

## Abstract 19

### A Randomized Controlled Trial of the Effect of Hostility Reduction on Cardiac Autonomic Regulation

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**Objective:** To test whether reduction in hostility increases autonomic regulation of the heart.

**Methods:** In this randomized controlled trial, participants were 158 healthy adults, 20 to 45 years of age, who were one standard deviation (SD) above national norms on the Cook-Medley Hostility Scale and the Spielberger Trait Anger Index. Participants also were interviewed using the Interpersonal Hostility Assessment Technique (IHAT). They were randomly assigned to a 12-week cognitive behavior therapy (CBT) program for hostility reduction or a wait-list control condition. The main outcome measure was cardiac autonomic modulation, measured as

RR interval variability (RRV) derived from 24-ECG recordings.

**Results:** In a MANOVA assessing psychological outcomes of hostility, anger, and IHAT scores, there was a significant treatment effect with an average reduction across the three outcomes that was approximately 0.7 SD (ES = 0.685, se = 0.184,  $P < 0.001$ ) greater for the intervention group than for the control group.

In contrast, the change in HR was  $-0.14$  bpm (95% CI,  $-2.43$  to  $2.14$ ) in treatment participants and  $-1.36$  bpm (95% CI,  $-3.28$  to  $0.61$ ) in wait-list participants. HF RRV, an index of cardiac parasympathetic modulation, increased by  $0.07$  ln msec<sup>2</sup> (95% CI,  $-0.10$  to  $0.24$ ) for participants in the treatment condition and decreased by  $0.04$  ln msec<sup>2</sup> (95% CI,  $-0.18$  to  $0.10$ ) for participants in the wait-list condition. These differences were not significant. The findings for other indices of RRV were similar.

**Conclusions:** Reduction of hostility and anger was not accompanied by increases in cardiac autonomic modulation. These findings raise questions about the status of disordered ANS regulation of the heart as a pathophysiological mechanism underlying the hostility–heart disease relationship and about whether hostility itself is a mechanism or merely a marker of elevated risk of heart disease.