Implications of life-history strategies for obesity

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The association between low socioeconomic status (SES) and obesity is well documented. In the current research, a life history theory (LHT) framework provided an explanation for this association. Derived from evolutionary behavioral science, LHT emphasizes how variability in exposure to unpredictability during childhood gives rise to individual differences in a range of social psychological processes across the life course. Consistent with previous LHT research, the current findings suggest that exposure to unpredictability during childhood (a characteristic common to low SES environments) is associated with the adoption of a fast life-history strategy, one marked by impulsivity and a focus on short-term goals. We demonstrate that a fast life-history strategy, in turn, was associated with dysregulated weight-management behaviors (i.e., eating even in the absence of hunger), which were predictive of having a high body mass index (BMI) and being obese. In both studies, findings held while controlling for participants' current socioeconomic status, suggesting that obesity is rooted in childhood experiences. A serial mediation model in study 2 confirmed that effects of childhood SES on adult BMI and obesity can be explained in part by exposure to unpredictability, the adoption of a fast life-history strategy, and dysregulated-eating behaviors. These findings suggest that weight problems in adulthood may be rooted partially in early childhood exposure to unpredictable events and environments. LHT provides a valuable explanatory framework for understanding the root causes of obesity.

evolutionary psychology | evolutionary medicine | life history theory | health behavior | obesity

More than one third of American adults and about one third of children and adolescents are currently obese, and an even greater number are overweight (1). Obesity is a risk factor for many negative health outcomes including heart disease, type 2 diabetes, hypertension, liver disease, cancer, and stroke (2). Consequently, identifying the behavioral factors that increase obesity is a key goal for research.

This article provides an evolutionary perspective on obesity by using a life history theory (LHT) approach. Derived from evolutionary biology, LHT suggests that adaptive psychological processes are calibrated early in life to help people maximize their reproductive potential across the lifespan, given the contingencies they encounter in their local childhood environment (3, 4).

Although existing epidemiological research has used a life-course perspective to understand how childhood psychosocial variables contribute to adult disease (5), no studies to our knowledge have adopted a LHT approach. Recognizing developmental factors suggested by LHT not only enhances our theoretical understanding of obesity but also generates insight into possible interventions.

Work in the LHT tradition finds that the degree of unpredictability in an organism's early-life environment influences behavior in a variety of domains throughout the life course (6). LHT is rooted in the idea that organisms have a finite energy budget that must be allocated in a way that maximizes overall reproductive fitness. LHT emphasizes that an organism's fitness-maximizing strategy is adaptively calibrated to the level of unpredictability it encounters early in its development. A high level of unpredictability signals that the future is uncertain, increasing the extent to which the organism invests in short-term pursuits tied to immediate reproduction. A low level of unpredictability signals that the future is relatively certain and that the organism can afford to adopt a strategy marked by a longer time horizon and greater investment in somatic effort over the long term. These responses to early developmental unpredictability are referred to as "fast" and "slow" life-history strategies, respectively. Hence, life-history strategies vary on a continuum from fast to slow and serve to optimize an organism's reproductive success given the level of unpredictability encountered early in development.

Faster life-history strategies are characterized by behaviors that increase immediate reproduction, such as having more sexual partners earlier in life and a larger number of total offspring and displaying an orientation toward impulsivity, risk-taking, and short-term rewards (7, 8). Conversely, slower life-history strategies are characterized by behaviors that emphasize long-term investment in fewer offspring, such as delaying reproduction until later in life, having fewer sexual partners, and delaying gratification in favor of long-term rewards (9).

LHT provides insight into the psychological processes underlying obesity. Growing up in an environment characterized by unpredictability means not knowing when resources will be available in the future and so activates psychological processes that promote the consumption of resources when they are immediately available. This mindset may lead people to consume food when it is available, regardless of whether they currently require the nourishment. If the availability of food in the future is uncertain, it may behoove one to consume as much as possible when one has access to it. This behavior would have been highly

Significance

The prevalence of obesity reflects a major public health challenge in the United States and many other parts of the world. The current studies adopt a life history theory (LHT) perspective to explain in part the root causes of obesity. This research identifies exposure to unpredictable environments during childhood as a distal factor that contributes to obesity in adulthood. Exposure to childhood unpredictability is linked to the development of a fast life-history strategy, one marked by impulsivity and a focus on short-term goals. LHT provides a valuable conceptual framework for understanding the behavioral mechanisms that underlie dysregulated patterns of eating and, ultimately, the development of weight-management problems in adulthood. This research also provides insight into pathways for obesity prevention.

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functional in ancestral times, when attaining enough food was a persistent challenge (10).

The potential role of life-history strategies in obesity is indicated by the well-known association between low socioeconomic status (SES) and obesity (11–13). Conditions associated with low SES include uncertainty and resource scarcity, reflecting the type of harsh and unpredictable conditions that promote the development of fast life-history strategies. Indeed, recent work showed that low childhood SES is associated with a tendency among adults to eat even when nourishment is not currently needed (14; also see ref. 15). Although such behavior may have been adaptive in ancestral environments, it sets the stage for dysfunctional patterns of overeating in modern environments. Moreover, even when individuals from low-SES environments improve their circumstances and transition into more stable, higher-SES environments, their faster life-history strategy may persist, promoting a continued tendency to eat in the absence of current energy needs.

Despite recent laboratory evidence documenting overeating among low-SES people (14), we are aware of no previous studies examining the role of LHT in predicting a tendency to be overweight or obese. Moreover, although previous research is consistent with hypotheses derived from LHT, studies often have used low SES as a proxy for childhood unpredictability and have fallen short of directly examining the role of unpredictable childhood environments. The current research addresses these important gaps in the literature.

Study 1 leveraged data from a longitudinal study of an ethnically and racially diverse sample of 226 individuals (113 married couples) to examine the association between childhood SES and participants' weight and obesity status in adulthood. We predicted that lower childhood SES would be associated with higher weight and a greater likelihood of being obese in adulthood. Study 2 used a large online sample ($n = 400$) to test a serial mediation model accounting for the associations among childhood SES, childhood unpredictability, life-history strategy, the presence of dysregulated eating behaviors, and obesity. That study also allowed us to test a critical theoretical assumption that to our knowledge has been left untested—that effects of low SES on weight problems are caused in part by exposure to unpredictability in childhood.

In both studies, we conducted analyses controlling for current SES. These analyses allowed us to isolate the role of childhood experiences and rule out the possibility that weight problems in adulthood simply reflect low current levels of SES. Other studies have shown that the associations between childhood SES and many chronic health conditions are eliminated when controlling for current SES (16). The current research sought to show that current SES does not account for the link between childhood unpredictability and adult weight.

Study 1: Results and Interim Discussion

Before testing our primary prediction, we first used growth-curve modeling to estimate participants' body mass index (BMI) trajectories. We estimated the following first level of a three-level model using the Hierarchical Linear Modeling 7 computer program (17):

$$\mathrm{Y_{tic}(BMI)} = \pi_{0ic} + \pi_{1ic}(\mathrm{Time}) + e_{tic}. \quad [1]$$

in which (*i*) *t* indexes time, *i* indexes individuals, and *c* indexes couples; (*ii*) time represents the wave of assessment and was coded from 0 to 6 (so that the intercept represented initial BMI at the start of marriage); (*iii*) the autocorrelation from repeated assessments was controlled in level 2, and the shared variance between husbands' and wives' data was controlled in level 3; and (*iv*) all level 2 estimates and the level 3 intercept were allowed to vary randomly. We used restricted maximum likelihood estimation and placed no restrictions on the autoregressive error structures.

Both men and women reported mean BMI levels that fell in the overweight range at the beginning of the study (for men, $\pi = 28.02$, SE = 0.68; for women, $\pi = 27.50$, SE = 0.78) and, on average, experienced significant increases in BMI over the three-and-a-half-year study (for men, $\pi = 0.25$, SE = 0.06; for women, $\pi = 0.28$, SE = 0.06). Men and women reported similar initial BMIs [$b = -0.52$, SE = 0.79, $t(78) = -0.65$, $P = 0.515$] and similar increases in BMI over the course of the study [$b = 0.03$, SE = 0.09, $t(78) = 0.33$, $P = 0.740$].

Notably, we observed substantial between-subjects variability in both initial BMI [χ^2 (2) = 481.99, $P < 0.001$] and change in BMI [χ^2 (2) = 10.41, $P = 0.006$]. The primary analysis examined whether childhood family income partially accounted for this variability. We predicted that individuals whose families had relatively few financial resources during childhood (controlling for current income) would report higher levels of BMI and steeper increases in BMI over time. To test these predictions, parameters estimated in Eq. **1** (initial BMI and change in BMI) were regressed onto childhood family income in level 2. We controlled (*i*) participants' current income (grand-centered) at level 1 to isolate the effect of childhood family income and (*ii*) a dummy code of whether the childhood income variable was assessed at baseline (coded 0) or at time 4/time 6 (coded 1) at the level-2 intercept (Table 1).

Consistent with predictions, individuals whose families had less (compared with more) money during childhood had higher BMIs at the start of their first marriages. Further, childhood family income was marginally associated with changes in BMI over time, such that those from lower-income childhoods reported greater increases in BMI over the 3.5 y of the study. Notably, these associations (*i*) were not moderated by participant sex [for initial BMI: $b = -0.30$, SE = 0.69, $t(73) = -0.43$, $P = 0.667$; for change in BMI: $b = 0.16$, SE = 0.17, $t(73) = 0.92$, $P = 0.360$] and (*ii*) remained unchanged when we no longer included participants' current income as a level 1 covariate or the dummy code for time of childhood income assessment as a level 2 covariate [for initial BMI: $b = -0.01$, SE = 0.00, $t(78) = -2.44$, $P = 0.017$; for change in BMI: $b = -0.00$, SE = 0.00, $t(78) = -1.84$, $P = 0.070$].

We conducted similar analyses that replaced BMI as the dependent variable with a dichotomous variable reflecting participants'

Table 1. Relationship between childhood family income and BMI

Variable	Coefficient	Standard error	*P* value	Effect size, *r*
Intercept	27.85	0.63		
Time of income assessment	−0.80	1.00	0.424	0.09
Childhood family income	−0.01	0.00	0.014	0.28
Time	0.25	0.04	<0.001	0.54
Childhood family income	−0.00	0.00	0.074	0.20
Current income	0.00	0.00	0.429	0.07

Level 1 coefficients are πs; level 2 coefficients are *b*s; *df* = 102 for intercept; *df* = 77 for time, time of income assessment, and childhood family income variables; *df* = 135 for current income variable.

Table 2. Zero-order correlations among key variables in study 2

Key variables	1	2	3	4	5	6	7
1. Childhood SES	—						
2. Current SES	0.23***	—					
3. Unpredictability	−0.29***	−0.06	—				
4. Life-history strategy	0.10*	0.29***	−0.32***	—			
5. Dysregulated eating	−0.04	−0.09^{+}	0.004	−0.13**	—		
6. BMI	−0.13*	−0.14**	0.05	−0.04	0.32***	—	
7. Obesity	−0.14**	−0.18***	0.09^{+}	−0.09^{+}	0.31***	0.78***	—

Significance: ***$P < 0.001$; **$P < 0.01$; *$P < 0.05$; $^{+}P < 0.10$.

obesity status. Results mirrored those for BMI scores. Childhood family income was associated with obesity status at study initiation [$b = -0.00$, SE = 0.00, $t(77) = -2.00$, $P = 0.049$]. Although childhood family income was unassociated with changes in participants' obesity status over the course of the study [$\beta = -0.00$, SE = 0.00, $t(77) = -0.94$, $P = 0.351$], childhood family income remained associated with participants' obesity status at the end of the study [$b = -0.00$, SE = 0.00, $t(77) = -3.38$, $P = 0.001$]. That is, individuals whose families had less money during childhood were more likely to be obese at the start of their first marriages, and this difference remained 3.5 y later. These intercept and slope associations (*i*) were not moderated by participant sex [for initial obesity status: $b = -0.18$, SE = 0.30, $t(73) = -0.59$, $P = 0.557$; for change in obesity status: $b = -0.09$, SE = 0.14, $t(73) = -0.67$, $P = 0.507$] and (*ii*) remained unchanged when we no longer included participants' current income as a level 1 covariate or the dummy code for time of childhood income assessment as a level 2 covariate [for initial obesity status: $b = -0.01$, SE = 0.00, $t(78) = -1.95$, $P = 0.054$; for change in obesity status: $b = -0.00$, SE = 0.00, $t(78) = -0.99$, $P = 0.324$].

Thus, in a demographically diverse sample, childhood family income predicted participants' weight and obesity status in adulthood. Moreover, childhood family income predicted increases in BMI over 3.5 y. Importantly, these associations emerged independent of current income, indicating that they do not simply reflect the well-known association between current SES and obesity. Rather, findings are consistent with the hypothesis that early childhood experiences calibrate behavior such that those exposed to unpredictable environments are more likely than those from predictable environments to be obese in adulthood.

Several limitations of this study warrant caution, however. First, rather than measuring childhood unpredictability directly, we used the measure of childhood SES. Although the two constructs are related, a more rigorous test would include a measure of childhood unpredictability. In fact, our theoretical framework suggests a measure of unpredictability should account for the association between childhood SES and weight/obesity. Second, we did not directly examine the role of life-history strategy, instead basing our interpretation on the assumption that lower SES promotes a faster life-history strategy (7). Third, we did not examine the role of dysregulated-eating behaviors, which presumably serve as proximate causes of obesity in those pursuing a fast life-history strategy. Study 2 addressed each of these limitations.

Study 2 Results

Table 2 presents zero-order correlations among all variables.

To assess the predicted relationships among childhood SES, childhood unpredictability, life-history strategy, dysregulated eating, and obesity status, we used the Process macro (18) within a multiple regression framework to conduct a serial mediation analysis (Fig. 1). We entered childhood unpredictability, life-history strategy, and dysregulated-eating behavior as putative mediators of the relationship between SES and obesity. (Similar analyses treating BMI as the outcome variable closely mirrored those reported here and can be found in *SI Study 2*, *Mediational Model of BMI*.) As predicted, lower childhood SES was associated with significantly higher unpredictability [$b = -0.31$, $t(398) = -6.11$, $P < 0.001$]. We also observed the expected association between greater childhood unpredictability and a faster life-history strategy [$b = -0.18$, $t(397) = -6.42$, $P < 0.001$]. Consistent with the idea that childhood unpredictability accounts for the effect of childhood SES on life-history strategy, there was no direct effect of childhood SES on life-history strategy [$b = 0.003$, $t(397) = 0.10$, $P = 0.917$]. Next, entering childhood SES, childhood unpredictability, and life-history strategy as predictors of dysregulated eating confirmed that a faster life-history strategy was associated with more dysregulated eating [$b = -0.24$, $t(396) = -2.80$, $P = 0.005$]; consistent with the idea that life-history strategy is the more proximal predictor of dysregulated eating, neither childhood SES [$b = -0.04$, $t(396) = -0.86$, $P = 0.392$] nor childhood unpredictability [$b = -0.05$, $t(396) = -1.03$, $P = 0.302$] had a direct effect on dysregulated eating. Finally, entering childhood SES, childhood unpredictability, life-history strategy, and dysregulated eating as predictors of obesity revealed an association between dysregulated eating and obesity [$b = 0.58$, $z = 5.71$, $P < 0.001$] and a direct effect of childhood

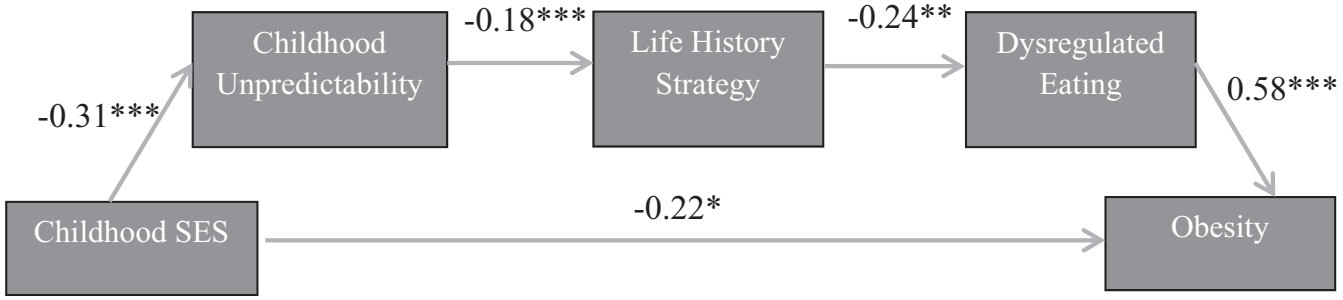

Fig. 1. Study 2: Path model of a serial mediation analysis linking childhood SES to obesity via childhood unpredictability, life-history strategy, and dysregulated-eating behaviors. Numbers refer to unstandardized regression weights. *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

SES [$b = -0.22$, $z = -2.27$, $P = 0.023$]; consistent with the idea that dysregulated eating is the more proximal predictor of obesity, neither life-history strategy [$b = -0.12$, $z = -0.72$, $P = 0.470$] nor childhood unpredictability [$b = 0.90$, $z = 0.98$, $P = 0.325$] had a direct effect on obesity.

To provide a direct test of serial mediation (i.e., the indirect effect of childhood SES on obesity, as mediated through childhood unpredictability, life-history strategy, and dysregulated eating, respectively), a bootstrapping analysis with 5,000 resamples indicated a significant indirect effect, with a point estimate of −0.008 and a 95% bias-corrected CI of (−0.020, −0.002). The data thus are consistent with the hypothesis that participants who experienced lower childhood SES experienced greater levels of childhood unpredictability, which was associated with a faster life-history strategy, which in turn was associated with dysregulated eating, which was ultimately associated with an increased likelihood of obesity in adulthood.

We tested three alternative models, none of which demonstrated good fit to the data. First, childhood SES was not indirectly associated with likelihood of obesity in adulthood merely through dysregulated-eating behaviors [point estimate = −0.025, 95% bootstrap CI = (−0.094, 0.037)]. Second, childhood SES was not indirectly associated with likelihood of obesity merely through childhood unpredictability [point estimate = −0.027, 95% bootstrap CI = (−0.105, 0.023)]. Third, and finally, childhood SES was not indirectly associated with likelihood of obesity merely through life-history strategy [point estimate = −0.0004, 95% bootstrap CI = (−0.015, 0.010)]. These null effects are important because they help rule out alternative models that otherwise would be plausible given the cross-sectional nature of the data. Only the full mediational model fit the data well.

Again suggesting that results are unique to childhood SES, findings remained unchanged when controlling for participants' current SES: All reported paths remained significant, and a bootstrapping analysis with 5,000 resamples indicated a significant indirect effect, with a point estimate of −0.007 and a 95% bias-corrected CI of (−0.018, −0.0008). Full results of the serial mediation analysis controlling for current SES can be found in *SI Study 2*, *Mediational Model of BMI Controlling for Current SES*.

Discussion

The high prevalence of obesity represents a major public health challenge. LHT provides a potentially valuable conceptual framework for understanding the behavioral mechanisms that underlie high rates of obesity. The current work suggests that effects of low SES on adult health outcomes can be partially explained by exposure to unpredictable environments in childhood. We observed a higher likelihood of obesity among adults from low SES childhoods, even after controlling for current SES (study 1), and our findings directly implicate the role of childhood unpredictability as it pertains to the development of a fast life-history strategy and ultimately to dysregulated eating (study 2). Taken together, these studies suggest that exposure to unpredictable environments in childhood may calibrate people's behavior in ways that promote dysregulated eating and obesity in adulthood.

Even though life-history strategies are calibrated in childhood to help people maximize the benefits of their current environments, those strategies can be maladaptive when the environment changes, as it often does when people grow into adulthood. With regard to obesity, even if food (and other social and material resources) is readily available in adulthood, behavior nevertheless may be calibrated to the availability and predictability of resources experienced as a child. Moreover, those strategies ultimately have been designed through evolution to optimize outcomes in the context of ancestral environments and so may not be well-calibrated to modern environments in which high-calorie food is often abundant. Thus, although fast life-history strategies may be adaptive at some level, they can be quite dysfunctional in many current contexts.

LHT provides a theoretical framework for understanding a range of developmental variables that appear to underlie adult eating dysregulation. For example, research has documented a robust link between childhood trauma (e.g., abuse, neglect, parental alcoholism) and obesity in adulthood (19). Viewed through the lens of the current research, such findings can be explained by the fact that forms of childhood trauma are likely to instill a sense of unpredictability and uncertainty in children. Also consistent with a LHT approach are findings suggesting that early menarche in women—an indicator of early sexual maturation and a correlate of fast life-history strategies (20)—is associated with higher BMI during adolescence and into adulthood (11). Thus, LHT provides a unifying framework for understanding a range of early developmental factors associated with obesity.

As such, LHT helps generate predictions about additional factors that might contribute to adult obesity. For example, growing up in a household in which the father leaves the family (e.g., because of divorce) has been identified as a determinant of fast life-history strategies in women (3, 21). Similarly, frequent residential changes and changes in parents' employment status serve as determinants of fast life-history strategies in both sexes (22). Consequently, research may benefit from investigating the link between these factors and adult obesity. Moreover, research suggests that it is unpredictability in childhood, rather than living in a harsh or stressful environment, that determines life-history strategies (6, 22). This finding highlights the importance of differentiating between factors characterized by stressful but predictable conditions (e.g., consistently lacking wealth) and those marked by uncertainty, turbulence, and frequent change. Thus, the current research has important implications for identifying factors that place children at risk for experiencing obesity later in life.

The current research also has implications for developing childhood interventions to combat obesity and identifying the persons most likely to benefit from them. People's life-history strategies appear to be calibrated mainly by exposure to unpredictability during the first 5 y of life (6). Interventions may benefit from targeting children and early adolescents who experience unpredictable environments in those early years. Such interventions could target social-structural factors, in particular sources of uncertainty or unpredictability, that serve as early developmental precursors to obesity later in life. As noted earlier, LHT implies that fast life-history strategies are produced not by low SES or by a lack of resources per se but rather by unpredictable life events (6). Therefore, interventions aimed at addressing some of the antecedents to obesity may benefit from increasing the presence of structure, certainty, and stability in the lives of at-risk children and adolescents.

Such interventions could also help reduce a larger constellation of behavioral problems characteristic of fast life-history strategies, including risk-taking, competitiveness, impulsivity, and low-investment parenting behaviors (23). Moreover, the same forms of childhood trauma known to promote obesity also have been shown to promote other negative health outcomes, including risky sexual behavior (24) and drug use (25). Therefore aside from their implications for reducing obesity, interventions that reduce childhood unpredictability could ameliorate a range of problematic behavioral outcomes known to reflect fast life-history strategies.

The current findings are limited by their reliance on cross-sectional data and retrospective self-report measures. This research is also limited by its lack of focus on other factors shown to contribute to obesity, including structural and lifestyle factors such as the availability of calorie-dense foods, the use of exercise as a weight-management strategy, and cultural norms pertaining

to weight (26). Indeed, results of study 2 show that, even after accounting for people's life-history strategy, SES still exhibited a direct relationship with obesity status, and that relationship might be accounted for by the presence of such factors. Future research would benefit from examining how life-history strategies might interact with such factors. For example, the link between fast life-history strategies and dysregulated eating might be exacerbated by the presence of cultural standards that normalize being overweight or by a lack of access to nutritious foods and means of exercise. Considering such factors within the broader framework of LHT provides an integrative framework with which to understand both proximate and developmental factors that contribute to obesity in adulthood.

The current work is also limited by its lack of focus on genetic and prenatal factors. Some personality traits (e.g., impulsivity) associated with life-history strategies are partially heritable (27), and heritable factors could work in concert with developmental factors to shape eating behavior over the lifespan. Future work would profit from delineating heritable causes underlying adult obesity from developmental causes. Likewise, prenatal calibration of fetal development by the mother could play a role in shaping offspring behavior. Moreover, low birth weight could be correlated with both prenatal development and childhood unpredictability, and the potential role of low birth weight in adult obesity should be a focus of future research. Indeed, future work may benefit from examining the independent (and possibly interactive) roles played by genetic, prenatal, and developmental processes. Future research should also consider alternative theoretical perspectives in addition to the LHT viewpoint that childhood ecological conditions prepare people to operate effectively, assuming that conditions are similar later in life (28, 29). For example, harsh and unpredictable childhood conditions might also influence behavior because those conditions negatively impact people's health, and people may compensate for that impact by speeding up their life course because of the risk of early mortality (30, 31).

Future empirical work would also benefit from including biometric indicators of life-history strategies (e.g., age of menarche, age at the birth of the first child) in addition to psychometric indicators. Psychometric indicators reveal the psychological processes underlying life-history strategies, whereas biometric indicators reveal life-history outcomes, and thus the two types of measures complement one another in important ways (32).

Future research also should continue to examine relationships among particular psychological processes and outcomes thought to reflect fast versus slow life histories. For example, although variables such as valuing long-term committed relationships are generally thought to reflect slow life-history strategies, long-term relationships also afford biparental care and would have been especially adaptive in ancestral environments thought to promote fast life-history strategies (e.g., high prevalence of natural hazards such as predators). Attending carefully to specific psychological processes in future research may reveal relationships with fast versus slow strategies that are nuanced and potentially moderated by context (e.g., the harshness of the environment). In turn, understanding those relationships may shed additional light on links between childhood variables and adult outcomes related to health and well-being.

Finally, it is worth noting that some of the associations documented in this research reflect relatively small effect sizes. Nevertheless, it is also worth noting that even small associations can have considerable importance for public health, particularly for health problems that are highly prevalent in the population (e.g., ref. 33). Given the very high prevalence and public health costs associated with obesity, if even a small proportion of the variance in weight can be accounted for by factors in our model, interventions designed to reduce obesity could be meaningfully improved by taking those factors into consideration.

Materials and Methods

Study 1 Method.

Participants. Participants were 226 first-married newlywed spouses (113 couples) participating in a broader longitudinal study investigating relationship processes. Participants were recruited through invitations sent to couples who had applied for marriage licenses in counties near the study location. Three hundred eighty-nine couples were screened via telephone interviews to ensure the couples met the following criteria, given broader goals of the study: (*i*) they had been married for less than 4 mo, and both partners could attend a laboratory session within the first 4 mo of marriage; (*ii*) neither partner had been previously married; (*iii*) they were at least 18 y of age; and (*iv*) they spoke English (to ensure comprehension of the questionnaires). One hundred fifty-nine couples did not meet eligibility criteria. Of the 226 individuals who participated, 51 failed to provide complete data [21 husbands (18.6%) and 22 wives (19.5%) failed to provide an estimate of childhood family income; two (1.8%) husbands and four wives (3.5%) failed to provide height and weight at baseline and all follow-up assessments]. Thus, the final sample consisted of 90 husbands and 87 wives (of 103 couples).

At baseline, husbands were 27.76 (SD = 5.25) years of age and had completed 15.68 (SD = 2.74) years of education. Seventy-seven percent were employed full time, and 13% were full-time students. Mean income of husbands was $46,696 (SD = $49,455) per year. Wives were 26.93 (SD = 4.13) years of age and had completed 16.40 (SD = 2.59) years of education. Sixty percent were employed full time, and 13% were full-time students. Mean income of wives was $36,731 (SD = $37,649) per year. The sample was relatively diverse; 55% of husbands and 58% of wives self-identified as Caucasian, 19% of husbands and 20% of wives self-identified as Black or African American, 17% of husbands and 12% of wives self-identified as Hispanic or Latino/a, 3% of husbands and 6% of wives self-identified as Asian, and 5% of husbands and 4% of wives self-identified as another race/ethnicity (one husband and one wife did not report their race/ethnicity). Twenty-one percent of the couples had children. Approximately 27% of the sample (n = 61) was obese at baseline; an additional 24% was overweight at baseline.

Procedure. Couples were either mailed a packet of surveys to complete at home or were emailed a link to complete their surveys online. Surveys included an informed consent form approved by the Florida State University Institutional Review Board, measures of childhood family income, current income, height and weight (from which BMI was calculated), and other measures beyond the scope of the current analyses (see *SI Study 1* for a full list of measures). At their laboratory sessions, participants completed a variety of tasks beyond the scope of the current analyses and were compensated $100 for completing the surveys and session.

At ∼6-mo intervals subsequent to the initial assessment, couples were contacted by telephone and were mailed measures assessing height and weight (and, at the fourth and sixth follow-up assessments, an additional measure assessing childhood family income). Spouses completed six follow-up assessments, and thus the study spanned the first 3.5 y of marriage. After completing each follow-up assessment, couples were mailed a $30 check for participating.

Materials.

Childhood family income. To assess childhood family income at baseline, spouses provided a numerical estimate in response to the question: "How much money did your parents earn (combined income) while you were growing up (if you were raised by only one parent who did not receive child support, what was the income of that parent)?" Sixty-nine spouses failed to respond to this question, many of whom indicated that they did not know how much money their parents earned. Thus, in an effort to reduce missing data, we again assessed childhood family income at the fourth and sixth follow-up assessments (∼18 mo and 30 mo, respectively, into the marriages) using a different format. Specifically, spouses responded to the question: "What was your household income when you were growing up?" using an eight-point scale on which 1 = $15,000 or less, 2 = $15,001–$25,000, 3 = $25,001–$35,000, 4 = $35,001–$50,000, 5 = $50,001–$75,000, 6 = $75,001–$100,000, 7 = $100,001–$150,000, and 8 = $150,001 and above. Twenty-six spouses who had failed to provide a numerical estimate at baseline responded to this question (24 additional spouses provided data at the fourth assessment; two additional spouses provided data at the sixth assessment). Responses were converted by averaging the endpoints of the ranges provided. For example, spouses who indicated that their parents earned approximately $25,001–$35,000 yearly were coded $30,000. Responses provided at the first assessment were used when available, and responses provided at the fourth and sixth assessments were used when data were missing from the first assessment. Although this methodology may have introduced some degree of measurement error, such error did not pose a problem for detecting the hypothesized effect.

Body mass. At baseline and all subsequent follow-up assessments, we calculated indices of body mass by converting the self-reported height and weight into a standard index, the BMI (kg/m^2).

Obesity status. Obesity status was defined based on guidelines provided by the Centers for Disease Control (34). Participants with BMI scores above 30.0 were categorized as obese. Participants with BMI scores above 25.0 were categorized as overweight.

Current income. To ensure that any effects of childhood family income were independent of participants' current income, we assessed and controlled for current income in all analyses. At baseline and all subsequent follow-up assessments, participants provided a numerical estimate in response to the question: "How much money did you earn last year, before taxes?"

Study 2 Method.

Participants. We recruited 400 participants from Amazon's Mechanical Turk (211 women, 188 men, 1 other/unspecified; mean age = 35.66 y, SD_e = 11.34 y). In this sample, 77.8% self-identified as Caucasian, 8.0% self-identified as African American or Black, 7.2% self-identified as Asian, 5.3% self-identified as Hispanic, and 1.8% self-identified as other. Approximately 21.5% of the sample (n = 86) was obese; an additional 30% of the sample was overweight.

Design and procedure. After providing online informed consent (the consent procedure was approved by the Northwestern University Institutional Review Board), participants completed measures including items assessing their childhood environment and their life-history strategy (full measures are provided in *SI Study 2, Additional Measures*). Participants completed a four-item measure of childhood SES (e.g., "My family usually had enough money for things when I was growing up": 1 = strongly disagree, 7 = strongly agree; α = 0.86, mean = 3.69, SD = 1.49) (35) and a three-item measure of current SES (e.g., "I have enough money to buy the things I want": 1 = strongly disagree, 7 = strongly agree; α = 0.85, mean = 3.72, SD = 1.59) (34). Participants completed an eight-item measure of childhood unpredictability (e.g., "My family life was generally inconsistent and unpredictable from day-to-day": 1 = strongly disagree, 7 = strongly agree; α = 0.93, mean = 2.63, SD = 1.56) (36). To assess participants' life-history strategy, each participant completed the 20-item Mini-K scale which assesses a single factor that differentiates between fast (low-K) and slow (high-K) life-history strategies (e.g., "I avoid taking risks," "I would rather have one than several sexual relationships at a time": 1 = strongly disagree, 7 = strongly agree; α = 0.84, mean = 4.74, SD = 0.87) (37). The Mini-K shows good internal consistency and convergent validity with other measures aimed at assessing life-history strategy (38, 39).

Participants also reported their height in inches (mean = 67.58, SD = 3.98) and current weight in pounds (mean = 172.37, SD = 42.54), which we used to calculate their BMI (mean = 26.43, SD = 5.79) and obesity status. Participants also completed a three-item measure of dysregulated-eating behavior that assessed the extent to which they regulated their eating behavior based on their hunger levels ["I only eat when I'm hungry" (reverse-coded), "I rely on my levels of hunger to decide when and how much to eat" (reverse-coded), and "I eat even when I'm not hungry": 1 = strongly disagree, 7 = strongly agree; α = 0.79, mean = 3.57, SD = 1.41]. Higher values indicate a greater tendency to eat regardless of current hunger levels.

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