



Published in final edited form as:

Ann Behav Med. 2017 April ; 51(2): 189–198. doi:10.1007/s12160-016-9842-4.

Attachment Orientations, Respiratory Sinus Arrhythmia, and Stress are Important for Understanding the Link between Childhood Socioeconomic Status and Adult Self-Reported Health

Kyle W. Murdock, PhD¹ and Christopher P. Fagundes, PhD^{1,2,3}

¹Department of Psychology, Rice University

²Department of Symptom Research, The University of Texas MD Anderson Cancer Center

³Department of Psychiatry, Baylor College of Medicine

Abstract

Background—Low childhood SES is reliably associated with poor adult health. Social environments early in life and physiological stress responses are theorized to underlie this link; however, the role of attachment orientations is relatively unknown.

Purpose—In this study, we examined whether attachment orientations (i.e., attachment anxiety and attachment avoidance) and self-reported stress were mediators of the association between childhood SES and self-reported health in adulthood. Furthermore, we examined whether parasympathetic nervous system functioning was a moderator of associations between attachment orientations and self-reported stress.

Methods—Participants ($N = 213$) provided self-reports of childhood SES, attachment orientations, stress, and self-rated health. Respiratory sinus arrhythmia (RSA) was measured at rest, as well as during an acute social stressor.

Results—Low childhood SES was associated with poor self-reported health via the serial pathway from attachment anxiety to general stress. Moreover, attachment avoidance was associated with self-reported health via general stress, but only among those with high stress-induced RSA. Findings were independent of participant age, sex, race, body mass index, baseline RSA, and adult SES.

Conclusions—Attachment theory is useful for understanding why those from low SES backgrounds are at greater risk of negative health outcomes in adulthood. Findings extend our

Corresponding Author: Kyle W. Murdock, Rice University, 6100 Main Street, BRC 780Q, Houston, Texas 77005. kyle.w.murdock@rice.edu; Telephone: 713-348-8163; Fax: 713-348-5221.

Conflicts of interest: The authors do not have any conflicts of interest to declare.

Adherence to ethical standards: All authors have agreed upon the content and form of the manuscript. The manuscript is not under consideration for publication elsewhere, and the manuscript has not been published previously. All authors declare that there are no conflicts of interest. The authors have full control of all primary data and agree to allow the journal to review the data if requested. No data has been fabricated or manipulated to support conclusions. Consent to submit has been received explicitly from all co-authors, and all authors have contributed sufficiently to the manuscript and share accountability for the results.

knowledge of how interpersonal relationships in childhood can shape emotional and physical health outcomes in adulthood.

Keywords

attachment theory; respiratory sinus arrhythmia; stress; self-rated health; child development

Low early life socioeconomic status (SES) is reliably associated with poor adult health (1–4). Individuals from low early life SES backgrounds have shorter life spans in comparison to those from high early life SES backgrounds (5). Low early life SES is associated with an increased number of hospital visits each year in adulthood (6). Parasympathetic nervous system reactivity to stress is one hypothesized mechanism for the association between childhood SES and adult health (7). Developing a better understanding of the mechanisms underlying the association between childhood SES and adult health is important for prevention and treatment efforts. The present study examined how attachment orientations, parasympathetic nervous system functioning, and general stress may underlie the association between early life SES and self-reported health in adulthood.

The family stress (8) and risky families models (9) purport that early life social conditions lead to differential health outcomes. Consistent with the family stress and risky families models, low early life SES is associated with increased risk of mental and physical health problems in adulthood (10). In particular, harsh, inconsistent, and uninvolved parenting (often due to economic pressures) are highlighted as key mechanisms underlying the association between early life SES and poor well-being (11). Support for the role of harsh, inconsistent, and uninvolved parenting as a mechanism linking low childhood SES to poor well-being has been identified across a range of samples from diverse racial/ethnic backgrounds (11). For example, high SES parents tend to be more child centered and sensitive in their approach to parenting whereas low SES parents tend to be less sensitive and more authoritarian (12). As a result, children in low SES environments may be more likely develop insecure attachment orientations than children in high SES environments (13). Factors such as poor nutrition (14), heightened stress (15), and lack of social support (16) are factors that may enhance a child's vulnerability for being exposed to less sensitive parenting behaviors that promote attachment insecurity in low SES families. This is important given that low SES environments and insecure attachments promote unhealthy behaviors by continuing to affect the quality of parenting and other close relationships that are developed across the lifespan. Indeed, obesity (17) and engagement in risky health behaviors (e.g., smoking, lack of seatbelt use; 18) are more common among children and adults with insecure attachment orientations.

Attachment orientations (also referred to as attachment styles or patterns) were originally described by developmental psychologists as trait-like patterns of attachment that develop via the quality of one's relationship with their caregiver and the responsiveness of this caregiver to one's bids for attention (19). Infants were originally described as being secure or insecure in their attachment orientation (20), with insecure attachment being comprised of both anxious-avoidant and anxious-ambivalent attachment orientations (20). Children with an anxious-avoidant attachment orientation were described as being those who did not

receive sensitive care from their attachment figure and learned not to seek them when distressed. Children with an anxious-ambivalent attachment orientation were thought to receive inconsistent care from their attachment figure, which is more common among low SES families in comparison to high SES families (13). Children with sensitive and responsive caregivers were thought to view their attachment figure as distress-alleviating and a secure base, hence the term secure attachment.

Based on the pioneering early work by social psychologists and others interested in adult relationships, convincing evidence demonstrated that the same individual differences in attachment orientations identified in caregiver-child relationships also characterize adults' attachment orientations towards others in adulthood (21). A large literature has confirmed these predictions and psychometric research has demonstrated the benefits of adopting a two-dimensional model as opposed to a categorical model for measuring attachment orientations (22). One of the dimensions is attachment anxiety or the degree to which one is concerned about the availability and responsiveness of close others (22). The other dimension, attachment avoidance, reflects the extent to which one is uncomfortable using others for security (22). Those low on attachment anxiety and avoidance are labeled as secure. Social psychologists, and others interested in adult attachment have largely focused on assessing attachment orientations using this dimensional approach to close relationships.

Importantly, both theoretical traditions highlight the role of attachment orientations for emotion regulation and see early childhood experiences as the most important for establishing attachment orientations. Secure individuals are able to buffer stressful life events and the negative emotions they can generate by drawing upon their felt security (23, 24). Supportive early life environments promote effective emotion regulation later in life (25). Those with insecure attachment orientations tend to demonstrate poor emotion regulation, leading to increased risk of experiencing chronic stress (26). When chronic, stress can dysregulate immune functioning and promote disease (27). High attachment anxiety is associated with increased risk of immune and endocrine dysfunction (28), in addition to chronic pain, heart disease, stroke, asthma, bronchitis, emphysema, diabetes, and hypertension (29, 30). Accordingly, attachment theory is important for understanding the link between childhood SES and adult health outcomes.

Evidence for an association between attachment and health outcomes is consistent with findings indicating that attachment anxiety leads to enhanced stress responses. Indeed, attachment anxiety is associated with increased cortisol reactivity when discussing a relationship concern with one's partner (31). Individuals high in attachment anxiety and avoidance evidence greater increases in heart rate and blood pressure when confronted with a stressful situation if their partner is present, but not when their partner is absent (32). Furthermore, attachment anxiety is associated with self-reported distress during a stressful cognitive task, whereas attachment avoidance is associated with less parasympathetic nervous system activity (33).

Attachment avoidance is less reliably associated with stress and physical health problems than attachment anxiety. For example, attachment avoidance was not associated with cortisol production and cellular immunity (28,34), but was associated with subjective and behavioral

responses to stress (35). Furthermore, higher attachment avoidance was associated with greater inflammation following a marital conflict, but attachment avoidance was unrelated to inflammation after a social support interaction (36). Accordingly, there may be other factors that are important for understanding a potential association between attachment avoidance and health. For instance, attachment avoidance was associated with well-being via perceived social support, whereas attachment anxiety and well-being were directly associated (37). More broadly, high emotional support has also been found to reduce the negative impact of attachment insecurity on health (38), suggesting that one's emotional environment is important to consider when evaluating the association between attachment and health.

When avoidantly attached individuals experience stress, they often use suppression techniques that attempt to shift their attention away from unwanted thoughts (39). Experimental work indicates that suppression is associated with the ability to prevent reactivation of unwanted, and previously suppressed, thoughts among avoidant individuals in many circumstances; however, when under high cognitive load, avoidantly attached individuals are unable to suppress such thoughts (40). In other words, attachment avoidance is associated with self-regulatory failure when individuals are under high cognitive load (i.e., when one's working memory capacity is overextended; 41). Self-regulatory failure due to rumination is associated with increased psychological stress and depression (42,43). Moreover, suppression is associated with increased cardiovascular reactivity and hypertension risk (44), as well as inflammation (45). Such findings are consistent with prior work indicating that self-regulatory processes are important for understanding the association between attachment avoidance and well-being (46).

There is considerable theoretical work demonstrating that early life SES interacts with one's physiological capacity for coping with stress to predict mental and physical health outcomes throughout the lifespan (9, 47). However, relatively few empirical studies have evaluated how the synergistic relationship between attachment orientations and physiological stress is associated with mental and physical health outcomes. In such work, attachment avoidance, but not attachment anxiety, interacted with stress-induced respiratory sinus arrhythmia (RSA) to predict post-cancer treatment quality of life (48), and loss adjustment relationship (49,50).

RSA is the variability in heart rate that is due to respiration and is a marker of parasympathetic nervous system activity (51). RSA is a marker of one's self-regulatory capacity (also known as self-regulatory strength) or the degree to which one can utilize self-regulation before being susceptible to self-regulation failure (52). Normatively, RSA goes down when one is confronted with a stressor (52–55). According to the neurovisceral integration model and polyvagal theories, RSA is important for regulation of emotion (56,57), and high stress-induced RSA relative to baseline has been described as representing self-regulatory capacity (52). Given prior work showing that stress-induced RSA is important for understanding why only some individuals with high attachment avoidance are susceptible to stress and negative health outcomes (48–50), as well as work indicating that attachment anxiety is reliably associated with stress and health, we hypothesized that: (1) Individuals with low childhood SES would report greater attachment anxiety and avoidance in comparison to those with high childhood SES. (2) Greater attachment anxiety would be

serially associated with increased stress, and this serial association would underlie the association between low early life SES and poor adult health. (3) Stress-induced RSA would change the association between attachment avoidance and stress such that those with low attachment avoidance and high stress-induced RSA would report lower stress than those with high attachment avoidance and/or low stress-induced RSA. (4) Childhood SES would be indirectly associated with poor adult health through the attachment avoidance and stress only when the role of stress-induced RSA in changing the association between attachment avoidance and stress is included.

Methods

The data utilized for the present study was from the Pittsburgh Cold Study 3, which is publicly available and has been described previously (58,59). Healthy individuals ($N = 213$) from the Pittsburgh, Pennsylvania area were recruited to participate in the present study between 2008 and 2011. The study was approved by the Carnegie Mellon University and University of Pittsburgh institutional review boards, and all participants provided informed consent. As compensation for their time and efforts, participants were given \$1,000 for completing the full study protocol.

For the present study, participants completed self-report measures of childhood SES, attachment orientations, stress, and self-reported health. Furthermore, RSA was measured at rest and during engagement in the Trier Social Stress Test (TSST; 60). The TSST is a well validated stress paradigm in which participants were told that they would be delivering a five-minute speech in which they were to defend against an alleged transgression (i.e., shoplifting or a traffic violation). Participants were given five minutes to prepare before delivering a video recorded speech during which RSA was measured.

Measures

Childhood socioeconomic status—The MacArthur Scale of Subjective Social Status USA ladder version (61) was utilized to measure childhood and adult SES. In regards to childhood SES, participants were asked to separately indicate their mother's and father's social status on an illustration of a nine step ladder in which the top represents those with the most education, money, and respected jobs. Conversely, the bottom step of the ladder represents those with the least education, money, and respected jobs. Participants were asked to place an "X" on the ladder that best represented each parents' position. Scores range from 1 (lowest status) to 9 (highest status), and scores for maternal and paternal SES were averaged to form an overall indicator of childhood SES. No instructions were given about how to respond for those with more than two parents. All participants provided a response for their mothers' SES, while four participants did not provide a response for their fathers' SES. Participants were also asked to complete the measure in reference to their own SES as an adult, which was utilized as a covariate in the analyses described below. Acceptable psychometric characteristics have been identified using the Scale of Subjective Social Status (62). One's subjective social status is associated with objective indicators of SES such as income, educational attainment, and employment status (61,63).

Attachment orientations—The Experiences in Close Relationship Scale (ECR)-short form (64) was utilized to measure attachment orientations. Six-items measure each attachment dimension (i.e., attachment anxiety and attachment avoidance). Participants were asked to indicate the degree to which item (e.g., “I am very uncomfortable being close to people.”) is true for them on a scale from 1 (disagree strongly) to 7 (agree strongly). The original 36-item ECR was developed using a factor analysis of all items available from 14 self-report measures of attachment (42). Two dimensions were identified and labeled as anxiety and avoidance. There is a general consensus from experts in the field that attachment consists of these two dimensions (39). The ECR-short form has demonstrated similar reliability and construct and criterion validity as the original measure (64). Cronbach’s alpha was .83 for attachment avoidance and .89 for attachment anxiety in the present sample.

Respiratory sinus arrhythmia—RSA was recorded using a respiration band and three electrocardiogram leads (Vernier Software & Technology, Beaverton, OR). The baseline period lasted 20 minutes and participants were instructed to sit upright in a chair and rest quietly. An automated algorithm was utilized to record interbeat interval (IBI) sequences (Mindware Version 2.51, Mindware Technologies, LTD) and a 250 Hz sampling frequency (65). Measurement of RSA during baseline was separated into five minute epochs, which were averaged to form an overall indicator of baseline RSA. Spectral analysis of IBIs was conducted using a Fast Fourier transform algorithm (66). High frequency (HF) band power was calculated as the sum of the powers associated with any peaks in the range of 0.12 Hz to 0.40 Hz. An identical procedure was utilized to measure stress-induced RSA during the TSST, which consisted of a single five minute epoch. Valid baseline RSA data was obtained from 199 participants, whereas valid stress-induced RSA was measured from 190 participants.

General Stress—Participants completed the Perceived Stress Scale (PSS; 67). On the PSS, participants are asked to indicate the degree to which they have experienced 10 symptoms of general stress on a scale ranging from 0 (never) to 4 (very often). Cronbach’s alpha was .81 for the PSS in the present study.

Self-reported health—Participants were asked to indicate their general health on a scale ranging from 1 (excellent) to 5 (poor) on the widely utilized single item from the RAND SF-36 questionnaire (68). The single-item on the SF-36 has evidenced strong psychometric characteristics (68).

Demographics—Participants provided self-reported information about their age, gender, and race/ethnicity. Height and weight were measured in order to generate a body mass index (BMI) for each participant. These demographic variables were utilized as covariates in the analyses described below.

Analytic strategy—We utilized multiple imputation to handle random missing data, which is superior to listwise deletion (69). Consistent with prior work demonstrating the utility of evaluating stress-induced RSA relative to baseline RSA in predicting stress and health outcomes (48–50, 70), we adjusted for baseline RSA in our analyses. We also controlled for participant age, gender, race/ethnicity, BMI, and adult SES. EQS structural

equation modeling software was utilized (version 6.1; 71). Attachment orientations and self-reported general stress were examined as mediators of the association between childhood SES and self-reported health (see Figure 1). Moreover, stress-induced RSA was examined as a moderator of the association between attachment avoidance and general stress. 5,000 bias corrected bootstrap samples were utilized to examine indirect effects to test for mediation and serial mediation (i.e., mediation in sequence), which is consistent with modern approaches to mediation analysis (72).

Results

Descriptive statistics and bivariate correlations for primary study variables are presented in Table 1. In regards to covariates, current SES was associated with self-reported health ($r = .39, p < .001$), while BMI ($r = -.17, p = .01$) and current SES ($r = -.16, p = .02$) were associated with attachment anxiety. Current SES ($r = -.17, p = .01$) was also associated with attachment avoidance. Furthermore, participant age ($r = -.73, p < .001$), BMI ($r = -.33, p < .001$), and baseline RSA ($r = .72, p < .001$) were associated with stress-induced RSA. Current SES was the only covariate associated with general stress ($r = -.16, p = .02$), while participant BMI ($r = -.17, p = .02$) and current SES ($r = .16, p = .02$) were associated with self-reported health.

The information depicted in Figures 2 and 3 was tested within one overall model (see Figure 1; $F^2 = .14$) in which attachment anxiety and avoidance were associated ($r = .49, p < .001$); however, we separated our description of the results for each attachment dimension in order to improve clarity given that childhood SES was not associated with attachment avoidance ($\beta = .02, p = .75$). Additionally, childhood SES was not associated with baseline RSA ($\beta = .06, p = .25$) or stress-induced RSA ($\beta = -.08, p = .12$). As seen in Figure 2, higher childhood SES was associated with less attachment anxiety, which, in turn, was associated with less general stress. Higher general stress was associated with worse self-reported health. Using 5,000 bootstrap samples to test indirect effects, neither attachment anxiety nor general stress independently mediated the association between childhood SES and self-reported health; however, support for serial mediation was identified such that the pathway from childhood SES to self-reported health was partially explained by the serial pathway from attachment anxiety to general stress. Partial mediation was supported given that a significant association between childhood SES and self-reported health was significant in the overall model.

Attachment avoidance and the interaction between attachment avoidance and stress-induced RSA were associated with general stress (see Figures 3 & 4). Using the Johnson-Neyman technique (73), attachment avoidance was associated with general stress when the transformed value for stress-induced RSA was greater than 5.45, which represented 75.59% of the present study sample. An indirect effect was identified for general stress mediating the association between attachment avoidance and self-reported health. As such, support for moderated-mediation was identified.

Discussion

The present study is one of the first to examine attachment orientations as mediators of the link between early adversity and adult health. This link has been suggested in prior work (8–9); however, it has not been tested empirically. In the present study, childhood SES was associated with adult self-reported health via the serial pathway from attachment anxiety to general stress as expected. In contrast to expectations, childhood SES was not associated with attachment avoidance; however, attachment avoidance was associated with self-reported health via general stress among those with high stress-induced RSA as expected. Results were independent of participant age, gender, BMI, adult SES, and baseline RSA. Such findings shed light on possible mechanisms linking childhood SES and adult health, which is important given that self-reported health is linked to subsequent morbidity and mortality across various age ranges (74,75), ethnic groups (76,77), and patient populations (78–80).

The family stress and risky families models (8,9) highlight that early social environments shape one's social behaviors, emotional processing, and stress responses, which ultimately impact mental and physical health outcomes in adolescence and adulthood. Attachment theory provides a useful framework to understand the association between early social environments and mental and physical health throughout the lifespan. Individuals who are insecurely attached experience greater stress than secure individuals (35), which has a profound impact on health via psychological, behavioral, and biological processes (81). For instance, stress negatively impacts the immune systems which reduces the strength of immune responses to vaccines, enhances inflammation, slows wound healing, and shortens telomeres (82). Social stressors are more likely to enhance inflammation in comparison to non-social stressors (83,84), suggesting that attachment may have particularly important connections to immune dysregulation. Future research should consider this potential pathway. Childhood SES has been linked to immune dysregulation (2), supporting a hypothesized association between attachment anxiety and immunity given present study data.

Present study findings also indicate that individual differences in parasympathetic nervous system activity, as indexed by RSA, are important for describing associations between attachment, stress, and self-reported health. Specifically, attachment avoidance was associated with self-reported health through stress among those with high, but not low, stress-induced RSA. Such findings may reflect that individuals who have high attachment avoidance are ineffective at regulating their emotions over time, regardless if stress-induced RSA is low or high, given that chronic interpersonal stressors consistently lead to self-regulatory failure (85). However, they may be able to effectively regulate their emotions during such stressors if they have the regulatory strength (i.e., stress-induced RSA withdrawal) to do so.

The risky families model indicates that chronic or recurrent exposure to family based stressors early in life may lead to deficiencies in one's ability to demonstrate an adaptive parasympathetic nervous system response to stress (9). Our results do not support an association between childhood SES and parasympathetic nervous system functioning,

consistent with findings indicating that chronic stress may not change RSA over time (86). However, stress-induced RSA changed the association between attachment avoidance and stress, indicating that RSA is important for determining how individuals regulate the negative thoughts or feelings they may have about relying on others within interpersonal relationships (i.e., attachment avoidance). Therefore, it will be important to integrate aspects of the risky families model (9), the neurovisceral integration model (57), and polyvagal theories (56), to get a better understanding of the association between childhood environments and adult health in future theoretical and empirical work.

Prior work has indicated that attachment related processes may change (or moderate) associations between childhood SES and adult health. For example, individuals with warm mothers were less likely to experience heightened inflammation in adulthood in comparison to those with less warm mothers (87). Similarly, high maternal nurturance was found to reduce the risk of developing metabolic syndrome in adulthood among those with low childhood SES (88). Present study findings enhance theory by demonstrating that low childhood SES places one at risk of experiencing greater attachment anxiety, which places one at risk of experiencing poor health via stress. Therefore, it may be beneficial to integrate findings demonstrating the importance of parental warmth with present study findings in future work. Specifically, in line with the family stress and risky families models (8,9), it may be that low SES is associated with low parental warmth among some, but not all, families, which may place children at risk of developing insecure attachment orientations that are associated with poor health in adulthood. Future empirical work is needed to elucidate such a possibility.

Consistent with present study findings, those with low childhood SES demonstrate enhanced physiological reactivity when faced with stressors (89). However, when in the presence of a supportive figure, the association between low childhood SES and high physiological reactivity was attenuated. This is important given that insecure attachment orientations are associated with smaller social network sizes and less relationship reciprocity than attachment security (90). Therefore, present study findings may relate to the degree to which individuals experience social support. As mentioned previously, social support has been found to attenuate the association between attachment insecurity and health (91). In the present study, participants engaged in a stressful task that is social in nature, but inherently unsupportive, which may have augmented the association between childhood SES and self-reported health. Future work would benefit from measuring social support and examining if present study findings may differ if an individual has a supportive individual accompanying them during exposure to a stressful task.

The cross-sectional design of the study limited our ability to examine associations over time; however, findings are grounded in theory (8,9) and are consistent with the expected time course in which childhood SES would be associated with attachment, stress, and health. Future work using longitudinal designs are needed to establish temporal precedence and enhance causal inference. The study is also limited by the predominantly white sample. Further research with more racially diverse groups is needed given identified differences in self-reported health by ethnicity (92). The study is also limited given that dyadic processes, which are important for understanding how relationships may influence health (93), were

not measured. It would be interesting to examine how attachment patterns among couples interacting to predict stress and health outcomes for each partner. Additionally, self-reports of childhood SES, attachment orientations, stress, and health were utilized in the present study indicating that there may have been shared method bias (94). Importantly, self-reported health is associated with broad health relevant outcomes such as immune dysregulation (95) and all-cause mortality (96). Although self-reported health is a strong predictor of broad health outcomes, it does not provide specific information about the health outcomes that attachment orientations may be indirectly associated with. Future work would benefit from evaluating specific health outcomes to move the field forward; however, given the association between attachment orientations and stress, it is likely that attachment orientations are associated with broad health outcomes (97).

Conclusions

Attachment theory is useful for understanding why low childhood SES is associated with poor health outcomes in adulthood. In the present study, childhood SES was associated with adult self-reported health via the serial pathway from attachment anxiety to general stress. Findings also provide insight into why attachment avoidance is less consistently associated with general stress and health in comparison to attachment anxiety by demonstrating that attachment avoidance was associated with self-reported health through general stress only when stress-induced RSA was high.

Acknowledgments

Data collection was supported by the National Institute of Allergy and Infectious Diseases (AI066367) and the National Center for Complementary and Integrative Health (RC1AT005799). Preparation of the manuscript was supported by the National Heart, Lung, and Blood Institute (1R01HL127260-01; 1F32HL131353).

References

1. Aber JL, Bennett NG, Conley DC, Li J. The effects of poverty on child health and development. *Annu Rev Publ Health*. 1997; 18:463–483.
2. Cohen S, Doyle WJ, Turner RB, Alper C, Skoner DP. Childhood socioeconomic status and host resistance to infection illness in adulthood. *Psychosom Med*. 2004; 66(4):553–558. [PubMed: 15272102]
3. Cohen S, Janicki-Deverts D, Chen E, Matthews KA. Childhood socioeconomic status and adult health. *Ann N Y Acad Sci*. 2010; 1186:37–55. [PubMed: 20201867]
4. Fagundes CP, Way B. Early-life stress and adult inflammation. *Curr Dir Psychol Sci*. 2014; 23(4): 277–283.
5. Braveman PA, Cubbin C, Egerter S, Williams DR, Pamuk E. Socioeconomic disparities in health in the United States: What the patterns tell us. *Am J Public Health*. 2010; 100(1):186–196.
6. National Center for Health Statistics. *Health statistics: Measuring our nation's health*. Hyattsville, MD: 2015.
7. Steptoe A, Feldman PJ, Kunz S, Owen N, Willemsen G, Marmot M. Stress responsivity and socioeconomic status: A mechanism for increased cardiovascular risk? *Eur Heart J*. 2002; 23:1757–1763. [PubMed: 12419295]
8. Conger RD, Ge X, Elder GH, Lorenz FO, Simons RL. Economic stress, coercive family process, and developmental problems of adolescents. *Child Dev*. 1994; 65(2):541–561. [PubMed: 8013239]
9. Repetti RL, Taylor SE, Seeman TE. Risky families: Family social environments and the mental and physical health of offspring. *Psychol Bull*. 2002; 128(2):330–366. [PubMed: 11931522]

10. Wickrama KAS, Conger RD, Lorenz FO, Jung T. Family antecedents and consequences of trajectories of depressive symptoms from adolescence to young adulthood: A life course investigation. *J Health Soc Behav.* 2008; 49(4):468–483. [PubMed: 19181050]
11. Conger RD, Conger KJ, Martin MJ. Socioeconomic status, family processes, and individual development. *J Marriage Fam.* 2010; 72(3):685–704. [PubMed: 20676350]
12. Hoff, E., Laursen, B., Tardif, T. Socioeconomic status and parenting. In: Bornstein, MH., editor. *Handbook of parenting Volume 2: Biology and ecology of parenting.* Mahwah, New Jersey: Lawrence Erlbaum Associates, Publishers; 2002. p. 231-252.
13. Vaughn, BE., Bost, KK. Attachment and temperament: Redundant, independent, or interacting influences on interpersonal adaptation and personality development?. In: Cassidy, J., Shaver, PR., editors. *Handbook of attachment: Theory, research, and clinical applications.* New York, New York: Guilford Press; 1999. p. 198-225.
14. Valenzuela M. Maternal sensitivity in developing society: The context of urban poverty and infant chronic undernutrition. *Dev Psychol.* 1997; 33(5):845–855. [PubMed: 9300217]
15. Lovejoy MC, Graczyk PA, O'Hare E, Neuman G. Maternal depression and parenting behavior: A meta-analytic review. *Clin Psychol Rev.* 2000; 20(5):561–592. [PubMed: 10860167]
16. Wilson AL. Poverty and children's health. *Child Youth Fam Serv Q.* 1993; 16:14–16.
17. Mazzeschi C, Pazzagli C, Laghezza L, Radi G, Battistini D, De Feo P. The role of both parents' attachment pattern in understanding childhood obesity. *Front Psychol.* 2014; 5:791. [PubMed: 25120507]
18. Ahrens KR, Ciechanowski P, Katon W. Associations between adult attachment style and health risk behaviors in an adult female primary care population. *J Psychosom Res.* 2012; 72(5):364–370. [PubMed: 22469278]
19. Ainsworth, MS., Blehar, MC., Waters, E., Wall, S. *Patterns of attachment: A psychological study of the Strange Situation.* Hillsdale, NJ: Lawrence Erlbaum Associates, Inc; 1978.
20. Bowlby, J. *Attachment and loss.* 3rd. New York, New York: Basic books; 1980.
21. Hazan C, Shaver PR. Romantic love conceptualized as an attachment process. *J Pers Soc Psychol.* 1987; 59:511–524.
22. Fraley RC, Waller NG, Brennan KA. An item-response theory analysis of self-report measures of adult attachment. *J Pers Soc Psychol.* 2000; 78:350–365. [PubMed: 10707340]
23. Fagundes CP, Diamond LM, Allen KP. Adolescent attachment insecurity and parasympathetic functioning predict future loss adjustment. *Pers Soc Psychol Bull.* 2012; 38:821–832. [PubMed: 22399361]
24. Murray SL, Holmes JG, Griffin DW. Self-esteem and the quest for felt security: How perceived regard regulates attachment processes. *J Pers Soc Psychol.* 2000; 78(3):478–498. [PubMed: 10743875]
25. Karreman A, Vingerhoets JJM. Attachment and well-being: The mediating role of emotion regulation and resilience. *Pers Individ Dif.* 2012; 53:821–826.
26. Mikulincer, M., Shaver, PR. Adult attachment and affect regulation. In: Caddidy, J., Shaver, PR., editors. *Handbook of attachment: Theory, research, and clinical applications.* second. New York, New York: Guilford Press; 2008. p. 503-531.
27. Glaser R, Kiecolt-Glaser JK. Stress-induced immune dysfunction: implications for health. *Nat Rev Immunol.* 2005; 5(3):243–251. [PubMed: 15738954]
28. Jaremka LM, Glaser R, Loving TJ, Malarkey WB, Stowell JR, Kiecolt-Glaser JK. Attachment anxiety is linked to alterations in cortisol production and cellular immunity. *Psychol Sci.* 2013; 24(3):272–279. [PubMed: 23307944]
29. McWilliams LA, Bailey SJ. Associations between adult attachment ratings and health conditions: Evidence from the National Comorbidity Survey Replication. *Health Psychol.* 2010; 29(4):446–453. [PubMed: 20658833]
30. Puig J, Englund MM, Simpson JA, Collins WA. Predicting adult physical illness from infant attachment: A prospective longitudinal study. *Health Psychol.* 2013; 32(4):409–417. [PubMed: 22823067]
31. Brooks KP, Robles TF, Schetter CD. Adult attachment and cortisol responses to discussions with a romantic partner. *Personal Relationships.* 2011; 18(2):302–320.

32. Carpenter EM, Kirkpatrick LA. Attachment style and presence of a romantic partner as moderators of psychophysiological responses to a stressful laboratory situation. *Personal Relationships*. 1996; 3(4):351–367.
33. Maunder RG, Lancee WJ, Nolan RP, Hunter JJ, Tannenbaum DW. The relationship of attachment insecurity to subjective stress and autonomic function during standardized acute stress in healthy adults. *J Psychosom Res*. 2006; 60:283–290. [PubMed: 16516661]
34. Fagundes CP, Jaremka LM, Glaser R, Alfano CM, Povoski SP, Lipari AM, Agnese DM, Yee LD, Carson WE, Farrar WB, Malarkey WB, Chen M, Kiecolt-Glaser JK. Attachment anxiety is related to Epstein-Barr virus latency. *Brain Behav Immun*. 2014; 41:232–238. [PubMed: 24945717]
35. Dewitte M, De Houwer J, Goubert L, Buysse A. A multi-modal approach to the study of attachment related distress. *Biol Psychiatry*. 2010; 85(1):149–162.
36. Gouin JP, Glaser R, Loving TJ, Malarkey WB, Stowell J, Houts C, Kiecolt-Glaser JK. Attachment avoidance predicts inflammatory responses to marital conflict. *Brain Behav Immun*. 2009; 23(7): 898–904. [PubMed: 18952163]
37. Konstantinos K, Sideridis GD. Attachment, social support and well-being in young and older adults. *J Health Psychol*. 2006; 11:863–876. [PubMed: 17035259]
38. Merz EM, Consedine NS. Attachment security moderates the links between emotional and instrumental family support and wellbeing in later life. *Attach Hum Dev*. 2009; 11:203–221. [PubMed: 19266366]
39. Mikulincer M, Shaver PR, Pereg D. Attachment theory and affect regulation: The dynamics, development, and cognitive consequences of attachment-related strategies. *Motiv Emotion*. 2003; 27(2):77–102.
40. Mikulincer M, Dolev T, Shaver PR. Attachment-related strategies during thought suppression: Ironic rebounds and vulnerable self-representations. *J Pers Soc Psychol*. 2004; 87(6):940–956. [PubMed: 15598116]
41. Sweller J. Cognitive load theory, learning difficulty, and instructional design. *Learn Instr*. 1994; 4(4):295–312.
42. Brennan, KA., Clark, CL., Shaver, PR. Self-reported measurement of adult attachment: An integrative overview. In: Simpson, JA., Rholes, WS., editors. *Attachment Theory and Close Relationships*. Guilford Press; New York: 1998. p. 46-76.
43. Diamond LM, Fagundes CP. Psychobiological research on attachment. *J Soc Pers Relat*. 2010; 27(2):218–225.
44. Vögele C, Jarvis A, Cheeseman K. Anger suppression, reactivity, and hypertension risk: Gender makes a difference. *Ann Behav Med*. 1997; 19(1):61–69. [PubMed: 9603679]
45. Appleton AA, Buka SL, Loucks EB, Gilman SE, Kubzansky LD. Divergent associations of adaptive and maladaptive emotion regulation strategies with inflammation. *Health Psychol*. 2013; 32(7):748–756. [PubMed: 23815767]
46. Karreman A, Vingerhoets JJM. Attachment and well-being: The mediating role of emotion regulation and resilience. *Pers Individ Dif*. 2012; 53:821–826.
47. Diamond LM, Fagundes CP, Cribbett MR. Individual differences in adolescents' sympathetic and parasympathetic functioning moderate associations between family environment and psychosocial adjustment. *Dev Psychol*. 2012; 48(4):918–931. [PubMed: 22268602]
48. Fagundes CP, Jaremka LM, Malarkey WB, Kiecolt-Glaser JK. Attachment style and respiratory sinus arrhythmia predict post-treatment quality of life in breast cancer survivors. *Psychooncology*. 2014; 23:820–826. [PubMed: 24532423]
49. Fagundes CP, Diamond LM, Allen KP. Adolescent attachment insecurity and parasympathetic functioning predict future loss adjustment. *Pers Soc Psychol Bull*. 2012; 38(6):821–832. [PubMed: 22399361]
50. Sbarra DA, Borelli JL. Heart rate variability moderates the association between attachment avoidance and self-concept reorganization following marital separation. *Int J Psychophysiol*. 2013; 88(3):253–260. [PubMed: 22542651]
51. Hayano J, Yasuma F, Okada A, Mukai S, Fujinami T. Respiratory sinus arrhythmia: A phenomenon improving pulmonary gas exchange and circulatory efficiency. *Circulation*. 1996; 94:842–847. [PubMed: 8772709]

52. Segerstrom SC, Nes LS. Heart rate variability reflects self-regulatory strength, effort, and fatigue. *Psych Sci.* 2007; 18(3):275–281.
53. Beauchaine TP. Vagal tone, development, and Gray’s motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Dev Psychopathol.* 2001; 13:183–214. [PubMed: 11393643]
54. Gentzler AL, Santucci AK, Kovacs M, Fox NA. Respiratory sinus arrhythmia reactivity predicts emotion regulation and depressive symptoms in at-risk and control children. *Biol Psychol.* 2009; 82(2):156–163. [PubMed: 19596044]
55. Rottenberg J, Salomon K, Gross JJ, Gotlib IH. Vagal withdrawal to a sad film predicts subsequent recovery from depression. *Psychophysiology.* 2005; 42:277–281. [PubMed: 15943681]
56. Porges SW. The polyvagal theory: Phylogenetic substrates of a social nervous system. *Int J Psychophysiol.* 2001; 42(2):123–146. [PubMed: 11587772]
57. Thayer JF, Lane RD. A model of neurovisceral integration in emotion regulation and dysregulation. *J Affect Disord.* 2000; 61(3):201–216. [PubMed: 11163422]
58. Cohen S, Janicki-Deverts D, Turner RB, Casselbrant ML, Li-Korotky HS, Epel ES, Doyle WJ. Association between telomere length and experimentally induced upper respiratory viral infection in healthy adults. *JAMA.* 2013; 309(7):699–705. [PubMed: 23423415]
59. Janicki-Deverts D, Cohen S, Doyle WJ, Marsland AL, Bosch J. Childhood environments and cytomegalovirus serostatus and reactivation in adults. *Brain Behav Immun.* 2014; 40:174–181. [PubMed: 24675032]
60. Kirschbaum C, Pirke K, Hellhammer DH. The ‘Trier Social Stress Test’: A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology.* 1993; 28(1–2):76–81. [PubMed: 8255414]
61. Adler NE, Epel ES, Castellazzo G, Ickovics JR. Relationship of subjective and objective social status with psychological and physiological functioning: preliminary data in healthy white women. *Health Psychol.* 2000; 19(6):586–592. [PubMed: 11129362]
62. Operario D, Adler NE, Williams DR. Subjective social status: Reliability and predictive utility for global health. *Psychol Health.* 2004; 19(2):237–246.
63. Goodman E, Adler NE, Kawachi I, Frazier AL, Huang B, Colditz GA. Adolescents’ perceptions of social status: Development and evaluation of a new indicator. *Pediatrics.* 2001; 108:1–8. [PubMed: 11433046]
64. Wei M, Russell DW, Mallinckrodt B, Vogel DL. The experiences in Close Relationship Scale (ECR)-Short Form: Reliability, validity, and factor structure. *J Person Assess.* 2007; 88:187–204.
65. Malik M. Heart rate variability: Standards of measurement, physiological interpretation and clinical use. Task force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *Circulation.* 1996; 93(5):1043–1065. [PubMed: 8598068]
66. Duhamel P, Vetterli M. Fast Fourier transforms: A tutorial review and state of the art. *Signal Process.* 1990; 19:259–299.
67. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. *J Health Soc Behav.* 1983; 24:385–396. [PubMed: 6668417]
68. Ware JE, Sherbourne CD. The MOS36-item short-form health survey (SF-36). I. Conceptual framework and item selection. *Med Care.* 1992; 30(6):473–483. [PubMed: 1593914]
69. Schafer JL, Olsen MK. Multiple imputation for multivariate missing-data problems: A data analyst’s perspective. *Multivar Behav Res.* 1998; 33(4):545–571.
70. Cacioppo JT, Uchino BN, Berntson GG. Individual differences in the autonomic origins of heart rate reactivity: The psychometrics of respiratory sinus arrhythmia and preejection period. *Psychophysiology.* 2007; 31(4):412–419.
71. Bentler, PM. EQS (Version 6.1). Encino, CA: Multivariate Software; 2004.
72. Hayes AF. Beyond Baron and Kenny: Statistical mediation analysis in the new millennium. *Commun Monogr.* 2009; 76(4):408–420.
73. Johnson PO, Fay LC. The Johnson-Neyman Technique, its theory and application. *Psychometrika.* 1950; 15(4):349–367. [PubMed: 14797902]

74. Larsson D, Hemmingsson T, Allebeck P, Lundberg I. Self-rated health and mortality among young men: What is the relation and how may it be explained? *Scand J Public Health*. 2002; 30(4):259–266. [PubMed: 12680501]
75. Nybo H, Petersen HC, Gaist D, Jeune B, Andersen K, McGue M, Vaupel JW, Christensen K. Predictors of mortality in 2,249 nonagenarians - the Danish 1905-cohort survey. *J Am Geriatric Soc*. 2003; 51(10):1365–1373.
76. Nielsen AB, Siersma V, Hiort LC, Drivsholm T, Kreiner S, Hollnagel H. Self-rated general health among 40-year-old Danes and its association with all-cause mortality at 10-, 20-, and 29 years' follow-up. *Scand J Public Health*. 2008; 36(1):3–11. [PubMed: 17853002]
77. Yu ES, Kean YM, Slymen DJ, Liu WT, Zhang M, Katzman R. Self-perceived health and 5-year mortality risks among the elderly in Shanghai, China. *Am J Epidemiol*. 1998; 147(9):880–890. [PubMed: 9583719]
78. Bosworth HB, Siegler IC, Brummett BH, Barefoot JC, Williams RB, Clapp-Channing NE, Mark DB. The association between self-rated health and mortality in a well-characterized sample of coronary artery disease patients. *Med Care*. 1999; 37(12):1226–1236. [PubMed: 10599604]
79. Jylhä M. What is self-rated health and why does it predict mortality? Towards a unified conceptual model. *Soc Sci Med*. 2009; 69(3):307–316. [PubMed: 19520474]
80. Kaplan GA, Camacho T. Perceived health and mortality: A nine-year follow-up of the Human Population Laboratory Cohort. *Am J Epidemiol*. 1983; 117(3):292–304. [PubMed: 6829557]
81. Schneiderman N, Ironson G, Siegel SD. Stress and health: Psychological, behavioral, and biological determinants. *Annu Rev Clin Psychol*. 2005; 1:607–628. [PubMed: 17716101]
82. Glaser R, Kiecolt-Glaser JK. Stress-induced immune dysfunction: implications for health. *Nat Rev Immunol*. 2005; 5(3):243–251. [PubMed: 15738954]
83. Dickerson SS, Gable SL, Irwin MR, Aziz N, Kemeny ME. Social-evaluative threat and proinflammatory cytokine regulation: An experimental laboratory investigation. *Psychol Sci*. 2009; 20(10):1237–1244. [PubMed: 19754527]
84. Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychol Bull*. 2004; 130(3):355–391. [PubMed: 15122924]
85. Wagner, DD., Heatherton, TF. Emotion and self-regulation failure. In: Gross, JJ., editor. *Handbook of Emotion Regulation*. Second. New York, New York: The Guilford Press; 2014. p. 613-628.
86. Schubert C, Lambert M, Nelesen RA, Bardwell W, Choi JB, Dimsdale JE. Effects of stress on heart rate complexity: A comparison between short-term and chronic stress. *Biol Psychol*. 2009; 80(3):325–332. [PubMed: 19100813]
87. Chen E, Miller GE, Kobor MS, Cole SW. Maternal warmth buffers the effects of low early-life socioeconomic status on pro-inflammatory signaling in adulthood. *Mol Psychiatry*. 2011; 16(7):729–737. [PubMed: 20479762]
88. Miller GE, Lachman ME, Chen E, Gruenewald TL, Karlamangla AS, Seeman TE. Pathways to resilience: Maternal nurturance as a buffer against the effects of childhood poverty on metabolic syndrome at midlife. *Psychol Sci*. 2011; 22(12):1591–1599. [PubMed: 22123777]
89. John-Henderson NA, Stellar JE, Mendoza-Denton R, Francis DD. Socioeconomic status and social support: Social support reduces inflammatory reactivity for individuals whose early-life socioeconomic status was low. *Psychol Sci*. Advance online publication.
90. Fiori K, Consedine NS, Merz EM. Attachment, social network size, and patterns of social exchange in later life. *Res Aging*. 2011; 33:465–493.
91. Merz EM, Consedine NS. The association of family support and wellbeing in later life depends on adult attachment. *Attach Hum Dev*. 2009; 11:203–221. [PubMed: 19266366]
92. Saxena S, Eliahoo J, Majeed A. Socioeconomic and ethnic group differences in self reported health status and use of health services by children and young people in England: Cross sectional study. *BMJ*. 2002; 325(7363):520–523. [PubMed: 12217992]
93. Pietromonaco PR, Uchino B, Schetter CD. Close relationship processes and health: Implications of attachment theory for health and disease. *Health Psychol*. 2013; 32(5):499–153. [PubMed: 23646833]

94. Podsakoff PM, MacKenzie SB, Podsakoff NP. Sources of method bias in social science research and recommendations on how to control it. *Annu Rev Psychol.* 2012; 63:539–569. [PubMed: 21838546]
95. Murdock KW, Fagundes CP, Peek MK, Vohra V, Stowe RP. The effect of self-reported health on latent herpesvirus reactivation and inflammation in an ethnically diverse sample. *Psychoneuroendocrinology.* 2016; 72:113–118. [PubMed: 27398881]
96. DeSalvo KB, Muntner P. Discordance between physician and patient self-rated health and all-cause mortality. *Ochsner J.* 2011; 11:232–240. [PubMed: 21960756]
97. Fraley RC, Shaver PR. Adult romantic attachment: Theoretical developments, emerging controversies, and unanswered questions. *Rev Gen Psychol.* 2000; 4(2):132–154.

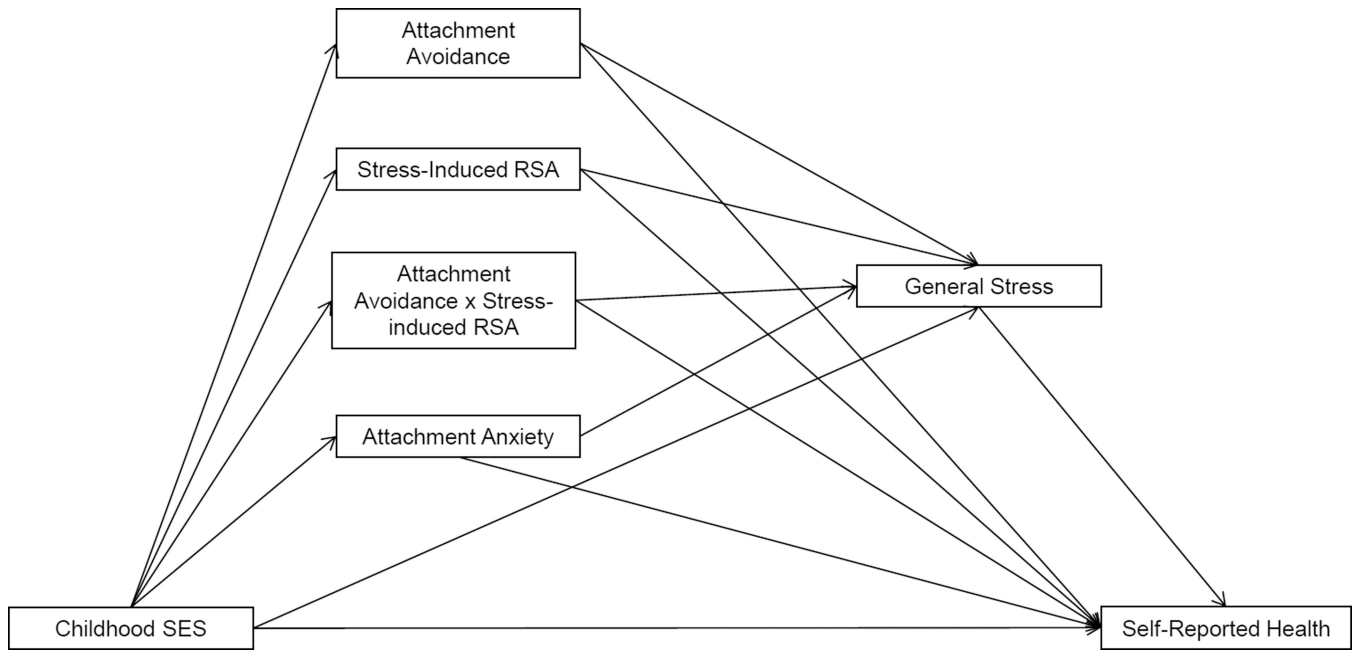


Fig 1. Full model tested. SES = socioeconomic status; RSA = respiratory sinus arrhythmia.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

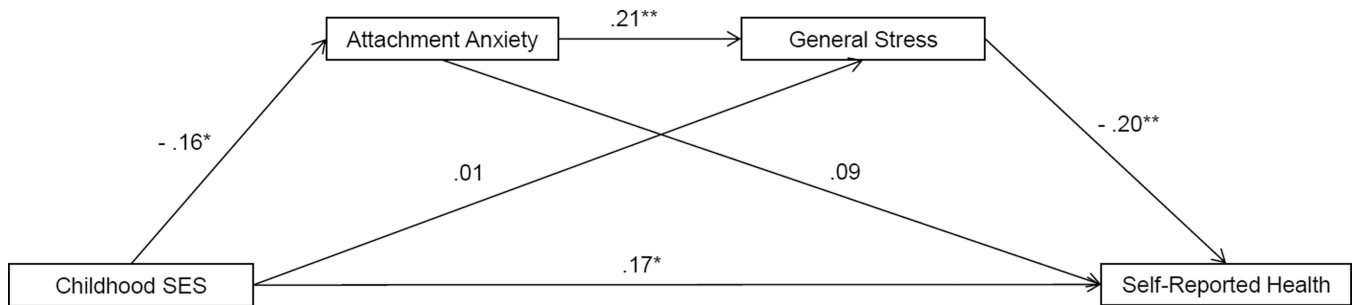


Fig 2.

A serial mediation model of associations between childhood socioeconomic status, attachment anxiety, general stress, and self-reported health. SES = socioeconomic status. Values represent standardized regression coefficients. Indirect effects using 5,000 bootstrap samples: attachment anxiety ($-.0144$, $SE = .0155$, 95% $CI = -.0548, .0088$), general stress ($-.0039$, $SE = .0133$, 95% $CI = -.0341, .0206$), and serial mediation (i.e., mediation in sequence; $.0068$, $SE = .0046$, 95% $CI = .0013, .0217$). Control variables included participant age, sex, ethnicity, body mass index, adult SES, attachment avoidance, baseline respiratory sinus arrhythmia (RSA), stress induced RSA, and the interaction between attachment avoidance and stress induced RSA. * $p < .05$; ** $p < .01$; *** $p < .001$.



Fig. 3.

A moderated-mediation model of associations between attachment avoidance, general stress, and self-reported health. SES = socioeconomic status; RSA = respiratory sinus arrhythmia. Values represent standardized regression coefficients and 95% confidence intervals are depicted in parentheses. Indirect effect using 5,000 bootstrap samples = $-.0186$, $SE = .0035$, 95% $CI = -.0786, -.0035$. Control variables included participant age, sex, ethnicity, body mass index, baseline RSA, attachment anxiety, and adult socioeconomic status. * $p < .05$.

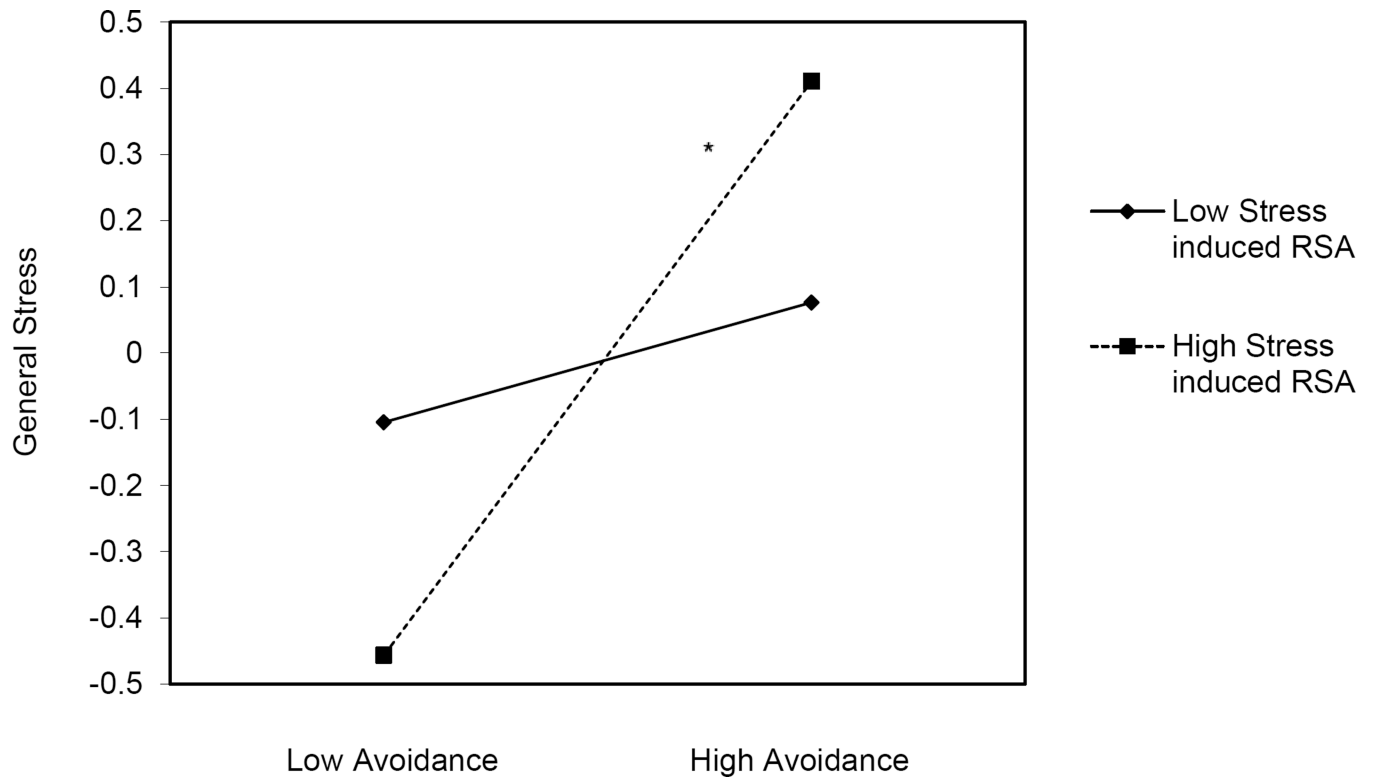


Fig. 4. The interaction between attachment avoidance and stress induced respiratory sinus arrhythmia (RSA) in predicting general stress. * = significant slope.

Table 1

Descriptive statistics and bivariate correlations for primary study variables

Variable	<i>M</i>	<i>SD</i>	1	2	3	4	5
1. Childhood SES	5.07	1.93	--				
2. Attachment anxiety	3.43	1.34	-.21**	--			
3. Attachment avoidance	2.88	1.33	-.11	.46**	--		
4. Stress-induced RSA [†]	6.16	1.14	-.10	-.04	.12	--	
5. General stress	12.04	5.65	-.07	.32**	.38**	.06	--
6. Self-reported health	2.92	0.71	.20**	-.03	-.11	.03	-.21**

Notes.[†]Represents the log transformed value. SES = socioeconomic status; RSA = respiratory sinus arrhythmia.