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

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Abstract

Life history theory predicts that developmental exposure to conditions typical of low socioeconomic status (SES) should calibrate development in ways that promote survivability in harsh and unpredictable ecologies. Guided by these insights, the current research tested the hypothesis that low childhood SES would 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 childhood and adult SES. Results revealed that people with higher childhood SES regulated food intake based on immediate energy need; they ate more when need was high than when need was low. This relationship was not observed among those with lower childhood SES. These individuals consumed comparably high amounts of food whether current energy need was high or low. Childhood SES may have a lasting impact on food regulation.

Keywords: life history theory, childhood socioeconomic status, energy regulation, evolutionary-developmental psychology, thrifty phenotype, eating behavior

Word Count (1988)

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

Obesity is a growing problem in the U.S. and around the world (Flegal, 2005; Organization, 2014). An important factor that contributes to obesity risk is childhood socioeconomic status (SES). Several studies find low childhood SES to be a major predictor of obesity and insulin resistance in adulthood (Gonzalez et al., 2012; Kestila, Rahkonen, Martelin, Lahti-Koski, & Koskinen, 2009; Poulton et al., 2002; Tamayo, Herder, & Rathmann, 2010; Wells, Evans, Beavis, & Ong, 2010), even among those 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, less is known about the mechanisms that drive this association (Laitinen, Power, & Jarvelin, 2001). The explanations that have been proposed 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, Power, & Jarvelin, 2001). Although these factors undoubtedly contribute to the low childhood SES / obesity link, 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 survivability in environments that are resource scarce, but promote obesity in those that are food rich.

Life history theory predicts that conditions in early-life serve as a blueprint for the environments one is likely to encounter in adulthood (Belsky, Steinberg, & Draper, 1991; Ellis, Figueredo, Brumbach, & Schlomer, 2009; Kaplan & Gangestad, 2005). Researchers have therefore hypothesized that developmental 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; West-Eberhard, 2003). Consistent with this hypothesis, research finds that exposure to resource scarcity in utero and early childhood encourages the development of a thrifty phenotype, characterized by a smaller body size, slower metabolism, and a reduced level of behavioral activity [see e.g. (Barker, 1997; Bateson & Martin, 1999; Bateson et al., 2004; Gluckman & Hanson, 2004; Gluckman et al., 2009)].

Here, we build on these insights, examining whether one's early-life environment may also have a lasting impact on the mechanisms that guide food intake. Mechanisms of homeostatic 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. Developmental exposure to the conditions typical of low SES may therefore undermine the role that bodily signals of hunger and satiety play in guiding food regulation, promoting the 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 survivability in those that are resource-scarce.

The Current Research

Here, we present the results of three studies that test the hypothesis that low childhood SES will predict 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 higher SES

environments would regulate food intake based on their immediate physiological energy need. Specifically, we predicted that they would consume more calories when need was high than when need was low. For those growing up in lower SES environments, however, we predicted that physiological energy need would have a considerably lower impact on food intake. In these individuals, we predicted that food intake would be comparably high, regardless of bodily energy need.

Study 1

Study 1 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 higher SES environments would eat more when energy need was high than when need was low. For those growing up in lower SES environments, however, we predicted that participants would eat a comparably high number of calories regardless of their current energy need.

Methods and Materials

Participants. Thirty-one[1] female students at a North American university (M_{age} = 19.21 years, *SD* = 1.26, range 18-22) participated in exchange for partial course credit. Because those from low SES environments are more likely to be obese (e.g., Gonzalez et al., 2012) and obesity impairs energy regulation (see e.g., Galic, Oakhill, & Steinberg, 2010), participants were screened in advance to be non-obese (BMI < 30) and to be devoid of food allergies and 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: (1) number of hours since they had last eaten and (2) how hungry they

felt (rated on a 7-point scale; 1: *very full*, 7: *very hungry*). 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 .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?"* We included this item both to buttress the cover story and so that we could control for it 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 two-minute waiting period, participants were directed to complete a survey that asked questions about their age, height, weight, and childhood SES. Childhood SES was measured using an established measure of relative childhood socioeconomic status (Griskevicius, Delton, Robertson, & Tybur, 2011; Hill, Rodeheffer, DelPriore, & Butterfield, 2013), which we used as a proxy measure of exposure to harshness and unpredictability in childhood (Chen & Miller, 2012; Griskevicius et al., 2011, 2013; White et al., 2013). This proxy was chosen because research indicates that individuals growing up in lower-SES environments experience higher levels of morbidity-mortality, have greater exposure to premature disability and death, and have less stability in their day-to-day life and have more chaotic and unpredictable home

environments (Adler et al., 1994; Miller, Chen, & Parker, 201; Evans, 2004; Evans, Gonnella, Marcynyszyn, Gentile, & Salpekar, 2005; Jensen, James, Boyce, & Hartnett, 1983; Matheny, Wachs, Ludwig, & Phillips, 1995). To assess childhood SES, participants were therefore asked to think about their childhood before age 12 and rate their agreement | disagreement with the following statements on seven-point rating scales: 1) My family had enough money for things growing up, 2) I grew up in a relatively wealthy neighborhood, and 3) I felt relatively wealthy compared to others my age. The 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 of childhood SES are prone to error, past studies have documented a strong link between adults' retrospectively reported childhood SES and their actual SES in childhood (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. To determine this, 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 respective starting weight. We then used the nutrition information provided on each product's nutrition label to calculate the total number of calories consumed during the laboratory session.

Results

See Table 1 for descriptive statistics. We used multiple regression to test our predictions. In each of our analyses, food intake was regressed on childhood SES and current energy need (both centered) in the first step and the interaction between these variables in the second step. Participants' body weight and ratings of food liking (both centered)^2 were also entered into 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 (Naish & Harris, 2012; Spiegel, Shrager, & Stellar, 1989; Yeomans et al.,1997).

Total Calories Consumed. We first 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 our predictors on this outcome, $b = 32.09$ ($SE = 11.48$), $t(24) = 2.80$, $p = .01$, semipartial $r^2 = .13$. 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 (1 *SD* above the mean), there were no differences between those from high- and low- SES environments in the number of calories consumed, $b = 17.07$, $p = .27$. However, when energy need was low (1 *SD* below the mean), 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 higher SES environments, $b = -47.10$ ($SE = 16.51$), $t(24) = -2.85$, $p = .009$, semipartial $r^2 = .14$ (see Figure 1).

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

Table 1.
Descriptive Statistics for Study 1

	M	*SD*	*Min*	*Max*
Age	19.21	1.26	18	22
Weight	140.61	22.98	102	211
Childhood SES	5.24	1.54	1.33	7
Hours Since Eaten	3.97	3.84	0	10
Hunger	4.23	1.28	2	6
Cookie Liking	5.58	1.29	2	7
Pretzel Liking	5.45	0.89	4	7
Total Calories Consumed	199.59	119.62	42.69	396.51

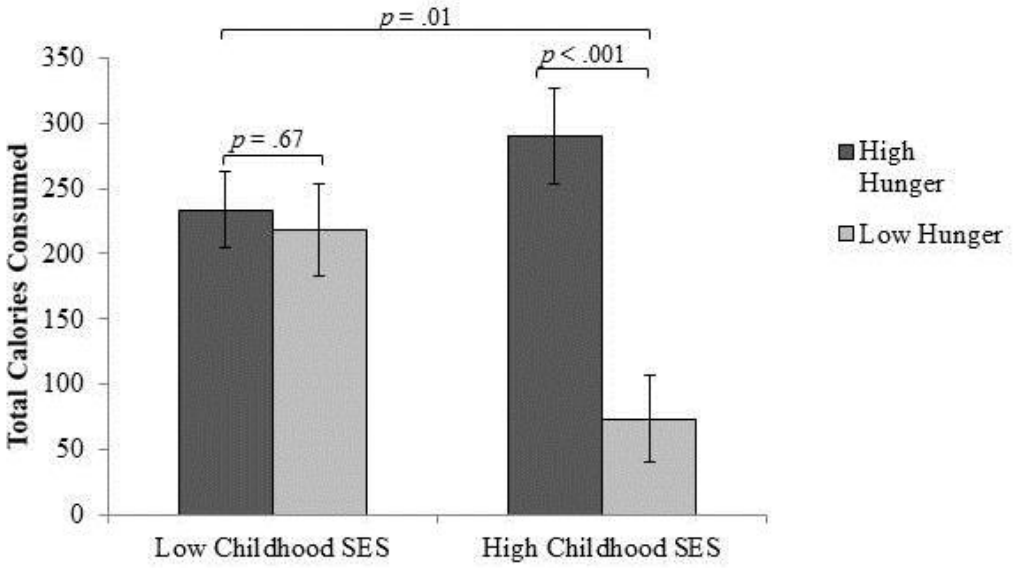

Fig. 1. Total calories consumed by participants as a function of hunger level and childhood socioeconomic status (Study 1). Plotted means represent one standard deviation above and below the mean of childhood socioeconomic status and mean hunger level. Error bars reflect the standard error of the mean.

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 versus relatively low calorie pretzels) consumed by participants. All covariates were the same as those described in the target analysis, with the exception of the "liking" variable. Instead of controlling for both cookie- and pretzel-liking (as we did in the target analysis), we only controlled for the hedonic response to the food we were examining (e.g., we controlled for cookie- but not pretzel- liking 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 on cookie intake, $b = 5.99$ ($SE = 1.75$), $t(25) = 3.43$, $p = .002$, semipartial $r^2 = .18$. First, 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 those from high- and low- SES environments 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 higher 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 higher SES environments (1 *SD* above the mean), higher self-reported energy need predicted greater cookie consumption compared to when energy need was low, $b = 17.50$ ($SE = 4.02$), $t(25) = 4.36$, $p < .001$, 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 lower SES environments, however (b = -.95, p = .80).

Lastly, we looked at the impact of childhood SES and energy need on the number of grams of pretzels that participants consumed. Results revealed no interaction between childhood SES and energy need on pretzel intake, b = 1.31, p = .19.

Study 2

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

We predicted that participants in the water condition would eat a comparably high number of calories across levels of childhood SES. However, we predicted that participants' childhood SES would moderate the impact of the sugar-sweetened drink on subsequent calorie consumption. Specifically, we predicted that individuals with higher childhood SES would eat less after consuming the sugar sweetened drink compared to those who consumed water. For individuals reared in lower SES environments, however, we predicted that they would eat comparably high amounts of food regardless of whether or not they had received a prior blood glucose boost from the sugar-sweetened drink. Additionally, we predicted that these effects would be specific to childhood SES and would not emerge in response to lower adult SES.

Methods and Materials

Participants. Fifty-five female students at a North American university (M_{age} = 19.33 years, *SD* = 1.55, range 18-25; 27 in the Sprite Condition) participated in exchange for partial course credit. Participants were non-obese (BMI < 30) and screened in advance for food allergies and diabetes. Participants were instructed to avoid eating or drinking anything other than water for at least 5 hours prior to their study session. Five participants (4 from the Sprite Condition) were removed due to their failure to comply with the fasting procedure (i.e., they ate or drank something other than water less than 5 hours prior to the testing session) or failing to finish their drink.

Procedure. Participants came into 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, a research assistant gave each participant an unmarked, red plastic cup containing 12 ounces of either a sucrose-sweetened soda (Sprite) or an unsweetened sparkling mineral water (La Croix). Participants were given two minutes to drink the beverage and were then asked follow-up questions about their enjoyment of the beverage to buttress the cover story. Participants then completed a 10-minute filler task (listing consumer brand names) to allow time for changes in blood glucose to occur (Aaroe & Petersen, 2013; Wang & Dvorak, 2010).

Following the 10-minute 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 one ounce bag of mini Oreo cookies), which were served in a white bowl. After tasting and evaluating the cookies, participants were told that that they could eat as much of the remaining food as they liked while they finished the survey. The dependent measure consisted of the total number of calories consumed, as measured by the difference between the starting weight and the weight of

the uneaten cookies and calculating calories using the information provided on the nutrition label.

At end of the session participants were asked to provide information about whether they had complied with the fasting procedure, the number of hours it had been since they had last eaten, their weight, and their childhood and current socioeconomic status. In addition to the three-item measure of relative childhood SES used in Study 1, participants also responded to a more objective single-item measure of childhood SES ["Based on your best estimate, what was your family's socioeconomic status during your early childhood (age 12 and earlier)?"], as well as an analogous measure of their adult SES ["Based on your best estimate, what is you / your family's socioeconomic status currently?"]. Participants responded to each item on a seven-point scale (endpoints: 1 = *very poor*, 7 = *very wealthy*). We included these two additional SES measures because we wanted to test whether we could replicate our pattern of results using a more objective measure of childhood SES (as well as the subjective measure used in Study 1). We also sought to examine whether the predicted shifts are specific to childhood SES, as our theory would predict, or whether they also emerge in response to low adult SES.

Results

See Table 2 for descriptive statistics. 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 the interaction between these variables in the second step. As in Study 1, participants' body weight and ratings of food liking (centered) were also entered into the first step to control for differences in consumption based on energy need and hedonic response to the cookies. We also included the number of hours it had been since participants had last eaten as a covariate, as the length of the pre-session fasting period would

also impact the degree to which the fixed number of calories administered (via the soft drink) would impact one's current energy needs.

Table 2.
Descriptive Statistics for Study 2.

	Water		Sprite			
	M	*SD*	*M*	*SD*	*Min*	*Max*
Weight	136.61	20.40	135.00	20.51	90	190
Childhood SES (three item)	5.10	0.98	5.35	1.20	2	6.67
Childhood SES (single item)	4.54	0.96	4.26	1.02	2	7
Adult SES (single item)	5.04	0.84	4.74	1.06	2	7
Hours Since Eaten	9.39	1.07	9.74	0.71	6	10
Hunger	2.64	0.68	2.81	0.68	1	4
Cookie Liking	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

We first examined the impact of energy need and childhood SES on food intake using the single-item childhood SES measure. The results of this analysis revealed a significant interaction between drink condition and childhood SES on the number of calories consumed, b = -22.99 (SE = 11.28), $t(48)$ = -2.04, p = .05, semipartial r^2 =.05 (see Figure 2). Simple slopes tests revealed that for participants whose energy needs were high (i.e., those in the water condition), childhood SES was not predictive of how much food was consumed (b = 5.29, p = .52). When energy need was low, however (i.e., for 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 higher childhood SES environments (1 SD above the mean) consumed a significantly greater number of calories when energy need was high (water condition) compared to when energy need was low (sweetened drink condition), b = -47.38 (SE = 15.85), $t(48)$ = -2.99, p = .004, semipartial r^2 =.11 (see Figure 2). We did not observe an effect of drink condition on those who grew up in lower childhood SES

environments, however (b = -1.73, p = .91). These individuals consumed comparable amounts of food across testing conditions.

We also tested whether we could replicate these results using the three-item measure of relative childhood SES as the moderator. As in the analysis with the one-item childhood SES measure, the results using the three-item childhood SES measure [α = .79] revealed a significant interaction between drink condition and childhood SES 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 needs were high (i.e., those in the water condition), childhood SES was not predictive of how much food was consumed (b = 11.55, p = .18). When energy need was low (i.e., 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, with those from lower SES childhood environments eating more than those with higher childhood SES. Moreover, regions of significance tests revealed that participants who grew up in higher SES environments (1 SD above the mean) consumed a significantly greater number of calories when energy need was high (water condition) compared to when energy need was low (Sprite condition), b = -48.32 (SE = 16.80), $t(48)$ = -2.88, p = .006, partial r^2 =.11. We did not observe an effect of drink condition among those who grew up in lower SES environments, however (b = 2.28, p = .89).

Lastly, we examined the impact of adult SES on the relationship between energy need and food intake using a model analogous to the one described above but with adult – rather than childhood – SES as the critical moderator. The results revealed a significant main effect of drink condition on the number of calories consumed, indicating that those in the water condition consumed more calories than those in the Sprite condition [M_{Sprite} = 53.14, SD = 44.02; M_{Water} =

71.30, *SD* = 51.11; *b* = -25.41 (*SE* = 11.53), *t*(49) = -2.19, *p* = .03, semipartial r^2 =.07]. However, there was no main effect of adult SES on food intake (*b* = -11.25, *p* = .25), nor did adult SES interact with drink condition to influence calorie consumption (*b* = 5.25, *p* = .67).

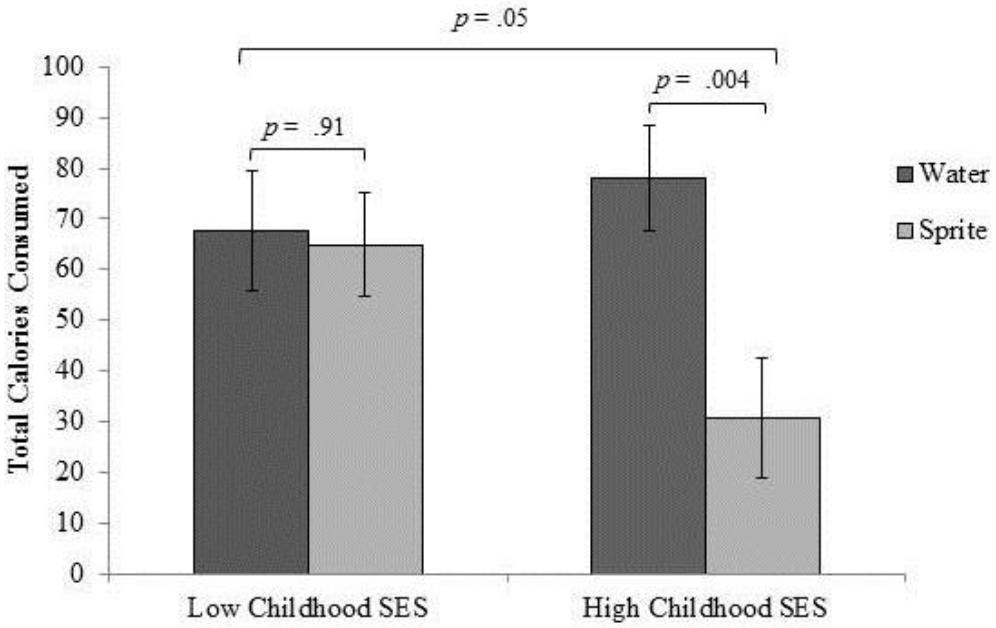

Fig.2. Total calories consumed as a function of drink condition and childhood SES (Study 2). Plotted means represent one standard deviation above and below the mean of childhood SES. Error bars reflect the standard error of the mean.

Study 3

Study 3 was designed to replicate and extend the results of Study 2 in two ways. First, we included men in our sample to ensure that our results replicate 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, conceptually replicating the pattern observed in Studies 1 and 2. In addition, we predicted that participants' post-drink blood glucose would mediate the effects of drink condition on calorie consumption for those from higher childhood SES environments but not for those from lower childhood SES environments.

Methods and Materials

Participants. Seventy-seven students at a North American university (21 men, 56 women; 35 in the Sprite condition) (M_{age} = 20.18 years, *SD* = 1.98, range 18-27) participated in exchange for partial course credit. Participants were non-obese (BMI < 30) and screened in advance for food allergies and diabetes. Six participants (6 in the Sprite Condition) were excluded due to their failure to comply with the fasting or procedure or failing to finish their drink.

Procedure. The procedure and cover story were the same as in Study 2. The primary difference was the inclusion of a direct measure of blood glucose, which participants were told was being taken so that we could control for its impact on the responses they provide during the next phase of the study. The blood glucose measure was taken 10 minutes after participants consumed their assigned beverage using a OneTouch Ultrasoft lancet. A trained research associate read the results using a TrueResult glucometer and recorded them using the participants' ID number. 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 childhood and adult SES measures used in Study 2.

Results. See Table 3 for descriptive statistics. We first tested whether the impact of childhood SES and energy need on food intake differed between men and women. To test this possibility, we used multiple regression. In our first model, participant sex, blood glucose, and childhood SES included as predictors of food intake in the first step, all two-way interactions in the second step, and the three-way interaction between these variables in the third step. As in each of our prior models, cookie liking was included in the first step to control for its impact on

food intake. Participant 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' post-drink energy needs is already accounted for by measuring energy need directly via blood glucose. Although the results revealed a main effect of participant sex on food intake (men ate significantly more than women: $b = 5.75$ ($SE = 2.36$), $t(72) = 2.44$, $p = .02$, semipartial $r^2 = .06$), all two-way ($ps \geq .57$) and three way ($p = .19$) interactions with participant sex were not significant. Running the analogous model with drink condition (rather than blood glucose) as the predictor also failed to find an interaction between participant gender and any of our other predictors (all $ps \geq .45$).

Next, to test our predictions about the relationship between drink condition, blood glucose, and childhood SES on food intake, we used Hayes' (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 for each indirect effect (Preacher & Hayes, 2004). In our model, drink condition (Sprite vs. water) was the independent variable, blood glucose was the mediator, and food intake was the dependent measure. Childhood SES was also entered as a moderator in the path between the mediator and the dependent measure. Lastly, food liking and participant sex were entered as covariates to control for their impact on food intake.

Table 3.
Descriptive Statistics for Study 3.

	Water		Sprite			
	M	*SD*	*M*	*SD*	*Min*	*Max*
Weight	143.71	27.05	141.34	27.63	90	225
Childhood SES	4.33	1.20	4.77	1.00	2	7
Adult SES	4.74	1.08	5.03	1.12	2	7
Blood Glucose	82.38	9.39	115.46	22.68	2	119
Cookie Liking	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

As seen in Figure 3, the results demonstrated a significant relationship between drink condition and blood glucose levels, b = -33.06 (SE = 3.90), t = -8.49, p < .001, 95% CI [-40.82, -25.30], showing that those who consumed the Sprite had significantly higher blood glucose than those who consumed water (M_{Sprite} = 115.46, SD = 22.68; M_{Water} = 82.38, SD = 9.39). Additionally, the model revealed that the relationship between blood glucose and food intake was moderated by childhood SES, b = -.14 (SE = .06), t = -2.33, p = .02, 95% CI [-.26, -.02]. For participants from higher SES environments, food intake was statistically mediated by post-drink levels of blood glucose, b = 7.35 (SE = 3.25), 95% CI [1.29, 13.55]. No such relationship was found for those from lower SES environments, however, b = -3.11(SE = 3.51), 95% CI [-9.55, 4.31].

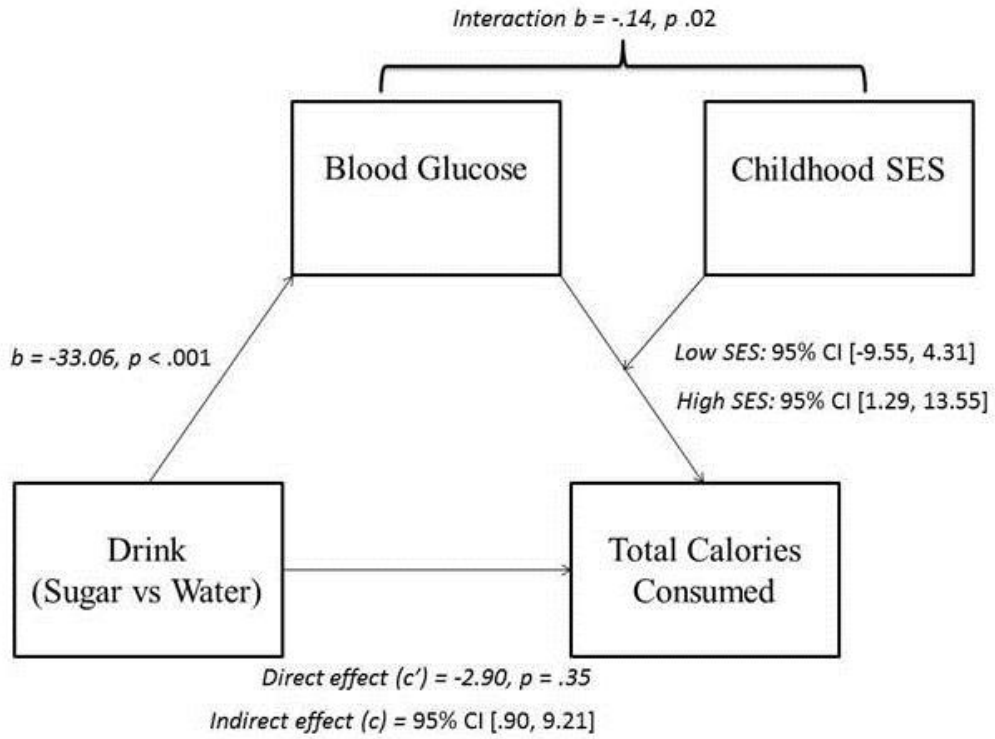

Fig. 3. Moderated mediation model looking at the indirect effect of drink condition on participants' calorie consumption via changes in blood glucose (Study 3). Results demonstrate that blood glucose statistically mediates food intake in response to drink condition for participants with higher childhood SES (95% CI [1.29, 13.55]) but not for participants with lower childhood SES (95% CI [-9.55, 4.31]).

We next used multiple regression to probe the interaction between blood glucose and childhood SES found in our moderated mediation model. As in previous models, blood glucose and childhood SES were included as the predictors in the first step, the interaction between these variables was entered into the second step, and cookie liking and gender were included as covariates. Results revealed that when measured 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 for whom blood glucose was high (1 *SD* above the mean), however, those who grew up in lower childhood SES environments consumed a greater number of calories than those from higher SES environments, $b = -4.90$ ($SE = 2.10$), $t(70) = -2.34$, $p = .02$, semipartial $r^2 = .05$.

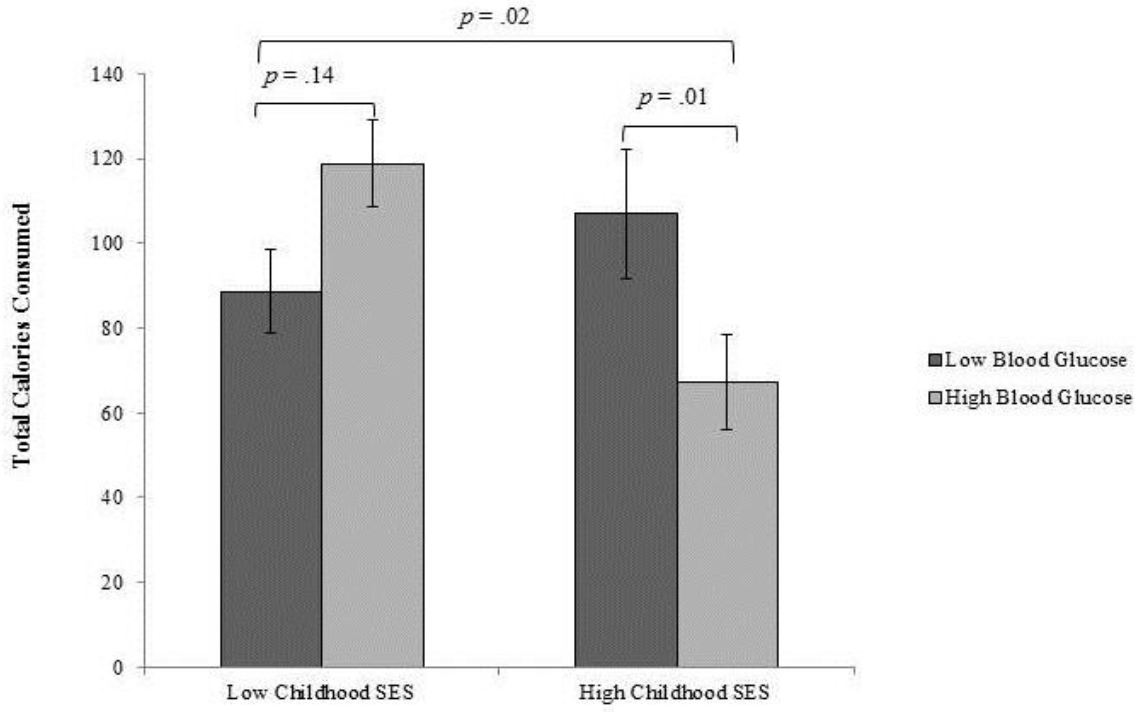

Fig. 4. Total calories that participants consumed as a function of blood glucose and CSES (Study 3). Plotted means represent one standard deviation above and below the mean of blood glucose and childhood SES. Error bars reflect the standard error of the mean.

Lastly, we ran a moderated mediation model similar in kind to that described above, but with adult SES (rather than childhood SES) entered as the critical moderator. The results of this model revealed that adult SES was predictive of the number of calories consumed, with lower SES predicting greater food intake, b = -2.44 (SE = .99), $t(69)$ = -2.47, p = .02, 95% CI [-4.42, -.47]; however, adult SES did not interact with participants' blood glucose to influence food intake, b = -.06 (SE = .04), $t(69)$ = -1.55, p = .13, 95% CI [-.13, .02].

General Discussion

Research indicates that low childhood SES is a major predictor of obesity in adulthood (Gonzalez et al., 2012; Poulton et al., 2002; Wells et al., 2010). We proposed that – in addition to the sociological factors known to contribute to this association (Baltrus et al., 2007; Laitinen et al., 2001) – low childhood SES may also calibrate the mechanisms that guide food intake such that they motivate eating in the absence of hunger. Although such consumptive patterns promote survivability in resource scarce environments, they predict overeating and obesity in environments that are food-rich (Fisher & Birch, 2002; Francis, Granger, & Susman, 2013).

We found support for our hypothesis across three studies. Among individuals who grew up in higher SES environments, food intake varied according to immediate physiological energy need. These individuals consumed more calories when current energy need was high than low. For those growing up in lower childhood SES environments, however, the relationship between physiological need and food intake was decoupled. For these individuals, food intake appeared to be guided primarily by opportunity. They ate comparably high amounts of food regardless of whether current energy need was high or 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, with no such results emerging for adult SES (Studies 2 and 3). These results are consistent with the hypothesis that early developmental exposure to low socioeconomic status may become biologically-embedded in one's energy regulation systems in ways that can encourage weight gain, even among those able to escape these conditions in adulthood. Moreover, these results suggest that one's childhood environment – in addition to playing an important role in calibrating responses to external, environmental stressors in adulthood (e.g., Griskevicius et al., 2011; Hill et al., 2013) – may also 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 could be dysregulated among those from lower SES environments, including ghrelin/leptin, sensory-specific satiety, or motivation to regulate caloric intake. 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, 2004; West-Eberhard, 2003) and may have implications for their health and disease risk in adulthood (Barker, Eriksson, Forsen, & Osmond, 2002; Nettle, Frankenhuis, & Rickard, 2013). Moreover, it provides an important starting point for new lines of research into the development of obesity among those from low childhood SES environments. For example, the current research raises questions regarding the ontogeny and etiology of the observed patterns of energy intake such as what are the critical dimensions of low SES environments that promote eating in the absence of energy

need? Additionally, 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, developmental exposure to low childhood SES may impact insulin or leptin signaling or NPY (neuropeptide Y) expression 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)]. Lastly, the current research raises important questions about precisely how individuals from low childhood SES regulate their energy intake, if not based on need. Although the current research provides evidence that low childhood SES promotes food intake in the absence of need, we did not systematically vary external factors that those with low childhood SES might use to guide their energy intake, such as food availability or palatability. Each of these lines of research has the potential to offer critical new insights into the development of unhealthy weight gain and obesity among those with low childhood SES.

Footnotes

1 - Studies 1 and 2 were comprised of females only to minimize between-participant variance on our dependent measure (Rolls, Fedoroff, & Guthrie, 1991; Geary & Lovejoy, 2007). Sample sizes were chosen based on the recommendation by Cohen that researchers have 30 participants within each testing condition to achieve 80% power (the minimum suggested power for an ordinary study) in cases where the expected effect size is medium to large (Cohen, 1988 (e.g., Cohen, 1988). We increased target *n* to 45 per testing condition in Study 3 since it was the first study in the series to include men and we anticipated greater variability in food intake. Additionally, post hoc power analyses (reported in the online supplemental materials) revealed that each of our experiments were sufficiently powered.

2 – For each of the Studies 1 – 3, we also ran our statistical models without the inclusion of the covariates. The results were consistent with those reported in the text and can be found in the supplemental online materials.

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Material for Online Supplemental Materials

Post-hoc Power Analyses

We conducted a post-hoc power analysis using G Power (version 3.1; Faul, Erdfelder, Buchner, & Lang, 2009) on our results to determine whether our studies were adequately powered. For study 1, we found that with our effect size f^2 = .33, we would need a sample size 31 to achieve .7 power and a sample size 38 to achieve .8 power. For study 2, we found that with our effect sizes for our two SES measures ranged from f^2 = .26 to f^2 = .33, we would need 46 participants to achieve .8 power for the smallest effect size (f^2 = .26). For study 3, we found that with our effect sizes for our two energy need measures (blood glucose and drink condition) ranged from f^2 = .30 to f^2 = .69, we would need 41 participants to achieve .8 power for the smallest effect size (f^2 = .30). Accordingly, each of our studies (Study 1: N = 31, Study 2: N = 55, Study 3: N = 77) is sufficiently powered to detect the predicted effects.

Study 1

Relationship between the covariates and dependent variables

We first examined the relationship between each of our chosen covariates and our dependent measure. To this end, we entered Ps body weight and their self reported hedonic responses to each the pretzels and the cookies as predictors into a regression model that had food intake (total calories consumed) as the dependent measure. Results revealed a main effect of cookie liking as a significant predictor of calorie intake, b = 37.80 (SE = 16.65), $t(30)$ = 2.27, p = .03, semipartial r^2 = .14. There were no main effects of pretzel liking or of participant weight upon calorie consumption (ps > .27).

Target analyses re-run without covariates included

We next re-ran each of our target analyses (see Study 1 in the main paper) without the addition of any covariates in the model. For our first model, we entered childhood SES, energy need, and the interaction between the two as variables into a multiple regression model. Results revealed a significant main effect of energy need upon calorie consumption, $b = 67.07$ ($SE =$ .18.46), $t(30) = 3.63$, $p < .001$, semipartial $r^2 = .31$. This main effect was qualified by a 2-way interaction between childhood SES and energy need, $b = 33.69$ ($SE = 10.01$), $t(30) = 3.37$, $p =$ .002, semipartial $r^2 = .19$.

First, we probed this interaction by examining the impact of childhood SES on calorie consumption at different levels of hunger. When hunger was high (1 *SD* above the mean), there were no differences between those from high and low SES environments in the number of calories consumed, $b = 19.54$, $p = .15$. However, when hunger was low (1 *SD* below the mean), those who grew up in lower SES environments consumed a significantly greater number of calories than those from higher SES environments, $b = -47.84$ ($SE = 15.53$), $t(30) = -3.08$, $p = .005$, semipartial $r^2 = .16$.

We next examined the impact of hunger on calories consumed at different levels of childhood SES. For individuals growing up in higher SES environments (1 *SD* above the mean), higher self-reported hunger predicted greater calorie consumption compared to when hunger was low, $b = 126.31$ ($SE = 23.64$), $t(30) = 5.34$, $p < .001$, semipartial $r^2 = .49$. We did not observe an effect of hunger on the number of calories consumed by participants who grew up in lower SES environments (1 *SD* below the mean), however ($b = 22.57$, $p = .28$).

Study 2

Relationship between the covariates and dependent variables

As with Study 1, we first examined the relationship between each of our chosen covariates and our dependent measure. We entered Ps hedonic response to the cookies, the length of time since they had previously eaten, and P's body weight into a regression model with calories consumed as the dependent measure. Results revealed a main effect of cookie liking as a significant predictor of calorie intake, $b = 21.73$ ($SE = 7.03$), $t(54) = 3.09$, $p = .003$[1], semipartial $r^2 = .14$. Neither participant body weight nor the number of hours since Ps had last eaten was predictive of calorie consumption (ps > .19).

Target analyses re-run without covariates included

As in Study 1, we next re-ran each our target analyses (see Study 2) without the addition of the covariates.

Single item measure of childhood SES. In our first model we examined the impact of drink condition and childhood SES on food intake using our single-item measure of childhood SES. We did this by entering childhood SES (centered), condition (dummy coded; sprite coded as the reference group), and the interaction between the two into a multiple regression model. There was no significant interaction between drink condition and childhood SES on the number of calories consumed, $b = 19.51$ ($SE = 12.97$), $t(54) = 1.51$, $p = .14$, partial $r^2 = .04$. Although the interaction was no longer conventionally significant without the addition of the covariates, we probed it further to examine the simple effects. Simple slope tests revealed that in the Sprite condition, there was a significant effect of childhood SES on food intake, $b = -18.46$ ($SE = 8.97$), $t(54) = -2.06$, $p = .045$, semipartial $r^2 = .07$, indicating that lower SES was predictive of eating a greater number of calories. No such relationship emerged among Ps in the water condition, $b =$

[1] Given that cookie liking was found to be such a strong predictor of food intake, we re-ran the analyses with only this covariate in the model and results are nearly identical to the results presented in the main paper. All predicted interactions and simple effects using both childhood SES moderators had p values falling below the threshold of significance $p \leq .05$.

1.05, p = .91. Regions of significance tests revealed that for those participants who grew up in higher SES environments, participants ate more when energy need was high than when energy need was low, b = 40.00 (SE = 18.09), t(54) = -2.21, p = .03, semipartial r^2= .09. For those who grew up in lower SES environments, however, there were no differences in food intake among those for whom energy needs were high or low, b = 1.25, p = .95.

Composite measure of childhood SES. In our second model, we examined the impact of drink condition and childhood SES on food intake using our three-item measure of childhood SES. We did this by entering childhood SES (centered), condition (dummy coded; sprite coded as the reference group), and the interaction between the two into a second multiple regression model. Results revealed no significant interaction between drink condition and childhood SES on the number of calories consumed, b= 9.35 (SE = 12.27), t(54) = .76, p = .45, partial r^2 =.01. Although our interaction was not significant in this alternative model, we probed the interaction following the procedure used in primary model. Simple slope tests revealed that in the sprite condition, although childhood SES was negatively related to food intake, this difference was not statistically significant, b = -9.94, p = .21. Follow up analyses looking at individuals who were relatively high or low in childhood SES (1 *SD* above or below mean childhood SES) found that, although not statistically significant, participants who grew up in in higher SES environments ate less when energy need was high than when energy need was low, b = 27.01, p = .16. As expected, no such trend was found among those from lower SES environments, however, b = 6.65, p = .72.

Adulthood SES. Lastly, we conducted a regression model using adult SES (instead of childhood SES) as our critical moderator. As in the model reported in the paper, this model

revealed no significant interaction between condition and adult SES on calorie intake, *b*= -6.47 (*SE* = 14.10), *t*(54) = -.46, *p* = .65, partial r^2 < .01.

Study 3

Relationship between the covariates and dependent variables

As in Studies 1 and 2, we first examined the impact of our chosen covariates on food intake. Results revealed Ps gender and cookie liking by entering each of these variables into a regression model. Results revealed that cookie liking was a significant predictor of calorie intake, *b* = 5.12 (SE = 1.29), *t*(75) = 3.97, *p* < .001, semipartial r^2 = .08. Gender was also found to be a strongly significant predictor of calorie intake, *b* = -6.99 (SE = 2.53), *t*(75) = -2.76, *p* = .007, semipartial r^2 = .17.

Target analyses re-run without covariates included

Moderated Mediation. We next re-ran each of the target analyses without the addition of the covariates. Wetested for moderated mediation without including the covariates from our main paper, using Hayes' PROCESS SPSS macro [Model 14 (Hayes, 2013)]. Ten thousand bootstrap resamples were collected to generate a bias-corrected 95% confidence interval for each indirect effect (Preacher & Hayes, 2004). In our model, drink condition (Sprite vs. water) was the independent variable, blood glucose was the mediator, and food intake was the dependent measure. Childhood SES was also entered as a moderator in the path between the mediator and the dependent measure. The results revealed a significant relationship between drink condition and blood glucose levels, *b* = -33.07 (*SE* = 3.84), *t*(77) = -8.62, *p* < .001, 95% CI [-40.72, -25.43], showing the same pattern as in our main analyses- that those who consumed the Sprite had significantly higher blood glucose than those who consumed water. Further, the relationship between blood glucose and food intake was moderated by childhood SES, *b* = -.14 (*SE* = .07),

t(77) = -2.05, *p* = .04, 95% CI [-.27, -.004]. For participants from higher SES environments, food intake was fully mediated by post-drink levels of blood glucose, *b* = 7.06 (*SE* = 3.70), 95% CI [.67, 14.43]. No such relationship was found among those from lower SES environments, *b* = -3.28(*SE* = 3.05), 95% CI [-9.49, 2.66].

Blood glucose by childhood SES. We next unpacked the moderated using multiple regression, with blood glucose (centered) and childhood SES (centered) entered in the first step and their interaction entered in the second step. Results revealed a significant interaction between blood glucose level and childhood SES, *b* = -.14 (SE = .07), *t*(76) = -2.08, *p* = .04, semipartial r^2 = .06. When blood glucose was low (1 *SD* below mean blood glucose levels), there were no childhood SES-based differences in calorie consumption, *b* = 1.43, *p* = .36. When blood glucose was high (1 *SD* above the mean), however, those who grew up in lower childhood SES environments consumed a greater number of calories than those from higher SES environments, *b* = -5.18 (*SE* = 2.37), *t*(76) = -2.19, *p* = .03, semipartial r^2 = .06. We next unpacked the interaction by looking at individuals of relatively high or low childhood SES (1 *SD* above and below mean childhood SES). Results demonstrated that for those from low childhood SES background, there was no effect of blood glucose upon eating habits, *b* = .15, *p* = .15. For those from higher SES environments, however, there was a significant effect of blood glucose, *b* = -.17 (*SE* = .08), *t*(76) = -2.02, *p* = .048, semipartial r^2 = .05, with individuals eating more when blood glucose levels were lower.

Adulthood SES. As in the paper, we ran one last moderated mediation model similar to that described above, but with adult SES (rather than childhood SES) entered as the moderator. The results of this model revealed that adult SES was predictive of the number of calories consumed, with lower SES being predictive of greater food intake, *b* = -3.02 (*SE* = 1.10), *t*(77) =

-1.56, p = .007, 95% CI [-5.20, -.83]; however, it did not interact with participants' blood glucose to influence food intake, b = -.06 (SE = .04), $t(69)$ = -1.56, p = .12, 95% CI [-.15, .02].

Supplemental Materials

Post-hoc Power Analyses

We conducted a post-hoc power analysis using G Power (version 3.1; Faul, Erdfelder, Buchner, & Lang, 2009) on our results to determine whether our studies were adequately powered. For study 1, we found that with our effect size f^2 = .33, we would need a sample size 31 to achieve .7 power and a sample size 38 to achieve .8 power. For study 2, we found that with our effect sizes for our two SES measures ranged from f^2 = .26 to f^2 = .33, we would need 46 participants to achieve .8 power for the smallest effect size (f^2 = .26). For study 3, we found that with our effect sizes for our two energy need measures (blood glucose and drink condition) ranged from f^2 = .30 to f^2 = .69, we would need 41 participants to achieve .8 power for the smallest effect size (f^2 = .30). Accordingly, each of our studies (Study 1: N = 31, Study 2: N = 55, Study 3: N = 77) is sufficiently powered to detect the predicted effects.

Study 1

Relationship between the covariates and dependent variables

We first examined the relationship between each of our chosen covariates and our dependent measure. To this end, we entered Ps body weight and their self reported hedonic responses to each the pretzels and the cookies as predictors into a regression model that had food intake (total calories consumed) as the dependent measure. Results revealed a main effect of cookie liking as a significant predictor of calorie intake, b = 37.80 (SE = 16.65), $t(30)$ = 2.27, p = .03, semipartial r^2 = .14. There were no main effects of pretzel liking or of participant weight upon calorie consumption (ps > .27).

Target analyses re-run without covariates included

We next re-ran each of our target analyses (see Study 1 in the main paper) without the addition of any covariates in the model. For our first model, we entered childhood SES, energy need, and the interaction between the two as variables into a multiple regression model. Results revealed a significant main effect of energy need upon calorie consumption, $b = 67.07$ ($SE =$ .18.46), $t(30) = 3.63$, $p < .001$, semipartial $r^2 = .31$. This main effect was qualified by a 2-way interaction between childhood SES and energy need, $b = 33.69$ ($SE = 10.01$), $t(30) = 3.37$, $p =$ .002, semipartial $r^2 = .19$.

First, we probed this interaction by examining the impact of childhood SES on calorie consumption at different levels of hunger. When hunger was high (1 *SD* above the mean), there were no differences between those from high and low SES environments in the number of calories consumed, $b = 19.54$, $p = .15$. However, when hunger was low (1 *SD* below the mean), those who grew up in lower SES environments consumed a significantly greater number of calories than those from higher SES environments, $b = -47.84$ ($SE = 15.53$), $t(30) = -3.08$, $p = .005$, semipartial $r^2 = .16$.

We next examined the impact of hunger on calories consumed at different levels of childhood SES. For individuals growing up in higher SES environments (1 *SD* above the mean), higher self-reported hunger predicted greater calorie consumption compared to when hunger was low, $b = 126.31$ ($SE = 23.64$), $t(30) = 5.34$, $p < .001$, semipartial $r^2 = .49$. We did not observe an effect of hunger on the number of calories consumed by participants who grew up in lower SES environments (1 *SD* below the mean), however ($b = 22.57$, $p = .28$).

Study 2

Relationship between the covariates and dependent variables

As with Study 1, we first examined the relationship between each of our chosen covariates and our dependent measure. We entered Ps hedonic response to the cookies, the length of time since they had previously eaten, and P's body weight into a regression model with calories consumed as the dependent measure. Results revealed a main effect of cookie liking as a significant predictor of calorie intake, $b = 21.73$ ($SE = 7.03$), $t(54) = 3.09$, $p = .003$[2], semipartial $r^2 = .14$. Neither participant body weight nor the number of hours since Ps had last eaten was predictive of calorie consumption (ps > .19).

Target analyses re-run without covariates included

As in Study 1, we next re-ran each our target analyses (see Study 2) without the addition of the covariates.

Single item measure of childhood SES. In our first model we examined the impact of drink condition and childhood SES on food intake using our single-item measure of childhood SES. We did this by entering childhood SES (centered), condition (dummy coded; sprite coded as the reference group), and the interaction between the two into a multiple regression model. There was no significant interaction between drink condition and childhood SES on the number of calories consumed, $b = 19.51$ ($SE = 12.97$), $t(54) = 1.51$, $p = .14$, partial $r^2 = .04$. Although the interaction was no longer conventionally significant without the addition of the covariates, we probed it further to examine the simple effects. Simple slope tests revealed that in the Sprite condition, there was a significant effect of childhood SES on food intake, $b = -18.46$ ($SE = 8.97$), $t(54) = -2.06$, $p = .045$, semipartial $r^2 = .07$, indicating that lower SES was predictive of eating a greater number of calories. No such relationship emerged among Ps in the water condition, $b =$

[2] Given that cookie liking was found to be such a strong predictor of food intake, we re-ran the analyses with only this covariate in the model and results are nearly identical to the results presented in the main paper. All predicted interactions and simple effects using both childhood SES moderators had p values falling below the threshold of significance $p \leq .05$.

1.05, p = .91. Regions of significance tests revealed that for those participants who grew up in higher SES environments, participants ate more when energy need was high than when energy need was low, b = 40.00 (SE = 18.09), $t(54)$ = -2.21, p = .03, semipartial r^2= .09. For those who grew up in lower SES environments, however, there were no differences in food intake among those for whom energy needs were high or low, b = 1.25, p = .95.

Composite measure of childhood SES. In our second model, we examined the impact of drink condition and childhood SES on food intake using our three-item measure of childhood SES. We did this by entering childhood SES (centered), condition (dummy coded; sprite coded as the reference group), and the interaction between the two into a second multiple regression model. Results revealed no significant interaction between drink condition and childhood SES on the number of calories consumed, b= 9.35 (SE = 12.27), $t(54)$ = .76, p = .45, partial r^2 =.01. Although our interaction was not significant in this alternative model, we probed the interaction following the procedure used in primary model. Simple slope tests revealed that in the sprite condition, although childhood SES was negatively related to food intake, this difference was not statistically significant, b = -9.94, p = .21. Follow up analyses looking at individuals who were relatively high or low in childhood SES (1 SD above or below mean childhood SES) found that, although not statistically significant, participants who grew up in in higher SES environments ate less when energy need was high than when energy need was low, b = 27.01, p = .16. As expected, no such trend was found among those from lower SES environments, however, b = 6.65, p = .72.

Adulthood SES. Lastly, we conducted a regression model using adult SES (instead of childhood SES) as our critical moderator. As in the model reported in the paper, this model

revealed no significant interaction between condition and adult SES on calorie intake, *b*= -6.47 (*SE* = 14.10), *t*(54) = -.46, *p* = .65, partial r^2 < .01.

Study 3

Relationship between the covariates and dependent variables

As in Studies 1 and 2, we first examined the impact of our chosen covariates on food intake. Results revealed Ps gender and cookie liking by entering each of these variables into a regression model. Results revealed that cookie liking was a significant predictor of calorie intake, *b* = 5.12 (SE = 1.29), *t*(75) = 3.97, *p* < .001, semipartial r^2 = .08. Gender was also found to be a strongly significant predictor of calorie intake, *b* = -6.99 (SE = 2.53), *t*(75) = -2.76, *p* = .007, semipartial r^2 = .17.

Target analyses re-run without covariates included

Moderated Mediation. We next re-ran each of the target analyses without the addition of the covariates. Wetested for moderated mediation without including the covariates from our main paper, using Hayes' PROCESS SPSS macro [Model 14 (Hayes, 2013)]. Ten thousand bootstrap resamples were collected to generate a bias-corrected 95% confidence interval for each indirect effect (Preacher & Hayes, 2004). In our model, drink condition (Sprite vs. water) was the independent variable, blood glucose was the mediator, and food intake was the dependent measure. Childhood SES was also entered as a moderator in the path between the mediator and the dependent measure. The results revealed a significant relationship between drink condition and blood glucose levels, *b* = -33.07 (*SE* = 3.84), *t*(77) = -8.62, *p* < .001, 95% CI [-40.72, -25.43], showing the same pattern as in our main analyses- that those who consumed the Sprite had significantly higher blood glucose than those who consumed water. Further, the relationship between blood glucose and food intake was moderated by childhood SES, *b* = -.14 (*SE* = .07),

t(77) = -2.05, *p* = .04, 95% CI [-.27, -.004]. For participants from higher SES environments, food intake was fully mediated by post-drink levels of blood glucose, *b* = 7.06 (*SE* = 3.70), 95% CI [.67, 14.43]. No such relationship was found among those from lower SES environments, *b* = -3.28(*SE* = 3.05), 95% CI [-9.49, 2.66].

Blood glucose by childhood SES. We next unpacked the moderated using multiple regression, with blood glucose (centered) and childhood SES (centered) entered in the first step and their interaction entered in the second step. Results revealed a significant interaction between blood glucose level and childhood SES, *b* = -.14 (SE = .07), *t*(76) = -2.08, *p* = .04, semipartial r^2 = .06. When blood glucose was low (1 *SD* below mean blood glucose levels), there were no childhood SES-based differences in calorie consumption, *b* = 1.43, *p* = .36. When blood glucose was high (1 *SD* above the mean), however, those who grew up in lower childhood SES environments consumed a greater number of calories than those from higher SES environments, *b* = -5.18 (*SE* = 2.37), *t*(76) = -2.19, *p* = .03, semipartial r^2 = .06. We next unpacked the interaction by looking at individuals of relatively high or low childhood SES (1 *SD* above and below mean childhood SES). Results demonstrated that for those from low childhood SES background, there was no effect of blood glucose upon eating habits, *b* = .15, *p* = .15. For those from higher SES environments, however, there was a significant effect of blood glucose, *b* = -.17 (*SE* = .08), *t*(76) = -2.02, *p* = .048, semipartial r^2 = .05, with individuals eating more when blood glucose levels were lower.

Adulthood SES. As in the paper, we ran one last moderated mediation model similar to that described above, but with adult SES (rather than childhood SES) entered as the moderator. The results of this model revealed that adult SES was predictive of the number of calories consumed, with lower SES being predictive of greater food intake, *b* = -3.02 (*SE* = 1.10), *t*(77) =

-1.56, p = .007, 95% CI [-5.20, -.83]; however, it did not interact with participants' blood glucose to influence food intake, b = -.06 (SE = .04), $t(69)$ = -1.56, p = .12, 95% CI [-.15, .02].