhood stress—a more important driver of long-term BMI increase than adult stress—reverberates throughout the life course to foster cumulative disadvantage in body mass, and how such processes differ for men and women. Results highlight the importance of considering sex-specific social contexts of early childhood in order to design effective clinical programs that prevent or treat overweight and obesity later in life.

Keywords

Body Mass Index; stress; childhood; adulthood; gender

Stress has well-documented adverse effects on a range of health outcomes including mental health, physical health, health behaviors and mortality risk (Kruger & Chang, 2008; Umberson, Liu & Reczek, 2008). Recent work has directed particular attention to the role of

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stress in influencing excessive weight gain (Block et al., 2009; Pine et al., 2001; Udo, Grilo, & McKee, 2014; Wardle et al., 2011). The impact of stress on weight gain is important because excessive body weight (in this study, body weight refers to body mass, taking into account weight relative to height) is associated with increased morbidity and mortality risk (Breeze, Clarke, Shipley, Marmot & Fletcher, 2006; Lewis et al., 2009; Winter et al., 2014).

Recent longitudinal evidence suggests that stress in adulthood is associated with weight gain over time (Block et al., 2009; Harding et al., 2013; Wardle et al., 2011), with potentially stronger effects on women than men (Udo, Grilo & McKee, 2014). A separate literature on childhood stress suggests that higher levels of childhood stress are associated with greater weight gain from childhood to adolescence (Gundersen et al., 2011; Shankardass et al., 2014; Stenhammar et al., 2010; Wells et al., 2010), with limited and mixed evidence for gender differences in this association (see Hernandez et al., 2014; Hernandez & Pressler, 2015; Vanaelst et al., 2014). Previous studies have not considered whether stress trajectories that begin in childhood and fluctuate throughout adulthood are connected in ways that have long-term consequences for stability and change in body mass throughout the life course, or whether these processes unfold in different ways by gender (Gundersen et al., 2011).

Theoretical work on social factors that contribute to cumulative (dis)advantage in health over the life course suggests that childhood experiences shape adult experiences to have cumulative effects on health outcomes over time (Ferraro & Kelley-Moore, 2003; Scott, Melhorn & Sakai, 2012; Ziol-Guest, Duncan & Kalil, 2009). We draw on a stress and life course perspective and analyze longitudinal data from a national panel survey spanning a fifteen-year period to assess how stress exposure in childhood and adulthood work together to shape trajectories of change in body mass over time. Moreover, the life course experiences of men and women differ in fundamental ways that are reflected in health behaviors and health outcomes (Williams & Umberson, 2004). Extensive theoretical and empirical work has pointed to gender differences in weight gain processes (Udo, Grilo & McKee, 2014) as well as stress exposure and ways of coping with stress (Rosenfield & Mouzon, 2013; Turner & Avison, 2003). However, gender differences in the link of stress and BMI are less clear, with some studies reporting a stronger effect for women (Grunberg & Straub, 1992; Udo, Grilo & McKee, 2014), some suggesting a stronger effect for men (Wardle et al., 2011), and others revealing little gender difference (Harding et al., 2013). Therefore, we further examine whether the impacts of childhood and adulthood stress on body mass trajectories over time operate in different ways for men and women.

Stress and Body Mass: Previous Empirical Evidence

Many scholars have considered the link between stress and weight gain. Much of this research is based on theoretical models, clinical samples, and/or research conducted in laboratory settings that rely on simulated stressors (Adam & Epel, 2007; Björntorp, 2001; Dallman et al., 2003; Dallman et al., 2006; Jastreboff et al., 2011; Scott, Melhorn & Sakai, 2012). These studies generally conclude that elevated stress is significantly associated with weight gain but results are limited in applicability to general populations. Using nationally representative longitudinal data, recent studies have also linked various domains of stress in adulthood (including job stress and relationship strain) to weight gain over time (Block et

al., 2009; Brunner, Chandola & Marmot, 2007; Kouvonen et al., 2011; Nyberg et al., 2012). A separate literature also shows that childhood stress (e.g., family stress, peer problems, childhood poverty) is associated with weight gain at least into adolescence (Evans, Fuller-Rowell, & Doan, 2012; Gundersen et al., 2011; Vanaelst et al., 2014; Wells et al., 2010; Ziol-Guest et al., 2009). An explicit focus on stress in childhood and body mass in adulthood is rare. As we will discuss shortly, stress exposures in childhood and adulthood may work together to influence change in body mass over the life course, a process that unfolds in different ways for men and women.

A Stress and Life Course Framework

According to a stress and life course perspective (Elder, Johnson, & Crosnoe, 2003), the developmental tasks and challenges of life change as individuals age, and each person has his/her own life-history trajectory of stress within a specific social, cultural and economic context. The life course perspective on stress thus directs attention to change in levels of stress over time as well as the long-term effects of stress on individuals throughout the life course to influence a wide range of health behaviors and health outcomes, including weight change (Ferraro & Kelley-Moore, 2003; Hayward & Gorman, 2004). More importantly, a life course perspective points to the importance of considering how the effects of childhood stress may reverberate throughout the life course to shape trajectories of change in weight over time, and in addition to any weight change in response to stress exposure in adulthood (Pine et al., 2001; Stunkard, Faith & Allison, 2003). Stress processes launched in childhood clearly undermine health years and even decades later (Haas, 2008; Hatch, 2005; Hayward & Gorman, 2004; Miller, Chen & Parker, 2011), and this advantage or disadvantage may have a cumulative effect on weight change over time (Dannefer, 2003). Stress exposure may shape trajectories of change in body mass over time, with sustained or escalating stress contributing to an even faster increase in body mass. We work from this perspective to hypothesize that *both childhood stress and ongoing stress in adulthood promote weight gain over time (Hypothesis 1a), and the effect of childhood stress on BMI trajectories occurs, in part, by contributing to stress in adulthood (Hypothesis 1b).*

Gender Difference

Gender is a fundamental determinant of life course context. Cumulative stress processes unfold in response to social circumstances that vary for men and women throughout life (e.g., women experience more financial strain throughout life than do men) (Matud, 2004; Thorne, 2010). Recent research suggests that, compared to men, women are exposed to higher levels of stress during both childhood and adulthood, and women also report more physical and emotional symptoms of stress (American Psychological Association, 2010; Hankin & Abramson, 2001; Matud, 2004; Turner & Avison, 2003). Moreover, men and women tend to respond to stress in different ways (Rosenfield & Mouzon, 2013). For example, women are more likely than men to exhibit internalizing responses to stress, such as depressive symptoms, whereas men are more likely than women to exhibit externalizing responses to stress, such as alcohol consumption (Rosenfield & Mouzon, 2013). There is additional evidence that women eat more than men, especially fatty foods, in response to

stress (Wardle, Steptoe, Oliver & Lipsey, 2000), whereas men are more likely to play sports or listen to music as a way of managing stress (American Psychological Association, 2010).

American women and men have different rates of overweight and obesity. Generally, men are more likely than women to be overweight (39.6% versus 27.3%) and women are more likely than men to be obese (34.9% versus 31.8%) (U.S. Dept. of Health and Human Services, 2011). The social conditions that promote weight gain are likely to vary for men and women (Block et al., 2009). Although current evidence is mixed (Harding et al., 2013; Wardle et al., 2011), several longitudinal studies point to stronger effects of stress in adulthood on weight gain for women than for men (Grunberg & Straub, 1992; Udo, Grilo & McKee, 2014). Any gender difference in stress and body mass may have its origins in childhood but evidence on gender differences in stress and weight gain in childhood is limited and unclear. For example, a few studies suggest that family stress, financial stress and peer problems experienced in childhood are more likely to lead to weight gain for girls than boys (Hernandez et al., 2014; Hernandez & Pressler, 2015; Vanaelst et al., 2014), yet childhood exposure to maternal risky health behaviors is more likely to promote boys' weight gain than girls' (Hernandez & Pressler, 2015). Moreover, potential gender differences in childhood stress effects that are not apparent by adolescence may emerge later in life although empirical evidence on this point is rare. For example, adults who experienced high levels of childhood stress appear to be more emotionally reactive to stress in adulthood (Umberson et al., 2005). Given extensive theoretical and empirical work on gender differences in stress responses (Rosenfield & Mouzon, 2013; Taylor et al., 2000; Turner & Avison, 2003) and body mass (U.S. Dept. of Health and Human Services, 2011), as well as gendered experiences in relation to stress-related food consumption (Wardle et al., 2000), we expect that *stress in childhood and adulthood leads to greater weight gain for women than for men over time (Hypothesis 2)*.

METHODS

Data

We analyzed national longitudinal data covering a 15 year period. Our data were from the Americans' Changing Lives Survey conducted by the Institute for Social Research (House, 2003). Data were collected at four time points from 1986 to 2001. The original sample (ages 24–96 in 1986) was obtained using multistage stratified area probability sampling with an oversample of black Americans, persons over 59 years of age, and married women whose husbands were over the age of 64. Face-to-face interviews lasting approximately 90 minutes each were conducted with individuals in 1986 (N=3,617), 1989 (N=2,867) and 1994 (N=2,398), with a follow-up phone interview in 2001 (N=1,787).

Measures

Contemporary stress research distinguishes different types of stressors as well as the importance of composite measures of stress (Turner, Wheaton & Lloyd, 1995). Individuals may be exposed to major traumatic events, stressful life events, and chronic strains over the life course, and one's overall stress level reflects all of these exposures. We followed the

practice of previous studies (e.g., Umberson et al., 2005) to calculate childhood and adulthood stress using the ACL data.

Childhood Stress—Childhood stress was based on seven family-related stressors that occurred at age 16 years or younger: 1) at least one parent died, 2) parents divorced, 3) parents had marital problems, 4) at least one parent had a mental health problem, 5) at least one parent had an alcohol problem, 6) never knew father, and 7) family economic hardship. Family economic hardship was coded 1 for those respondents who report that their family was “somewhat worse off” or “a lot worse off” than the average family in their community. Each of the other childhood stressors was coded 0 if it did not occur and 1 if it did occur, and scores on all these dichotomous variables were summed to create an indicator of the number of childhood stressors experienced. Because the childhood stress measure was only collected at Time 2, there are 783 missing values for this measure including 33 due to missing reports and 750 due to survey attrition at Time 2. We used Full Information Maximum Likelihood (FIML) methods to handle those missing values (see more details of this approach in the “Statistical Methods” section). Additional analysis (not shown) excluding those missing cases revealed similar results.

Adulthood Stress—Following stress research recommendations (Thoits, 1995; Turner et al., 1995), the adulthood stress measure was based on five recent life events and five potential sources of chronic strain in various life domains. We considered five significant life events that occurred in the three years prior to Time 1 and in the periods between Times 1, 2 and 3 (1 = yes, 0 = no): 1) Death of a significant other included the death of a child, close friend, or relative other than a parent; 2) Death of a parent referred to the death of a mother or a father; 3) Provide or arrange care referred to providing or arranging care for an impaired friend or relative; 4) Involuntary job loss measured whether the respondent involuntarily lost a job for reasons other than retirement; 5) Residential move referred to moving to a new residence.

We included five chronic strains in parenting, financial, job, health, and caregiver domains, measured at Times 1, 2, and 3. 1) Parental role stress was measured with three items that tapped into overall satisfaction with being a parent, frequency of feeling upset or bothered as a parent, and degree of happiness with the way children had turned out to this point ($\alpha = .63$ at Time 1; $\alpha = .68$ at Time 2; $\alpha = .66$ at Time 3; nonparents were coded as 0). 2) Financial stress was comprised of three items including difficulty meeting monthly payments on bills, how well finances usually work out at the end of the month (some left over, just enough, not enough to make ends meet), and satisfaction with present financial situation (completely satisfied, very satisfied, somewhat satisfied, not very satisfied, not at all satisfied; $\alpha = .77$ at Time 1, $\alpha = .80$ at Time 2, $\alpha = .73$ at Time 3). 3) Job stress referred to the frequency of feeling bothered or upset at work (never, rarely, sometimes, often, always; respondents who were not employed in a paid job were coded as 0). 4) Health-related stress was reflected in activity limitations due to health conditions (scored 1–5, with higher score indicating more limitations). 5) Care provider stress referred to how stressful it was to provide/arrange care for an impaired friend or relative (not at all stressful, not too stressful, somewhat stressful,

quite stressful, and very stressful; respondents who did not provide or arrange care for others were coded as 0).

We first standardized each dichotomized life event variable, and then summed the standardized life event variables and the standardized chronic strain variables into the measure of adulthood stress. Standardization ensured that the measures were weighted equally (Turner et al., 1995). The final adulthood stress measure was standardized with an unweighted mean of 0 and an unweighted standard deviation of 1 at each wave. We used only the first three waves of adulthood stress information because some survey questions were altered or became unavailable in the fourth wave. There were a small number of missing cases for the adulthood stress measure with less than 1% at each wave. Excluding those missing cases (results not shown) did not change our results.

Body-Mass-Index—Body weight was measured using the Body Mass Index (BMI), which was calculated as weight (in kilograms) divided by height-squared (in meters) (World Health Organization, 1995). We examined the BMI as a continuous variable in growth curve analyses because we were focusing on trajectories and degrees of change in body mass over time. A number of studies point to the importance of even small amounts of change in BMI for health (Breeze et al., 2006; Ogden, 2007). There are 79 (Time 1), 55 (Time 2), 44 (Time 3) and 46 (Time 4) missing values for BMI in the data. ACL protocol involved regression-based imputation for missing weight and imputation for missing height based on sex-specific mean height, and then calculating values for missing BMI based on imputed weight and height (House, 2003). We used the ACL-provided missing-imputed values for BMI in the analysis. Additional analysis (not shown) excluding those missing cases revealed similar results.

Gender and Covariates—Gender (1 = male, 0 = female) was included primarily as a moderator. Other covariates included age (in continuous years, centered at the mean), education (years of schooling, centered at the mean), income (in dollars, centered at the mean), race (1 = black, 0 = others), previously divorced (1 = yes, 0 = no), and previously widowed (1 = yes, 0 = no)—all measured at Time 1. We controlled for marital continuity and transitions across waves, including continuously married T1-T4 (reference group), continuously unmarried T1-T4, and any marital transitions T1-T4. We also included a time-varying covariate for birth of a child, which was indicated by the presence of a child younger than 3 years in the household at Time 1 or the birth of a child between subsequent survey waves.

Statistical Methods

We conducted latent growth curve analyses using a Structural Equation Modeling (SEM) approach to assess the effects of stress on BMI trajectories. Each individual began the study period with different initial levels of stress and BMI. Moreover, each individual had a unique history of change in adulthood stress and BMI over the study period. Two growth trajectories were modeled for changes in adulthood stress and BMI respectively. We used information from the first three waves on adulthood stress change to predict four waves of change in BMI. Initial level and rate of change in both BMI and adulthood stress were

viewed as growth parameters that varied randomly among respondents. Because experiences of childhood stress might influence both adulthood stress and BMI change, we included childhood stress as a potential predictor for both trajectories of body mass and adulthood stress. The structural parameters from the model provided the basis for assessing effects of stress on BMI trajectories. The linear growth curve model we used was specified as:

$$Y_{ij} = \pi_{0i} + \pi_{1i}T_{ij} + \varepsilon_{ij}$$

$$\pi_{0i} = X_i' A_0 + b_0 C_i + \gamma_{00} \phi_{0i} + \zeta_{0i}$$

$$\pi_{1i} = X_i' A_1 + b_1 C_i + \gamma_{10} \phi_{0i} + \gamma_{11} \phi_{1i} + \zeta_{1i}$$

$$S_{ij} = \phi_{0i} + \phi_{1i}T_{ij} + v_{ij}$$

$$\phi_{0i} = X_i' A_2 + b_2 C_i + v_{0i}$$

$$\phi_{1i} = X_i' A_3 + b_3 C_i + v_{1i}$$

Where Y_{ij} represented BMI of individual i at Time j . S_{ij} represented the adulthood stress of individual i at Time j . T_{ij} was the time factor indicating the number of years since the beginning of the study. π_{0i} and π_{1i} represented the latent intercept and latent slope of body mass trajectory respectively, which were affected by the growth parameters of the stress trajectory indicated by ϕ_{0i} and ϕ_{1i} . γ_{00} , γ_{10} , and γ_{11} were the structural parameters for the effects of stress trajectory on body mass trajectory. Childhood stress (indicated by C_i) along with vectors of other covariates (indicated by X_i) predicted both growth trajectories of BMI and adulthood stress. b_0 , b_1 , b_2 and b_3 were the corresponding coefficients for childhood stress, and A_0 , A_1 , A_2 and A_3 were corresponding coefficients vectors for the other covariates. ε_{ij} , ζ_{0i} , ζ_{1i} , v_{ij} , v_{0i} , and v_{1i} were residual terms. We also estimated covariances between residuals in growth parameters within growth trajectories.

To fully understand possible gender variation in the patterns of weight change in response to stress, we conducted multiple group analysis (i.e., stratified by men and women) with the specified growth curve model. Additional analysis using Z-tests (results not shown but available upon request) suggested that gender differences in all of our key findings are statistically significant. We employed multiple goodness-of-fit indices developed for SEM for purposes of model evaluation and revision. The smaller the value of the Root Mean Square Error of Approximation (RMSEA), the better the model fit, with a value lower than 0.08 indicating a good model fit (Browne & Cudeck, 1993). For Comparative Fit Index

(CFI), a value of greater than 0.90 indicates a reasonably good model fit (Hu & Bentler, 1999). Similarly, Tucker–Lewis Index (TLI) indicates a good model fit with a value greater than 0.90 (Sharma, Mukherjeeb, Kumarc, & Dillond, 2005). All models were estimated using Mplus software (Muthen & Muthen, 2007). Because the original ACL sample included a wide age range (24–96 in 1986), we conducted additional analysis (results not shown but available upon request) using narrowed age ranges such as 24–64 and 24–54. These results revealed similar patterns as we reported based on the full sample.

We used Full Information Maximum Likelihood (FIML) methods to handle missing values due to survey attrition or missing reports. The FIML approach maximizes a casewise likelihood function using only those observed variables with the assumption of missing at random (MAR) (Little & Rubin, 2002; Muthen & Muthen, 2007). To address the concern that sample attrition due to mortality is not random, we further apply the approach developed by Heckman (Heckman, 1979) to adjust sample selection bias due to mortality selection. This approach consists of modeling the hazard that a respondent would die during the study period using Cox regression models, conditional on a set of predictors. Then, in the final SEM analysis, BMI is modeled as a function of a set of independent variables, including the estimated death hazard. Following this Heckman-type correction, estimates of BMI should be interpreted as adjusted for factors that may affect that risk, as well as for mortality risk. This approach has been applied in previous longitudinal investigations to adjust for sample selection bias due to mortality (e.g., Liu, 2012; Liu & Waite, 2014).

RESULTS

Descriptive Results

Table 1 shows weighted descriptive statistics for all analyzed variables for the total sample as well as by gender. From Table 1, we can see that on average, men had greater BMI than women at each wave; women experienced higher levels of childhood stress than did men, although gender difference in adulthood stress was not significant. Compared with men, women were older, had less education and lower income, were more likely to be previously divorced/widowed, continuously unmarried, and experience marital transitions; and they were less likely to be continuously married or experience new births.

Estimated Stress Trajectories for Men and Women

We began by assessing whether and how the experience of childhood stress differed for men and women. Panel A of Table 2 shows the Ordinary Least Squares regression results for gender differences in childhood stress, and suggests that men experienced significantly lower levels of childhood stress than did women ($b = -.109$, $p < .05$) after controlling for socio-demographic covariates.

Next, we discuss how typical trajectories of adulthood stress unfolded over time and whether these trajectories differed for men and women. Panel B of Table 2 shows growth curve results for the estimated adulthood stress trajectories depicted by the initial level, or latent intercept, and the rate of change, or the latent slope, in adulthood stress over time with childhood stress and socio-demographic covariates controlled. The mean values of the latent

intercept (intercept = .224, $p < .001$) and slope (intercept = $-.012$, $p > .05$) indicate population average growth trajectories of adulthood stress, and suggest an average stable stress pattern over time for the total sample. Results in Panel B of Table 2 also suggest significant individual variation in stress trajectories in adulthood after covariates were included—indicated by significant variances in the random intercept (variance = .380, $p < .001$) and random slope (variance = .005, $p < .001$) for stress trajectories over time.

Panel B of Table 2 also shows the estimated effects of gender and childhood stress along with other socio-demographic covariates on the initial level and rate of change in adulthood stress over time. Childhood stress was positively associated with initial levels of adulthood stress ($b = .104$, $p < .001$) but childhood stress did not have significant effects on the rate of change in adulthood stress over time ($b = -.002$, $p > .05$). Thus, it seems that higher levels of childhood stress may translate into higher levels of stress throughout adulthood but these higher levels of stress in adulthood, due to childhood stress, appear to be stable rather than continuing to escalate over time. Gender differences in trajectories of stress in adulthood were not statistically significant.

Estimated Effects of Stress on BMI

After exploring stress trajectories, we examined how these trajectories were associated with respondents' BMI trajectories. To better illustrate the key results for the effects of stress on BMI trajectories from the multiple group growth curve analysis, we drew structural path diagrams for men and women in Figure 1. All model-fit indexes (CFI = .987; TLI = .980; RMSEA = .022) indicate the model has a good fit. In Figure 1, the “BMI intercept” and “adulthood stress intercept” are latent constructs indicating initial levels of BMI and stress in adulthood, respectively, at the beginning of the study period; the “BMI slope” and “adulthood stress slope” reflect the changing rate of BMI and stress in adulthood, respectively, over the study period. Figure 1 shows that, for women, childhood stress was positively associated with initial levels of stress in adulthood ($b = .103$, $p < .001$), and initial levels of stress in adulthood were then positively associated with initial body mass ($b = 1.316$, $p < .001$). Moreover, childhood stress was directly and positively associated with the changing rate of BMI over time ($b = .016$, $p < .05$). That is, women who experienced higher levels of childhood stress tended to have higher initial levels of stress in adulthood, and thus heavier body mass at the beginning of the study period, and they also gained body mass more rapidly over time in comparison to women who experienced lower levels of childhood stress. In addition, although initial levels of stress in adulthood were positively associated with initial BMI ($b = 1.316$, $p < .001$), initial levels of stress in adulthood were not significantly related to BMI change over time ($b = .006$, $p > .05$). There was no evidence that change in adulthood stress was associated with BMI change for women ($b = .007$, $p > .05$). Therefore, we see that childhood stress appears to be more important than adulthood stress in affecting women's BMI trajectories over time. However, these linkages of stress with BMI trajectories were not apparent for men, as shown in Figure 1. It is also noteworthy that for both women and men, there was a significant negative correlation between initial levels of and change in adulthood stress; and also for women but not for men, there was a significant negative correlation between initial levels of and change in BMI.

DISCUSSION

The link between stress and weight change has been well documented (Block et al., 2009; Pine et al., 2001; Udo, Grilo, & McKee, 2014; Wardle et al., 2011). However, few studies have examined how stress trajectories that begin in childhood and fluctuate throughout adulthood are connected to have long-term consequences for weight change throughout the life course (Gundersen et al., 2011). We use a stress and life course perspective to view weight change over the life course as a developmental process that reflects past and present exposure to stress. The classic stress and coping model suggests that stress results in psychological and physiological arousal and individuals may engage in a range of health behaviors, including overeating or eating high fat foods, in an attempt to cope with or reduce that arousal (Umberson, Liu & Reczek, 2008). We suggested that this process might lead to an increase in body mass over time that is reflected in population-level weight disparities. If patterns of stress response are established in childhood, the effects may carry over into adulthood and manifest as higher levels of body mass that are sustained over time or as an escalating pattern of BMI increase that continues throughout life. We analyzed national longitudinal data to assess body mass trajectories as continuous processes in response to earlier childhood stress as well as ongoing stress levels during adulthood for men and women.

Our results suggest that childhood stress has stronger effects on BMI increases over time than does stress in adulthood, especially for women (partly consistent with Hypothesis 1a). At the beginning of the study period, women who reported higher levels of childhood stress also reported higher levels of stress in adulthood; in turn, higher baseline levels of adulthood stress are associated with higher initial BMI (partly consistent with Hypothesis 1b). However, over time, childhood stress has a direct and long-lasting effect driving a more rapid increase in women's BMI over time, regardless of how women's stress levels fluctuated in adulthood (partly inconsistent with Hypothesis 1b). This is mainly because change in adulthood stress and change in BMI are not significantly associated with each other over time (discussed below). Change in body mass is a process that unfolds throughout life, and childhood may be a critical life period for establishing patterns that have a long term impact on trajectories of change in body mass over the entire life course (Dietz, 1994; Glavin et al., 2014). This finding fits with a life course perspective on cumulative (dis)advantage processes positing that stress processes launched in childhood reverberate throughout the life course to shape trajectories of change in health years and even decades later (Hayward & Gorman, 2004). Our results suggest that childhood stress also sets a long-term trajectory of weight gain into motion, having a cumulative effect on body mass over time, *but only for women*.

We found that not only did women experience higher levels of childhood stress than did men, but childhood stress also had a stronger effect on women's BMI increases than on men's (consistent with Hypothesis 2). Indeed, stress experienced in either childhood or adulthood has little impact on change in body mass for men. These findings fit with prior work suggesting that men and women respond to stress in different ways (Rosenfield & Mouzon, 2013). It may be that women are more likely to increase the amount they eat as a way of coping with stress and thus experience weight gain in response to stress, whereas

men are more likely to engage in other less weight-related coping strategies such as isolating themselves or drinking alcohol in response to stress, possibilities that should be investigated in future research. Our findings add to recent work suggesting that different social locations associated with gender may lead to different ways of coping with stress (Bird & Rieker, 2008; Matud, 2004), and these ways of coping have implications for health and mortality outcomes throughout life.

Finally, we did not find evidence that changes in stress trajectories during adulthood led to BMI change over time for either men or women (partly inconsistent with Hypothesis 1a). Previous studies that focus only on stress in adulthood and BMI trajectories may overestimate the effect of adulthood stress because the origins of adult stress levels are found in childhood. It is childhood stress that launches a lifelong pattern of weight gain, especially for women. This result may also reflect the relatively stable trajectories of adulthood stress over the study period we examined. However, we found that higher initial levels of stress in adulthood (which is predicted by childhood stress and remain quite stable over time) are related to greater BMI for women throughout the life course, and greater body mass due to initial adulthood stress persists for women over time regardless of how stress levels fluctuate in adulthood. These results suggest that the overall level of stress in adulthood is more important than change in adulthood stress in contributing to women's weight gain. Some studies suggest that eating may help to alleviate arousal evoked by exposure to excessive stress and to regulate mood state, at least temporarily (Dallman et al., 2006). Through this process, levels of stress may shape women's body mass, with sustained consequences for body mass over time.

Note, for both women and men, we find a negative correlation between initial levels of and change in adulthood stress—indicating that those with higher initial levels of adulthood stress experience a less rapid increase in stress over time than those with lower initial levels of stress. For women but not for men, we also find a negative correlation between initial levels of and change in BMI—indicating that women with higher initial BMI tend to increase BMI at a slower rate than women with lower initial BMI. These results may be related to a ceiling effect in that people with very high levels of stress/BMI may have less capacity for further increase in stress/BMI when compared with people with lower levels of stress/BMI.

Possible Explanatory Mechanisms

Future research should direct particular attention to testing the possible explanatory mechanisms through which childhood and adulthood stress work together to influence sex-specific patterns of weight gain throughout the life course. We have largely drawn on a stress and life course approach to ground our study of gendered patterns of stress and weight gain. The classic stress and coping model suggests that stress results in psychological (e.g., anxiety, depression) and physiological (e.g., increased heart rate and blood pressure) arousal (Amirkhan, 2012) and individuals may engage in a range of health behaviors, including overeating, in an attempt to cope with or reduce that arousal (Umberson, Liu & Reczek, 2008). Indeed, some studies suggest that eating may help to alleviate arousal and to regulate mood state, at least temporarily (Dallman et al., 2006; Kassel, Stroud & Paronis, 2003).

Stress research also shows that women are more likely than men to engage in emotion-focused coping in response to stress (Matud, 2004), and this type of coping may be associated with emotion-driven food consumption (Spoon, et al., 2006).

In addition to a stress and coping explanation, other potential mechanisms for our finding of a female-specific stress/BMI relationship should be considered. For example, stress may evoke sex-specific hormonal responses that influence eating behavior. A fight/flight response to stress that is more common among men may evoke glucose production that suppresses appetite (Camilleri et al., 2014). In contrast, if women are more emotionally reactive to stress and stress triggers cortisol production that promotes appetite (particularly for calorie-dense sweet foods) (Beydoun, 2014), this may provide a partial explanation for the sex-specific weight gain we find for women in response to stress. This pattern might be exacerbated for women because they are more likely than men to seek social affiliation in response to stress (Taylor et al., 2000). In turn, social affiliation is associated with more social eating and calorie consumption (Herman, Roth & Polivy, 2003).

Another possibility is that childhood stress produces neurobiological effects on brain development that differ for males and females and, in turn, these neurobiological manifestations have sex-specific functional consequences throughout life (Teicher et al., 2003), perhaps including consequences for weight-related stress responses. Gender differences in depression may also help explain the female-specific stress/BMI relationship in adult populations. Stress contributes to depression and females are more likely than males to be depressed after adolescence (Bird & Rieker, 2008). Previous studies show that depression is associated with weight gain in childhood (Pine et al., 2001) and adulthood (Singh et al., 2013). Moreover, depression has been associated with emotion-driven eating and consumption of energy-dense foods (Camilleri, et al., 2014), as well as greater weight gain for women than for men in adulthood (Singh et al., 2013).

Limitations

We note several limitations of the present study. First, our measures of childhood stress as well as stressful life events are based on retrospective reports which may involve biases and measurement errors due to missing reports (Hardt & Rutter, 2004). However, previous studies provide evidence to support the validity and reliability of retrospective reports of childhood and prior life experiences (Hardt & Rutter, 2004; Laursen & Little, 2012; Schwarz & Sudman, 2012). Second, our stress measures do not take into account perceived severity of stressful life events, which could explain some of the gender difference in the association of stress and BMI. Third, our measure of BMI is calculated based on self-reported weight and height that may be subject to reporting biases. Yet, studies have demonstrated the validity and accuracy of self-reported weight and height as measures for BMI (Stommel & Schoenborn, 2009). Fourth, although we control for a number of socio-demographic covariates, we may not include all potential confounders in the statistical models due to data limitations. Fifth, we suggested that stress affects weight change. However, weight change may also affect subsequent stress levels (Singh, Jackson, Dobson & Mishra, 2013). Although our strategy of using the first three waves of stress to predict BMI change throughout the 15-year study period aimed to control for some effects of

temporal order, we cannot rule out the possibility that BMI change alters subsequent stress levels in adulthood. This limitation points to the need for future research to rely on longitudinal data to further assess the potential endogeneity of stress using alternative models that take into account the potential reciprocal relationships between stress and BMI change over time. Finally, various social, psychological and behavioral pathways work together to forge the links between stress and BMI change. Future research should identify the specific mechanisms that underlie these linkages.

CONCLUSION

The present study adds important evidence concerning patterns of a female-specific impact of stress on weight gain. Our analysis suggests that stress, especially as experienced in childhood, is linked to increasing body mass over time, but only for women. Because the increase in body mass may be cumulative over a long period of time, much of the health risk associated with childhood stress and weight gain may not become apparent until later in adulthood. These findings highlight the need for interventions and policies designed to reduce stress in childhood, with an eye to reducing the sex-specific and long-term adverse consequences of childhood stress for stress levels in adulthood as well as population patterns of weight gain. Clinical care and intervention programs addressing obesity often focus on more proximate behavioral factors such as healthy eating and physical exercise in adulthood to reduce obesity risk. Our study highlights the importance of considering sex-specific social contexts of early childhood in order to design effective clinical programs that prevent or treat overweight and obesity later in life. Given the importance of body mass for health and disability, it is imperative that we develop theoretical and analytical models to shed additional light on the ways in which stress maps onto body mass trajectories throughout the life course with special attention to gender differences in these processes.

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How childhood and adulthood stress works together to affect weight change over time is unclear.

We use growth curve models to examine the linkages of childhood, adulthood stress and weight change.

Childhood stress has stronger effects on weight change over time than does stress in adulthood.

Childhood stress has a stronger effect on women's weight gain than on men's.

Sex-specific childhood context is important for designing clinical programs.

FIGURE 1. Structural Path Diagram of Childhood Stress, Adulthood Stress and BMI Trajectories for Women (N=2259) and Men (N=1358) from the Multiple Group Growth Curve Model

* $p < .05$, ** $p < .01$, *** $p < .001$. CFI = .987; TLI = .980; RMSEA = .022.

Model controlled for age (T1), age-squared (T1), black (T1), education (T1), family income (T1), previously divorced (T1), previously widowed (T1), marital transitions from T1 to T4, a time-varying covariate of birth as well as death hazard.

Bolded lines indicate significant paths. Numbers are unstandardized regression coefficients in the diagram. The output with standardized coefficients is not available in Mplus for

random effects estimates due to the multiple variance/covariance matrix (Muthen & Muthen, 2007)

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TABLE 1

Weighted Descriptive Statistics for the Variables Analyzed

	Total (N=3617)		Women (N=2259)		Men (N=1358)	
	Mean/%	S.D.	Mean/%	S.D.	Mean/%	S.D.
Body Weight						
Body Mass Index at T1	25.52	4.66	25.11	5.07	25.98	4.10 *
Body Mass Index at T2	26.04	4.86	25.56	5.23	26.62	4.32 *
Body Mass Index at T3	26.56	5.03	26.09	5.31	27.11	4.63 *
Body Mass Index at T4	27.44	5.33	27.00	5.63	27.97	4.90 *
Stress						
Adulthood Stress at T1	-0.02	1.01	-0.01	0.98	-0.03	1.05
Adulthood Stress at T2	-0.05	1.00	-0.06	0.97	-0.03	1.04
Adulthood Stress at T3	-0.00	1.00	0.00	0.98	-0.01	1.03
Childhood stress T1	0.84	1.21	0.91	1.27	0.75	1.13 *
Socio-demographic Covariates						
Age T1	47.11	16.45	48.12	16.85	45.98	15.91 *
Male T1 (%)	47.09					
Black T1 (%)	10.97		11.84		9.99	
Education T1	12.37	3.14	12.19	3.00	12.56	3.27 *
Family Income (\$1000s) T1	30.45	24.04	28.06	23.69	33.13	24.16 *
Previously Divorced T1 (%)	24.51		25.88		22.98	* *
Previously Widowed T1 (%)	11.81		16.98		6.01	* *
Marital Transitions T1-T4						
Continually Married (%)	48.83		42.19		56.30	* *
Continually Unmarried (%)	16.34		20.53		11.64	* *
Any Marital Transitions (%)	34.82		37.28		32.06	* *
Birth of New Baby Within 3 Years at T1 (%)	10.51		9.06		12.15	* *
Birth of New Baby T1-T2 (%)	7.08		6.15		8.13	* *
Birth of New Baby T2-T3 (%)	4.36		3.17		5.69	* *
Birth of New Baby T3-T4 (%)	0.40		0.47		0.33	

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T1: Time 1; T2: Time 2; T3: Time 3; T4: Time 4.
* Gender difference is significant at the level of $p < 0.05$.

TABLE 2

Estimated Unstandardized Regression Coefficients for Childhood Stress from Ordinary Least Squares Regression Model and for Adulthood Stress from Linear Growth Curve Model (N=3617)

	A. Childhood Stress ^a		B. Adulthood Stress Trajectories	
	b	(s.e.)	Latent Intercept	Latent Slope
Men	-0.109*	(0.044)	0.034	(0.033)
Age	-0.009***	(0.001)	-0.012***	(0.001)
Age-squared	0.000	(0.000)	0.000	(0.000)
Black	0.067	(0.046)	0.113***	(0.034)
Education			-0.010*	(0.005)
Family Income			-0.008***	(0.001)
Previously Divorced			0.191***	(0.037)
Previously Widowed			0.006	(0.046)
Marital Transitions T1-T4 (ref: Continually Married)				-0.010 (0.008)
Continuously Unmarried			0.113*	(0.046)
Marital Transitions			0.144***	(0.038)
Birth of New Baby			-0.062	(0.038)
Childhood Stress			0.104***	(0.016)
Intercept	0.860***	(0.039)	-0.224***	(0.037)
Residual Variance	1.252***	(0.033)	0.380***	(0.028)
R-squared/Chi-square Value	0.023		150.660	

* p < .05,
** p < .01,
*** p < .001

^aIn the model to predict childhood stress, we only controlled for gender, age and race due to concerns of the time order since other covariates were all referred to adulthood experience. Our additional analysis (not shown) with all those covariates controlled revealed similar patterns as we reported in this table.