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Self-Esteem Variability Predicts Arterial Stiffness Trajectories in Healthy Adolescent Females

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Objective: There is mounting evidence that high levels of self-esteem are associated with better health outcomes, particularly in older adults dealing with serious medical illnesses. Much less is known about how this linkage unfolds developmentally, particularly during times like adolescence, when youngsters' self-views are typically in flux. Here we explore the self-esteem of adolescent females over a 2.5-year period, and how it covaries with trajectories of vascular function assessed over the same timeframe. ***Method:*** One-hundred and thirty adolescent females completed the Rosenberg Self-Esteem scale every 6 months for 2.5 years. Vascular function was measured three times over the same period, using peripheral artery tonometry. Indices of endothelial function and arterial stiffness were derived from these measurements. ***Results:*** Hierarchical Linear Modeling revealed an association between self-esteem variability and arterial stiffness trajectories, $\beta = 9.0 \times 10^{-3}$, $SE = 4.4 \times 10^{-3}$, $p = .04$. To the extent that their self-esteem fluctuated over the 2.5-year study, participants showed increasing trajectories of arterial stiffness, independent of various demographic and biobehavioral confounders. This association was also independent of participants' trait-like self-esteem over the same period of time. Neither trait self-esteem nor self-esteem variability was related to endothelial function. ***Conclusion:*** These findings suggest that fluctuating self-esteem may accelerate the early stages of vascular stiffening in young women, regardless of whether self-views are generally positive or negative.

Keywords: self-esteem, self-esteem variability, vascular function, arterial stiffness, adolescence

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Self-esteem (SE), generally defined as one's feelings of self-worth (Rosenberg, 1986), has long been considered an important determinant of psychological well-being (Taylor & Brown, 1988; but see Baumeister, Campbell, Krueger, & Vohs, 2003 for a contrary view). Given SE's presumptive influence on psychological well-being, researchers have speculated that it might also have consequences for physical health. Indeed, some research suggests that in patients with established medical illnesses, higher SE presages better adaptation, fewer complications, speedier recovery, and longer survival (Helgeson & Fritz, 1999; Scheier et al., 1999). There is also some evidence for a role of SE in the onset of medical problems in the general population. One large study of Finnish males assessed SE at baseline and tracked mortality for an average of 9.8 years thereafter (Stamatakis et al., 2003). Although SE predicted mortality in univariate analyses, this association became nonsignificant when other psychosocial characteristics, such as hopelessness, were entered into the model (Stamatakis et al., 2003).

Most of the extant research on SE and health problems focuses on later life stages, with little attention to how these dynamics play out developmentally. This is a significant oversight because the earlier decades of life are increasingly recognized as instrumental in establishing both SE and health life-course trajectories. Adolescence is a particularly rich time of life to study SE, as youths are actively engaged in forming identities (Markus & Nurius, 1986). Adolescents go through cycles that entail adopting new goals, confronting the reality that they are unattainable, and then adjusting expectations. Self-views, and well-being more generally, shift as these cycles progress (Wrosch & Miller, 2009). Adolescence is also an important period for establishing life span health trajectories. Many of the chronic illnesses of aging, particularly cardiovascular disease (CVD), are now recognized as life-course conditions. Autopsy studies indicate that atherosclerosis, the pathologic condition that underlies many clinical manifestations of CVD, begins during childhood and adolescence (Kavey, 2003). Youngsters also display risk factors for CVD, such as high blood pressure, altered blood lipids, and central obesity, that track into adulthood (Morrison, Friedman, & Gray-McGuire, 2007). These observations have spurred interest in identifying psychosocial conditions that, in childhood and adolescence, might contribute to the early stages of the atherosclerotic process (Matthews, 2005).

To our knowledge, one study has examined SE and health problems from a developmental perspective. Trzesniewski et al.

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(2006) assessed SE three times over a 4-year period in a sample of youth, at ages 11, 13, and 15, and then followed those participants to 26 years of age. Average SE ratings over the 4 years were prospectively inversely associated with adult health indicators: poorer cardiorespiratory fitness, larger waist-to-hip (WHR) ratios, and lower self-perceived fitness. These findings were independent of other known risk factors for adult health problems, including demographics, socioeconomic status (SES), depressed mood, and body mass index (Trzesniewski et al., 2006).

These are intriguing findings based on rigorous methodology. However, they do not address an important and interesting question: Given that SE fluctuates as part of normative adolescent development (Savin-Williams & Demo, 1983), to what extent do the vacillations themselves matter for health? The idea that SE variability (SEV) may be important for well-being, independent of trait SE, dates to at least the 1980s (Kernis, Grannemann, & Barclay, 1989). This work indicates that SEV is associated with a tendency to experience negative mood states (Kernis et al., 1989), such as depression (Franck & De Raedt, 2007), and lower subjective well-being, as reflected by indicators like autonomy, environmental mastery, purpose in life, and interest in goals (Kernis, Paradise, Whitaker, Wheatman, & Goldman, 2000; Paradise & Kernis, 2002). In addition to having direct effects, SEV appears to interact with trait SE to influence well-being. For example, individuals with high but unstable SE appear to be especially predisposed to negative psychosocial outcomes, such as lower self-acceptance and fewer positive relations with others (Paradise & Kernis, 2002). These findings have led some researchers to argue that SEV may be as or more important to well-being as trait SE (Kernis et al., 1989; Rosenberg, 1986).

Despite the wealth of evidence suggesting that SEV is important for well-being, its link to physical health outcomes, or the processes that give rise to them, has to our knowledge not yet been explored. Here we fill this gap by exploring SE dynamics in adolescence and how they relate to markers of vascular function that potentially reflect the early stages of atherosclerosis (for background on vascular function, see Holewijn, den Heijer, Stalenhoef, & de Graaf, 2010). We followed a sample of healthy young women over 2.5 years, during which SE was measured every 6 months and vascular function annually. Vascular dysfunction, as marked by endothelial function (EF) and arterial stiffness (AS), is one of the earliest noninvasive indicators of the pathogenic processes that ultimately give rise to CVD (Holewijn et al., 2010). EF captures the arterial endothelium's ability to control vasodilation through nitric oxide secretion; it is a measure of how quickly and efficiently the endothelium can respond to changing conditions (Davignon & Ganz, 2004). AS refers to the elasticity of the arterial walls or the ability to "bounce back" following a pressure wave (Safar, Levy, & Struijker-Boudier, 2003). Both low EF and high AS can reflect the early phases of atherosclerosis (Davignon & Ganz, 2004; Safar et al., 2003), and are predictive of clinical outcomes above and beyond other cardiovascular risk factors, such as blood pressure (Vlachopoulos, Aznaouridis, & Stefanadis, 2010). On the basis of the psychosocial evidence discussed above, we expected SE indices to predict trajectories of vascular function. Specifically, we expected that vascular function would decline most rapidly in adolescents with lower trait SE and more SEV. We also expected an interaction between these two facets of SE, such that persons with a combination of high but unstable SE would show increasing AS and declining EF over time.

Method

Participants

Data were collected as part of a larger project on depression and atherosclerosis in young women at risk for affective disorders. The participants were recruited from Vancouver, BC, through various advertisements. Eligibility criteria were (a) female, 15–19 years old, and fluent in English; (b) free of acute illness and standing medication in the past 2 weeks, other than oral contraceptives; and (c) without a history of chronic medical or psychiatric disorders. A total of 147 participants recruited to be at risk for an initial major depressive episode were enrolled. High-risk was defined as having a first-degree relative with a history of affective disorder and/or scoring in the top quartile of the population on cognitive vulnerability to depression. In order to be included in the present analyses, we specified a priori that participants had to complete three of the six SE assessments and two of the three vascular function assessments. These were the minimum number of data points we needed to compute SEV and model vascular function trajectories. When these criteria were imposed, 17 of the 147 participants were considered lost to attrition (12.0%): seven participants withdrew, five moved away, and seven lost contact. These participants did not differ from those who met inclusion criteria for our analyses on any variable included, such as average SE, $t(155) = 0.89$, $p = .37$, SEV, $t(143) = -1.17$, $p = .24$, average AI, $t(143) = 0.45$, $p = .66$, and average RHI, $t(143) = 0.29$, $p = .79$. The final analytic sample consisted of 130 young, healthy female participants. All procedures and methods were approved by the University of British Columbia Research Ethics Board. Written consent was obtained from all participants prior to participation. For participants younger than 18 years of age, parent or guardian consent was also obtained.

Procedure

Participants visited the research center every 6 months over a 2.5 year period. To control for diurnal variations, sessions always occurred between 8 a.m. and 11 a.m., following an overnight fasting period. Participants were asked not to engage in any exercise or smoke cigarettes the morning of each visit. After obtaining informed consent, participants completed a series of interviews and questionnaires, described in more detail below, which provided information regarding demographics, mood states, health practices, and biobehavioral characteristics. Vascular function assessments were performed at visits 2, 4, and 6.

Measures

Self-esteem variability and trait self-esteem. At each visit, participants completed the Rosenberg Self-Esteem Scale (Rosenberg, 1965), which consists of 10 statements about the self that participants respond to using a 7-point scale (*strongly agree* to *strongly disagree*). Items include such statements as "I wish I could have more respect for myself." Possible scores range from 10 to 70. The SE scale was internally consistent, with an average

Cronbach's alpha of 0.91 across visits, and a range of 0.90–0.92. Trait SE was calculated by taking the average across all visits. SEV was calculated by taking the standard deviation (SD) of each participant's SE scores across all visits. SEV and trait SE were inversely correlated, $r = -.22$, $p = .01$.

To clarify what high SEV might reflect, we also calculated the maximum "rise" and "dip" in SE that each participant experienced over the project. SE rise was calculated by subtracting each participant's average SE from her maximum SE. SE dip was calculated by taking the absolute value of the difference between the participants' average SE and minimum SE. SEV was associated with both rise, $r = .89$, $p < .01$ and dip scores, $r = .91$, $p < .01$.

Vascular function. Vascular function was assessed using the EndoPAT2000 (Itamar Medical Ltd, Caesarea, Israel), a noninvasive technology that provides a beat-to-beat plethysmographic recording of the finger arterial pulse-wave amplitude (PWA) using pneumatic probes (Bonetti et al., 2004). EndoPAT finger PWAs are reliably associated with radial and carotid artery pressure waveforms (Millasseau et al., 2000), and are highly related to other arm-based and centrally derived vascular function recordings (Dhindsa, Barnes, DeVan, Sugawara, & Tanaka, 2011; Haller, Silverstein, & Shuster, 2007; Heffernan, Patvardhan, Karas, & Kuvin, 2011). EndoPAT assessments occurred at 6 months, 1.5, and 2.5 years after study entry using the protocol recommended by the manufacturer (Itamar Medical Ltd, Caesarea, Israel). Participants were seated with arms placed at heart level. A pneumatic probe was placed on the index finger of each hand and, after the PWA signal had been acquired, the participant rested quietly for 5 minutes, during which resting or baseline EF was assessed. Reactive hyperemia was induced when a blood-pressure cuff, placed around the participant's nondominant arm, was inflated to 60 mmHg above their systolic blood pressure. After a 5-minute occlusion period the cuff was rapidly deflated, inducing reactive hyperemia. The PWA signal was recorded for the next 5 minutes on both the occluded and nonoccluded arms.

The data were analyzed with a computerized, operator-independent, automated algorithm supplied by Itamar Medical (Version 2.3.2) that standardizes artifact detection and computational procedures. The software computed a measure of EF: the reactive hyperemia index (RHI), as described elsewhere (Tomfohr, Martin, & Miller, 2008). RHI is calculated from baseline PWA, defined as the average for the final 2.5 minutes of the baseline period, and postocclusion PWA, defined as 1.5 to 2.5 minutes after cuff deflation. Additional information on the RHI and its calculation can be found in this article's online supplementary material.

The EndoPAT software also provides an indirect measure of AS, the augmentation index (AI). It captures the contribution of wave reflections to arterial pressure waveforms, and is defined as the ratio of the difference between the first and second systolic peaks of the waveform assessed during the 5 minute baseline period.[1] The computerized algorithm provided by Itamar identifies peak volume and inflection points, calculating them from a fourth-order derivative as described elsewhere (Kelly, Hayward, Avolio, & O'Rourke, 1989). Higher AI scores are indicative of greater AS. Concurrent validity studies indicate that EndoPAT AI is highly correlated with other AI assessment techniques, including those measured from the aorta and radial artery (Dhindsa et al., 2011; Haller et al., 2007; Heffernan et al., 2011). EndoPAT AI also has high correlations with ventricular-vascular coupling, systemic vascular function (Heffernan et al., 2011; Heffernan et al., 2010), age-related changes in vascular function, and target organ damage (Heffernan, Patvardhan, Kapur, Karas, & Kuvin, in press). EndoPAT AI also demonstrates high levels of reproducibility in healthy adults (ICC = 0.84; McCrea, Skulas-Ray, Chow, & West, 2012).

Alternative explanations. Any link between SE and vascular function could reflect the influence of confounds. To minimize the odds of detecting a spurious association, we statistically controlled for variables that are known correlates of both SE and vascular function: demographic characteristics (age, ethnicity, SES), WHR, depressed mood (as measured by the Beck Depression Inventory, see below), and health behaviors: daily physical activity, alcohol consumption, and cigarette use. SES was indexed by the highest years of education completed by either the participant's mother or father.

Waist-to-hip ratio (WHR). WHR is a marker of central adiposity and is associated with atherosclerosis (Turkbey et al., 2010), and low SE during adolescence (Strauss, 2000). Waist circumference was measured from the side at the midpoint between the upper iliac crest and lower costal margin at the midaxillary line using a standard measuring tape. Hip circumference was taken at the hips on the widest part of the buttocks. Measurements were taken at least twice, until a consistent reading was obtained. The ratio was calculated by dividing the waist by the hip measurement.

Depressed mood. Low SE and high SEV have been associated with depression (Franck & De Raedt, 2007), and mood problems increase risk of CVD (Seldenrijk et al., 2011). To minimize the chances that depression may inflate any observed association, we had participants complete the 21-item Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) at all six visits. The BDI is a widely used measure of depressive symptom intensity experienced during the past week. The BDI was internally consistent in our sample, with an average Cronbach's alpha of 0.93 across visits. Besides controlling for average levels of depressive symptoms, we also examined the potential confounding role of mood variability. This was done by calculating the SD of each participant's BDI scores across the study.

Health behaviors. Health behaviors, such as physical activity, alcohol consumption, and cigarette use, may also impact vascular function and SE. Self-reported physical activity has been associated with both AS (Boreham et al., 2004) and SE (Calfas & Taylor, 1994) in young adults. Physical activity was assessed at each visit using the Paffenbarger Activity Scale (Paffenbarger, Blair, Lee, & Hyde, 1993). Participants were asked if at least once a week they engaged "in regular activity akin to a brisk walking, jogging, bicycling, and so forth long enough to work up a sweat" and, if so, how many times a week and for how long each time. Average weekly physical activity was calculated by multiplying "how many times per week" by "for how long each time." The Paffenbarger Scale has been validated in previous research (Paffenbarger et al., 1993).

Alcohol consumption and smoking habits are associated with increased risk of CVD and arterial stiffness (Kannel, D'Agostino,

[1] This is recorded and expressed as a percentage: (P2 − P1)/P1 × 100

& Belanger, 1987; Steinberg, Pearson, & Kuller, 1991), and low SE (Glendinning, 2002; Glindemann, Geller, & Fortney, 1999). Smoking status was assessed at each visit by asking participants if they smoked (yes/no) and, if "yes," to provide the number of cigarettes smoked per day. Over the study only three of our 147 participants could be classified as smokers (defined as someone who uses at least 10 cigarettes a day), and so smoking was excluded from our analyses. Alcohol consumption was recorded as the number of beverages consumed in a typical week-long period. A drink was considered one glass of wine, one 12-ounce beer, or one shot of hard liquor.

To simplify the statistical models, WHR, BDI and health behavior variables were modeled as between-subjects covariates, with values based on average responses over the six waves of assessment. Models in which these variables were treated as time-varying yielded identical conclusions.

Results

Preliminary Analyses

Sample characteristics are presented in Table 1. The sample consisted of healthy adolescent females who were primarily of Asian and European descent. Participants' parents had on average a university education, indicating that as a whole the sample was of moderate to high SES. On average, RHI scores were within the normal range established by the manufacturer (> 1.67), suggesting healthy EF. As of yet, no normal value cut-offs have been provided for AI.

To examine how SE fluctuated over the study we computed an intraclass coefficient (ICC) for SE, which was estimated to be .64. This indicates that roughly two-thirds of the SE score variance over time was attributable to between-person sources. The other one-third of the variance derives from within-person fluctuations, which are a combination of stable intraindividual changes and person by time interactions. Conceptually, this ICC value suggests it is appropriate to view SE as a construct that fluctuates around what is for most people a fairly stable trait. ICCs were also calculated for vascular function indices, .45 and .34 for AI and RHI respectively, indicating that there was a substantial amount of within-participant variability in these indicators over the follow-up. These figures suggest that there is sufficient variability in vascular health trajectories within the sample to justify exploring the potential influence of SE indices.

Table 1
*Characteristics of the Sample (*N *= 130)*

Variable	Mn +/− *SD* or Number (%)
Ethnicity	
European descent	64 (49)
Non-European descent	66 (51)
Age (years)	17.2 +/− 1.4
SES (highest parental years of education)	15.9 +/− 3.3
Average WHR	0.75 +/− 0.04
Average BDI score	7.1 +/− 5.6
Mean self-esteem	46.3 +/− 4.6
Self-esteem variability (*SD*)	5.1 +/− 2.8
Average weekly brisk physical activity (minutes)	123 +/− 125
Average alcohol consumption (drinks/week)	1.5 +/− 5.4
Arterial stiffness	
Visit 1 AI (Range)	−0.78 +/− 14 (−26–58)
Visit 2 AI (Range)	−2.8 +/− 11 (−22–43)
Visit 3 AI (Range)	−2.0 +/− 14 (−26–35)
Endothelial function	
Visit 1 RHI (Range)	2.0 +/− 0.56 (1.1–4.0)
Visit 2 RHI (Range)	2.0 +/− 0.65 (1.0–4.3)
Visit 3 RHI (Range)	1.9 +/− 0.48 (1.1–3.3)

Primary Analyses

To model relations between indices of SE and trajectories of vascular function, we estimated a series of multilevel models with the HLM 6.08 software package (Raudenbush, Bryk, & Congdon, 2004). Separate models were constructed for RHI and AI scores. The within-person (Level 1) models included a time since study entry variable (in months) and a random error term. The between-person (Level 2) models included relevant SE indices and four standard control variables: age, ethnicity (European descent vs. non-European descent), parental education, and mean WHR. In follow-up analyses we examined alternative explanations by adding depressive symptoms and health behaviors to Level 2. In all cases we estimated random slope models, in which Level 2 error terms were allowed to vary freely. Models were estimated with restricted maximum likelihood and robust standard errors.

Standard control variables. To estimate the extent to which standard control variables explained changes in vascular function over time, we constructed models that included time from study entry at Level 1, and age, ethnicity, parental education, and WHR at Level 2. Of the control variables, only ethnicity was associated with AI at baseline, $\beta = 0.55$, $SE = 0.18$, $p < .01$, with participants of non-European descent showing higher arterial stiffness scores at study entry. None of the control variables were associated with AI trajectory, and all control variables were unrelated to RHI scores at baseline and over follow-up (see Supplemental Table 1).

Trait self-esteem. Next, we evaluated whether trait SE was predictive of vascular function trajectories. The models included the standard control variables at level 2 along with a trait SE variable (see Table 2). Ethnicity continued to predict AI at baseline, $\beta = .55$, $SE = .18$, $p < .01$, but no other control variable was associated with AI or RHI either at baseline or over time. Trait SE was unrelated to baseline levels of both AI, $\beta = 5.2 \times 10^{-3}$, $SE = 0.02$, $p = .79$, and RHI, $\beta = 7.9 \times 10^{-3}$, $SE = 0.01$, $p = .50$. It was also unrelated to trajectories of AI, $\beta = 7.0 \times 10^{-5}$, $SE = 9.7 \times 10^{-4}$, $p = .94$, and RHI, $\beta = -4.5 \times 10^{-4}$, $SE = 5.3 \times 10^{-4}$, $p = .40$, over the follow-up period.

Self-esteem variability. Next we ran models where SEV was included at Level 2, to examine its association with vascular function trajectories. SEV was not associated with RHI scores at baseline, $\beta = 1.5 \times 10^{-2}$, $SE = 8.2 \times 10^{-2}$, $p = .86$, or over time, $\beta = 4.4 \times 10^{-3}$, $SE = 4.5 \times 10^{-3}$, $p = .33$ (see Table 3).

In models predicting AI trajectories, none of the covariates was a significant predictor, other than the previously discussed association between ethnicity and baseline AI, $\beta = 0.57$, $SE = 0.18$, $p < .01$. However, SEV was positively associated with AI trajectories, $\beta = 8.6 \times 10^{-3}$, $SE = 4.4 \times 10^{-3}$, $p = .05$, such that participants who had more SEV also had increasing AI over the 2-year follow-up (see Figure 1). Conversely, participants with less SEV

Table 2
Results of HLM Model Predicting Changes in AI and RHI Over Time From Control Variables and Trait Self-Esteem

Variable	Intercept (Baseline) β	*SE*	*p*	Slope (Trajectory) β	*SE*	*p*
Arterial stiffness (AI)						
Ethnicity	0.55	0.18	<.01	1.9×10^{-3}	9.6×10^{-3}	.85
Age	4.0×10^{-2}	6.7×10^{-2}	.55	2.6×10^{-3}	3.5×10^{-3}	.45
SES	1.1×10^{-2}	2.6×10^{-2}	.66	5.1×10^{-4}	1.4×10^{-3}	.72
WHR	−1.7	2.0	.39	-2.8×10^{-2}	0.10	.79
Trait self-esteem	5.2×10^{-3}	1.9×10^{-2}	.79	7.0×10^{-5}	9.7×10^{-4}	.94
Endothelial function (RHI)						
Ethnicity	-2.4×10^{-2}	0.10	.82	5.0×10^{-3}	5.6×10^{-3}	.37
Age	-3.1×10^{-2}	3.9×10^{-2}	.43	1.1×10^{-3}	1.9×10^{-3}	.55
SES	-6.8×10^{-3}	1.4×10^{-2}	.82	-7.9×10^{-4}	6.9×10^{-4}	.25
WHR	1.2	0.99	.22	-2.8×10^{-2}	6.5×10^{-2}	.67
Trait self-esteem	7.9×10^{-3}	0.01	.50	-4.5×10^{-4}	5.3×10^{-4}	.40

showed a decline in AI over time. Further analysis indicated that this association was independent of trait SE: After trait SE scores were added to Level 2, SEV continued to predict AI trajectories in the same manner, $\beta = 9.0 \times 10^{-3}$, $SE = 4.4 \times 10^{-3}$, $p = .04$ (see Table 3). A local effect size was calculated by determining the proportional reduction in variance (PRV) when SEV was and was not included in the model (Peugh, 2010). The PRV value for SEV was .24, indicating that, above and beyond the standard control variables, SEV explained 24% of the between-person variance in AI trajectories over the follow-up period. To ensure that the covariates were not driving this association, we also ran an unadjusted model wherein SE indices alone were used to predict AI trajectories. The results were identical, such that SEV alone predicted changes in AI over time, $\beta = 9.4 \times 10^{-3}$, $SE = 4.3 \times 10^{-3}$, $p = .03$, but SE did not, $\beta = 1.4 \times 10^{-3}$, $SE = 3.4 \times 10^{-3}$, $p = .68$.

To clarify the meaning of these results, we modeled links between maximum SE rise and dip for each participant and AI trajectories using the same analytic strategy. Largest SE dips were positively associated with AI trajectories over time, $\beta = 1.2 \times 10^{-2}$, $SE = 5.6 \times 10^{-3}$, $p = .03$, but largest rises were not, $\beta = -3.1 \times 10^{-3}$, $SE = 6.4 \times 10^{-3}$, $p = .63$. These findings suggest that the SEV results are a reflection of largest within-person dips in SE.

Interaction. Previous research indicates that high, unstable SE is associated with psychological distress (Paradise & Kernis, 2002). Thus, we considered whether the interaction between SE and SEV might predict vascular function trajectories (see Supplementary Table 2). However, a product term reflecting the interaction of these variables was not associated with baseline levels or trajectories of either RHI or AI (p's $> .30$).

Alternative Explanations

The final models evaluated alternative explanations. SEV's association with trajectories of AI remained statistically significant after we included depressive symptoms and variability in depression scores over the follow-up, $\beta = 1.2 \times 10^{-2}$, $SE = 5.4 \times 10^{-3}$, $p = .03$, and health practices, like alcohol consumption, $\beta = 1.2 \times 10^{-2}$, $SE = 5.0 \times 10^{-3}$, $p = .02$, and physical activity, $\beta = 1.2 \times 10^{-2}$, $SE = 5.0 \times 10^{-3}$, $p = .02$, in the Level 2 models (see Supplementary Table 3).

Table 3
Results of HLM Model Predicting Changes in AI and RHI Over Time From SEV and Control Variables (Including Trait Self-Esteem)

Variable	Intercept (Baseline) β	*SE*	*p*	Slope (Trajectory) β	*SE*	*p*
Arterial stiffness (AI)						
Ethnicity	0.57	0.18	<.01	-2.6×10^{-4}	9.7×10^{-3}	.98
Age	0.04	0.08	.61	3.2×10^{-3}	4.1×10^{-3}	.44
SES	0.01	0.03	.62	1.3×10^{-4}	1.3×10^{-3}	.93
WHR	−1.8	1.6	.27	−0.03	0.08	.72
Trait self-esteem	4.4×10^{-3}	0.02	.81	4.3×10^{-4}	7.7×10^{-4}	.57
SEV	−0.02	0.07	.78	9.0×10^{-3}	4.4×10^{-3}	.04
Endothelial function (RHI)						
Ethnicity	-1.4×10^{-2}	0.10	.89	4.2×10^{-3}	5.7×10^{-3}	.46
Age	-2.7×10^{-2}	4.0×10^{-2}	.49	1.2×10^{-3}	1.9×10^{-3}	.54
SES	-5.3×10^{-3}	1.4×10^{-2}	.70	-9.5×10^{-4}	7.1×10^{-4}	.18
WHR	1.1	1.0	.28	-2.4×10^{-2}	6.4×10^{-2}	.71
Trait self-esteem	8.6×10^{-3}	1.0×10^{-2}	.39	-3.7×10^{-4}	5.2×10^{-4}	.48
SEV	1.5×10^{-2}	4.5×10^{-2}	.74	2.2×10^{-3}	2.5×10^{-3}	.37

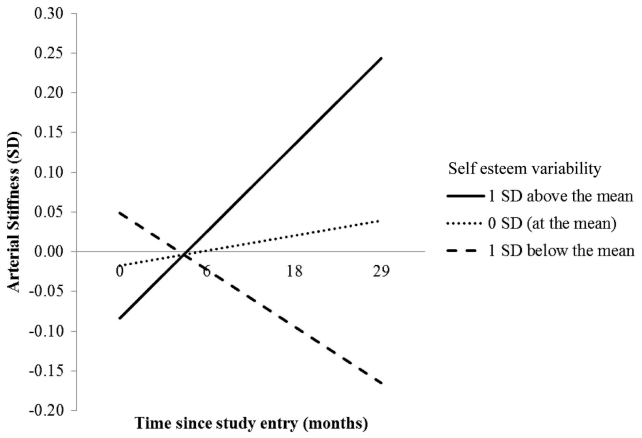

Figure 1. Trajectories of arterial stiffness over 2 years as a function of self-esteem variability. Estimated arterial stiffness values are plotted at the sample's mean level of SEV, as well as one standard deviations (*SD*s) above and below it. The values are adjusted for age, ethnicity, SES, WHR, and trait self-esteem.

Discussion

The purpose of this study was to examine how patterns of SE and vascular function covary during the transition into adulthood. We followed adolescent females over 2.5 years, tracking SE every 6 months and vascular function annually. SEV was associated with trajectories of AS over time, over and above trait SE, with participants who had more SEV exhibiting increased AS over the follow-up. Conversely, participants with less SEV had decreasing AS. Further analyses indicated that drops in SE drove this association, with participants who had the largest dips from their trait level displaying the most upward trajectories of AI. By contrast, trait SE was not predictive of vascular function trajectories over time, and there was no evidence that high but unstable SE was either.

These findings have interesting theoretical implications. They suggest that during the late adolescent years, fluctuations in SE covary with trajectories of vascular health, but absolute levels of self-worth do not. Why might this be the case? We believe it is because adolescents are actively forming personal identities. As part of doing so, they often experience both successes and failures with corresponding fluctuations in SE that would not be captured by aggregating SE scores across multiple assessments. By contrast, SEV captures these fluctuations, and thus, provides a reflection of teenagers' success in navigating toward self-relevant goals. It is possible that the relative importance of these SE indices varies across the life span, with SEV acting as an effective barometer for adolescents in the middle of identity formation, and trait SE taking on this function in adulthood, when people have already solidified their identities and established stable views of the self (Trzesniewski, Donnellan, & Robins, 2003).

The mechanism(s) responsible for the association between SEV and AS remain unclear. From the covariance analyses presented above, we can be reasonably confident that health-related behaviors, like smoking, drinking, and exercise, are not primary mediators. Variations in SE may be triggered by psychosocial stressors, like interpersonal difficulties (Cambron, Acitelli, & Steinberg, 2010) or by normative developmental challenges associated with goal pursuit (Wrosch & Miller, 2009). Biologically, these variations may influence outflow of both sympathetic and parasympathetic nervous systems. These systems are known to regulate vascular tone, both through direct innervation of the vascular muscle, causing vasoconstriction or dilation, respectively (Martens, Greenberg, & Allen, 2008), and through action of the parasympathetic neurotransmitter acetylcholine on vascular endothelial cells, producing endothelium-dependent vasodilation (Harris & Matthews, 2004). Thus, variations in SE could have acted through autonomic mechanisms (Martens et al., 2008) to modify AS trajectories in our sample. This hypothesis will need to be evaluated in future research.

That said, if SE fluctuations trigger the autonomic mechanism(s) discussed above, they would also be expected to affect EF trajectories. It is not clear why we do not see such a relationship in this sample. In general, the same processes that cause AS to increase also cause EF to decline (Zieman, Melenovsky, & Kass, 2005). There remains, however, ambiguity regarding the temporal ordering of these changes, and it may differ across individuals. EF and AS are only moderately correlated in adults (Nigam, Mitchell, Lambert, & Tardif, 2003), and their progression differs across individuals, with evidence for each preceding and following the other (Zieman et al., 2005). In this sample of healthy young women, we may have captured an early stage of vascular dysfunction that is manifested in variations in AS but is not yet evident in EF. If this is the case, SEV may forecast declines in EF over longer periods of time. Future research is needed to explore this possibility.

There were several limitations in this study. First, our sample consisted of adolescent females who were enrolled because they were at high risk for depression. It remains unclear how well the findings will generalize to the population at large, particularly to males. However, past work on SEV has predicted psychosocial adjustment in both sexes (Kernis et al., 1989), so there is precedent for hypothesizing the findings will prove generalizable. Regardless, further study is needed to assess this. Second, we used a fairly new method that assessed AS in the finger. Most previous research has focused on central or aortic stiffness (e.g., Weber et al., 2004). Research suggests that indices of finger AS relate to central pulse wave velocity in both healthy and patient samples (Woodman et al., 2005), but more research is needed with this specific methodology before any conclusions about prognostic significance are possible. Finally, the structure of our analyses leaves open questions about cause and effect. Consistent with our hypotheses, SEV covaried with AS over the 2.5-year study period, but we cannot be certain about the direction of causality or whether this association persists beyond the study end. Additional long-term follow-ups with repeated vascular function assessments would be necessary to sort out this issue.

In the meantime, these findings suggest that variations in SE, and in particular dips, may accelerate the early stages of vascular dysfunction in otherwise healthy young women, and do so regardless of how positively or negatively these women typically view themselves. These results converge with previous research linking SEV to well-being, and extend this phenomena to a biological process implicated in the development of CVD. Our findings also highlight the utility of adopting a dynamic

approach to the conceptualization and assessment of SE in behavioral medicine research, particularly when such work is being conducted in a developmental context like adolescence.

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