Published in final edited form as:
Psychophysiology. 2009 July ; 46(4): 862–869. doi:10.1111/j.1469-8986.2009.00829.x.

Positive Affect is Associated with Cardiovascular Reactivity, Norepinephrine Level, and Morning Rise in Salivary Cortisol

B.H. Brummett, PhD.[1], **S.H. Boyle, PhD.**[1], **C.M. Kuhn, Ph.D.**[2], **I.C. Siegler, Ph.D., M.P.H.**[1], and **R.B. Williams, M.D.**[1]

[1]Departments of Psychiatry and Behavioral Sciences, Duke University Medical Center, Durham, North Carolina, 27710, USA

[2]Department of Pharmacology and Cancer Biology, Duke University Medical Center, Durham, North Carolina, 27710, USA

Abstract

Positive affect was examined as a predictor of: 1] cardiovascular reactivity during a sadness and an anger recall task, and recovery following the protocol, 2] epinephrine (EPI) and norepinephrine (NOREPI) reactivity and level during the recall protocol, and 3] the diurnal pattern of salivary cortisol. Sample was 328 individuals. Negative affect, age, race, sex, smoking status, income, and BMI were adjusted. During sadness recall positive affect was inversely related to SBP (p=.007) and DBP (p=.049) reactivity, and unrelated to HR (p =.226). Positive affect was unrelated to reactivity during anger recall (p's >.19), and was unrelated to recovery at the end of the recall protocol. Positive affect was inversely related to the mean level of NOREPI (p=.046), and unrelated to EPI (p=.149). Positive affect was inversely related to the increase in cortisol 30 minutes post awakening (p=.042), and unrelated to the evening decline in cortisol levels (p=.174). Positive emotions may be relevant to good health.

Keywords

Positive affect; cardiovascular reactivity; salivary cortisol; norepinephrine; epinephrine

Introduction

The proposition that emotional experiences can have consequences for health is supported by epidemiologic evidence and studies demonstrating its physiological plausibility [1-5]. Research on this topic has been largely centered on negative emotions, particularly depression, anxiety, and anger. A strong contrasting case is growing, however, for the health benefits of positive emotions per se [6-14]. Positive emotions have been linked, for example, to lower cortisol output [15-17], lower rates of hypertension and diabetes mellitus [18], and longevity [19]. Early studies in this area sometimes failed to simultaneously examine the effects of negative and positive emotion—a critical element for evaluation of the independent impact of positive emotion. However, recent studies such as those cited above have examined negative and positive emotion together and effects for positive emotion have remained statistically significant.

Basic motivational theories of emotion provide a general model that helps link the experience of emotion, both positive and negative, with health-related outcomes. The

Corresponding author: Beverly H. Brummett, Box 2969 Duke University Medical Center, Durham, North Carolina, 27710. Phone: 919 (684-6129), FAX: 919 (681-8960) e-mail: brummett@duke.edu.

underlying premise of these models is that emotions exist in the service of motivating behavior, or promoting what are referred to as action tendencies, e.g., [20,21]. These behavioral action tendencies may influence health in numerous ways, some of which may be negative and some positive. Because emotions direct behaviors, they have the ability to influence health habits and lifestyle choices, e.g., physical activity and social engagement. The experience of emotion, and accompanying action tendencies, also stimulates the central nervous system resulting in a cascade of physiological responses that can affect health. Certain action tendencies, such as those that often accompany negative emotions, may lead to behavioral responses that are associated with biological arousal, thus, the experience of these emotions may lead to CNS activation of autonomic, neuroendocrine, and immune-related responses that can have a detrimental impact on health. Alternatively, it has been hypothesized that one benefit of positive affect is that the resulting actions and biological responses may provide a respite from the stress associated with negative affectivity [22,23].

Studies designed to examine mechanisms underlying the associations among positive emotion, better health, and longevity, have focused on cardiovascular reactivity and recovery—an appropriate area in which examine emotion-health relations for at least two reasons. First, individuals with heightened cardiovascular reactivity and/or delayed recovery are at increased risk for coronary disease, e.g., [24,25]. Second, emotional reactions are associated with changes in cardiovascular arousal, and importantly, negative and positive emotions have been associated with differing patterns of arousal, e.g. [26,27]. For example, research has shown that systolic blood pressure responses are significantly higher during tasks with a negative emotional valance, as compared to those with a positive emotional valance, e.g., [26]. Related research has demonstrated that the experience of positive emotion, and processes related to positive emotion such as social connectedness, diminish blood pressure reactivity and enhance recovery from negative emotional states [28,29].

Other studies evaluating mechanisms that may account for associations between positive emotion and health have examined measures of neuroendocrine function [17]. Norepinephrine, epinephrine, and cortisol are stress related neuroendocrine hormones that influence numerous bodily processes. Norepinephrine in the CNS acts to regulate behavioral and physiologic responses to the environment including alerting, attention, and autonomic nervous system regulation. Norepinephrine and epinephrine are the peripheral mediators of “fight-or-flight” sympathoadrenal response that increases heart rate and mobilizes other bodily responses [30]. Similarly, release of cortisol increases blood pressure and blood sugar levels in response to stress [31]. Chronically elevated levels of cortisol have been associated with negative health outcomes such as reduced bone density and lean mass, and atrophy of hippocampal cells [32], as well as to abdominal obesity, Type 2 diabetes, hypertension, and autoimmune conditions [33,34]. Prolonged neuroendocrine response to stress has also been associated with depressed mood [35], whereas, lower levels of salivary cortisol have been associated with measures of well-being [36]. Thus, the association between neuroendocrine function and emotional responding is also a potential mechanism linking positive emotion to physical health outcomes.

In the present study we extend the literature on the health effects of positive emotion by examining a factor score representing the tendency to experience positive affect, adjusted for a similar trait measure of negative affect, as a predictor of simultaneously assessed cardiovascular and neuroendocrine responses during a laboratory protocol in 328 community volunteers. Additionally, we assessed these trait emotion ratings as predictors of cortisol in a natural setting. Specifically, we hypothesized that positive affect would be significantly related to: 1) lower blood pressure and heart rate reactivity during a sadness and an anger recall task, 2) faster blood pressure and heart rate recovery during the recall protocol, and 3) lower epinephrine and norepinephrine reactivity during this protocol. In

addition, we predicted that positive affect would be associated with lower diurnal salivary cortisol levels, as measured by the morning rise and the evening decline in cortisol. Importantly, we predict that these associations will be independent of the tendency to experience negative affect, age, race, sex, smoking status, income level, and body mass index (BMI).

Methods

Participants and Procedures

Participants were recruited to take part in the Family Heart Study. The study was designed to identify genetic variants that interact with the environment to affect expression and clustering of psychosocial and biobehavioral characteristics (endophenotypes) that increase risk of cardiovascular disease (CVD), with a focus on the characteristic of hostility. Sibling pairs were recruited via community based ads. Individuals who reported that they were currently suffering from any major medical condition (e.g., cancer, heart disease, arthritis, diabetes) or psychiatric disorder (e.g., bipolar disorder, schizophrenia, memory loss) or who were pregnant or planning to become pregnant were excluded from the study. Participants who were the first family member to volunteer were screened for their level of hostility and individuals who were high or low on hostility, according to predefined criteria, were further recruited to participate in the study. Next, participants were asked to contact their brother(s) and/or sister(s) who might also qualify for and be interested in the study. If they agreed, they were told they should contact us by phone and arrangements were made for them to enroll in the study. Participants who did not have a family member who qualified to take part in the study were not enrolled. The study was conducted at Duke University Medical Center, and all subjects gave informed consent prior to their participation in the study using a form approved by the Duke University Medical Center Institutional Review Board and were compensated $275 for study completion. The sample consisted of 328 subjects (159 participants and 169 siblings) who had complete data on measures of positive affect as well as each covariate included in all analyses (i.e., negative affect, age, race, sex, smoking status, income level, and BMI) as of July 2007. For analyses of specific measures (i.e., cardiovascular reactivity, plasma catecholamines, and salivary cortisol) the total number of participants ranged from 268 to 328. The current hypotheses do not involve genetic analyses, and our hypotheses are not concerned with sib-pair relation. The potential within sib-pair correlation, however, was accounted for in a mixed models approach using sib-pair as a clustering identifier for the random effect in all analyses.

Measures

Positive Affect and Negative Affect—The tendency to experience positive and negative affect were represented by factor scores comprised of ratings from the following measures of emotion: the Profile of Mood States (POMS) [37]; the Visual Analog Scale (VAS), and the NEO-PI-R [38]. Prior research has established the validity of the POMS [39], the NEO-PI-R [38], and the VAS [40]. By including both POMS and VAS ratings, along with NEO ratings, the resulting factor scores reflect a general tendency, or trait assessment, regarding the experience of negative and/or positive emotional responses during periods of emotional expression (as those felt during the recall of such experiences). These scores are indicative of positive and negative emotional traits, and are likely to be a better representation of general emotional reactions than that of VAS/POMS or NEO ratings alone. In attempt to adequately assess an underlying trait, a valid approach for increasing reliability is through use of multiple measures sampled at multiple time points. Thus, our factor scores represent the shared variance among the different measures of emotion assessed at different times, creating a score that represents a trait measure of the tendency to experience positive and negative emotion under varying conditions.

The factor score for positive affect was comprised of 4 separate ratings for “happy” from the Visual Analog Scale (VAS) that were gathered during the emotional recall protocol (pre-anger recall, post-anger recall, pre-sadness recall, and post sadness recall); 2 separate ratings for the Profile of Mood States (POMS) [37] component score “friendly” that were gathered at the beginning of the protocol (prior to baseline ratings of blood pressure), and again at the conclusion of the protocol; and finally the E-6 facet score from the NEO-PI-R [38] representing “positive emotions”. Similarly, the factor score for negative affect was comprised of 4 separate ratings for “depressed” from the Visual Analog Scale (VAS) that were gathered during the emotional recall protocol (pre-anger recall, post-anger recall, pre-sadness recall, and post sadness recall); 2 separate ratings for the Profile of Mood States (POMS) [37] component score “depression-dejection” that were gathered at the beginning of the protocol and again at the conclusion of the protocol; and finally the N-3 facet score from the NEO-PI-R [38] representing “depression”. These constructs representing positive affect and negative affect were modeled using principal axes factor analysis. The factor loadings for positive affect and negative affect are presented in Table 1.

Our approach for the selection of specific affective measures to assess positive and negative emotion was both empirically and conceptually driven. Regarding the positive emotion factor—the primary focus of the paper—there were no additional items from the VAS, apart from “happy”, that were conceptually appropriate. With regard to a negative emotion factor, for both conceptual and empirical concerns we did not create a factor that attempted to represent anger, anxiety, and depression. Conceptually, we view these as distinct emotions. This notion was empirically supported, as our attempt to use anxiety, anger-hostility, and depression measures from the POMS, VAS, and NEO did not result in a satisfactory 1-factor solution. Whereas, the measures used for our depression-dejection factor addressed both our conceptual and empirical concerns very well.

Cardiovascular Reactivity (CVR) and Recovery—Minute to minute blood pressure (BP) readings were gathered via a Dynamap XL 9300 (Johnson & Johnson Health Care System, Inc.), see e.g.,[41] for similar use of this CVR protocol. Baseline systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) were represented by the mean of the last 5 minutes of a 10 minute resting baseline. Anger and sadness recall periods lasted 5 minutes with a 10 minute rest period between. Anger and sadness recall blood pressure and heart rate measures were represented by the mean of the readings during each of the 5 minute recall periods. Blood pressure and heart rate were also assessed during a 5 minute recovery period at the end of the protocol, and recovery measures were represented by the mean of the readings during this 5 minute period.

For the emotion recall periods, participants were asked to think of an incident that made them very angry toward another person, and that still makes them angry right now when they think about it. They were then asked to visualize that experience in their mind, recalling in detail what happened. Next participants were asked to verbally recount the entire story, including how they felt at that time. For the first minute, they were asked to visualize the (anger or sadness) incident and then for the next four minutes they were asked to describe the situation. Blood pressures were taken throughout the five minute period; one during the first visualization minute and four during the telling of the episode. The same protocol was followed for the sadness task that followed, substituting the recall of an incident that made them extremely sad.

Per the instructions and tasks included for the full study protocol, no caffeine, nicotine, nor alcohol was consumed prior to the emotion recall task, which took place between 11:30 a.m. and 12:30 p.m. for all participants. With very few deviations all subjects had virtually the same level of physical activity during the morning prior to the reactivity protocol.

Epinephrine (EPI) and Norepinephrine (NOREPI)—Prior to the start of the recall procedure, an in-dwelling catheter was inserted for all participants. Blood was sampled for the assessment of EPI and NOREPI levels immediately following the 10 minute baseline period, immediately following the completion of the anger recall task, and again at the end of the protocol (blood was not sampled immediately following the sadness recall). Blood samples were spun for 15 minutes in a refrigerated centrifuge and plasma was transferred into polypropylene tubes containing .050 ml Glutathione and then frozen at -70c. All blood samples were processed at the Clinical Research Center at DUMC. Plasma levels of EPI and NOREPI were measured by high-pressure liquid chromatography (HPLC) followed by electrochemical detection. Data were collected with a computer-based system, and quantitated with the use of internal standard and external standard curves. See [42] for similar methodology measuring plasma EPI and NOREPI.

Salivary Cortisol—Saliva was gathered the day following the recall task (using salivetttes with swabs) upon awakening, 30 minutes post awakening, and at bedtime. Participants were instructed not to eat, drink, or brush their teeth 30 minutes prior to collecting the salivary samples. Saliva was centrifuged for 10 minutes at 3000 rpms at room temperature in centrifuge, then frozen at -70c. Salivary cortisol was assayed by ELISA using a kit from Oxford Biomedical Research. See [17] for similar methodology measuring salivary cortisol.

Age, Race, Sex, Smoking Status, Income, and Body Mass Index (BMI)—Selection of sociodemographic covariates was guided by a recent line of work examining the association between positive affect and physiological outcomes [15-17]. Age was represented in years. Race was coded African American=0, Caucasian=1; sex Female=0, 1=Male; and smoking status 0=not currently smoking, 1=current smoker. Income was scaled in increments of $5,000 from 0 to 19, with 0=less than $10,000, and 19=$100,000 or more. BMI was calculated as weight in kilograms divided by height in meters squared. Table 2 presents descriptive data for the sample.

Statistical Analyses

All analyses were carried out using SAS (Cary, NC) PROC MIXED with maximum likelihood estimation. Importantly, mixed models allowed for us to account for potential within-sib-pair correlation, using sib-pair as a clustering identifier for the random effect in all analyses. The variance-covariance structure was selected *a priori* as first-order autoregressive (AR1), a structure that was used throughout all analyses.

BP and HR during the emotion tasks and the recovery period were represented by the mean of each of these measures collected during the 5 minute recall and recovery periods. Separate mixed models were estimated for each CV outcome during both the anger and sadness recall tasks and recovery period. In each model, positive affect was examined as a predictor of SBP, DBP, or HR levels during the anger and sadness recall tasks. Negative affect, age, race, sex, income, smoking status, BMI, and corresponding baseline cardiovascular measures were included as covariates in all models. Thus, the parameter estimate for positive affect in these models can be interpreted as the prediction of residualized change in BP and HR, independent of the effects of negative affect and other covariates.

Similar to CVR, positive affect was examined as a predictor of residualized change in EPI and NOREPI from the mean baseline level to the mean level during the anger recall task (blood was not drawn following the sadness task). As above, the mixed models included negative affect, age, race, sex, income, smoking status, BMI, and the corresponding baseline

plasma catecholamine measure as covariates. Values for EPI and NOREPI were log transformed for use in analyses in order to achieve appropriate distributional properties.

Finally, positive affect was examined as a predictor of 1) the rise in cortisol from awakening to 30 minutes post awakening, and 2) the fall in cortisol from 30-minutes post waking to bedtime. Mixed models, adjusted for negative affect, age, race, sex, BMI, income, and smoking status were used to assess the morning rise and the evening decline in cortisol.

Results

Cardiovascular Reactivity and Recovery

During sadness recall positive affect was significantly related to change in SBP ($p = .007$) and DBP ($p = .049$), with higher levels of positive affect associated with lower SBP and DBP reactivity. Results of the regression models examining these associations are presented in Table 3. Figure 1 displays the adjusted means for SBP and DBP reactivity during sadness recall for participants one standard deviation above and below the mean for positive affect. With regard to covariates, participants with a higher BMI and who reported being African American had higher SBP reactivity; and those with lower income, who reported being African American and older had higher DBP reactivity. Positive affect was unrelated to HR reactivity during sadness recall ($p = .226$), and to BP and HR reactivity during anger recall ($p's > .25$). Positive affect was unrelated to BP and HR recovery at the end of the recall protocol ($p's > .11$).

EPI and NOREPI

Positive affect was unrelated to the change in NOREPI ($p = .695$) or EPI ($p = .987$) from baseline to anger recall. Given the lack of an association of positive affect with reactivity of these measures, we were interested in the association of positive affect with the overall level of EPI and NOREPI throughout the protocol. Mixed models with repeated measures were used to assess positive affect as a predictor of the repeated measures of EPI and NOREPI at baseline, anger recall, and at the end of the procedure. Positive affect was a significant predictor of NOREPI as a main effect, indicating it was associated with the average level of NOREPI throughout the protocol ($p = .047$). Table 4 provides the results of this model. The form of the effect was such that higher levels of positive affect were related to lower levels of NOREPI. Figure 2 displays the adjusted means for levels of NOREPI at each time period for people who are one standard deviation above and below the mean for positive affect. As indicated in Table 4, being female was associated with lower levels of NOREPI, and being older was associated with higher levels of NOREPI. Positive affect was not significantly related to the level of EPI as a main effect ($p = .149$).

Cortisol

Higher positive affect was significantly related to a smaller rise in cortisol from awakening to 30-minutes post awakening ($p = .042$). Table 5 presents results of a model examining positive affect as a predictor of change in cortisol from awakening to 30-minutes post awakening. None of the covariates were significantly related to the morning increase in cortisol. Figure 3 displays the adjusted means for change in levels of salivary cortisol from awakening to 30-minutes post awakening for people one standard deviation above and below the mean for positive affect. Positive affect was unrelated to the decline in cortisol from morning to bedtime ($p = .174$).

Discussion

The main hypothesis, that positive affect is related to physiological measures relevant to health over and above the effects of negative affect, received support across multiple measures. The present findings for cardiovascular reactivity indicated that blood pressure reactivity during a sadness recall task was higher for those that have lower levels of positive affect, as compared to those with higher levels of positive affect. Others have reported similar inverse associations between positive emotion, blood pressure, and/or heart rate e.g., [15-17]. Such lower levels of blood pressure reactivity in those with higher levels of positive affect may, over time, result in decreased rates of cardiovascular disease e.g., [43,44]. The emotional experiences of anger and sadness are closely related, thus it was somewhat unexpected that positive affect was not significantly associated with CVR during the anger recall task. The present results would suggest that positive affect is more protective against rises in blood pressure during periods of sadness, as compared to incidents that are associated with the emotional reaction of anger. Although speculative, it is possible that individuals high in positive affect might show lower levels of reactivity because their recall of sad events is less salient than those who are low in positive affect, and this may not apply for circumstances involving anger.

The current results suggest that the tendency to experience positive affect is associated with lower levels of norepinephrine. Throughout the three measurement periods for plasma catecholamines during the reactivity protocol, norepinephrine was lower among individuals who experience higher levels of positive affect, as compared to those who experience lower levels of positive emotion. Flory et al., 2004 found that maximal prolactin response to fenfluramine was positively associated with ratings of positive mood, suggesting that individuals who experience higher levels of positive emotions have greater CNS serotonergic tone. Recently presented results [42] from our lab indicate that enhancing CNS serotonin levels via tryptophan infusion results in lowered levels of norepinephrine, as compared to a sham infusion day. These lower levels of norepinephrine following infusion continued through a reactivity protocol (unpublished data). However, infusion of tryptophan did not significantly affect norepinephrine reactivity. That is, norepinephrine increased as much from baseline to emotion recall tasks on the infusion day as it did on the sham infusion day, and across the protocol norepinephrine levels were lower on infusion day. Thus, although highly speculative, it is possible that an association between positive affect and norepinephrine is accounted for by higher levels of CNS serotonin. We had also predicted that levels of epinephrine would have a similar relation to positive affect as that of norepinephrine, but, in the present data this was not the case. Related research examining catecholamine response to acute psychological stress has also shown discrepant patterns of responding for measures of NOREPI and EPI. Specifically, scoring higher on the Beck Depression Inventory was associated with slower recovery for EPI, but not NOREPI, following an acute psychological stressor [35]. It is thought that varying responses for epinephrine and norepinephrine may reflect different aspects of specific stressors used [45]. Although speculative, the differential effects for measures of NOREPI and EPI in the present study may be partially accounted for by the fact that the experience of positive emotion may be coupled with less muscle sympathetic activity—perhaps leading to lower release of NOREPI, but not EPI.

The present data also suggest that the rise in salivary cortisol upon awakening is significantly lower for individuals who more frequently experience positive affect. A larger cortisol increase upon awakening has been associated with chronic stress e.g., [46], and depression e.g., [47]. More broadly, increased cortisol levels are known to be important for the development of numerous health problems e.g. [48], including cardiovascular disease [49]. Similar inverse relations between positive affect and cortisol have been reported in

several studies. Steptoe et al. [17] have reported associations between positive affect and salivary cortisol in a study using ecological momentary assessments (EMA) of happiness, as well as questionnaire ratings of affect. Specifically, in 72 healthy males EMA positive affect ratings were negatively associated with cortisol levels early in the day and with the increases in cortisol after awakening. Similarly, in another study Steptoe and Wardle [15] have shown that salivary cortisol is inversely associated with positive affect and the effect occurred primarily early in the day, as with the present findings. Finally, Lindfors and Lundberg [36] have shown that lower cortisol release during the morning hours, but not later in the day, is associated with a measure of positive well-being—again, a pattern of findings that parallel the present results.

Positive affect was unrelated to blood pressure and heart rate recovery. However, there was a trend ($p < .11$) for SBP recovery such that those with higher positive affect had an increased recovery. We would note that the limited period of recovery in the present study (i.e., only 5 minutes) may have contributed to the lack of an association. Indeed, a sizeable number of participants failed to fully recover, i.e., their recovery measures remained higher than those of the baseline period.

It is worth noting that in the present study negative affect was not associated with our physiological outcomes. This lack of association may have occurred, in part, due to the nature of the specific negative emotion we measured (i.e., depression-dejection). The present results might have varied somewhat if the negative emotion factor would have been a measure representing anger and/or hostility. However, we were unable to create a satisfactory negative emotion factor for anger/hostility using assessments from the POMS, VAS, and NEO-PI-R.

Apart from the measure of positive emotion, certain covariates were related to increased CVR and higher levels of NOREPI. In the present study African Americans were more likely to have increases in SBP and DBP during sadness recall as compared to Caucasian participants. Similar findings have been reported in other studies e.g., [50,51], a fact worth noting given the high prevalence of hypertension and cardiovascular disease in African Americans [52]. In the current study CVR was also associated with higher BMI and lower income, findings that also concur with results from prior studies e.g., [53,54], respectively. Finally, level of NOREPI was associated with age and gender. Although we would not have necessarily predicted these relations, results from animal models have reported age and gender associations with NOREPI function[55].

There are limitations that should be noted with regard to interpretation of the findings in the current study. Given the significant findings demonstrating a relation between cardiovascular reactivity and positive emotion during the sadness, but not the anger recall, a similar relationship between positive affect and sadness-induced catecholamine changes might have been expected. Unfortunately, catecholamine measures were not collected during the sadness recall. According to the design of the present study, approximately half of the participants were selected to be high or low on a measure of trait hostility. This selection criteria may have limited the generalizability of the present findings. The participants were in their early thirties and were selected to be in good health. Therefore, it is also possible that our findings may not generalize well to older or less well individuals. Likewise, the findings concerning CVR and norepinephrine were demonstrated in the laboratory portion of this study and results from ambulatory studies may better reflect relations between positive emotion and daily emotional stress. Finally, measures of blood pressure and heart rate reactivity were taken while participants were verbally recalling an emotional event. Thus, speaking is likely to have somewhat inflated the present measurement of reactivity.

Research from our laboratory [19] demonstrated that the positive emotion facet of the NEO-PI was a significant predictor of mortality in a sample of cardiac patients; and a measure related to positive emotions, specifically, expectations for successful recovery following a cardiac event, was associated with increased longevity in a model adjusted for depressive affect [56]. Others have demonstrated associations between measures of positive emotion and longevity, independent of negative emotion [57,58]. These associations between positive affect and the physiological measures relevant to good health examined in the present study suggest plausible biological mechanisms that may account for at least part of these demonstrated associations between positive emotion and greater longevity.

Acknowledgments

We would like to thank Dr. Michael Babyak for advice regarding the methodology used in the present manuscript.

This research was supported by the by National Heart, Lung, and Blood Institutes grant 3P01 HL036587; the Clinical Research Unit grant M01RR30l; NHLBI grant P01HL36587; and by the Behavioral Medicine Research Center Fund.

Acronyms

BMI	Body Mass Index
DBP	Diastolic Blood Pressure
SBP	Systolic Blood Pressure
HR	Heart Rate
EPI	Epinephrine
NOREPI	Norepinephrine
CVD	Cardiovascular Disease
CVR	Cardiovascular Reactivity

References

1. Smith TW. Hostility and health: Current status of a psychosomatic hypothesis. Health Psychology. 1992; 11(3):139–150. [PubMed: 1618168]
2. Smith, TW.; Gallo, LC. Personality Traits as Risk Factors for Physical Illness. In: Baum, A.; Revenson, T.; Singer, J., editors. Handbook of Health Psychology. Lawrence Erlbaum; Hillsdale: 2001. p. 139-172.
3. Sanderman R, Ranchor AV. The predictor status of personality variables: Etiological significance and their role in the course of disease. European Journal of Personality. 1997; 11:359–382.
4. Kaplan GA. Where do shared pathways lead? Some reflections on a research agenda. Psychosomatic Medicine. 1995; 57(3):208–212. [PubMed: 7652121]
5. Hemingway H, Marmot M. Psychosocial factors in the etiology and prognosis of coronary heart disease: systematic review of prospective cohort studies. BMJ. 1999; 318:1460–7. [PubMed: 10346775]
6. Argyle, M. The psychology of happiness. 2nd ed.. Routledge; New York, NY: 2001.
7. Chesney MA, et al. Positive emotions: exploring the other hemisphere in behavioral medicine. International Journal of Behavioral Medicine. 2005; 12:50–8. [PubMed: 15901213]
8. Diener E. Subjective well-being: The science of happiness and a proposal for a national index. American Psychologist. 2000; 55:34–43. [PubMed: 11392863]
9. Fredrickson BL. What good are positive emotions? Review of General Psychology. 1998; 2:300–319.

10. Folkman S, Moskowitz JT. Positive affect and the other side of coping. American Psychologist. 2000; 55:647–654. [PubMed: 10892207]
11. Lyubomirsky S, King L, Diener E. The benefits of frequent positive affect: Does happiness lead to success? Psychological Bulletin. 2005; 131:803–855. [PubMed: 16351326]
12. Mroczek DK, Kolarz CM. The effect of age on positive and negative affect: A developmental perspective on happiness. Journal of Personality and Social Psychology. 1998; 75:1333–1349. [PubMed: 9866191]
13. Scheier MF, et al. Optimism and rehospitalization after coronary artery bypass graft surgery. Archives of Internal Medicine. 1999; 159:829–35. [PubMed: 10219928]
14. Seligman MEP, et al. Positive psychology progress: Empirical validation of interventions. American Psychologist. 2005; 60:410–421. [PubMed: 16045394]
15. Steptoe A, Wardle J. Positive affect and biological function in everyday life. Neurobiology of Aging. 2005; 26S:S108–S112.
16. Steptoe A, Wardle J, Marmot M. Positive affect and health-related neuroendocrine, cardiovascular, and inflammatory processes. PANS. 2005; 102:6508–6512.
17. Steptoe A, et al. Neuroendocrine and cardiovascular correlates of positive affect measured by ecological momentary assessment and by questionnaire. Psychoneurodndocrinology. 2007; 32:56–64.
18. Richman L, et al. Positive emotion and health: Going beyond the negative. Health Psychology. 2005; 24:422–9. [PubMed: 16045378]
19. Brummett BH, et al. Ratings of positive and depressive emotion as predictors of mortality in coronary patients. International Journal of Cardiology. 2005; 20:213–6. [PubMed: 15823627]
20. Frijda, NH. The emotions. Cambridge University Press; Cambridge: UK: 1986.
21. Lazarus, RS. Emotion and adaptation. Cambridge University Press; Cambridge: UK: 1991.
22. Lazarus, RS.; Kanner, AD.; Folkman, S.; P. R.; Kellerman, H., editors. Theories of emotion. Academic Press; New York: 1980. Emotions: A cognitive- phenomenological analysis; p. 189-217.
23. Fredrickson BL, et al. The undoing effect of positive emotions. Motivation & Emotion. 2000; 24:237–258.
24. Gerin W, Pickering TG. Association between delayed recovery of blood pressure after acute mental stress and parental history of hypertension. Journal of Hypertension. 1995; 13:603–610. [PubMed: 7594416]
25. Linden W, et al. Physiological stress reactivity and recovery: Conceptual siblings separated at birth. Journal of Psychosomatic Research. 1997; 42:117–135. [PubMed: 9076640]
26. Newmann SA, Waldstein SR. Similar patterns of cardiovascular response during emotional activation as a function of affective valence and arousal and gender. Journal of Psychosomatic Research. 2001; 50:245–253. [PubMed: 11399281]
27. Warner RM, Strowman SR. Cardiovascular reactivity and positive/negative affect during conversations. J Behav Med. 1995; 18:141–159. [PubMed: 7563043]
28. Fredrickson BL, Levenson RW. Positive emotions speed recovery from the cardiovascular sequelae of negative emotions. Cognition and Emotion. 1998; 12:191–220.
29. Ong AD, Allaire JC. Cardiovascular intraindividual variablity in later life: the influence of social connectedness and positive emotions. Psychology and Aging. 2005; 20:476–485. [PubMed: 16248706]
30. Jacobs GD. The physiology of mind-body interactions: The stress response and the relaxation response. The Journal of Alternative and Complementary Medicine. 2001; 7:83–92. [PubMed: 11246939]
31. Fraser R, et al. Cortisol effects on body mass, blood pressure, and cholesterol in the general population. Hypertension. 1999; 33:1364–8. [PubMed: 10373217]
32. Epel, e.S.; Burke, HM.; Wolkowitz, OM. The psychoneuroendocrinology of aging: Anabolic and catabolic hormones. In: Aldwin, CM.; Park, CL.; Spiro, A., editors. Handbook of health psychology and aging. Guilford Press; New York: 2007. p. 119-141.

33. McEwen BS, et al. The role of adrenocorticoids as modulators of immune function in health and disease: neural, endocrine and immune interactions. Brain Research Reviews. 1997; 23:79–133. [PubMed: 9063588]
34. Bjorntorp P. Do stress reactions cause abdominal obesity and comorbidities? Obes Rev. 2001; 23:79–133.
35. Gold SM, et al. Higher Beck depression scores predict delayed epinephrine recovery after acute psychological stress independent of baseline levels of stress and mood. Biological Psychology. 2004; 67:261–273. [PubMed: 15294385]
36. Lindfors P, Lundberg U. Is low cortisol release an indicator of positive health? Stress and Health. 2002; 18:153–160.
37. McNair DM. Lorr M. An analysis of mood in neurotics. Journal of Abnormal and Social Psychology. 1964; 69:620–627.
38. Costa, PT.; McCrae, RR. Revised NEO Personality Inventory (NEO PI-R) and NEO Five-Factor Inventory (NEO-FFI). Psychological Assessment Resources; Odessa, FL: 1992.
39. Nyenhuis DL, et al. Adult and geriatric normative data and validation of the profile of mood states. J Clin Psychol. 1999; 55:79–86. [PubMed: 10100834]
40. Wewers ME, Lowe NK. A critical review of visual analogue scales in the measurement of clinical phenomena. Research in Nursing and Health. 2007; 13:227–236. [PubMed: 2197679]
41. Williams RB, et al. Childhood socioeconomic status and serotonin transporter gene polymorphism enhance cardiovascular reactivity to mental stress. Psychosomatic Medicine. 2008; 70:32–39. [PubMed: 18158371]
42. Williams, RB., et al. American College of Neuropsychopharmacology. Boca Ratan, Fl: 2007. Effects of Increased and Decreased Centeral Nervous System Serotonin Activity on Plasma Catecholamine Levels in Humans.
43. Williams RB. Blood pressure reactivity to psychological stress. A new risk factor for coronary disease? Hypertension. 2006; 47:329–330. [PubMed: 16461851]
44. Treiber FA, et al. Cardiovascular reactivity and development of preclinical and clinical disease states. Psychosomatic Medicine. 2003; 65:46–62. [PubMed: 12554815]
45. Biondi M, Picardi A. Psychological stress and neuroendocrine function in humans: the last two decades of research. Psychotherapy Psychosomatics. 1999; 68:114–150.
46. Schulz P, et al. Increased free cortisol secretion after wakening in chronically stressed individuals due to work overload. Stress Med. 1998; 14:91–97.
47. Pruessner M, et al. Self-reported depressive symptoms and stress levels in healthy young men: associations with the cortisol response to awakening. Psychososom Med. 2003; 65:92–99.
48. Luecken LJ, et al. Alterations in morning cortisol associated with PTSD in women with breast cancer. J Psychosom Res. 2004; 56:13–15. [PubMed: 14987959]
49. Girod JP, Brotman JD. Does altered glucocorticoid homeostasis increase cardiovascular risk? Cardiovasc Res. 2004; 64:217–226. [PubMed: 15485680]
50. Richman LS, et al. Discrimination, dispositions, and cardiovascular responses to stress. Health Psychology. 2007; 26:675–83. [PubMed: 18020838]
51. Covelli MM. Prevalence of behavioral and physiological risk factors of hypertension in African American adolescents. Pediatr Nurs. 2007; 33:323–4. 327–32. [PubMed: 17907733]
52. Stewart D, Johnson W, Saunders E. Hypertension in black Americans as a special population: why so special? Curr Cardio Rep. 2006; 8:405–10.
53. Steptoe A, Wardle J. Cardiovascular stress responsivity, body mass and abdominal adiposity. Int J Obes (Lond). 2005; 29:1329–37. [PubMed: 15953935]
54. Wilson DK, et al. Socioeconomic status and blood pressure reactivity in healthy black adolescents. Hypertension. 2000; 35:496–500. [PubMed: 10642348]
55. Snyder DL, et al. Effect of age, gender, rat strain, and dietary restriction, on norepinephrine release from cardiac synaptosomes. J Gerontol Series A: Biological Sciences and Medical Sciences. 1998; 53:B33–B41.
56. Barefoot, JC., et al. American Psychosomatic Society. Baltimore, MD: 2008. Recovery expectations of cardiac patients as predictors of survival. American Psychosomatic Society.

57. Blazer DG, Hybels CF. What symptoms of depression predict mortality in community-dwelling elders? Journal of American Geriatric Society. 2004; 52:2052–2056.

58. Ostir GV, et al. Emotional well-being predicts subsequent functional independence and survival. Journal of American Geriatric Society. 2000; 48:473–478.

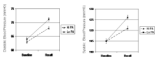

Figure 1.
Adjusted means for SBP and DBP reactivity during sadness recall: participants one standard deviation above and below the mean for positive affect (PA).

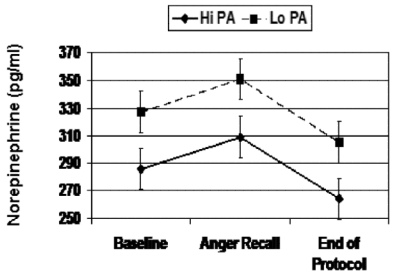

Figure 2.
Adjusted means from repeated measures analyses for norepinephrine during recall protocol: participants one standard deviation above and below the mean for positive affect (PA).

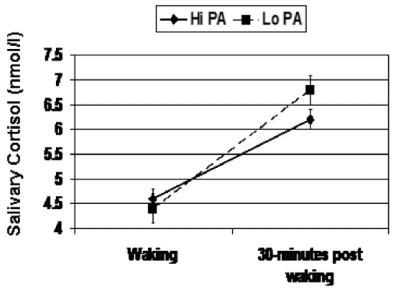

Figure 3.
Adjusted means for rise in salivary cortisol from awakening to post 30-minutes awakening: participants one standard deviation above and below the mean for positive affect (PA).

Table 1

Principal axes factor analysis: Loadings for positive affect and negative affect

Positive affect components	factor loadings
VAS “Happy” 1	0.66
VAS “Happy” 2	0.73
VAS “Happy” 3	0.66
VAS “Happy” 4	0.80
POMS “Friendly” 1	0.77
POMS “Friendly” 2	0.76
NEO-E6 Positive Emotion	0.50

Negative Affect components	factor loadings
VAS “Depressed” 1	0.67
VAS “Depressed” 2	0.73
VAS “Depressed” 3	0.68
VAS “Depressed” 4	0.80
POMS “Depression-Dejection” 1	0.75
POMS “Depression-Dejection” 2	0.80
NEO-N3 Depressed	0.51

Note: Correlation between positive and negative emotion factors r = -.34

Table 2

Sample Characteristics

Characteristic	Value
Age, mean (SD)	31.4 (8.8)
BMI, mean (SD)	26.4 (5.7)
Smoking Status, n (%) current smoker	38 (11.6%)
Annual Income level, median	$35,000-40,000
Resting SBP, mean (SD)	114.0 (12.6)
Resting DBP, mean (SD)	63.6 (7.8)
Resting HR, mean (SD)	70.0 (10.4)
Awakening Salivary Cortisol (nmol/l), mean (SD)	4.8 (2.5)
Baseline Epinephrine (pg/ml), mean, (SD)	27.4 (20.9)
Baseline Norepinephrine (pg/ml), mean (SD)	297.9 (132.9)
NEO-PI-R E-6 Facet "Positive Emotions", mean (SD)	54.1 (10.0)
NEO-PI-R N-3 Facet “Depression”, mean (SD)	50.3 (10.1)

Table 3

Results of models examining positive affect as a predictor of systolic and diastolic blood pressure reactivity during a sadness recall.

	Systolic Blood Pressure			Diastolic Blood Pressure		
	Standardized Estimate	**(SE)**	**p =**	**Standardized Estimate**	**(SE)**	**p =**
Intercept	4.6	6.7	0.498	10.8	3.5	0.002
Baseline (SBP OR DBP)	12.1	0.7	0.001	6.9	0.4	0.001
BMI	1.3	0.7	0.049	0.3	0.4	0.412
Income	-0.1	0.1	0.543	-0.2	0.1	0.002
Smoking Status (0)	2.3	1.9	0.214	0.4	1.2	0.758
Race (Caucasian)	-4.7	1.4	0.001	-2.8	0.9	0.002
Sex (F)	2.2	1.4	0.117	0.2	0.8	0.796
Age	0.8	0.7	0.249	0.1	0.4	0.042
Positive affect	-1.9	0.7	0.007	-0.9	0.4	0.049
Negative affect	0.3	0.7	0.618	0.1	0.4	0.960

Note: Income modeled as Categorical Rank Order.

Table 4

Results of model examining positive affect as a predictor of the average level of norepinephrine at baseline, during an anger recall task, and at the conclusion of the recall protocol.

	Level of Norepinephrine		
	Standardized Estimate	**(SE)**	**p =**
Intercept	13.4	0.3	0.001
BMI	-0.1	0.1	0.091
Income	0.0	0.0	0.834
Smoking Status (0)	-0.1	0.1	0.371
Race (Caucasian)	0.1	0.1	0.643
Sex (F)	-0.2	1.4	0.005
Age	0.3	0.1	0.001
Positive affect	-0.2	0.1	0.047
Negative Affect	-0.1	0.1	0.092

Table 5

Results of model examining positive affect as a predictor of rise in cortisol from awakening to 30-minutes post awakening.

	Rise in Cortisol		
	Standardized Estimate	**(SE)**	**p =**
Intercept	2.2	0.9	0.022
BMI	-0.0	0.2	0.996
Income	-0.0	0.1	0.370
Smoking Status (0)	-0.4	0.5	0.421
Race (Caucasian)	0.1	0.3	0.694
Sex (F)	0.4	0.3	0.235
Age	-0.0	0.2	0.939
Positive affect	-0.4	0.2	0.042
Negative affect	0.1	0.2	0.762