Low Income is Associated with High Baseline Levels and Low Stress Reactivity of Cortisol, but Not Alpha Amylase

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Summary

Does poverty have particular neurobiological consequences? We test here whether low-income individuals show higher baseline levels, or higher stress-induced reactivity, of the stress hormone cortisol and the sympathetic marker salivary alpha-amylase. A sample of 80 healthy university students was sampled both at baseline, and while performing the Trier Social Stress Test (TSST), a standard stress-inducing laboratory task. We find that poor individuals have higher baseline levels of cortisol, and lower cortisol reactivity to stress, than richer individuals. In contrast, salivary alpha amylase shows no relationship to income, neither in baseline levels nor stress reactivity. Together, these findings suggest that poverty is associated with dysregulation of cortisol; this may lead to adverse health outcomes and disadvantageous decision-making.

Keywords:

Hypothalamic-pituitary-adrenal (HPA) axis; sympathetic-adrenal-medullary (SAM) axis; cortisol; salivary alpha amylase (sAA); income; poverty; socioeconomic status; stress reactivity; Trier Social Stress Test (TSST)

Introduction

Does poverty get under your skin? A prominent hypothesis is that low-income environments may be characterized by both greater exposure to stressful events, and the absence of resources to deal with such stress (Baum et al., 1999; Steptoe et al., 2002; Brunner, 1997; Kristenson, 2004). This hypothesis predicts changes in physiological markers of stress: if low-income populations are more stressed, they should show higher levels, altered daily profiles, or different stress reactivity in stress hormones. In the following, we refer to this claim as the poverty-stress hypothesis. These changes could in turn lead to adverse consequences of their own, such as immunosuppression (Cacioppo et al., 2002) or altered decision-making (Porcelli & Delgado, 2009).

Do low-income populations indeed show differences in physiological markers of stress? A growing body of literature investigates this question; however, it will emerge in the following discussion that the answer remains unclear.

The physiological response to stress in humans is characterized by the joint activation of two systems: the hypothalamic-adrenal-pituitary (HPA) axis and its associated stress hormone cortisol, and the sympathetic-adrenal-medullary (SAM) axis and its associated stress hormone norepinephrine (NE). Cortisol can be measured directly in saliva, where it is a good indicator of levels in the blood (Kirschbaum & Hellhammer, 1994); sympathetic can be measured by proxy in saliva, through salivary alpha-amylase (sAA; van Stegeren et al., 2006; Ehlert et al., 2006).

Most attention in the literature has been devoted to cortisol. A number of studies show higher baseline levels of cortisol in populations of low socioeconomic status (SES), consistent with the poverty-stress hypothesis (Cohen et al., 2006a, 2006b; Evans & Kim, 2009; Evans & English,

2002; Li et al., 2007; Lupien et al., 2000; Arnetz et al., 1991; see Dowd et al., 2009, for a review). However, other studies show no associations, or relationships restricted to certain population groups or parts of the daily cortisol profile (Dowd et al., 2006; Gersten, 2008; Goodman et al., 2005; Ranjit et al., 2005; Rosmond & Bjorntorp, 2000; Steptoe et al., 2005; Decker, 2000; Rosero-Bixby & Dow, 2009). Finally, three studies even show a positive association between baseline cortisol and SES (Brandstadter et al., 1991; Chen & Paterson, 2006; Fiocco et al., 2007). Thus, it remains unclear to what extent low SES is in fact associated with higher baseline cortisol levels; the first purpose of the present study was to shed further light on this question.

Second, a question that has received much less attention is whether cortisol *reactivity* to stress is altered in people with low SES. In the few studies that have addressed this question, participants are typically presented with mildly stressful tasks in the laboratory, such as the well-known Trier Social Stress Task (TSST; Kirschbaum et al., 1993). The results are conflicting: Fiocco et al. (2007) find a negative association between cortisol reactivity and SES, Kristenson et al. (2001) find a positive association, Adler et al. (2000) no association between reactivity and SES, but one between SES and adaptation to repeated stress; and two other studies find no relationship (Steptoe et al., 2005; Kapuku et al., 2002). Thus, the second aim of this paper was to investigate further to what extent cortisol reactivity to stress is associated with income.

The literature on SES and sAA is smaller yet; two studies show higher sAA levels in low-SES children (Wolf et al., 2008; Granger et al., 2006), but one of these only obtained an effect in asthmatic and not healthy children (Wolf et al., 2008), and no study has investigated this link in adults. In addition, to our knowledge no study has investigated whether sAA reactivity to stress

is modulated by income. Thus, the final goals of this paper were to investigate whether baseline levels of sAA, and sAA reactivity to stress, are affected by income in adults.

To address these research questions, we measured both baseline levels of cortisol and sAA, and cortisol and sAA reactivity to a laboratory stressor, in a sample of Swiss university students. We find that lower income students have higher levels of baseline cortisol, and reduced cortisol reactivity to stress, compared to richer students. However, no relationships between income and baseline sAA levels were found; furthermore, income did not predict sAA reactivity to laboratory stress.

Methods

Participants

We recruited 81 health male participants form the subject pool of the University of Zürich. Their mean age was 21.31 ± 1.85 years. We excluded students of economics and psychology, and those who were acutely or chronically ill, took medications, drugs, smoked more than 5 cigarettes a day, regularly consumed more than 60g of alcohol per day, suffered from allergies or psychiatric disorders, were in psychological or psychiatric treatment at the time of the study, had previously participated in a TSST, or had a body mass index smaller than 18 or greater than 25. Participants were instructed to not consume medications, alcohol, or coffee, and not to engage in sexual intercourse, for 24h before the experiment. In addition, they were asked to get up at least 3h before the beginning of the experiment, and to not drink coffee, eat, smoke, or perform strenuous physical activity in the last 2h before the experiment. All participants were tested in the afternoon between 2pm and 8pm, when plasma cortisol levels are close to the circadian

trough. They gave written informed consent and were reimbursed for their participation. An experimental session lasted 2h.

Stress Manipulation

Psychosocial stress was induced with a grouped version of the Trier Social Stress Test (TSST-G; Kirschbaum et al., 1993; von Dawans et al., 2010). The procedure followed closely that described by Dawans et al. (2010), and involved a preparation period of 5 min, followed by a video- and audio- taped public speaking task of 12 min (a fictional job interview, see below), and a mental arithmetic task of 8 min, both in front of an evaluation committee (one man and one woman wearing white laboratory coats). A maximum of 4 and a minimum of 2 subjects were tested at the same time. In the job interview component of the task, each participant had 3 minutes to describe why their personal qualities qualified them for a job. The committee repeatedly interrupted the presentation with questions, following a pre-prepared script. In the arithmetic task, participants were asked to count backwards in steps of 16, starting at a random 4-digit number. Mistakes were corrected by the panel, and the participant had to start over. All Subjects first delivered their speech and after that performed the arithmetic task. Each subject was called at least twice and in random order for every task, to induce a feeling of unpredictability. Speaking time for every participant was kept constant.

To keep the cognitive load and circumstances of the control condition as comparable as possible, only lacking the component of social control, subjects in the control condition underwent the same conditions, with three important differences. First, subjects were not video- or audio-taped and there was no panel in laboratory coats, just a passive observer in a corner of the room. Second, while the mental arithmetic task was the same, the fictional job interview was replaced

by an account of a memorable experience with a good friend. The purpose of this task was toe require a similar amount of creativity and cognitive resources as the job interview, while not containing the same stressful element of social evaluation and having to "talk oneself up". Finally, all subjects performed their tasks simultaneously with the other participants; this made the individual contributions unintelligible to the passive observer and the other participants, thus further reducing the social evaluative element. Total duration of the task and speaking time for each participant were matched to the parameters of the stress condition.

Procedure

Subjects were randomly assigned to one of two conditions: control (N=41) and stress (N=40). Subjects were instructed not to talk to each other during the whole experiment. An overview of the study timeline is displayed in **Fig. 1**. Twenty minutes after subjects arrived in the laboratory, a first saliva sample was taken. Subjects were guided to a room where they received general instructions about the experiment, but not the TSST or Control tasks, to avoid inducing anticipatory stress before taking the second baseline saliva sample. After 20 min, second saliva sample was taken. Next, subjects received instructions for the TSST, and were given a 5 min preparation period for the stress or control task. They were then guided to another room, where they gave their speech. Before subjects were instructed to perform the arithmetic task, a third saliva sample was taken. Directly after the whole TSST or control procedure, a fourth saliva sample was taken. Next, participants were asked to sit at the chair placed behind them and performed economic choice tasks, the results of which are reported in a separate paper. After completing these tasks, participants remained seated, filled in a socioeconomic questionnaire, and read a neutral magazine. Additional saliva samples were taken 10 min, 20 min, and 50 min

after the end of the TSST or control task. After the last saliva sample was taken, participants were debriefed and paid for their participation.

Salivary Sampling and Biochemical Analysis

Salivary samples were obtained using Salivette sampling devices (Sarstedt, Nümbrecht, Germany) at 7 time points during the experiments (**Fig. 1**). Salivary samples were stored at -20°C until further analysis. Free cortisol levels were measured using a commercially available immunoassay (IBL, Hamburg, Germany). Salivary alpha-amylase levels were measured by a quantitative enzyme kinetic essay as described elsewhere (van Stegeren et al., 2006).

Income measure

Information about the individual incomes of participants was elicited with the following question: "How much money do you have available in an average month (excluding costs of rent and insurance)?" The purpose of this particular formulation was to elicit income data that was reflective of the financial liquidity of participants and independent from the source of the money, e.g. jobs vs. parents. Statistical analysis was performed on both the untransformed and log-transformed income data (see below).

Covariates

Our regressions control for the following covariates: *age*, measured in years; time between waking and the beginning of the experimental session (*timeawake*); body mass index (*bmi*), defined as weight (kg) / height^2 (cm); number of siblings (*numsibs*); political orientation (left = 0, right = 100; *polright*); and dummy variables for whether subjects had recently smoked

(*recentsmoke*), eaten (*recenteat*), or drunk coffee (*recentcoffee*) or alcohol (*recentalcohol*). To show the robustness of the effects, we report different specifications controlling for various subsets of these covariates.

Statistical Analysis

Baseline cortisol and sAA were determined by averaging the cortisol and sAA levels from the first two of the seven saliva samples. Stress responsivity of cortisol and sAA was defined as the area under the cortisol or sAA response curve (AUC) in those subjects exposed to the social stressor (stress group). The AUC was calculated with respect to the baseline to rule out effects stemming from baseline differences. The effectiveness of the stress task in raising hormone levels was assessed using a 7 (Sample Period: t0 vs. t20 vs. t30 vs. t40 vs. t50 vs. t60 vs. t90) x 2 (Stress: TSST-G vs. Control) General Linear Model (GLM) repeated measures ANOVA with Sample Period as a repeated measure.

The relationship between baseline hormone levels or hormone stress responsivity and income was assessed with ordinary least squares regression using heteroskedasticity-robust standard errors. Specifically, we regressed hormone levels (either baseline or AUC) on income (either linear or log), and varying sets of covariates as described above. When assessing stress reactivity, the regressions were restricted to those subjects who were exposed to the TSST. Different specifications include different subsets of covariates to show the robustness of our results.

Results

Effectiveness of TSST in raising cortisol and sAA levels

The experimental groups (stress vs. control) did not differ in age, BMI, or baseline cortisol and sAA (P's > 0.1). As expected, the stress manipulation significantly raised both cortisol and sAA levels: an ANOVA for cortisol showed a significant Sample Period x Stress interaction (**Fig. 1A**, $F_{1.6,124.4}$=34.70, P<0.001). Furthermore, a main effect of Sample Period ($F_{1.6,124.4}$=34.02, P<0.001) and a main effect of Stress ($F_{1,77}$=38.53, P<0.001) were found. Planned simple contrasts related to baseline showed that subjects in the stress condition had increased cortisol levels from t30 (during the TSST-G) until t90, i.e. at the end of the session (all P's<0.001). For alpha-amylase, a significant Sample Period x Stress interaction (**Fig. 1B**, $F_{5.1,395}$=8.89, P<0.001) and a significant main effect of Sample Period ($F_{5.1,395}$=23.12, P<0.001) were found. Planned simple contrasts compared to baseline showed that alpha-amylase levels were increased in the stress condition from t30, i.e. during the TSST-G, until after the early test condition at t50 (P's<0.05).

Cortisol and income

Our first main question was whether baseline cortisol is associated with income: do poorer people have higher cortisol levels? To answer this question, we regressed baseline cortisol levels on income, while controlling for various subsets of covariates, and using both linear and log specifications for the income variable. The results are shown in Table 1 (linear income variable) and Table 2 (log income variable). The income coefficient is significant in all specifications, with higher incomes associated with lower baseline cortisol levels, and low-income subjects showing higher baseline cortisol levels.

Second, we wished to test whether cortisol reactivity to the TSST was associated with income: do poorer people have higher or lower stress reactivity? To answer this question, we regressed cortisol AUC on income, again using both linear and log specifications, and controlling for various covariates. The results are shown in Table 3 (linear income variable) and Table 4 (log income variable). The coefficient on income is significantly positive in all specifications except that which does not control for the time elapsed between waking and testing, or any other covariates. Thus, higher-income subjects show higher stress reactivity in terms of cortisol AUC than lower-income participants.

sAA and income

Our third question was whether baseline sAA is associated with income. We proceeded as above for the relationship between baseline cortisol and income. The results are presented in Table 5 (linear income variable) and Table 6 (log income variable). None of the specifications resulted in a significant coefficient on income. Thus, baseline sAA does not appear to be related to income, whether it is specified linearly or as log.

Finally, we asked whether sAA reactivity to the TSST was associated with income. Again we proceeded as above for cortisol, using sAA AUC as the dependent variable. The results are shown in Tables 7 and 8. No relationship between income and sAA AUC was found, suggesting that income is not associated with stress reactivity in terms of sAA.

Discussion

The purpose of this paper was to assess whether low income is associated with high baseline cortisol or sAA levels, or differential stress reactivity of cortisol or sAA as a function of income. We found that low-income subjects indeed show higher baseline levels of cortisol, and blunted responsivity of cortisol to a laboratory stress test, compared to richer participants. In contrast, no effects on income on either baseline sAA levels or sAA responsivity to stress were found.

These results expand the existing literature in several ways. First, previous studies have yielded conflicting results regarding the association between baseline cortisol and income: some studies found a negative correlation, as we do here (Cohen et al., 2006a, 2006b; Evans & Kim, 2007; Evans et al., 2000; Li et al., 2007; Lupien et al., 2000; Arnetz et al., 1991), while others find no association or mixed results for different subgroups of participants or aspects of the diurnal cortisol profile (Dowd et al., 2006; Gersten, 2008; Goodman et al., 2005; Ranjit et al., 2005; Rosmond & Bjorntorp, 2000; Steptoe et al., 2005; Decker, 2000; Rosero-Bixby & Dow, 2009), or a positive correlation (Brandstadter et al., 1991; Chen & Paterson, 2006; Fiocco et al., 2007). Our study adds to this literature by showing that baseline levels of cortisol taken in the afternoon in healthy male undergraduate students strongly predict their income. Closer comparison of our study with those which produced conflicting results suggests potential explanations: we find our association between baseline cortisol and income in afternoon levels, and some previous studies reporting null results measured overnight cortisol (Dowd & Goldman, 2006; Gersten, 2008) or morning levels (Goodman et al., 2005). Also, some studies that report null results or associations in the other direction use children or teenage samples, replacing own income with parental education (Goodman et al., 2007) or neighborhood SES (Chen & Paterson, 2006); it may be the case that the association between cortisol and SES only emerges in adulthood when using own income as a predictor. Furthermore, instead of income, some studies use self-reported stress

about one's financial situation, occupational status, or a discretized income variable, such as quartiles, all of which are likely to be less fine-grained measures than the continuous income variable we use here (Dowd & Goldman, 2006; Gersten, 2008; Rosmond & Bjorntorp, 2000). Finally, one study reporting a null result has only 30 subjects (Decker, 2000), and two do not report cortisol as a separate outcome, but in combination with other measures such as epinephrine, or "at-risk" factors such as BMI (Gersten, 2008; Rosero-Bixby & Dow, 2009). Thus, it is likely that the significant relationship between baseline cortisol and income that we observe here is due to careful isolation of afternoon cortisol levels, together with our fine-grained income measure.

Second, this study is the first to rigorously show blunted cortisol reactivity to stress in low-income subjects. A previous study by Kristenson et al. (2001) found a similar result, but compared subjects from Lithuania (low SES) to subjects from Stockholm (high SES); this cross-country comparison naturally has a host of potential confounds and is thus less rigorous than the within-subject pool regression on monthly income we report here. Intriguingly, Fiocco et al. (2007) found a higher cortisol response to the TSST in subjects with low compared to high education; since education and income are usually positively correlated, this finding appears to contradict that of the present study. Since we used a sample of university students who were almost identical in educational achievement, we could not fruitfully address the respective relationships between income and cortisol reactivity vs. education and cortisol reactivity; further studies with more diverse samples will be required to answer this question.

Third, we find no associations between baseline sAA and income, or sAA reactivity to stress and income. To our knowledge, this is the first study of the relationship between baseline sAA and income in adults. In children, Granger et al. (2006) reported a negative correlation between

baseline sAA levels and SES, while Wolf et al. (2008) found a similar relationship in asthmatic but not healthy children. Since these results are conflicting and we are the first to have addressed this question with adults, and with a relatively small sample, we suggest that future studies revisit the relationship between baseline sAA and income.

Finally, this is the first report on the relationship between income and sAA stress reactivity; we find no significant association. However, in our view the hypothesis that sAA stress reactivity may be related to income remains plausible, as a number of previous studies have found blunted stress reactivity in terms of other sympathetic markers, in particular cardiovascular variables such as heart rate and blood pressure (Owens et al., 1993; Lynch et al., 1998; Carroll et al., 1997, 2000; Steptoe et al., 2002; Brydon et al., 2004). Thus, future studies might revisit the question of whether low-SES subjects may have altered sAA stress reactivity.

What is the physiological and behavioral significance of higher baseline cortisol levels, and blunted cortisol reactivity to stress, in low-income subjects? First, higher baseline cortisol levels in low-SES subjects may contribute to the higher rates or morbidity and mortality in this population. Specifically, chronically elevated cortisol levels have been shown to lead to hippocampal atrophy and memory deficits (Lupien et al., 1998), increased risk of cardiovascular disease and stroke (Rosmond & Bjorntorp, 2000), and immunosuppression (Cacioppo et al., 2002). Elevated baseline cortisol levels are also observed in chronically stressed rats (Katz, 1981) and monkeys (Sapolsky, 1993), as well as human patients suffering from depression (Holsboer, 2000; Checkley, 1996). Second, altered cortisol reactivity has been associated with a host of debilitating conditions, such as chronic stress (Kristenson et al., 1998; Benschop et al., 1994; van der Pompe, 1996), effort-reward imbalance (Siegrist et al., 1997), and vital exhaustion (Nicholson & van Diest, 2002); smokers and alcoholics also show attenuated cortisol reactivity

to stress (Errico et al., 1993; Kirschbaum et al., 1993; Roy et al., 1994). In sum, the cortisol baseline and reactivity differences that we here associate with low income are also associated with a host of adverse psychosocial and health outcomes. Of course, at present it remains unclear in which direction causality runs: the cortisol differences we observe could be a cause or a consequence of low income, or even of any of the psychosocial and socioeconomic factors mentioned above which are usually associated with poverty. What remains, however, is that once the cortisol differences have been established, they lead to adverse health consequences, and thus are one possible channel that may account for the link between low income and morbidity and mortality (Steptoe & Marmot, 2002; Baum et al., 1999; Brunner, 1997; Kristenson et al., 2004). A less well established, but potentially even more intriguing channel through which altered cortisol baselines and reactivity may affect long-term outcomes is through immediate behavioral consequences. It has long been known that chronically elevated cortisol levels have adverse consequences for memory processes (for reviews, see McEwen & Seeman, 1999; McEwen, 2004; McEwen & Sapolsky, 1995; de Kloet et al., 1999; Lupien et al., 2007, 2009; Kim & Haller, 2007). In line with these findings and the present results, Evans & Schamberg (2009) found that working memory performance in young adults was lower for individuals coming from poor families; moreover, the coefficient on poverty became non-significant when the authors controlled for allostatic load during childhood. Allostatic load was a composite measure which included overnight urine levels of cortisol, epinephrine, and norepinephrine, body mass index, and resting blood pressure. This finding suggests that altered cortisol levels may directly contribute to the adverse cognitive long-term outcomes that are typically observed in children from poor backgrounds.

Furthermore, a number of studies now show that economic decision-making is impaired by increased cortisol levels. Specifically, subjects with (experimentally) raised cortisol levels subjects perform worse on verbal analogy tasks (Keinan, 1987) and are less able to learn the optimal response among several available options (Gray, 1999; Preston et al., 2007; van den Bos et al., 2009). Recently, Porcelli and Delgado (2009) found that stressed subjects are less rational in risky decision-making, showing a greater degree of risk aversion in the gain domain, and of risk seeking in the loss domain, than non-stressed subjects. In our own work, we recently found that subjects with strongly increased cortisol levels became more present-biased in making choices between amounts of money available at different times (Cornelisse et al., in preparation). In sum, we have shown that poor individuals have higher baseline cortisol levels, and blunted cortisol reactivity to stress, than richer individuals. Salivary alpha amylase showed no relationship to income, neither in terms of baseline levels nor stress reactivity. Together, these findings substantiate the claim that poverty is characterized by particular neurobiological consequences; these consequences, in turn, may have adverse consequences for health and decision-making, and thus contribute to exacerbating the poverty that precipitated them.

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Figure Captions

Fig. 1: Timecourse of cortisol activation (**A**) and salivary alpha amylase (**B**) throughout the Trier Social Stress Test. The mean of samples 1 and 2 served as baseline samples. Cortisol reactivity to the Trier Social Stress test was defined as area under the curve in the "stress" group with respect to this baseline.

Tables

Table 1: Baseline Cortisol and Income

VARIABLES	(1) model1 cortbase	(2) model2 cortbase	(3) model3 cortbase	(4) model4 cortbase
income	-0.00420**	-0.00351**	-0.00324**	-0.00304**
	(0.00174)	(0.00171)	(0.00132)	(0.00143)
timeawake		0.000168	0.000132	0.000148
		(0.000113)	(0.000135)	(0.000155)
age			-0.723	-0.644
			(0.491)	(0.539)
bmi			-0.0350	0.0257
			(0.551)	(0.535)
polright			0.0185	0.00485
			(0.0421)	(0.0509)
numsibs			0.228	-0.104
			(1.138)	(1.458)
recentsmoke				-1.506
				(3.439)
recenteat				2.502
				(2.628)
recentalcohol				-1.080
				(3.259)
Constant	15.36***	19.00***	33.16	30.82
	(1.612)	(3.145)	(21.96)	(22.39)
Observations	79	73	72	70
R-squared	0.065	0.086	0.106	0.132

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 2: Baseline Cortisol and Log Income

VARIABLES	(1) model1 cortbase	(2) model2 cortbase	(3) model3 cortbase	(4) model4 cortbase
lnincome	-3.280**	-3.060*	-2.859*	-2.912*
	(1.632)	(1.732)	(1.699)	(1.674)
timeawake		0.000147	0.000126	0.000133
		(0.000114)	(0.000129)	(0.000148)
age			-0.568	-0.509
			(0.459)	(0.529)
bmi			-0.193	-0.117
			(0.563)	(0.546)
polright			0.00381	-0.00967
			(0.0442)	(0.0493)
numsibs			0.110	-0.182
			(1.052)	(1.309)
recentsmoke				-0.784
				(3.344)
recenteat				2.769
				(2.514)
recentalcohol				-1.074
				(3.140)
Constant	33.07***	35.18***	49.49*	47.25*
	(10.50)	(10.53)	(25.40)	(25.39)
Observations	79	73	72	70
R-squared	0.099	0.130	0.144	0.175

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table 3: Cortisol Stress Reactivity and Income

VARIABLES	(1) model1 cort_AUCi	(2) model2 cort_AUCi	(3) model3 cort_AUCi	(4) model4 cort_AUCi
income	0.150	0.233**	0.446***	0.434***
	(0.155)	(0.107)	(0.133)	(0.148)
timeawake		0.0234	0.0423*	0.0479*
		(0.0188)	(0.0232)	(0.0268)
age			37.21	15.99
			(105.8)	(116.0)
bmi			80.73	81.66
			(60.00)	(65.74)
polright			-23.09**	-23.69**
			(9.478)	(11.16)
numsibs			-206.2	-219.8
			(141.0)	(178.4)
recentsmoke				157.8
				(385.5)
recenteat				18.47
				(336.9)
o.recentalcohol				0
				(0)
Constant	673.5***	1,383**	98.29	724.7
	(182.4)	(530.4)	(2,389)	(2,700)
Observations	40	34	33	31
R-squared	0.009	0.056	0.285	0.281

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table 4: Cortisol Stress Reactivity and Log Income

VARIABLES	(1) model1 cort_AUCi	(2) model2 cort_AUCi	(3) model3 cort_AUCi	(4) model4 cort_AUCi
lnincome	98.60	216.1***	302.9***	299.2***
	(84.04)	(78.12)	(87.39)	(102.6)
timeawake		0.0301	0.0469*	0.0524*
		(0.0198)	(0.0235)	(0.0272)
age			3.016	-14.91
			(100.9)	(110.9)
bmi			111.0*	114.5
			(58.00)	(67.22)
polright			-19.80**	-20.90**
			(7.970)	(9.107)
numsibs			-158.6	-168.7
			(142.7)	(184.3)
recentsmoke				82.20
				(303.0)
recenteat				72.75
				(284.1)
o.recentalcohol				0
				(0)
Constant	163.5	401.6	-1,453	-943.4
	(502.8)	(456.3)	(2,174)	(2,531)
Observations	40	34	33	31
R-squared	0.011	0.094	0.322	0.319

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table5: Baseline sAA and Income

VARIABLES	(1) model1 saabase	(2) model2 saabase	(3) model3 saabase	(4) model4 saabase
income	0.00990	0.0102	0.0131	0.0122
	(0.0171)	(0.0174)	(0.0170)	(0.0151)
timeawake		0.000496	3.83e-06	-0.000666
		(0.00155)	(0.00181)	(0.00180)
age			-3.370	-6.481
			(7.779)	(9.179)
bmi			-9.048	-9.509
			(5.992)	(5.977)
polright			0.186	0.233
			(0.511)	(0.615)
numsibs			-1.676	3.821
			(16.58)	(15.71)
recentsmoke				68.68
				(63.04)
recenteat				-35.32
				(22.03)
recentalcohol				-36.40*
				(20.67)
Constant	109.7***	124.8***	384.7	441.7
	(15.07)	(44.23)	(233.9)	(268.2)
Observations	79	73	72	70
R-squared	0.003	0.003	0.037	0.118

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table 6: Baseline sAA and Log Income

VARIABLES	(1) model1 saabase	(2) model2 saabase	(3) model3 saabase	(4) model4 saabase
lnincome	6.314	6.958	5.983	4.598
	(13.01)	(13.77)	(11.97)	(11.57)
timeawake		0.000498	-0.000141	-0.000823
		(0.00154)	(0.00177)	(0.00174)
age			-3.414	-6.431
			(7.810)	(9.176)
bmi			-8.455	-8.997
			(5.892)	(5.903)
polright			0.239	0.289
			(0.524)	(0.613)
numsibs			-0.803	4.799
			(16.28)	(15.35)
recentsmoke				68.65
				(62.82)
recenteat				-35.97
				(22.17)
recentalcohol				-34.67*
				(19.95)
Constant	76.68	88.09	336.9	401.1
	(80.48)	(86.66)	(235.1)	(276.0)
Observations	79	73	72	70
R-squared	0.003	0.003	0.035	0.116

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 7: sAA Stress Reactivity and Income

VARIABLES	(1) model1 sAA_AUCi	(2) model2 sAA_AUCi	(3) model3 sAA_AUCi	(4) model4 sAA_AUCi
income	0.608	0.946	1.036	1.109
	(0.792)	(0.640)	(0.931)	(1.029)
timeawake		0.0900	0.121	0.137
		(0.0983)	(0.113)	(0.147)
age			-239.0	-241.7
			(486.5)	(583.8)
bmi			-376.2	-299.7
			(268.7)	(335.1)
polright			-29.54	-40.22
			(46.06)	(55.95)
numsibs			72.85	234.7
			(1,046)	(1,297)
recentsmoke				-1,491
				(1,753)
recenteat				1,456
				(1,537)
o.recentalcohol				0
				(0)
Constant	1,547*	3,779	18,840	17,371
	(836.5)	(2,846)	(14,152)	(19,473)
Observations	40	34	33	31
R-squared	0.007	0.036	0.124	0.147

Robust standard errors in parentheses

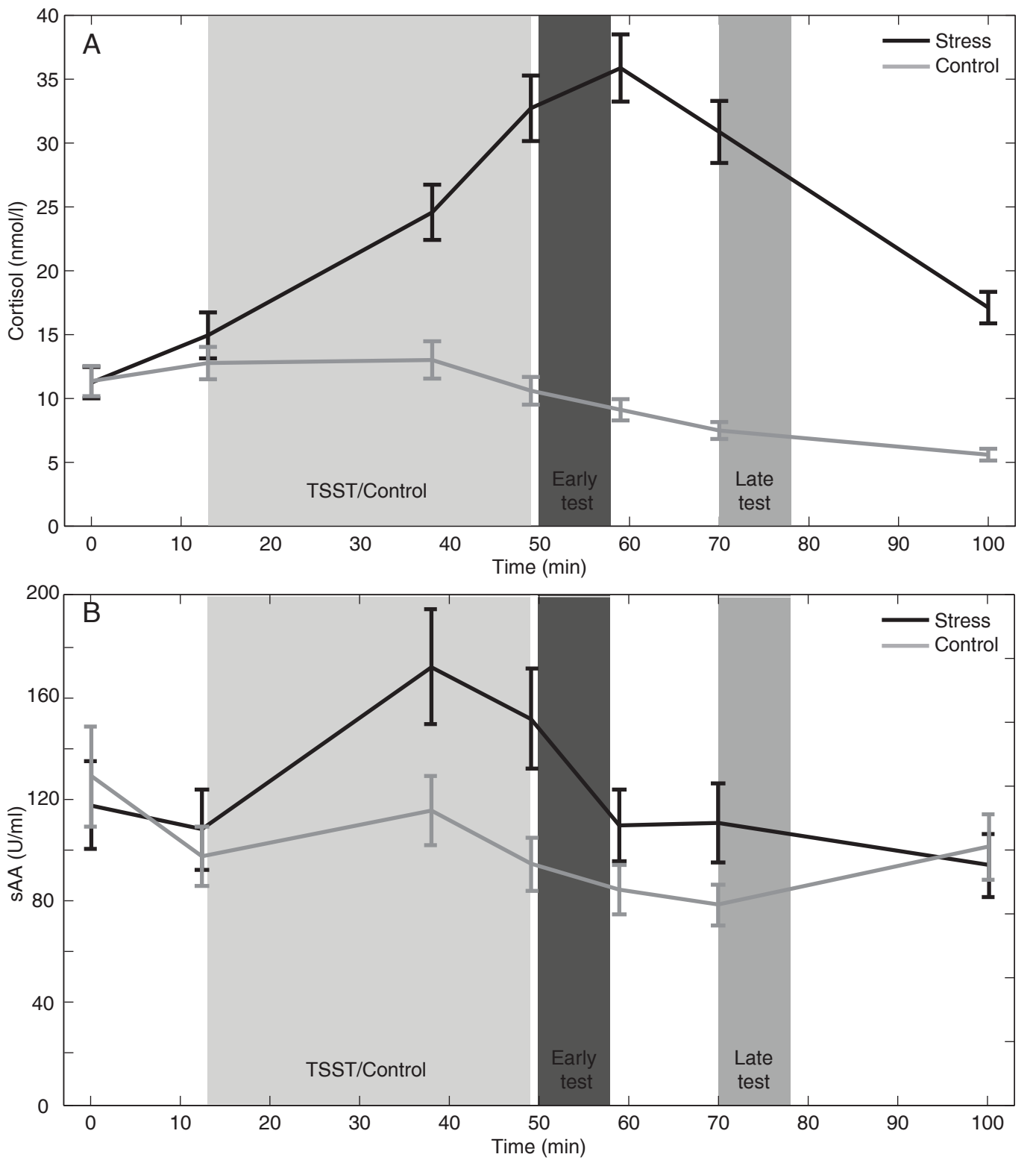
*** p<0.01, ** p<0.05, * p<0.1

Table 8: sAA Stress Reactivity and Log Income

VARIABLES	(1) model1 sAA_AUCi	(2) model2 sAA_AUCi	(3) model3 sAA_AUCi	(4) model4 sAA_AUCi
lnincome	27.32	252.3	60.03	93.34
	(573.8)	(620.1)	(697.3)	(778.7)
timeawake		0.0901	0.0969	0.109
		(0.103)	(0.123)	(0.161)
age			-296.1	-291.4
			(479.6)	(656.6)
bmi			-366.1	-297.3
			(289.9)	(358.8)
polright			-16.13	-22.58
			(45.38)	(57.34)
numsibs			280.0	462.2
			(1,048)	(1,345)
recentsmoke				-933.4
				(1,976)
recenteat				1,346
				(1,686)
recentcaffeine				-390.1
				(2,248)
o.recentalcohol				0
				(0)
Constant	1,735	2,813	18,770	16,928
	(3,465)	(3,703)	(14,801)	(21,063)
Observations	40	34	33	31
R-squared	0.000	0.019	0.104	0.126

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1


A
Stress
Control
Cortisol (nmol/l)
TSST/Control
Early test
Late test
Time (min)
B
Stress
Control
sAA (U/ml)
TSST/Control
Early test
Late test
Time (min)