Low Childhood Socioeconomic Status Promotes Eating in the Absence of Energy Need

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Abstract

Life-history theory predicts that exposure to conditions typical of low socioeconomic status (SES) during childhood will calibrate development in ways that promote survival in harsh and unpredictable ecologies. Guided by this insight, the current research tested the hypothesis that low childhood SES will predict eating in the absence of energy need. Across three studies, we measured (Study 1) or manipulated (Studies 2 and 3) participants' energy need and gave them the opportunity to eat provided snacks. Participants also reported their SES during childhood and their current SES. Results revealed that people who grew up in high-SES environments regulated their food intake on the basis of their immediate energy need; they ate more when their need was high than when their need was low. This relationship was not observed among people who grew up in low-SES environments. These individuals consumed comparably high amounts of food when their current energy need was high and when it was low. Childhood SES may have a lasting impact on food regulation.

Obesity is a growing problem in the United States and around the world (Flegal, Carroll, Kit, & Ogden, 2012). An important factor that contributes to obesity risk is childhood socioeconomic status (SES). Several studies have found low childhood SES to be a major predictor of obesity and insulin resistance in adulthood (Poulton et al., 2002; Wells, Evans, Beavis, & Ong, 2010), even among individuals who are able to improve their conditions later in life (Lawlor, Ebrahim, & Smith, 2002; Power, Manor, & Matthews, 2003).

Despite growing evidence that low childhood SES may increase obesity risk, little is known about the mechanisms that drive this association (Laitinen, Power, & Jarvelin, 2001). The proposed explanations typically focus on the environmental conditions of poverty that promote weight gain in childhood, such as lack of access to healthy foods and safe places to play (Baltrus, Everson-Rose, Lynch, Raghunathan, & Kaplan, 2007; Laitinen et al., 2001). Although these factors undoubtedly contribute to the link between low childhood SES and obesity, we propose that exposure to harsh and unpredictable early-life conditions may also become biologically embedded in one's energy-regulation mechanisms in ways that promote survival in environments that are scarce in resources, but promote obesity in those with a rich food supply.

A person's childhood environment provides a blueprint for the types of environments likely to be encountered in adulthood. Accordingly, life-history theory predicts that organisms calibrate their development in ways that promote survival and reproduction in their expected adult environments (Belsky, Steinberg, &

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Draper, 1991; Ellis, Figueredo, Brumbach, & Schlomer, 2009; Kaplan & Gangestad, 2005). Researchers have therefore hypothesized that exposure to harsh and unpredictable early-life environments should promote the development of an adult phenotype that is well adapted to survive in such conditions (Gluckman et al., 2007; Hales & Barker, 1992; Kuzawa, McDade, Adair, & Lee, 2010). Consistent with this hypothesis, research has found that exposure to resource scarcity in utero and during early childhood encourages the development of a thrifty phenotype, characterized by a small body, slow metabolism, and reduced level of behavioral activity (see, e.g., Barker, 1997; Bateson et al., 2004; Bateson & Martin, 1999; Gluckman et al., 2007).

Here, we build on these insights, examining whether early-life environments may also have a lasting impact on the mechanisms that guide food intake. Mechanisms of energy regulation typically develop such that current energy need plays an important role in regulating food intake (Havel, 1999; Woods, Seeley, Porte, & Schwartz, 1998). Indeed, people eat more when hungry than when full. However, in low-SES environments—where there is diminished access to resources that have historically provided a buffer from food shortages (Gurven & Kaplan, 2007)—it makes adaptive sense to eat when food is available, even if current energy need is low. Exposure to the conditions typical of low SES during development may therefore undermine the role that bodily signals of hunger and satiety play in guiding food regulation, promoting eating in the absence of bodily need. Although eating in the absence of bodily need is associated with obesity in contemporary food-rich environments (Fisher & Birch, 2002; Herman & Polivy, 1984), it would help promote survival in environments that are resource scarce.

The Current Research

Here, we present the results of three studies testing the hypothesis that low childhood SES predicts eating in the absence of energy need. In each of our studies, we either measured (Study 1) or manipulated (Studies 2 and 3) participants' energy need and gave them the opportunity to eat provided snacks. We predicted that people who grew up in high-SES environments would regulate their food intake on the basis of their immediate physiological energy need, consuming more calories when need was high than when need was low. In contrast, we predicted that physiological energy need would have a negligible impact on food intake among people who grew up in low-SES environments. Specifically, we predicted that their food intake would be comparably high when bodily energy need was high and when it was low.

Study 1

In Study 1, we assessed individuals' current energy need by measuring the length of time since their last meal and their current level of hunger. We then provided participants with an opportunity to eat snack foods (cookies and pretzels) and measured how many calories they consumed. We predicted that participants who grew up in high-SES environments would eat more when their energy need was high than when it was low. However, we predicted that participants who grew up in low-SES environments would eat comparably high numbers of calories when their current energy need was high and when it was low.

Method

Participants. Thirty-one[1] female students at a North American university (mean age = 19.21 years, *SD* = 1.26, range = 18–22) participated in exchange for partial course credit. Because people from low-SES environments, compared with those from high-SES environments, are more likely to be obese (e.g., Gonzalez et al., 2012), and because obesity impairs energy regulation (see, e.g., Galic, Oakhill, & Steinberg, 2010), we screened potential participants in advance and included only those who were not obese (body mass index < 30); we also excluded participants who had food allergies or diabetes.

Procedure and materials. Participants came to the laboratory individually and were told that they would be participating in a consumer research study. First, they filled out a survey that contained two key items: the number of hours since they had last eaten and how hungry they felt (rated on a scale from 1, *very full*, to 7, *very hungry*). Responses to these two items were highly correlated (*r* = .52) and were therefore transformed into *z* scores and averaged to form a measure of current energy need.

Next, participants were told that they would be evaluating some food products as part of a consumer taste-test study. They were presented with a 3-oz bag of chocolate-chip cookies (Famous Amos) and a 0.9-oz bag of pretzels (Snyder's). Each snack was presented to participants in a white Styrofoam bowl. Participants were instructed to sample each item and to evaluate its flavor by answering the question: "How much did you like this product?" (7-point rating scale from 1, *dislike extremely*, to 7, *like extremely*). We included this item both to buttress the cover story and to provide a control for liking of the food in our data analysis, as liking is a strong predictor of food intake (e.g., Spiegel, Shrager, & Stellar, 1989; Yeomans, Gray, Mitchell, & True, 1997). After tasting and evaluating each product, participants were told that it would take a

few moments to set up the next part of the study and that they could eat as much of the remaining food as they would like while waiting and while completing the remainder of the study.

After a 2-min waiting period, participants were directed to complete a survey that asked questions about their age, height, weight, and childhood SES. We used an established measure of relative childhood SES (Griskevicius, Delton, Robertson, & Tybur, 2011; Hill, Rodeheffer, DelPriore, & Butterfield, 2013) as a proxy measure of exposure to harshness and unpredictability in childhood. This proxy was chosen because research indicates that individuals who grow up in lower-SES environments experience higher levels of morbidity and mortality and have less stability in their day-to-day life (e.g., more chaotic and unpredictable home environments; Evans, Gonnella, Marcynyszyn, Gentile, & Salpekar, 2005; Jensen, James, Boyce, & Hartnett, 1983; Miller, Chen, & Parker, 2011). Participants were therefore asked to think about their childhood before age 12 and rate their agreement or disagreement with the following statements on 7-point rating scales (from 1, *strongly disagree*, to 7, *strongly agree*): "My family had enough money for things growing up," "I grew up in a relatively wealthy neighborhood," and "I felt relatively wealthy compared to others my age." Responses to these three items were aggregated to form an index of childhood SES ($\alpha = .87$), with higher scores reflecting higher childhood SES. Although it is possible that such retrospective accounts are prone to error, past studies have documented a strong link between adults' retrospectively reported and actual SES in childhood (S. Cohen, Janicki-Deverts, Chen, & Matthews, 2010; Duncan, Ziol-Guest, & Kalil, 2010).

The dependent measure consisted of the total number of calories consumed by each participant. At the end of each testing session, the uneaten cookies and pretzels were weighed separately, and the amount of food consumed was calculated by subtracting the remaining amount of each product from its starting weight. We then used the information provided on each product's nutrition label to calculate the total number of calories consumed during the laboratory session.

Results

Table 1 presents descriptive statistics for this study. We used multiple regression to test our predictions. In each of our analyses, calorie consumption was regressed on childhood SES and current energy need (both centered) in the first step and on the interaction between these variables in the second step. Participants' body weight and ratings of how much they liked the food (both centered)[2] were also entered in the first step, to control for differences in energy need based on body weight and

differences in food intake based on participants' hedonic responses to the food items (Spiegel et al., 1989; Yeomans et al., 1997).

Total calories consumed. In our target analysis, we examined the impact of childhood SES and energy need on the total number of calories participants consumed during their laboratory session. Results revealed a significant interaction between the two predictors, $b = 32.09$, $SE = 11.48$, $t(24) = 2.80$, $p = .01$, semipartial $r^2 = .13$. We probed this interaction by conducting regions-of-significance tests in which we examined the impact of each predictor on food intake at 1 standard deviation above (*high*) and below (*low*) the mean of the other predictor. First, we probed this interaction by examining the impact of childhood SES on calorie consumption at different levels of energy need. When energy need was high, there were no differences between participants from high- and low-SES environments in the number of calories consumed, $b = 17.07$, $p = .27$. However, when energy need was low, there was a negative relationship between childhood SES and food intake, with individuals from low-SES childhood environments consuming a significantly greater number of calories than those from high-SES environments, $b = -47.10$, $SE = 16.51$, $t(24) = -2.85$, $p = .009$, semipartial $r^2 = .14$ (see Fig. 1).

We next examined the impact of energy need on calories consumed at different levels of childhood SES. For individuals reared in high-SES environments, calorie consumption was greater when self-reported energy need was high than when it was low, $b = 108.66$, $SE = 26.61$, $t(24) = 4.08$, $p < .001$, semipartial $r^2 = .28$ (see Fig. 1). We did not observe an effect of energy need on the number of calories consumed by participants reared in low-SES environments, however, $b = 9.87$, $p = .67$.

Cookie and pretzel intake. We next examined separately the impact of childhood SES and energy need on the number of grams of each snack type (calorically dense cookies vs. relatively low-calorie pretzels) consumed by

Table 1. Descriptive Statistics for Study 1

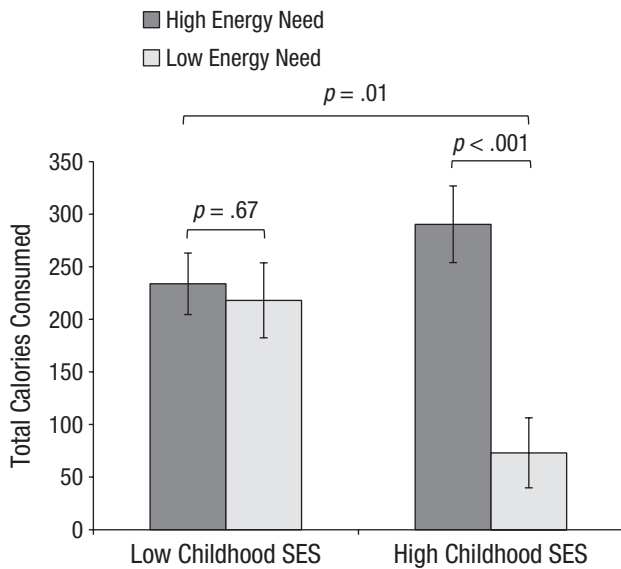| Variable	*M*	*SD*	Range
Age (years)	19.21	1.26	18–22
Weight (pounds)	140.61	22.98	102–211
Childhood socioeconomic status (1–7)	5.24	1.54	1.33–7.00
Hours since eating	3.97	3.84	0–10
Hunger (1–7)	4.23	1.28	2–6
Liking of the cookies (1–7)	5.58	1.29	2–7
Liking of the pretzels (1–7)	5.45	0.89	4–5
Total calories consumed	199.59	119.62	42.69–396.51

Fig. 1. Results from Study 1: total number of calories that participants consumed as a function of energy need and childhood socioeconomic status (SES). For both energy need and SES, *high* refers to values 1 standard deviation above the mean, and *low* refers to values 1 standard deviation below the mean. Error bars represent ±1 *SEM*.

participants. All covariates were the same as in the target analysis, except that instead of controlling for liking of both food items, we controlled only for the hedonic response to the food examined in each analysis (e.g., we controlled for liking of the cookies but not the pretzels in our analysis of cookie intake).

We first looked at the impact of childhood SES and energy need on the number of grams of cookies that participants consumed. Results revealed a significant interaction between childhood SES and energy need, $b = 5.99$, $SE = 1.75$, $t(25) = 3.43$, $p = .002$, semipartial $r^2 = .18$. We probed this interaction by examining the impact of childhood SES on cookie intake at different levels of energy need. When energy need was high (1 *SD* above the mean), there were no differences between participants from high- and low-SES environments (i.e., 1 *SD* above and 1 *SD* below the mean SES, respectively) in the number of grams of cookies consumed, $b = 3.18$, $p = .16$. However, when energy need was low (1 *SD* below the mean), there was a negative relationship between childhood SES and cookie intake, with individuals from low-SES childhood environments consuming a significantly greater number of grams of cookies than those from high-SES environments, $b = -8.80$, $SE = 2.58$, $t(25) = -3.41$, $p = .002$, semipartial $r^2 = .19$.

We next examined the impact of energy need on cookie intake at different levels of childhood SES. For individuals reared in high-SES environments, high self-reported energy need, compared with low energy need, predicted greater cookie consumption, $b = 17.50$,

$SE = 4.02$, $t(25) = 4.36$, $p < .001$, semipartial $r^2 = .31$. We did not observe an effect of energy need on the number of grams of cookies consumed by participants reared in low-SES environments, however, $b = -0.95$, $p = .80$.

Finally, we looked at the impact of childhood SES and energy need on the number of grams of pretzels that participants consumed. Our analysis revealed no interaction between childhood SES and energy need, $b = 1.31$, $p = .19$.

Study 2

Study 2 was designed to conceptually replicate and extend the findings of Study 1 using an experimental procedure to manipulate participants' energy need. After abstaining from eating or drinking for at least 5 hr prior to the study, participants were randomly assigned to drink either a beverage containing calories (Sprite) or a beverage devoid of calories (sparkling water). We then provided all participants with an opportunity to eat snacks and asked them to report on their childhood and current SES.

We predicted that participants who drank the water would consume comparably high numbers of calories across levels of childhood SES. However, we predicted that participants' childhood SES would moderate the impact of drinking Sprite on subsequent calorie consumption. Specifically, we predicted that among individuals from high-SES childhood environments, those who consumed the sugar-sweetened drink would eat less than those who consumed water. In contrast, we predicted that among individuals reared in low-SES environments, individuals who had received a glucose boost from the sugar-sweetened drink and those who had consumed water would eat comparably large amounts of food. Additionally, we predicted that the moderating effect of SES would be specific to childhood SES and would not emerge for current SES.

Method

Participants. Sixty female students at a North American university (mean age = 19.33 years, $SD = 1.55$, range = 18–24; 31 in the Sprite condition and 29 in the water condition) participated in exchange for partial course credit. We screened potential participants in advance and included only those who were not obese (body mass index < 30); we also excluded individuals who had food allergies or diabetes. Participants were instructed to avoid eating or drinking anything other than water for at least 5 hr prior to their study session. Five participants (4 in the Sprite condition, 1 in the water condition) were removed from analyses because they did not comply with the fasting procedure prior to their session or failed to finish their drink.

Procedure. Participants came to the laboratory individually and were told that they would be consuming a beverage as part of a consumer research study on taste preferences. Upon being seated, they were asked to indicate how many hours it had been since they had eaten and how hungry they felt (same 7-point rating scale as in Study 1). After responding to these initial questions, participants were given an unmarked, red plastic cup containing 12 oz of either a sucrose-sweetened soda (Sprite) or an unsweetened sparkling mineral water (La Croix). They were given 2 min to drink the beverage and were then asked follow-up questions about their enjoyment of it (to buttress the cover story). They then completed a 10-min filler task (listing consumer brand names) that allowed time for changes in blood glucose level to occur (Aarøe & Petersen, 2013; Wang & Dvorak, 2010).

Following the filler task, participants were informed that they would next be asked to evaluate a food item. They were presented with cookies (the contents of a 1-oz bag of mini Oreo cookies), which were served in a white bowl. After tasting and evaluating the cookies ("How much do you like this product?"; rating scale from 1, *dislike extremely*, to 7, *like extremely*), participants were told that that they could eat as much of the remaining food as they liked while waiting for the next survey to load on the computer and while they completed the survey questions. Participants were then asked to report their body weight, their childhood SES, and their current SES. Childhood SES was measured by the three-item measure of relative childhood SES used in Study 1, as well as by a more objective single-item measure: "Based on your best estimate, what was your family's socioeconomic status during your early childhood (age 12 and earlier)?" Current SES was measured with an analogous item, "Based on your best estimate, what is your family's socioeconomic status currently?" Participants responded to these additional SES items on a 7-point scale (1 = *very poor*, 7 = *very wealthy*). We included these two additional SES measures to test whether our pattern of results would be replicated using a more objective measure of childhood SES and to determine whether only childhood SES, and not adult SES, moderates the effect of energy need on calorie consumption, as our theory predicts. The study ended with a question probing participants' compliance with the fasting procedure. Participants read, "Please note that your response will not affect your ability to receive credit for participation in this study. How many hours has it really been since you last ate or drank anything other than water before today's study began?"

The dependent measure in Study 2 was the total number of calories consumed during the laboratory session, which we calculated by taking the difference between the starting weight of the cookies and the weight of the uneaten cookies and using the calorie information provided on the nutrition label.

Results

Table 2 presents descriptive statistics for this study. We used multiple regression to test our predictions. In our analysis, number of calories consumed was regressed on drink condition (dummy-coded) and SES (centered) in the first step and on the interaction between these variables in the second step. As in Study 1, participants' body weight and ratings of how much they liked the food (centered) were also entered in the first step to control for differences in consumption based on energy need and hedonic response. We also included the number of hours it had been since participants had last eaten as a covariate, as the length of the presession fasting period would also influence the degree to which the fixed number of calories administered (via the soft drink) affected participants' energy needs.

We first examined the effect of energy need and childhood SES on calorie consumption using the single-item measure of childhood SES. This analysis revealed a significant interaction between drink condition and childhood SES, $b = -22.99$, $SE = 11.28$, $t(48) = -2.04$, $p = .05$, semipartial $r^2 = .05$ (see Fig. 2). Simple-slopes tests revealed that for participants whose energy need was high (i.e., those in the water condition), childhood SES was not predictive of how much food was consumed, $b = 5.29$, $p = .52$. However, for participants whose energy need was low (i.e., those who received a glucose boost from a sugar-sweetened drink), there was a negative relationship between childhood SES and the amount of food consumed, $b = -17.70$, $SE = 7.80$, $t(48) = -2.27$, $p = .03$, semipartial $r^2 = .07$. Further, regions-of-significance tests revealed that participants who grew up in high-SES environments (i.e., SES 1 *SD* above the mean) consumed a significantly greater number of calories when their energy need was high (water condition) than when their energy need was low (Sprite condition), $b = -47.38$, $SE = 15.85$, $t(48) = -2.99$, $p = .004$, semipartial $r^2 = .11$ (see Fig. 2). We did not observe an effect of drink condition on participants who grew up in low-SES environments (i.e., SES 1 *SD* below the mean), however, $b = -1.73$, $p = .91$. These individuals consumed comparable amounts of food in the two conditions (see Fig. 2).

In a parallel analysis using the three-item measure of relative childhood SES ($\alpha = .79$), we again found that drink condition and childhood SES had a significant interactive effect on the number of calories consumed, $b = -23.23$, $SE = 10.90$, $t(48) = -2.13$, $p = .04$, partial $r^2 = .06$. Simple-slopes tests revealed that for participants whose energy need was high (i.e., those in the water condition), childhood SES was not predictive of how

Table 2. Descriptive Statistics for Study 2

Variable	Water condition		Sprite condition		Range
	M	*SD*	*M*	*SD*	
Weight (pounds)	136.61	20.40	135.00	20.51	90–190
Childhood socioeconomic status (three items; 1–7)	5.10	0.98	5.35	1.20	2.00–6.67
Childhood socioeconomic status (single item; 1–7)	4.54	0.96	4.26	1.02	2–7
Current socioeconomic status (single item; 1–7)	5.04	0.84	4.74	1.06	2–7
Hours since eating	9.39	1.07	9.74	0.71	6–10
Hunger (1–7)	2.64	0.68	2.81	0.68	1–4
Liking of the cookies (1–7)	6.00	0.94	6.00	0.83	3–7
Total calories consumed	71.30	51.11	53.14	44.02	9.29–143.93

much food was consumed, $b = 11.55$, $p = .18$. For participants whose energy need was low (i.e., those in the Sprite condition), however, there was a marginally significant negative relationship between childhood SES and the amount of food consumed, $b = -11.68$, $SE = 6.65$, $t(48) = -1.76$, $p = .086$, partial $r^2 = .04$; those from lower-SES childhood environments ate more than those from higher-SES childhood environments. Moreover, regions-of-significance tests revealed that participants who grew up in high-SES environments (i.e., 1 *SD* above the mean) consumed a significantly greater number of calories when their energy need was high (water condition) than when their energy need was low (Sprite

condition), $b = -48.32$, $SE = 16.80$, $t(48) = -2.88$, $p = .006$, partial $r^2 = .11$. In contrast, we did not observe an effect of drink condition among participants who grew up in low-SES environments (1 *SD* below the mean), $b = 2.28$, $p = .89$.

Finally, we examined the impact of current SES on the relationship between energy need and food intake using an analogous model in which current, rather than childhood, SES was the critical moderator. The analysis revealed a significant main effect of drink condition on the number of calories consumed; participants in the water condition consumed more calories ($M = 71.30$, $SD = 51.11$) than those in the Sprite condition ($M = 53.14$, $SD = 44.02$), $b = -25.27$, $SE = 11.53$, $t(49) = -2.19$, $p = .03$, semipartial $r^2 = .07$. However, there was no main effect of current SES on calories consumed, $b = -8.10$, $p = .18$, and current SES did not interact with drink condition to influence calorie consumption, $b = 5.25$, $p = .67$.
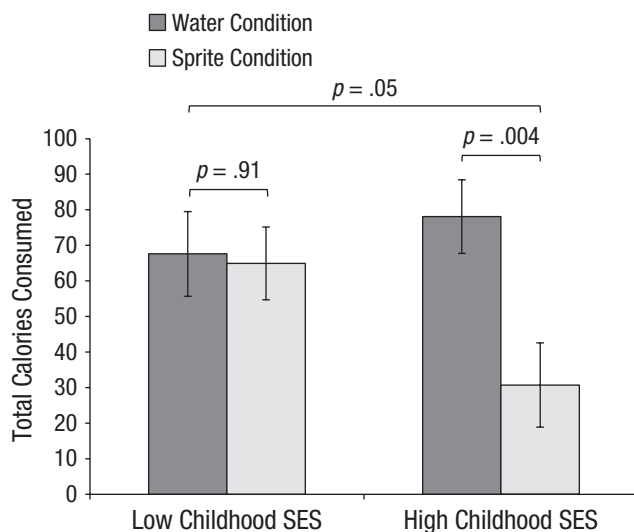
Study 3

Study 3 was designed to replicate and extend Study 2 in two ways. First, we included men in our sample to test whether our results would be replicated in a mixed-gender sample. Second, we measured participants' blood glucose following the drink manipulation to directly assess their energy needs. We predicted that childhood SES would moderate the impact of energy need on food intake among participants with low energy need, which would conceptually replicate the pattern observed in Studies 1 and 2. In addition, we predicted that blood glucose level after drinking would mediate the effects of drink condition on calorie consumption for participants from high-SES childhood environments but not for those from low-SES childhood environments.

Fig. 2. Results from Study 2: total number of calories that participants consumed as a function of drink condition and childhood socioeconomic status (SES) as indexed by the single-item measure. *High* and *low* SES refer to values 1 standard deviation above and below the mean, respectively. Error bars represent ±1 *SEM*.

Method

Participants. Eighty-three students at a North American university (21 men, 62 women; mean age = 20.18 years, SD = 1.98, range = 18–27) participated in exchange for partial course credit (41 in the Sprite condition, 42 in the water condition). We screened potential participants in advance and included only those who were not obese (body mass index < 30); we also excluded individuals who had food allergies or diabetes. Six participants (all women; all in the Sprite condition) were excluded from analyses because they did not comply with procedures or had abnormally high fasting blood glucose levels (> 99 mg/dl).

Procedure. The procedure and cover story were the same as in Study 2 except that we included a direct measure of blood glucose. Participants were told that we needed this measure to control for the effect of blood glucose level on their responses during the next phase of the study. Both at the beginning of the session[3] and 10 min after participants consumed their assigned beverage, blood glucose was measured using a OneTouch Ultrasoft lancet (Lifescan, Inc., Freemont, CA). A trained research associate read the results using a TrueResult glucometer (Nipro Diagnostics, Inc., Fort Lauderdale, FL) and recorded them using the participants' ID numbers. Participants were then presented with cookies using the same procedure and cover story as in Study 2. After tasting and evaluating the cookies, participants were allowed to eat as much of the remaining food as they liked while they finished the survey, which included the same single-item measures of childhood and current SES used in Study 2.

Results

Table 3 presents descriptive statistics for this study. We first tested whether the impact of childhood SES and energy need on calorie consumption differed between men and women, using multiple regression. In our first model, participant's sex, postmanipulation blood glucose level, and childhood SES were included as predictors in the first step, all two-way interactions were included in the second step, and the three-way interaction of these variables was entered in the third step. As in each of our prior models, participants' ratings of how much they liked the food was included as a control in the first step. Body weight and number of hours since having last eaten, which we controlled for in Study 2, were not included as covariates in Study 3 because the impact of these factors on participants' energy need was already accounted for by measuring energy need directly via postmanipulation blood glucose level. The results revealed a main effect of participant's sex on calorie consumption; men ate significantly more than women, $b = -6.84$, $SE = 2.54$, $t(71) = -2.29$, $p = .009$, semipartial $r^2 = .02$. However, all two-way (ps ≥ .40) and three way ($p = .33$) interactions with participant's sex were not significant. An analogous model in which drink condition replaced postmanipulation blood glucose level as a predictor also showed that participant's gender did not interact with any other predictors (all ps ≥ .45).

Next, to test our predictions about the interactive effects of drink condition, postmanipulation blood glucose level, and childhood SES on calorie consumption, we used Hayes's (2013) PROCESS SPSS macro (Model 14) to test for moderated mediation. Ten thousand bootstrap resamples were collected to generate a bias-corrected 95% confidence interval (CI) for each indirect effect (Preacher & Hayes, 2004). In our model, drink condition (Sprite vs. water) was the independent variable, postmanipulation blood glucose level was the mediator, and number of calories consumed was the dependent measure. Childhood SES was entered as a moderator in the path between the mediator and the dependent measure. Finally, hedonic rating of the cookies and participant's sex were entered as covariates to control for their impact on food intake.

As illustrated in Figure 3, there was a significant effect of drink condition on postmanipulation blood glucose level, $b = -33.06$, $SE = 3.90$, 95% CI = [−40.82, −25.30], $t(69) = -8.49$, $p < .001$; participants who consumed Sprite had

Table 3. Descriptive Statistics for Study 3

Variable	Water condition		Sprite condition		
	M	SD	M	SD	Range
Weight (pounds)	143.71	27.05	141.34	27.63	90–225
Childhood socioeconomic status (1–7)	4.33	1.20	4.77	1.00	2–7
Current socioeconomic status (1–7)	4.74	1.08	5.03	1.12	2–7
Blood glucose level (mg/dl)	82.38	9.39	115.46	22.68	2–119
Liking of the cookies (1–7)	6.14	0.90	6.21	0.85	3–7
Total calories consumed	91.43	47.49	92.73	53.33	9.14–146.29

significantly higher blood glucose levels (M = 115.46 mg/dl, SD = 22.68) than those who consumed water (M = 82.38 mg/dl, SD = 9.39). Additionally, the model revealed that the relationship between postmanipulation blood glucose level and calorie consumption was moderated by childhood SES, b = −0.14, SE = 0.06, 95% CI = [−0.26, −0.02], t(69) = −2.33, p = .02. For participants from high-SES environments (1 SD above the mean), calorie consumption was statistically mediated by postmanipulation level of blood glucose, b = 7.35, SE = 3.25, 95% CI = [1.29, 13.55]. No such relationship was found for participants from low-SES environments (1 SD below the mean), however, b = −3.11, SE = 3.51, 95% CI = [−9.55, 4.31].

We next used multiple regression to probe the interaction between postmanipulation blood glucose level and childhood SES found in our moderated-mediation model. Postmanipulation blood glucose level and childhood SES were included as the predictors in the first step, the interaction between these variables was entered in the second step, and hedonic rating of the cookies and gender were included as covariates. Results revealed that when postmanipulation blood glucose was low (1 SD below the mean), there were no childhood-SES-based differences in calorie consumption, b = 1.75, p = .20. Among participants with a high postmanipulation blood glucose level (1 SD above the mean), however, those who grew up in low-SES environments (1 SD below the mean) consumed a greater number of calories than did those who grew up in high-SES environments (1 SD above the mean), b = −4.90,

SE = 2.10, t(70) = −2.34, p = .02, semipartial r^2 = .05 (see Fig. 4).

Finally, we ran a moderated-mediation model similar to that described earlier, but with current SES, rather than childhood SES, entered as the critical moderator. The results revealed that current SES predicted the number of calories consumed; lower SES was associated with greater food intake, b = −2.44, SE = 0.99, 95% CI = [−4.42, −0.47], t(69) = −2.47, p = .02. However, current SES did not interact with postmanipulation blood glucose level to influence food intake, b = −0.06, SE = 0.04, 95% CI = [−0.13, 0.02], t(69) = −1.55, p = .13.

General Discussion

Research indicates that low childhood SES is a major predictor of obesity in adulthood (Poulton et al., 2002; Wells et al., 2010). We proposed that this association not only may be due to known sociological factors (Baltrus et al., 2007; Laitinen et al., 2001), but also may be due to developmental calibration to resource-scarce environments. Specifically, we proposed that growing up in low-SES environments can promote eating in the absence of hunger. Although such consumptive patterns predict overeating and obesity in food-rich environments (Fisher & Birch, 2002; Francis, Granger, & Susman, 2013), they promote survival in resource-scarce environments.

We found support for our hypothesis across three studies. Among individuals who grew up in high-SES
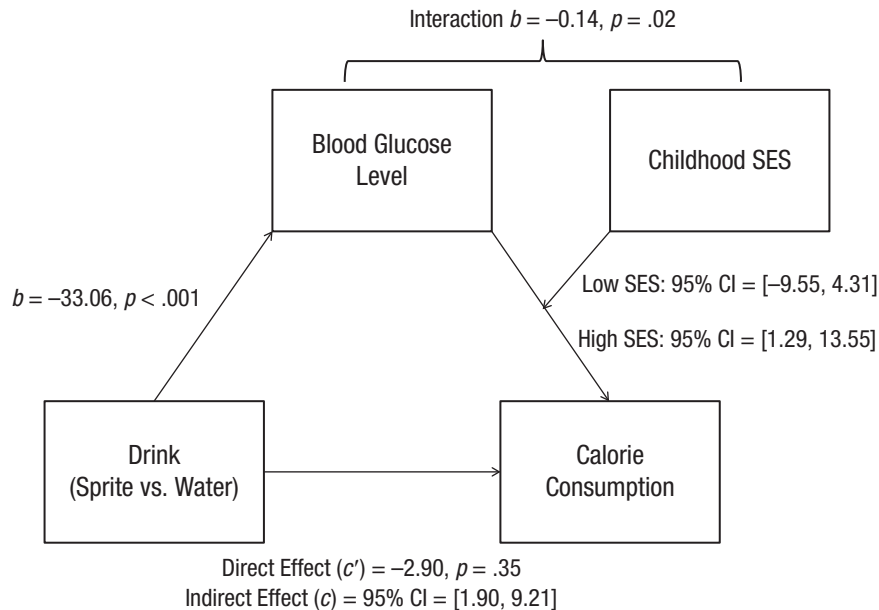

Interaction b = −0.14, p = .02

b = −33.06, p < .001

Low SES: 95% CI = [−9.55, 4.31]

High SES: 95% CI = [1.29, 13.55]

Direct Effect (c') = −2.90, p = .35
Indirect Effect (c) = 95% CI = [1.90, 9.21]

Fig. 3. Results from Study 3: effect of drink condition on participants' calorie consumption via postmanipulation blood glucose level, as moderated by childhood socioeconomic status (SES). *High* and *low* SES refer to values 1 standard deviation above and below the mean, respectively. CI = confidence interval.
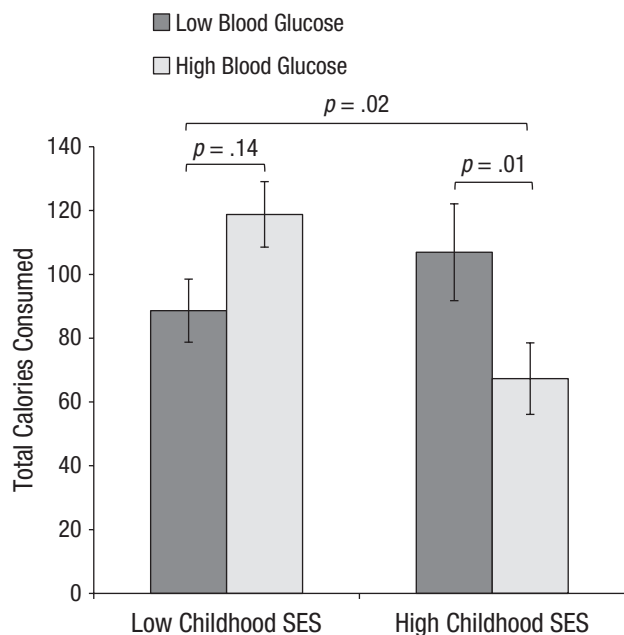
Fig. 4. Results from Study 3: total number of calories that participants consumed as a function of postmanipulation blood glucose level and childhood socioeconomic status (SES). For both postmanipulation blood glucose level and SES, *high* refers to values 1 standard deviation above the mean, and *low* refers to values 1 standard deviation below the mean. Error bars represent ±1 *SEM*.

environments, food intake varied according to immediate physiological energy need. These individuals consumed more calories when their current energy need was high than when it was low. For individuals who grew up in low-SES environments, however, the relationship between physiological need and food intake was decoupled. Their food intake appeared to be guided primarily by opportunity. They consumed comparably high numbers of calories when their current energy need was high and when it was low. This pattern was observed whether energy need was measured (Study 1) or manipulated (Studies 2 and 3) and whether childhood SES was measured using relative (Studies 1 and 2) or absolute (Studies 2 and 3) measures. Further, these results were found to be specific to childhood SES, as no such results emerged for current (adult) SES (Studies 2 and 3). Our results are consistent with the hypothesis that early developmental exposure to low SES may become biologically embedded in energy-regulation systems in ways that can encourage weight gain, even among individuals who are able to escape low-SES conditions in adulthood. Moreover, our results suggest that one's childhood environment—in addition to playing an important role in calibrating one's responses to external, environmental stressors in adulthood (e.g., Griskevicius et al., 2011; Hill et al., 2013)—may play an important role in how one responds to internal, physiological cues later in life.

It is important to note that the current results do not establish a causal relationship between low childhood SES and eating in the absence of energy need. Further, the results do not necessarily imply that lower SES is associated with decreased sensitivity to blood glucose fluctuations in adulthood. Indeed, any of the numerous factors that influence food intake, including ghrelin and leptin, sensory-specific satiety, and motivation to regulate caloric intake, could be dysregulated among individuals from low-SES environments. Research is needed to examine these possibilities. Despite these limitations, the current research contributes to a growing literature on life-history theory, which indicates that people's early life environments play an important role in calibrating their developmental pathways (Belsky et al., 1991; Ellis et al., 2009) and may have implications for their health and disease risk in adulthood (Barker, Eriksson, Forsen, & Osmond, 2002; Nettle, Frankenhuis, & Rickard, 2013). Moreover, our studies provide an important starting point for new lines of research into the development of obesity among individuals raised in low-SES environments. For example, these studies raise questions regarding the ontogeny and etiology of the observed patterns of energy intake: What are the critical dimensions of low-SES environments that promote eating in the absence of energy need? How and when do these conditions become biologically embedded in patterns of energy regulation? Do they emerge from fetal programming occurring in the uterine environment? Or do they emerge from learning or personal experiences with food insecurity?

The current research also raises questions about the biological mechanisms that promote eating in the absence of need. For example, exposure to low SES during childhood may affect insulin or leptin signaling or expression of neuropeptide Y in serum or relevant areas of the brain, such as the arcuate or paraventricular nuclei or the lateral hypothalamus (see, e.g., Danese et al., 2014). Finally, our studies raise important questions about precisely how individuals who grew up in low-SES environments regulate their energy intake, if not on the basis of need. Although we obtained evidence that low childhood SES promotes food intake in the absence of need, we did not systematically vary external factors that individuals raised in low-SES environments might use to guide their energy intake, such as food availability or palatability. Research exploring these questions has the potential to offer critical new insights into unhealthy weight gain and obesity among individuals exposed to low SES during childhood.

Author Contributions

S. E. Hill developed the hypothesis, designed the experiments, analyzed data for all three experiments, and wrote the

manuscript. M. L. Prokosch and D. J. DelPriore developed the research protocol, managed data collection, prepared data sets, analyzed data for all three experiments, created the figures, and helped with manuscript preparation. V. Griskevicius consulted on study design and assisted with manuscript preparation. A. Kramer conducted the experiments.

Declaration of Conflicting Interests

Supplemental Material

Additional supporting information can be found at http://pss .sagepub.com/content/by/supplemental-data

Open Practices

All data and materials have been made publicly available via Open Science Framework and can be accessed at https://osf .io/x8q4j/. The complete Open Practices Disclosure for this article can be found at http://pss.sagepub.com/content/by/ supplemental-data. This article has received badges for Open Data and Open Materials. More information about the Open Practices badges can be found at https://osf.io/tvyxz/wiki/ 1.%20View%20the%20Badges/ and http://pss.sagepub.com/ content/25/1/3.full.

Notes

1. We decided to include only females in Studies 1 and 2 in order to minimize between-participants variance in our dependent measure (Rolls, Fedoroff, & Guthrie, 1991). Sample sizes were chosen on the basis of J. Cohen's (1988) recommendation that 30 participants be tested within each condition to achieve 80% power (the minimum suggested power for an ordinary study) when the expected effect size is medium to large. We increased our target sample size to 40 to 45 participants per condition in Study 3 because it was the first study in the series to include men, and we anticipated greater variability in food intake. Post hoc power analyses (reported in the Supplemental Material available online) revealed that each of our experiments was sufficiently powered.
2. For each of the studies, we also ran our statistical models without the inclusion of the covariates. The results were consistent with those reported here and can be found in the Supplemental Material.
3. The premanipulation blood glucose measure was taken to screen out participants whose fasting blood glucose levels were consistent with having undiagnosed diabetes or misrepresenting the length of the fasting period (i.e., a fasting blood glucose level > 99 mg/dl; Mayo Clinic, 2014). One participant fell above this threshold.

References

Aarøe, L., & Petersen, M. B. (2013). Hunger games: Fluctuations in blood glucose levels influence support for social welfare. *Psychological Science*, *24*, 2550–2556.

Baltrus, P. T., Everson-Rose, S. A., Lynch, J. W., Raghunathan, T. E., & Kaplan, G. A. (2007). Socioeconomic position in childhood and adulthood and weight gain over 34 years: The Alameda County Study. *Annals of Epidemiology*, *17*, 608–614.

Barker, D. J. P. (1997). Maternal nutrition, fetal nutrition, and disease in later life. *Nutrition*, *13*, 807–813.

Barker, D. J. P., Eriksson, J. G., Forsen, T., & Osmond, C. (2002). Fetal origins of adult disease: Strength of effects and biological basis. *International Journal of Epidemiology*, *31*, 1235–1239.

Bateson, P., Barker, D., Clutton-Brock, T., Deb, D., D'Udine, B., Foley, R. A., . . . Sultan, S. E. (2004). Developmental plasticity and human health. *Nature*, *430*, 419–421.

Bateson, P. P. G., & Martin, P. (1999). *Design for a life: How behaviour develops*. London, England: J. Cape.

Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development*, *62*, 647–670.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

Cohen, S., Janicki-Deverts, D., Chen, E., & Matthews, K. A. (2010). Childhood socioeconomic status and adult health. *Annals of the New York Academy of Sciences*, *1186*, 37–55.

Danese, A., Dove, R., Belsky, D. W., Henchy, J., Williams, B., Ambler, A., & Arseneault, L. (2014). Leptin deficiency in maltreated children. *Translational Psychiatry*, *4*, Article e446. doi:10.1038/tp.2014.79

Duncan, G. J., Ziol-Guest, K. M., & Kalil, A. (2010). Early-childhood poverty and adult attainment, behavior, and health. *Child Development*, *81*, 306–325.

Ellis, B. J., Figueredo, A. J., Brumbach, B. H., & Schlomer, G. L. (2009). Fundamental dimensions of environmental risk. *Human Nature: An Interdisciplinary Biosocial Perspective*, *20*, 204–268.

Evans, G. W., Gonnella, C., Marcynyszyn, L. A., Gentile, L., & Salpekar, N. (2005). The role of chaos in poverty and children's socioemotional adjustment. *Psychological Science*, *16*, 560–565.

Fisher, J. O., & Birch, L. L. (2002). Eating in the absence of hunger and overweight in girls from 5 to 7 y of age. *American Journal of Clinical Nutrition*, *76*, 226–231.

Flegal, K. M., Carroll, M. D., Kit, B. K., & Ogden, C. L. (2012). Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999-2010. *Journal of the American Medical Association*, *307*, 491–497.

Francis, L. A., Granger, D. A., & Susman, E. J. (2013). Adrenocortical regulation, eating in the absence of hunger and BMI in young children. *Appetite*, *64*, 32–38.

Galic, S., Oakhill, J. S., & Steinberg, G. R. (2010). Adipose tissue as an endocrine organ. *Molecular and Cellular Endocrinology*, *316*, 129–139.

Gluckman, P. D., Lillycrop, K. A., Vickers, M. H., Pleasants, A. B., Phillips, E. S., Beedle, A. S., . . . Hanson, M. A. (2007). Metabolic plasticity during mammalian development is directionally dependent on early nutritional status. *Proceedings of the National Academy of Sciences, USA*, *104*, 12796–12800.

Gonzalez, A., Boyle, M. H., Georgiades, K., Duncan, L., Atkinson, L. R., & MacMillan, H. L. (2012). Childhood and family influences on body mass index in early adulthood: Findings from the Ontario Child Health Study. *BMC Public Health*, *12*, Article 755. doi:10.1186/1471-2458-12-755

Griskevicius, V., Delton, A. W., Robertson, T. E., & Tybur, J. M. (2011). Environmental contingency in life history strategies: The influence of mortality and socioeconomic status on reproductive timing. *Journal of Personality and Social Psychology*, *100*, 241–254.

Gurven, M., & Kaplan, H. (2007). Hunter-gatherer longevity: Cross-cultural perspectives. *Population and Development Review*, *33*, 321–365.

Hales, C. N., & Barker, D. J. P. (1992). Type 2 (non-insulin-dependent) diabetes mellitus: The thrifty phenotype hypothesis. *Diabetologia*, *35*, 595–601.

Havel, P. J. (1999). Mechanisms regulating leptin production: Implications for control of energy balance. *American Journal of Clinical Nutrition*, *70*, 305–306.

Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. New York, NY: Guilford Press.

Herman, C. P., & Polivy, J. (1984). A boundary model for the regulation of eating. *Research Publications – Association for Research in Nervous and Mental Disease*, *62*, 141–156.

Hill, S. E., Rodeheffer, C. D., DelPriore, D. J., & Butterfield, M. E. (2013). Ecological contingencies in women's calorie regulation psychology: A life history approach. *Journal of Experimental Social Psychology*, *49*, 888–897.

Jensen, E. W., James, S. A., Boyce, W. T., & Hartnett, S. A. (1983). The Family Routines Inventory: Development and validation. *Social Science & Medicine*, *17*, 201–211.

Kaplan, H. S., & Gangestad, S. W. (2005). Life history theory and evolutionary psychology. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 68–95). Hoboken, NJ: Wiley.

Kuzawa, C. W., McDade, T. W., Adair, L. S., & Lee, N. (2010). Rapid weight gain after birth predicts life history and reproductive strategy in Filipino males. *Proceedings of the National Academy of Sciences, USA*, *107*, 16800–16805.

Laitinen, J., Power, C., & Jarvelin, M. R. (2001). Family social class, maternal body mass index, childhood body mass index, and age at menarche as predictors of adult obesity. *American Journal of Clinical Nutrition*, *74*, 287–294.

Lawlor, D. A., Ebrahim, S., & Smith, G. D. (2002). Socioeconomic position in childhood and adulthood and insulin resistance: Cross sectional survey using data from British women's heart and health study. *British Medical Journal*, *325*, 805–807.

Mayo Clinic. (2014). *Diabetes: Tests and diagnosis*. Retrieved from http://www.mayoclinic.org/diseases-conditions/diabetes/basics/tests-diagnosis/con-20033091

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin*, *137*, 959–997.

Nettle, D., Frankenhuis, W. E., & Rickard, I. J. (2013). The evolution of predictive adaptive responses in human life history. *Proceedings of the Royal Society B: Biological Sciences*, *280*. doi:10.1098/rspb.2013.1343

Poulton, R., Caspi, A., Milne, B. J., Thomson, W. M., Taylor, A., Sears, M. R., & Moffitt, T. E. (2002). Association between children's experience of socioeconomic disadvantage and adult health: A life-course study. *The Lancet*, *360*, 1640–1645.

Power, C., Manor, O., & Matthews, S. (2003). Child to adult socioeconomic conditions and obesity in a national cohort. *International Journal of Obesity*, *27*, 1081–1086.

Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments, & Computers*, *36*, 717–731.

Rolls, B. J., Fedoroff, I. C., & Guthrie, J. F. (1991). Gender differences in eating behavior and body weight regulation. *Health Psychology*, *10*, 133–142.

Spiegel, T. A., Shrager, E. E., & Stellar, E. (1989). Responses of lean and obese subjects to preloads, deprivation, and palatability. *Appetite*, *13*, 45–69.

Wang, X. T., & Dvorak, R. D. (2010). Sweet future: Fluctuating blood glucose levels affect future discounting. *Psychological Science*, *21*, 183–188.

Wells, N. M., Evans, G. W., Beavis, A., & Ong, A. D. (2010). Early childhood poverty, cumulative risk exposure, and body mass index trajectories through young adulthood. *American Journal of Public Health*, *100*, 2507–2512. doi:10.2105/Ajph.2009.184291

Woods, S. C., Seeley, R. J., Porte, D., & Schwartz, M. W. (1998). Signals that regulate food intake and energy homeostasis. *Science*, *280*, 1378–1383.

Yeomans, M. R., Gray, R. W., Mitchell, C. J., & True, S. (1997). Independent effects of palatability and within-meal pauses on intake and appetite ratings in human volunteers. *Appetite*, *29*, 61–76.