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Early Life Socioeconomic Status and Metabolic Outcomes in Adolescents: The Role of Implicit Affect About One's Family

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Objectives: Previous research suggests that the quality of early family relationships may moderate the association between lower socioeconomic status (SES) and cardiovascular and other health outcomes. In this study, we investigated how implicit measures of early childhood environments (implicit anger, fear, or warmth about one's family) interacted with early life SES to predict metabolic outcomes in a sample of healthy adolescents. ***Method:*** Adolescents ($N = 259$) age 13 to 16 participated with 1 parent. Implicit family affect was measured with a computer-based implicit affect assessment tool. Early life SES was indexed by home crowding (e.g., number of people per bedroom) during the first 5 years of life. Metabolic indicators included resting blood pressure, total cholesterol, glycosylated hemoglobin, and waist circumference. ***Results:*** Early life SES significantly interacted with implicit negative family affect in resting systolic blood pressure and diastolic blood pressure levels, such that among those participants with higher early life SES, as implicit negative family affect increased, resting blood pressure also increased. Similarly, early life SES interacted with implicit family warmth to predict total cholesterol levels, such that among those participants with higher early life SES, as implicit family warmth decreased, total cholesterol increased. These patterns were not observed with current SES or with explicit measures of family relationships. ***Conclusions:*** These findings provide evidence that implicit family affect moderates the association between early life SES and adolescent metabolic outcomes in a way that suggests that implicit family affect may be more relevant among higher SES adolescents. The utility of implicit psychosocial measures in cardiovascular health studies, particularly for higher SES samples, is discussed.

Keywords: early life socioeconomic status, family environment, implicit affect, metabolic outcomes

Consistent evidence suggests that low early life socioeconomic status (SES) is a risk factor for chronic diseases across the life span (Cohen, Janicki-Deverts, Chen, & Matthews, 2010a; Galobardes, Lynch, & Davey Smith, 2004; Galobardes, Smith, & Lynch, 2006; Miller, Chen, & Parker, 2011a). However, not all individuals from low SES backgrounds experience poor health later in life (Chen & Miller, 2012), raising questions about factors that might moderate SES and health relationships (Miller, Chen, & Parker, 2011a). One important moderating childhood psychosocial factor that is often raised in the literature is the quality of family relationships (Dodge, Pettit, & Bates, 1994; Emery & Laumann-Billings, 1998; Pettit, Bates, & Dodge, 1997). However, many health studies assess early life family relationships retrospectively via self-reports, which can raise some questions about the accuracy of these data. In the present study, we adopt an alternative approach of using an implicit measure of early life family psychosocial environment and investigate how this measure affects associations between early life SES and metabolic outcomes in adolescents.

Early Life SES

Children from low early life SES backgrounds are vulnerable to numerous health problems later in life, including cardiovascular and respiratory diseases, certain cancers, and arthritis (Blane, Bartley, & Smith, 1997; Cohen, Janicki-Deverts, Chen, & Matthews, 2010a; Galobardes et al., 2004, 2006; Lawlor, Sterne, Tynelius, Davey Smith, & Rasmussen, 2006; Miller, Chen, & Parker, 2011a; Poulton et al., 2002; Power et al., 2007; Power, Manor, & Matthews, 1999). These individuals also show elevations in cardiovascular disease risk markers, including systemic inflammation and clustering of metabolic risk factors (Cohen, Doyle, Turner, Alper, & Skoner, 2004; Danese et al., 2009; Nazmi, Oliveira, Horta, Gigante, & Victora, 2010). Low early life SES is associated with changes across all physiological systems—autonomic, endocrine, and immune (Cohen et al., 2010a; Miller et al., 2011a). These associations are independent of SES in adulthood (Kittleson et al., 2006; Kuh, Hardy, Langenberg, Richards, & Wadsworth, 2002; Nazmi et al., 2010; Poulton et al., 2002), suggesting that there is something unique about the contribution of early life SES in terms of effects on health across the life span.

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Moderating Factors

Despite a robust association, many individuals do not fit the expected patterns of risk given their lower SES, leading researchers to investigate whether factors such as psychosocial characteristics may moderate SES-health associations. More specifically, when considering effects of early childhood circumstances, family relationships may play a role in attenuating risk factors for chronic diseases. For example, supportive family relationships have consistently been found to be a protective factor for disruptions in children's stress-related behavioral and biological regulatory systems (Masten & Shaffer, 2006; Pettit et al., 1997; Repetti, Taylor, & Seeman, 2002). Maternal warmth and sensitivity can attenuate stress responses often documented in children who grow up in lower SES environments (Cicchetti & Blender, 2006; Gunnar & Quevedo, 2005). Maternal warmth has also been shown to buffer those with low early life SES from higher allostatic load (Evans, Kim, Ting, Tesher, & Shannis, 2007), poor metabolic outcomes (Miller et al., 2011b), and from heightened proinflammatory signaling profiles (Chen, Miller, Kobor, & Cole, 2011), a key pathophysiological pathway for the development of diseases involving inflammation during the transition from early childhood to adolescence and adulthood.

Although the majority of the literature on SES-health moderators has pointed to the benefits for low SES, we also note that in some cases moderating factors have been found to be more relevant among those in higher SES circumstances. For example, the physiological impact of certain social stressors is more pronounced for individuals with higher status as they need to prepare for potential threats to their social standing (Kemeny, 2009). In addition, our previous study on implicit family affect in adults revealed that those from higher early life SES backgrounds but had greater implicit negative family affect had elevated resting blood pressure (Chan, Chen, Hibbert, Wong, & Miller, 2011). Hence, it is possible that certain family circumstances may be more relevant among individuals from higher SES backgrounds. In the present study, we used an implicit measure of family affect to examine potential moderating effects of family characteristics on the relationship between early life SES and metabolic outcomes in healthy adolescents.

Implicit Affect

Many studies in health psychology examining childhood family conditions have been constrained to using retrospective self-reports, which can be particularly vulnerable to measurement error and respondent biases (Hardt & Rutter, 2004; Metts, Sprecher, & Cupach, 1993). To address this issue—while still acknowledging the reality that most health research studies will not have life span longitudinal data available—we developed a behavioral paradigm to measure affect about one's early family conditions through implicit means (Chan et al., 2011). Implicit assessments are commonly used in social psychology when researchers wish to examine socially sensitive attitudes like prejudice (Fazio, Jackson, Dunton, & Williams, 1995; Greenwald, McGhee, & Schwartz, 1998; Wittenbrink, Judd, & Park, 1997), since the predictive validity of self-report measures tend to be impaired for socially sensitive topics compared with implicit measures (Greenwald et al., 2009). Implicit paradigms tap into automatic processing, capture attitudes that are expressed when individuals are unable to overtly monitor and control the influence of their attitudes on subsequent judgments of ambiguous stimuli and thus circumscribes respondent biases and social desirability motives (Fazio & Olson, 2003; Gawronski & Bodenhausen, 2006). We applied this paradigm to capture implicit affect toward early family environments using a computer paradigm that briefly presented an affective image (i.e., early childhood family photos) followed by an ambiguous stimulus (i.e., splatter paintings). Participants then rated the ambiguous stimulus on affect dimensions related to family environments (e.g., warmth, anger, fear). Attributed affect toward the ambiguous stimuli is then interpreted as affect stemming from the affective image (the childhood family photo). In an earlier study (Chan et al., 2011), we established the validity of this paradigm; in the present study, we investigate its ability to moderate the effects of SES on metabolic outcomes in adolescents.

Present Study

In a previous study, we documented the reliability and validity of this implicit measure across two samples of healthy adults (Chan et al., 2011). In the present study, we extend this research by examining the health implications of implicit family affect dimensions in a larger sample of adolescents. We examine metabolic outcomes—indicators including high blood pressure, obesity, insulin resistance, and elevated cholesterol—because elevations can be seen even among healthy adolescence, and these markers predict risk of developing cardiovascular disease (Lakka et al., 2002; Morrison, Friedman, & Gray-McGuire, 2007; Ridker, Buring, Cook, & Rifai, 2003). These indicators have also previously been linked with both lower early life SES (Danese et al., 2009) and family relationship buffers (Miller et al., 2011b). The value of assessing cardiovascular risk factors in childhood has been established previously, given that atherosclerotic lesions have been found even in adolescents and have been shown to increase in prevalence and extent in the young adult years (Berenson et al., 1992; Strong et al., 1999). We expected that early life SES would interact with implicit affect toward early family environments to predict metabolic outcomes in our sample of healthy adolescents. Some studies (Chen et al., 2011; Evans et al., 2007; Miller et al., 2011b) but not all (Chan et al., 2011; Kemeny, 2009) suggest a buffering role of positive family relationships on risks associated with lower SES. Thus, we expected that adolescents with lower early life SES but with positive implicit affect about their early family environment would have better metabolic outcomes relative to those adolescents with lower SES but with less positive implicit family affect. As a second goal, we included a self-report measure of one's childhood family environment and ratings of current relationships to compare the utility of our paradigm with explicit measures. Prior studies showed that correlations between implicit and explicit measures tend to vary widely and were especially weak when examining close relationships, suggesting that inconsistency between implicit and explicit measures are relatively common (Greenwald, Poehlman, Uhlmann, & Banaji, 2009). We included explicit measures as a covariate in our models, and also tested whether early life SES would interact with these explicit measures to predict metabolic outcomes. We expected that adolescents with lower SES but with positive self-reported family environments and relationships would have better metabolic out-

comes relative to those adolescents with lower SES and more negative family environments and relationships.

Method

Participants

Two hundred 59 adolescents were recruited through local public transportation and newspaper advertisements as well as community flyers as part of a larger study examining socioeconomic status, family life experiences, and cardiovascular disease risk. Adolescents were fluent in English and in good health, defined as being free of acute infections the 2 weeks preceding the study and having no history of chronic medical or psychiatric disorders. Adolescents ranged in age from 13 to 16 years ($M = 14.53$, $SD = 1.072$) and lived with the participating parent (who was interviewed about family SES). Participating parents ranged in age from 32 to 64 ($M = 45.83$, $SD = 5.50$). Fifty-three percent of adolescents were females, and 75.5% of parents were mothers. The ethnic breakdown of adolescents was 49.4% European-descent, followed by 15.3% Chinese-descent, which reflects the demographics in Vancouver where this study was conducted. The current study sample had a median household income in the $50,000–$74,999 CAD category, which is comparable to the national median household income in Canada in 2012 of $74,540 CAD (Statistics Canada, CANSIM, Table 111-0009). However, other studies that examined family relationship moderators on SES-health associations have focused on lower SES samples (Evans et al., 2007; Chen et al., 2011), while the present study sample ranges across the SES spectrum. See Table 1 for demographic characteristics of the sample.

Measures

Implicit affect. We used a previously validated affect attribution paradigm to capture adolescents' implicit affect toward their early family environment (Chan et al., 2011). Participants were asked to bring two personal photographs containing an image of them age 0–5 with at least one primary caregiver. This age range was selected based on previous research pointing to a potential sensitive period that impacts health later in life (Cohen et al., 2004; Miller & Chen, 2007).

Table 1
Descriptive Information

Variable	Minimum	Maximum	*M*	*SD*
Early life crowding (people/bedroom)	.50	7.25	1.53	.67
Risky Families measure	13.00	52.00	23.50	7.08
Resting SBP (mmHg)	80.33	129.67	102.50	9.49
Resting DBP (mmHg)	41.00	96.00	62.22	8.96
Total cholesterol (mmol/L)	2.27	6.45	3.86	.69
HbA1c (mmols/mol)	2.93	9.20	5.32	.40
Waist (cm)	26.00	123.00	76.73	11.82
Implicit anger (%)	.00	100.00	27.97	21.52
Implicit fear (%)	.00	100.00	26.64	22.37
Implicit warmth (%)	.00	100.00	56.85	26.97

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; HbA1c = glycosylated hemoglobin.

In this computerized implicit affect task, participants completed trials in which they were first shown either their family photograph or a control image with a neutral face (previously selected from a database of established neutral expression images; see Chan et al., 2011) for 75 ms. Following this image and a 125-ms blank screen, a splatter painting that had been randomly selected from a standard pool of neutral splatter paintings was shown for 100 ms. The splatter painting was then replaced by a black-and-white masking screen, and the adolescent was asked to indicate whether the splatter painting conveyed a target affect (either anger, fear, warmth, to which they replied *yes* or *no*). The trials varied in terms of the prime that was used (three possibilities: one of two family photographs or a control image) and the affect rating that was made (three possibilities: anger, fear, or warmth). There were a total of eight trials for each combination of prime and rating. Adolescents completed a total of 72 trials in this paradigm, which took on average 5 min to complete. Further details of the protocol and validity can be found in Chan et al. (2011).

Participants rated each splatter painting on three affect dimensions—anger, fear and warmth—because of the relevance of these emotions to childhood family psychosocial environments. These three dimensions were computed by summing up the number of times participants attributed the different emotions toward splatter paintings following a family photograph and following the control image. A percentage score was calculated (number of times participants endorsed an emotion divided by the number of trials probing that emotion) per each target affect for family photographs and for the control image. Implicit affect following the two different family photographs was averaged to obtain one percentage score per each affect dimension. Implicit affect toward the control image was subtracted out from implicit affect toward the early family environment to control for individual differences in the tendency to endorse the various affect descriptors. Final implicit affect values range from −1 to 1. Stability coefficients for this affect attribution paradigm ranged from .46 to .67 for a 6-month period, which is comparable to other implicit tasks (Cunningham, Preacher, & Banaji, 2001), and predicts negative psychosocial profiles in healthy samples of adults (Chan et al., 2011).

Early life SES. Household crowding is a measure of SES that captures resources and density (Evans & Lepore, 1992) and is useful when assessing SES retrospectively, as it is more easily recalled than other dimensions of SES, like family income and savings, over a lifetime. Hence, it is a commonly used retrospective measure of early childhood SES (Coggon, Barker, Inskip, & Wield, 1993). Parents were asked about the number of people living in their home and the number of bedrooms in their home for each year of their child's life. Crowding is computed as a ratio of the number of people living in the home to the number of bedrooms in the home. Higher scores indicate greater crowding in the home. This crowding ratio was averaged across the first 5 years of adolescents' lives to create an early life SES indicator. In this study, early life crowding was significantly correlated with current household income ($r = -.225$, $p < .001$), current crowding was significantly correlated with current household income ($r = -.336$, $p < .001$), and early life crowding was significantly correlated with current crowding ($r = .167$, $p = .007$). Household crowding has been associated with elevated stress responses (Brunner, 1997) and asthma severity (Weitzman, Gortmaker, Sobol, & Perrin, 1992).

Metabolic Outcomes

Blood pressure. Resting blood pressure was recorded with the VSM-100 BpTRU automatic blood pressure monitor using a standard occluding cuff on the participant's nondominant arm. Validation studies indicate that its measurements are within 10 mmHg 96.4% of the time (Mattu, Heran, & Wright, 2004). Following a 5-min period during which participants acclimated to the device, blood pressure readings were taken every 2 min over a 6-min period, totaling three readings that were averaged.

Total cholesterol. Blood samples were collected into EDTA-containing Vacutainer tubes (Becton-Dickinson, Oakville, Ontario, Canada) kept at 4° C and centrifuged within 12 hr. Total cholesterol was measured in a Hitachi 911 instrument (Kyowa Medex, Japan) in the Clinical Chemistry Lab at St. Paul's Hospital, Vancouver, British Columbia by using standard enzymatic colorimetric techniques with an interassay coefficient of variation of 0.9%. Total cholesterol values are expressed in mmol/L.

Glycosylated hemoglobin. Blood samples collected into EDTA-containing Vacutainer tubes (Becton-Dickinson, Oakville, Ontario, Canada). Glycosylated hemoglobin, an index of blood glucose control over a several month period, was measured with an ion exchange high-performance liquid chromatography technique (biorad, DIAMAT) in the Clinical Chemistry Lab and expressed as mmols/mol.

Waist circumference. As an index of central adiposity, waist circumference was measured twice using a steel measuring tape at the midpoint between the upper iliac crest and lower costal margin at the midaxillary line. Two measurements to the nearest .5 cm were recorded. The mean of the two closest measurements were calculated and expressed in cm.

Alternative Explanations

We assessed a number of alternative, competing explanations for potential interactions between early life SES and adolescents' implicit affect toward the family and metabolic outcomes. First, to test whether effects were specific to the early life period, we also included a measure of current SES to examine interactions of current SES with implicit affect. Current SES was indexed with the same crowding measure as early life SES (number of people per bedrooms in the home).

Second, to test whether effects were specific to an implicit measure of family environment, we included an explicit self-report measure of childhood family environment to examine interactions of early life SES with explicit reports of childhood family environments. The 13-item Risky Families (RF) measure is a commonly used self-report measure of the quality of the childhood family environment (Taylor, Lerner, Sage, Lehman, & Seeman, 2004). Respondents were asked to answer questions about their family life from early childhood. Items such as "How often did a parent or other adult in the household swear at you, insult you, put you down, or act in a way that made you feel threatened?" and "How often would you say you were neglected while you were growing up, that is, left on your own to fend for yourself?" were answered on a scale ranging from 1 (*not at all*) to 5 (*very often* or *very much*). Responses were reverse coded when necessary and summed, with higher scores indicating a harsher childhood family environment. The RF measure has been shown to have high agreement and reliability with clinical interviews conducted and coded by trained clinical interviews (Taylor et al., 2004).

Third, to test whether effects were specific to early life family relationships, we also included explicit measures of current interpersonal relationships to examine interactions of early life SES with current relationship quality. The quality of family relationships and friendships was measured using the Life Stress Interview, Youth Version (Hammen, 1991; Hammen, Adrian, & Hiroto, 1988). This is a semistructured interview that probes adolescents' chronic stress across different life domains during the last 6 months. In this study, we included the domains of family and friends, which probes for closeness, support, and conflict between adolescents and their family, and between adolescents and their peers. In each domain, a trained interviewer asked a series of open-ended questions and used the gathered information to rate the level of chronic, ongoing stress. Ratings range from 1 to 5, with higher numbers indicating more severe and persistent difficulties. Interrater reliabilities of over .85 have been documented and different domains of chronic stress captured by the interview have been shown to predict outcomes like depression (Adrian & Hammen, 1993; Rudolph & Hammen, 1999) and inflammatory markers (Chen, Fisher, Bacharier, & Strunk, 2003).

Procedure

After providing informed written assent from adolescents and obtaining parental consent, adolescents were seated in a private room. After a 5-min resting period, blood pressure readings were then taken by placing the occluding cuff on the upper aspect of the participant's nondominant arm with the microphone placed above an area where the brachial artery could be palpated. Following a series of anthropometric measures, adolescents were interviewed about their family and peer relationships, and were then seated at a computer to complete the affect attribution task and self-report questionnaires about the childhood family environment described above via MediaLab software, version 2008.1.22 (Jarvis, 2008). Parents answered demographic questions about the number of bedrooms and people in the home across the child's lifetime. Blood samples were collected through antecubital venipuncture into lithium-heparin Vacutainers (Becton-Dickinson, Oakville, Ontario, Canada). This study was approved by the Clinical Research Ethics Board of the University of British Columbia.

Analytical Method

First, we examined the individual main effects of early life SES, implicit negative family affect, and implicit family warmth on metabolic outcomes. Next, multiple regression analyses were conducted to investigate whether adolescents' implicit affect toward the early family environment interacted with early life SES to predict metabolic outcomes. Implicit anger and fear were aggregated to create one dimension of implicit negative affect as done in previous studies (Chan et al., 2011). Following the procedures recommended by Aiken and West (1991), implicit negative affect, implicit warmth, and early life SES were mean-centered and cross products were computed. The covariates of age, gender, and ethnicity were included. We did not include smoking as a covariate because only 7% of the 245 adolescents who answered questions about smoking indicated they smoked one or more cigarettes in the last 6 months, and only one indicated they smoked on a daily basis.

Results

Preliminary Results

Descriptive information is listed in Table 1. Adolescents attributed significantly less anger and fear after seeing their own early childhood family photograph compared with the control image ($M_{diff} > |11.897|$, $ts > |6.982|$, $ps < .001$). Adolescents also attributed significantly more warmth after a family photograph compared to the control image ($M_{diff} = 26.58577$, $t = 12.146$, $p < .001$). Implicit negative family affect was significantly correlated with implicit family warmth ($r = -.328$, $p < .001$). Early life SES was not significantly correlated with implicit negative affect ($r = -.063$, $p = .314$) or implicit warmth ($r = .077$, $p = .220$). Implicit family affect dimensions were not significantly correlated with the RF measure or ratings of current family and friend relationships ($rs < |.097|$, $ps > .121$). Metabolic indicator scores largely reflect variation within a normal range, as these adolescents were a young and healthy sample. Multiple regression results for the individual main effects of early life SES, implicit negative affect, and implicit warmth on metabolic outcomes, controlling for the covariates of age, gender, and ethnicity are presented in Table 2.

Early Life SES and Implicit Negative Affect

Resting blood pressure. With respect to systolic blood pressure (SBP), there was no main effect of early life SES on SBP ($\beta = .112$, $p = .110$). There was no main effect of implicit negative affect on SBP ($\beta = .047$, $p = .441$). However, there was a significant interaction between early life SES and implicit negative affect in predicting SBP ($\beta = -.136$, $p = .043$). This interaction indicated that resting SBP levels were lower for those from a higher early life SES background who also attributed less implicit negative affect toward their early family environment. Among those from a higher early life SES background, as implicit negative affect increased, SBP levels also increased (see Figure 1). This pattern holds even when controlling for waist circumference ($\beta = -.120$, $p = .061$). Simple slopes for the association between SES and SBP were tested for low (−1 *SD* below the mean), moderate (mean), and high (+1 *SD* above the mean) levels of implicit negative affect (Aiken & West, 1991). Among those with lower implicit negative affect, the traditional SES-health relationship was apparent, where higher early life SES was associated with lower SBP ($\beta = 3.384$, $p = .001$). In contrast, among those with higher implicit negative affect, implicit negative affect appears to trump SES, and there was no association between SES and SBP ($\beta = .390$, $p = .790$).

With respect to diastolic blood pressure (DBP), there was no main effect of early life SES on DBP ($\beta = .059$, $p = .404$). There was no main effect of implicit negative affect on DBP ($\beta = .061$, $p = .322$). However, there was a significant interaction between early life SES and implicit negative affect in predicting DBP ($\beta = -.172$, $p = .011$; see Figure 1). This interaction indicated that resting DBP levels were again lower for those from a higher early life SES background who also attributed lower implicit negative affect toward their early family environment. This pattern holds even when controlling for waist circumference ($\beta = -.170$, $p = .013$). Simple slope analyses (Aiken & West, 1991) revealed similar patterns as SBP, such that among those with lower implicit negative affect, higher early life SES was associated with lower DBP ($\beta = 2.750$, $p = .005$). In contrast, among those with higher implicit negative affect, there was no association between SES and DBP ($\beta = -1.106$, $p = .440$).

Total cholesterol. There was no main effect of early life SES on total cholesterol ($\beta = -.064$, $p = .368$). There was a significant main effect of implicit negative affect on total cholesterol ($\beta = -.135$, $p = .031$). There was no interaction between early life SES and implicit negative affect in predicting total cholesterol ($\beta = -.068$, $p = .316$).

Glycosylated hemoglobin. Glycosylated hemoglobin levels were not significantly predicted by the main effects of early life SES, implicit negative affect, or the interactions between early life SES and implicit negative affect ($\beta s < |.050|$, $ps > .494$).

Waist circumference. Waist circumference was not significantly predicted by the main effects of early life SES, implicit negative affect, or the interactions between early life SES and implicit negative affect ($\beta s < |.075|$, $ps > .224$).

Early Life SES and Implicit Warmth

Resting blood pressure. With respect to SBP, there was a significant main effect of early life SES on SBP ($\beta = .168$, $p = .014$). This main effect indicated that as early life crowding (e.g., the number of people per bedroom in the household) increased, adolescents' resting SBP also increased. There was no main effect of implicit warmth on SBP ($\beta = -.033$, $p = .601$). There was no interaction between early life SES and implicit warmth in predicting SBP ($\beta = -.006$, $p = .925$).

Table 2
Multiple Regression Main Effect Results for Early Life Crowding, Implicit Negative Affect, and Implicit Warmth Predicting Metabolic Outcomes, Controlling for the Covariates of Age, Gender, and Ethnicity

Dependent variable	β	*t*	*p*
Early life crowding			
Resting SBP (mmHg)	.163	2.513	.013
Resting DBP (mmHg)	.123	1.873	.062
Total cholesterol (mmol/L)	−.029	−.435	.664
HbA1c (mmols/mol)	.066	.988	.324
Waist (cm)	−.037	−.572	.568
Implicit negative affect			
Resting SBP (mmHg)	.036	.577	.565
Resting DBP (mmHg)	.048	.767	.444
Total cholesterol (mmol/L)	−.135	−2.172	.031
HbA1c (mmols/mol)	.028	.442	.659
Waist (cm)	−.076	−1.239	.216
Implicit warmth			
Resting SBP (mmHg)	−.017	−.281	.779
Resting DBP (mmHg)	−.110	−1.758	.080
Total cholesterol (mmol/L)	−.042	−.672	.502
HbA1c (mmols/mol)	−.084	−1.318	.189
Waist (cm)	.103	1.681	.094

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; HbA1c = glycosylated hemoglobin.

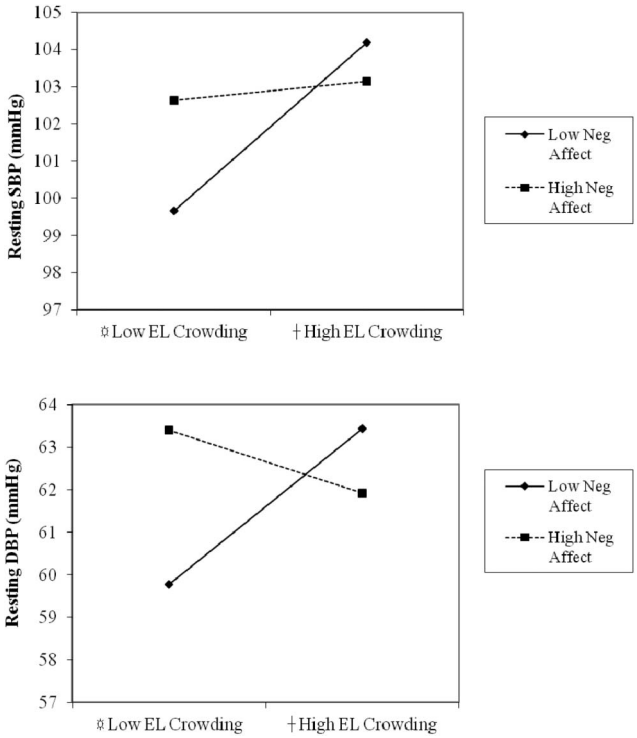

Figure 1. Interaction of early life (EL) socioeconomic status (SES; crowding) with implicit negative affect toward family predicting resting systolic blood pressure (SBP) and diastolic blood pressure (DBP) levels. The solid lines represents low implicit negative affect (−1 *SD*) and the dotted lines represents high implicit negative affect (+1 *SD*). Low EL crowding is −1 *SD* and high EL crowding is +1 *SD*.

With respect to DBP, there was a trend main effect of early life SES on DBP ($\beta = .119$, $p = .084$). There was also a significant main effect of implicit warmth on DBP ($\beta = -.128$, $p = .043$). This main effect indicated that as attribution of implicit warmth toward their early family environment increased, adolescents' resting DBP decreased. There was no interaction between early life SES and implicit warmth in predicting DBP ($\beta = .057$, $p = .383$).

Total cholesterol. There was no main effect of early life SES on total cholesterol ($\beta = -.061$, $p = .375$). There was no main effect of implicit warmth on total cholesterol ($\beta = -.054$, $p = .393$). There was a trending interaction between early life SES and implicit warmth in predicting total cholesterol ($\beta = .127$ $p = .054$). This pattern holds even when controlling for waist circumference ($\beta = .127$, $p = .054$). Similar to the above interactions, among those from a higher early life SES background, as warmth decreased, total cholesterol levels increased. Simple slope analyses (Aiken & West, 1991) did not reveal significant patterns at low (−1 *SD* below the mean), moderate (mean), and high (+1 *SD* above the mean) levels of warmth (βs < -1.106, ps $> .098$).

Glycosylated hemoglobin. Glycosylated hemoglobin levels were not significantly predicted by the main effects of early life SES, implicit warmth, or the interactions between early life SES and implicit warmth (βs $< |.094|$, ps $> .145$).

Waist circumference. Waist circumference was not significantly predicted by the main effects of early life SES, implicit warmth, or the interactions between early life SES and implicit warmth (βs $< |.108|$, ps $> .081$).

Alternative Explanations

Current SES. To test whether effects were specific to the early life period, or whether early life SES might be serving as a proxy for current family SES, we included current crowding as a covariate in all Early Life SES × Implicit Affect interaction analyses. All patterns remained the same (ps for interactions reported above range from .012 to .058). These results suggest that early life SES is not merely a proxy for current family SES.

To test whether effects were specific to early life SES, we also conducted interaction analyses with current SES and implicit family affect predicting metabolic outcomes. Current SES did not significantly interact with implicit negative affect or warmth to predict any outcomes of resting blood pressure, total cholesterol, glycosylated hemoglobin, or waist circumference (βs $< |.116|$, ps $> .073$). Thus, early life SES, but not current SES, interacts with an implicit measure of childhood family environment predicting adolescent metabolic outcomes.

Self-reported childhood family environment. To test whether findings were specific to implicit family environment measures or whether implicit measures may have merely served as a proxy for explicit family measures, we included the Risky Families measure as a covariate in all early life SES and implicit affect interaction analyses. All patterns remained the same (ps for interactions reported above range from .009 to .060), indicating that implicit family affect measures contribute to the interaction patterns uniquely above the contribution of explicit family measures.

To further test whether effects were specific to implicit measures, we also conducted interaction analyses with early life SES and the explicit Risky Families measure predicting metabolic outcomes. Early life SES did not significantly interact with the explicit RF measure to predict any outcomes of resting blood pressure, total cholesterol, glycosylated hemoglobin, or waist circumference (βs $< |.100|$, ps $> .101$). Thus, early life SES interacts with an implicit measure of childhood family environment, but not with an explicit measure of childhood family environment, in predicting adolescent metabolic outcomes.

Current family and friend relationships. Finally, to rule out the possibility that implicit affect toward the early family environment is simply a proxy for current explicit relationship quality, we included the current ratings of family and friendship relationship quality as a covariate in all early life SES and implicit affect interaction analyses. All patterns remained the same (ps for interactions reported above range from .009 to .057).

To test whether effects were specific to early childhood relationships or also emerged for current relationships, we conducted interaction analyses with early life SES and current ratings of family and friendship relationship quality predicting metabolic outcomes. Early life SES did not significantly interact with ratings of current family or current friendship relationship quality to predict resting blood pressure, total cholesterol, glycosylated hemoglobin, or waist circumference (βs $< |.093|$, ps $> .148$). This suggests that interactions of early life SES with early life implicit family affect are not better accounted for by current relationship ratings in either the family or friend domains.

Discussion

In a sample of healthy adolescents, we used a computerized affect attribution paradigm to capture implicit negative affect (e.g., anger, fear) and warmth toward the early family environment. We found that early life SES interacted with implicit affect toward family to predict blood pressure and cholesterol; however, contrary to our initial hypothesis, positive implicit family affect did not act as a buffer for lower SES adolescents. Rather, for those adolescents from a higher early life SES environment as implicit negative affect toward the early family environment increased, resting SBP and DBP levels also increased. Similarly, we also documented interactions between early life SES and implicit warmth toward the early family environment in total cholesterol levels, in a similar direction to BP. Furthermore, in testing alternative explanations, we demonstrated that these patterns held even when controlling for current SES and an explicit measure of the childhood family environment. Last, we did not observe the same interaction effects when using current SES in lieu of early life SES, when using an explicit, self-report measure of the childhood family environment, or when using ratings of current family or friend relationships in lieu of implicit family affect. Taken together, these patterns suggest that there is something specific to early life SES and to how adolescents implicitly feel about their early family environments that contribute to adolescent metabolic outcomes.

Although the findings from this study were inconsistent with our initial predictions about buffering effects for lower SES adolescents and with some previous studies (Chen et al., 2011; Evans et al., 2007; Miller et al., 2011b), there are several studies that have demonstrated moderation where effects are more pronounced in a higher SES or higher status group. For example, in a previous cognitive development study, SES was found to interact with the quality of the childhood home environment (e.g., availability of stimulation and support to a child), such that for higher SES children, an enriched home environment played a bigger role in boosting performance on cognitive development tests from Age 1 to Age 3, relative to lower SES children (Bradley et al., 1989). Further, physiological responses to an acute stressor are sometimes more pronounced in higher status individuals. Individuals who have higher subjective social status were the ones who showed the greatest adrenocortical response to a social evaluative stressor task in a controlled lab setting (Gruenewald et al., 2006). Youth who have higher subjective social status also display more pronounced inflammatory signaling following a recent targeted rejection life event compared with their lower status counterparts, and it has been speculated that higher status individuals are physiologically more sensitive to certain social stressors because of the need to defend their status (Murphy et al., 2013). Similarly, it may be that adolescents with higher SES are more sensitive to the effects that implicitly held (but not necessarily explicitly expressed) family affect has on metabolic processes. Consistent with this idea, in our previous study conducted in an adult sample (Chan et al., 2011), the interactions we found between early life SES and implicit family affect suggested that the effects of implicit affect were also stronger among those who are higher in SES, such that among those who were higher in early life SES, more negative implicit family affect was associated with higher BP levels. We speculate that one reason for this stronger effect in higher SES families is that negative implicit family affect may be more detrimental if adolescents from higher status communities have less broad social networks than adolescents from lower status communities. Previous research finds that individuals tend to form ties with peers of similar status and there are fewer individuals in each status group as status increases (McPherson, Smith-Lovin, & Cook, 2001). If higher status adolescents have fewer alternative network members to go to for support, the impact of negative implicit affect in the family may be stronger in this group.

Findings from simple slope analyses at different levels of implicit negative family affect revealed that the traditional association between early life SES and resting BP is only apparent among those adolescents with lower implicit negative family affect. In contrast, at higher levels of implicit negative family affect, no association was observed between early life SES and resting BP. This suggests that the expected benefits of higher SES on resting BP are only apparent when adolescents have less underlying implicit negative affect about their early environment and perhaps come from a less adverse early childhood family environment, while early life SES plays a smaller role in resting BP levels when adolescents have greater implicit negative family affect. This provides further evidence pointing to the role of adverse family characteristics in children's physical health (Repetti et al., 2002) and also points to the importance of considering both objective circumstances and subjective experiences (Greenwald et al., 2009) when assessing childhood psychosocial conditions.

It is interesting to note that the interactions patterns observed were different across metabolic outcomes. The effects of implicit negative family affect were observed for resting BP, and the effects of implicit warmth was observed for cholesterol levels. In line with previous studies documenting links between negative psychosocial factors (i.e., social threat) and physiological reactivity (Kemeny, 2009), we cautiously speculate that early childhood family relationship characteristics more closely related to the construct of social threat (i.e., implicit negative family affect) operates by altering autonomic processes more reactive to the immediate environment (Wager et al., 2009), and which would be expected to be associated with blood pressure. On the other hand, supportive early childhood family relationship characteristics (i.e., implicit family warmth) may contribute to cholesterol profiles via other pathways, such as the promotion of positive health behaviors (Uchino, 2006). However, these explanations are quite speculative, and further examinations of the specific physiological processes associated with the different implicit affect dimensions are warranted.

Finally, interaction patterns documented in this study were specific to early life SES and implicit family affect. We did not observe the same interaction effects when using current SES in lieu of early life SES. This finding adds to a growing literature documenting that early life represents a sensitive period that may have long-lasting effects on physiological systems (Blane et al., 1997; Cohen, Janicki-Deverts, Chen, & Matthews, 2010b; Galobardes et al., 2004, 2006; Miller, Chen, & Parker, 2011a; Poulton et al., 2002). Also, we did not observe the same interaction effects when using an explicit self-report measure of the childhood environment. This may be because the explicit measure tapped relationship quality items, whereas the implicit measure tapped affect. Or it may be because family moderation of SES effects in adolescence are more apparent for implicit than explicit measures (perhaps because adolescents are still living at home, and early child-

hood affect toward family members is more difficult to explicitly report on and separate out from current feelings during adolescence, relative to adults who are no longer living with their parents). Overall, these differing patterns point to the importance of measuring early childhood characteristics using tools other than self-report assessments, as implicit measures can capture more sensitive and sometimes unacknowledged feelings that are different from explicit measures (Greenwald et al., 2002). This pattern is consistent with previous social psychology work demonstrating that associations with implicit measures are different from associations with explicit measures (Greenwald et al., 2009). Our findings reveal that the current quality of family and friend relationships do not produce the same patterns as our implicit measure of early childhood family environment, suggesting that the implicit measure is not just a proxy for the state of one's current interpersonal relationships.

Limitations

There are several limitations to this study. First, findings were cross-sectional, and longitudinal studies are needed to determine whether implicit family warmth and negative affect truly precede change in metabolic or other physiologic processes. Second, our sample included healthy young participants. Future studies should conduct a broader assessment of other physiological pathways and disease outcomes across a diverse range of ages throughout the life span. Third, we are unable to directly verify that participant's implicit responses to family photos were specific to the early childhood period. Although we did rule out the idea that implicit family affect is simply a proxy for the explicit quality of current family relationships, it is possible that the measure also captures some spillover of current affect toward family. Fourth, given the multiple tests conducted, results would need to be replicated across other studies to be certain of the reliability of the findings. Fifth, although automatic affective responses are less explicitly biased than self-report measures, they are still retrospective measures, and where possible, prospective studies starting in early childhood would greatly help to shed light on family psychosocial moderating factors. Last, this study sample also did not focus on lower SES participants, and it is possible that the associations between implicit family affect and SES on adolescent metabolic outcomes may operate differently in a lower income sample. Given the mechanistic nature of this study, the sample was a convenience sample, rather than a population-based sample. While the general characteristics of this study's sample were in line with the broader community from which it was drawn, nonetheless, generalizability remains more of a question in smaller-scale, mechanistic studies compared to larger, epidemiological studies.

Conclusion

In a sample of healthy adolescents, implicit affect elicited by early family conditions interacted with a measure of early life SES to predict metabolic outcomes. The moderating effect of implicit family affect was specific to individuals higher in early life SES, such that higher SES individuals who had more implicit negative affect and less implicit warmth toward their early family environment displayed higher resting BP and cholesterol levels. Our findings also indicate that implicit characteristics about early childhood family relationships are specifically relevant to early life SES and not current SES. Implicit characteristics, but not explicitly reported characteristics, appear to be in some cases able to potentially override the effects of positive early life socioeconomic conditions on adolescents' resting SBP, DBP, and total cholesterol. These findings highlight the potential importance of incorporating implicit measures together with explicit measures when assessing early life factors and suggest that underlying implicit negative affect toward one's family may offset the benefits of material resources that higher SES children experience.

References

Adrian, C., & Hammen, C. (1993). Stress exposure and stress generation in children of depressed mothers. *Journal of Consulting and Clinical Psychology, 61,* 354–359.

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting Interactions*. Newbury, CA: SAGE.

Bates, T. C., Lewis, G. J., & Weiss, A. (2013). Childhood socioeconomic status amplifies genetic effects on adult intelligence. *Psychological Science, 24,* 2111–2116. http://dx.doi.org/10.1177/0956797613488394

Berenson, G. S., Wattigney, W. A., Tracy, R. E., Newman, W. P., III, Srinivasan, S. R., Webber, L. S., . . . Strong, J. P. (1992). Atherosclerosis of the aorta and coronary arteries and cardiovascular risk factors in persons aged 6 to 30 years and studied at necropsy (The Bogalusa Heart Study). *The American Journal of Cardiology, 70,* 851–858. http://dx.doi.org/10.1016/0002-9149(92)90726-F

Blane, D., Bartley, M., & Smith, G. D. (1997). Disease aetiology and materialist explanations of socioeconomic mortality differentials. *European Journal of Public Health, 7,* 385–391. http://dx.doi.org/10.1093/eurpub/7.4.385

Bradley, R. H., Caldwell, B. M., Rock, S. L., Ramey, C. T., Barnard, K. E., Gray, C., . . . Johnson, D. L. (1989). Home environment and cognitive development in the first 3 years of life: A collaborative study involving six sites and three ethnic groups in North America. *Developmental Psychology, 25,* 217–235. http://dx.doi.org/10.1037/0012-1649.25.2.217

Brunner, E. J. (1997). An empirical test of neutrality and the crowding-out hypothesis. *Public Choice, 92*(3/4), 261–279. http://dx.doi.org/10.1023/A:1017946112377

Chan, M., Chen, E., Hibbert, A. S., Wong, J. H. K., & Miller, G. E. (2011). Implicit measures of early-life family conditions: Relationships to psychosocial characteristics and cardiovascular disease risk in adulthood. *Health Psychology, 30,* 570–578. http://dx.doi.org/10.1037/a0024210

Chen, E., Fisher, E. B., Bacharier, L. B., & Strunk, R. C. (2003). Socioeconomic status, stress, and immune markers in adolescents with asthma. *Psychosomatic Medicine, 65,* 984–992. http://dx.doi.org/10.1097/01.PSY.0000097340.54195.3C

Chen, E., & Miller, G. E. (2012). "Shift-and-persist" strategies: Why low socioeconomic status isn't always bad for health. *Perspectives on Psychological Science, 7,* 135–158. http://dx.doi.org/10.1177/1745691612436694

Chen, E., Miller, G. E., Kobor, M. S., & Cole, S. W. (2011). Maternal warmth buffers the effects of low early-life socioeconomic status on pro-inflammatory signaling in adulthood. *Molecular Psychiatry, 16,* 729–737. http://dx.doi.org/10.1038/mp.2010.53

Cicchetti, D., & Blender, J. A. (2006). A multiple-levels-of-analysis perspective on resilience: Implications for the developing brain, neural plasticity, and preventive interventions. *Annals of the New York Academy of Sciences, 1094,* 248–258. http://dx.doi.org/10.1196/annals.1376.029

Coggon, D., Barker, D. J. P., Inskip, H., & Wield, G. (1993). Housing in early life and later mortality. *Journal of Epidemiology and Community Health, 47,* 345–348. http://dx.doi.org/10.1136/jech.47.5.345

Cohen, S., Doyle, W. J., Turner, R. B., Alper, C. M., & Skoner, D. P. (2004). Childhood socioeconomic status and host resistance to infectious illness in adulthood. *Psychosomatic Medicine, 66,* 553–558. http://dx.doi.org/10.1097/01.psy.0000126200.05189.d3

Cohen, S., Janicki-Deverts, D., Chen, E., & Matthews, K. A. (2010). Childhood socioeconomic status and adult health. *Annals of the New York Academy of Sciences, 1186,* 37–55. http://dx.doi.org/10.1111/j.1749-6632.2009.05334.x

Cunningham, W. A., Preacher, K. J., & Banaji, M. R. (2001). Implicit attitude measures: Consistency, stability, and convergent validity. *Psychological Science, 12,* 163–170. http://dx.doi.org/10.1111/1467-9280.00328

Danese, A., Moffitt, T. E., Harrington, H., Milne, B. J., Polanczyk, G., Pariante, C. M., . . . Caspi, A. (2009). Adverse childhood experiences and adult risk factors for age-related disease: Depression, inflammation, and clustering of metabolic risk markers. *Archives of Pediatrics & Adolescent Medicine, 163,* 1135–1143. http://dx.doi.org/10.1001/archpediatrics.2009.214

De Houwer, J., Teige-Mocigemba, S., Spruyt, A., & Moors, A. (2009). Implicit measures: A normative analysis and review. *Psychological Bulletin, 135,* 347–368. http://dx.doi.org/10.1037/a0014211

Dodge, K. A., Pettit, G. S., & Bates, J. E. (1994). Socialization mediators of the relation between socioeconomic status and child conduct problems. *Child Development, 65,* 649–665. http://dx.doi.org/10.2307/1131407

Emery, R. E., & Laumann-Billings, L. (1998). An overview of the nature, causes, and consequences of abusive family relationships. Toward differentiating maltreatment and violence. *American Psychologist, 53,* 121–135. http://dx.doi.org/10.1037/0003-066X.53.2.121

Evans, G. W., Kim, P., Ting, A. H., Tesher, H. B., & Shannis, D. (2007). Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Developmental Psychology, 43,* 341–351. http://dx.doi.org/10.1037/0012-1649.43.2.341

Evans, G. W., & Lepore, S. J. (1992). Conceptual and analytic issues in crowding research. *Journal of Environmental Psychology, 12,* 163–173. http://dx.doi.org/10.1016/S0272-4944(05)80068-4

Fazio, R. H., Jackson, J. R., Dunton, B. C., & Williams, C. J. (1995). Variability in automatic activation as an unobtrusive measure of racial attitudes: A bona fide pipeline? *Journal of Personality and Social Psychology, 69,* 1013–1027. http://dx.doi.org/10.1037/0022-3514.69.6.1013

Fazio, R. H., & Olson, M. A. (2003). Implicit measures in social cognition. research: Their meaning and use. *Annual Review of Psychology, 54,* 297–327. http://dx.doi.org/10.1146/annurev.psych.54.101601.145225

Galobardes, B., Lynch, J. W., & Davey Smith, G. (2004). Childhood socioeconomic circumstances and cause-specific mortality in adulthood: Systematic review and interpretation. *Epidemiologic Reviews, 26,* 7–21. http://dx.doi.org/10.1093/epirev/mxh008

Galobardes, B., Smith, G. D., & Lynch, J. W. (2006). Systematic review of the influence of childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood. *Annals of Epidemiology, 16,* 91–104. http://dx.doi.org/10.1016/j.annepidem.2005.06.053

Gawronski, B., & Bodenhausen, G. V. (2006). Associative and propositional processes in evaluation: An integrative review of implicit and explicit attitude change. *Psychological Bulletin, 132,* 692–731. http://dx.doi.org/10.1037/0033-2909.132.5.692

Greenwald, A. G., Banaji, M. R., Rudman, L. A., Farnham, S. D., Nosek, B. A., & Mellott, D. S. (2002). A unified theory of implicit attitudes, stereotypes, self-esteem, and self-concept. *Psychological Review, 109,* 3–25. http://dx.doi.org/10.1037/0033-295X.109.1.3

Greenwald, A. G., McGhee, D. E., & Schwartz, J. L. K. (1998). Measuring individual differences in implicit cognition: The implicit association test. *Journal of Personality and Social Psychology, 74,* 1464–1480. http://dx.doi.org/10.1037/0022-3514.74.6.1464

Greenwald, A. G., Poehlman, T. A., Uhlmann, E. L., & Banaji, M. R. (2009). Understanding and using the Implicit Association Test: III. Meta-analysis of predictive validity. *Journal of Personality and Social Psychology, 97,* 17–41. http://dx.doi.org/10.1037/a0015575

Gruenewald, T. L., Kemeny, M. E., & Aziz, N. (2006). Subjective social status moderates cortisol responses to social threat. *Brain, Behavior, and Immunity, 20,* 410–419. http://dx.doi.org/10.1016/j.bbi.2005.11.005

Gunnar, M., & Quevedo, K. (2005). The neurobiology of stress and development. *Annual Review of Psychology, 58,* 145–173. New York, NY: Oxford University Press.

Hammen, C. (1991). Generation of stress in the course of unipolar depression. *Journal of Abnormal Psychology, 100,* 555–561. http://dx.doi.org/10.1037/0021-843X.100.4.555

Hammen, C., Adrian, C., & Hiroto, D. (1988). A longitudinal test of the attributional vulnerability model in children at risk for depression. *The British Journal of Clinical Psychology, 27*(Pt 1), 37–46. http://dx.doi.org/10.1111/j.2044-8260.1988.tb00751.x

Hardt, J., & Rutter, M. (2004). Validity of adult retrospective reports of adverse childhood experiences: Review of the evidence. *Journal of Child Psychology and Psychiatry, and Allied Disciplines, 45,* 260–273. http://dx.doi.org/10.1111/j.1469-7610.2004.00218.x

Jarvis, B. G. (2008). MediaLab (Version 2008.1.22) [Computer Software]. New York, NY: Empirisoft Corporation.

Kemeny, M. E. (2009). Psychobiological responses to social threat: Evolution of a psychological model in psychoneuroimmunology. *Brain, Behavior, and Immunity, 23,* 1–9. http://dx.doi.org/10.1016/j.bbi.2008.08.008

Kittleson, M. M., Meoni, L. A., Wang, N.-Y., Chu, A. Y., Ford, D. E., & Klag, M. J. (2006). Association of childhood socioeconomic status with subsequent coronary heart disease in physicians. *Archives of Internal Medicine, 166,* 2356–2361. http://dx.doi.org/10.1001/archinte.166.21.2356

Kuh, D., Hardy, R., Langenberg, C., Richards, M., & Wadsworth, M. E. (2002). Mortality in adults aged 26–54 years related to socioeconomic conditions in childhood and adulthood: Post war birth cohort study. *British Medical Journal, 325,* 1076–1080. http://dx.doi.org/10.1136/bmj.325.7372.1076

Lakka, H.-M., Laaksonen, D. E., Lakka, T. A., Niskanen, L. K., Kumpusalo, E., Tuomilehto, J., & Salonen, J. T. (2002). The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. *Journal of the American Medical Association, 288,* 2709–2716. http://dx.doi.org/10.1001/jama.288.21.2709

Lawlor, D. A., Sterne, J. A. C., Tynelius, P., Davey Smith, G., & Rasmussen, F. (2006). Association of childhood socioeconomic position with cause-specific mortality in a prospective record linkage study of 1,839,384 individuals. *American Journal of Epidemiology, 164,* 907–915. http://dx.doi.org/10.1093/aje/kwj319

Masten, A. S., & Shaffer, A. (2006). How families matter in child development: Reflections from research on risk and resilience. In A. Clarke-Stewart & J. Dunn (Eds.), *Families count: Effects on child and adolescent development* (pp. 5–25). New York, NY: Cambridge University Press. http://dx.doi.org/10.1017/CBO9780511616259.002

Mattu, G. S., Heran, B. S., & Wright, J. M. (2004). Overall accuracy of the BpTRU—An automated electronic blood pressure device. *Blood Pressure Monitoring, 9,* 47–52. http://dx.doi.org/10.1097/00126097-200402000-00009

McPherson, M., Smith-Lovin, L., & Cook, J. M. (2001). Birds of a feather: Homophily in Social Networks. *Annual Review of Sociology, 27,* 415–444. http://dx.doi.org/10.1146/annurev.soc.27.1.415

Metts, S., Sprecher, S., & Cupach, W. R. (1993). Retrospective self-reports. In B. M. Montgomery & S. Duck (Eds.), *Studying interpersonal interaction* (p. 346). New York, NY: Guilford Press.

Miller, G., & Chen, E. (2007). Unfavorable socioeconomic conditions in early life presage expression of proinflammatory phenotype in adoles-

cence. *Psychosomatic Medicine, 69,* 402–409. http://dx.doi.org/10.1097/PSY.0b013e318068fcf9

Miller, G. E., Chen, E., & Parker, K. J. (2011a). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin, 137,* 959–997. http://dx.doi.org/10.1037/a0024768

Miller, G. E., Lachman, M. E., Chen, E., Gruenewald, T. L., Karlamangla, A. S., & Seeman, T. E. (2011b). Pathways to resilience: Maternal nurturance as a buffer against the effects of childhood poverty on metabolic syndrome at midlife. *Psychological Science, 22,* 1591–1599. http://dx.doi.org/10.1177/0956797611419170

Morrison, J. A., Friedman, L. A., & Gray-McGuire, C. (2007). Metabolic syndrome in childhood predicts adult cardiovascular disease 25 years later: The Princeton Lipid Research Clinics Follow-up Study. *Pediatrics, 120,* 340–345. http://dx.doi.org/10.1542/peds.2006-1699

Murphy, M. L. M., Slavich, G. M., Rohleder, N., & Miller, G. E. (2013). Targeted rejection triggers differential pro- and anti-inflammatory gene expression in adolescents as a function of social status. *Clinical Psychological Science, 1,* 30–40. http://dx.doi.org/10.1177/2167702612455743

Nazmi, A., Oliveira, I. O., Horta, B. L., Gigante, D. P., & Victora, C. G. (2010). Lifecourse socioeconomic trajectories and C-reactive protein levels in young adults: Findings from a Brazilian birth cohort. *Social Science & Medicine, 70,* 1229–1236. http://dx.doi.org/10.1016/j.socscimed.2009.12.014

Pettit, G. S., Bates, J. E., & Dodge, K. A. (1997). Supportive parenting, ecological context, and children's adjustment: A seven-year longitudinal study. *Child Development, 68,* 908–923.

Poulton, R., Caspi, A., Milne, B. J., Thomson, W. M., Taylor, A., Sears, M. R., & Moffitt, T. E. (2002). Association between children's experience of socioeconomic disadvantage and adult health: A life-course study. *Lancet, 360,* 1640–1645. http://dx.doi.org/10.1016/S0140-6736(02)11602-3

Power, C., Atherton, K., Strachan, D. P., Shepherd, P., Fuller, E., Davis, A., . . . Stansfeld, S. (2007). Life-course influences on health in British adults: Effects of socio-economic position in childhood and adulthood. *International Journal of Epidemiology, 36,* 532–539. http://dx.doi.org/10.1093/ije/dyl310

Power, C., Manor, O., & Matthews, S. (1999). The duration and timing of exposure: Effects of socioeconomic environment on adult health. *American Journal of Public Health, 89,* 1059–1065. http://dx.doi.org/10.2105/AJPH.89.7.1059

Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin, 128,* 330–366. http://dx.doi.org/10.1037/0033-2909.128.2.330

Ridker, P. M., Buring, J. E., Cook, N. R., & Rifai, N. (2003). C-reactive protein, the metabolic syndrome, and risk of incident cardiovascular events: An 8-year follow-up of 14,719 initially healthy American women. *Circulation, 107,* 391–397. http://dx.doi.org/10.1161/01.CIR.0000055014.62083.05

Rudolph, K. D., & Hammen, C. (1999). Age and gender as determinants of stress exposure, generation, and reactions in youngsters: A transactional perspective. *Child Development, 70,* 660–677.

Statistics Canada. Table 111-0009 - Family characteristics, summary, annual (number unless otherwise noted), CANSIM (database).

Strong, J. P., Malcom, G. T., McMahan, C. A., Tracy, R. E., Newman, W. P., III, Herderick, E. E., & Cornhill, J. F. (1999). Prevalence and extent of atherosclerosis in adolescents and young adults: Implications for prevention from the pathobiological determinants of atherosclerosis in youth study. *Journal of the American Medical Association, 281,* 727–735. http://dx.doi.org/10.1001/jama.281.8.727

Taylor, S. E., Lerner, J. S., Sage, R. M., Lehman, B. J., & Seeman, T. E. (2004). Early environment, emotions, responses to stress, and health. *Journal of Personality, 72,* 1365–1393. http://dx.doi.org/10.1111/j.1467-6494.2004.00300.x

Uchino, B. N. (2006). Social support and health: A review of physiological processes potentially underlying links to disease outcomes. *Journal of Behavioral Medicine, 29,* 377–387. http://dx.doi.org/10.1007/s10865-006-9056-5

Wager, T. D., van Ast, V. A., Hughes, B. L., Davidson, M. L., Lindquist, M. A., & Ochsner, K. N. (2009). Brain mediators of cardiovascular responses to social threat, part II: Prefrontal-subcortical pathways and relationship with anxiety. *NeuroImage, 47,* 836–851. http://dx.doi.org/10.1016/j.neuroimage.2009.05.044

Weitzman, M., Gortmaker, S. L., Sobol, A. M., & Perrin, J. M. (1992). Recent trends in the prevalence and severity of childhood asthma. *Journal of the American Medical Association, 268,* 2673–2677. http://dx.doi.org/10.1001/jama.1992.03490190073034

Wittenbrink, B., Judd, C. M., & Park, B. (1997). Evidence for racial prejudice at the implicit level and its relationship with questionnaire measures. *Journal of Personality and Social Psychology, 72,* 262–274. http://dx.doi.org/10.1037/0022-3514.72.2.262

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