Abstract 29

Trait Hostility Is Associated With Endothelial Cell Apoptosis in Healthy Adults

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Background: Trait hostility is associated with increased risk of incident cardiovascular disease (CVD) events. The underlying biologic mechanisms remain poorly characterized. Endothelial cell-derived microparticles (EMPs) are phospholipid-rich, submicron particles shed from the membranes of activated or apoptotic endothelial cells (ECs). EMPs play an important role in the pathobiology of atherosclerosis formation and thus CVD development by inhibiting nitric oxide bioavailability, promoting inflammation via leukocyte activation and transendothelial migration, and activating the coagulation cascade. The relation between trait hostility and EMPs in healthy adults without CVD is unknown.

Methods: Twenty-seven apparently healthy participants (age 37 ± 12 years, 63% female) without any clinical evidence of CVD, hypertension, diabetes, hypercholesterolemia, smoking, family history of premature CVD, rheumatologic disorders, active or recent infection, or any chronic medication use (including over-the-counter drugs or herbal medications) were recruited. Circulating EMPs in blood were assessed by flow cytometry. EMPs were defined as the number of particles with a diameter less than 1.5 μm that were negatively labeled by fluorescein isothiocyanate-conjugated monoclonal antibody to CD42b (specific to platelets) and positively labeled by phycoerythrin-conjugated monoclonal antibody to CD31 (EC apoptosis marker), CD51 (EC apoptosis and activation marker) or CD62E (EC activation marker). Hostility was assessed using the 50-item Cook-Medley Hostility scale, from which the Barefoot Hostility (Ho) 27-item score was calculated.

Results: There was a significant relationship between hostility and levels of EMPs expressing CD31 (beta = .409, P = .034) and CD51 (beta = .399, P = .039), but not CD62E (P = .170). The relationships were similar after adjusting for age, sex, and body mass index (for CD31, beta = .681, P = .007; for CD51, beta = .524, P = .036; for CD62E, P = .240).

Conclusion: These findings demonstrate that higher trait hostility scores are associated with greater circulating levels of EMPs, mostly phenotypic for EC apoptosis. Given the important role of EMPs in the pathobiology of atherosclerosis, these findings suggest that trait hostility may contribute to incident CVD events through EC injury and death.

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