Title

Aging in the Context of Chronic Disease: Premature, Accelerated, Normal, or Successful?

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cardiovascular emotional dampening may reduce threat appraisal and increase high risk behavior. This effect is most apparent in young men the blood pressure/risk relationship could be bidirectional. The effect of blood pressure on risk behavior could contribute to increased disease risk later in life.

Abstract 1688

ACTIVATION CONTROL: HEIGHTENED OR BLUNTED CARDIAC SYMPATHETIC REGULATION?

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Activation control (AC) describes the ability to mobilize a behavior against strong inclination to not do so and likely predicts reduced susceptibility to stress (Evans & Rothbart, 2007). The link between heightened cardiac sympathetic regulation (CSR) and chronic stress supports the notion that CSR is negatively coupled with AC (Cacioppo et al., 1998). Alternatively, augmented CSR may predict high AC, due to the role the former plays in effort mobilization (Gendolla et al., 2012). In the current study, the relation between CSR and AC was explored using a pooled cross-sectional approach. Eighty-four volunteers (mean age = 19.6, SD = 1.8) completed the AC scale of the Adult Temperament Questionnaire. ECG and impedance cardiography (ICG) were recorded while subjects completed a mental arithmetic, verbal fluency, and speech preparation task. Each task was preceded and followed by baseline and recovery periods. Interbeat interval (IBI) was derived from the ECG to index heart period, while pre-ejection period (PEP) was calculated from the ICG to index cardiac sympathetic activation. Reactivity and recovery scores were computed and entered into regression analyses. Results indicate that AC moderated the relation between PEP and IBI reactivity across tasks, $\beta = -.375$ $t(78) = -2.41$, $p = .018$. IBI was more strongly related to PEP reactivity at low levels of AC, $\beta = .634$ $t(78) = 4.19$, $p < .001$, compared to high levels of AC, $\beta = .132$ $t(78) = .956$, $p = .342$. These results suggest that AC is negatively related to CSR, supporting models linking behavioral regulation to inhibition of sympathetic activity (Thayer & Lane, 2000). These findings also suggest that increased sympathetic cardiac activity during effort in previous studies (Gendolla et al., 2012) may reflect approach tendencies independent of regulatory mechanisms (Derryberry & Rothbart, 1997).

Abstract 1514

DEPRESSIVE SYMPTOMS ARE ASSOCIATED WITH WORSENING OF ARTERIAL STIFFNESS WITH MENTAL STRESS

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Introduction: Arterial stiffness is an important marker of cardiovascular risk which worsens with acute mental stress. Psychological conditions, particularly depression, have been linked to recurrent cardiac events and death, but the mechanisms are unclear. We examined whether depressive symptoms, anxiety and anger worsen arterial stiffness induced by mental stress.

Methods: In 81 subjects with a history of MI in the previous 6 months, we used the SphygmoCor® Pulse Wave Velocity system at rest and 60 minutes after a standardized psychological stress (via speech task) and after a conventional physical (exercise or pharmacological) stress test. The central augmentation index (CAIx) was derived by pulse wave analysis software and the difference between CAIx after each stress condition and the respective resting phase was calculated. Depressive symptoms were assessed with the Beck Depression Inventory-II (BDI-II), state and trait anxiety with the State-Trait Anxiety Inventory (STAI), and state and trait anger with the State-Trait Anger Expression Inventory (STAXI-II). Linear regression models were used to model the association between change in CAIx with each stress (dependent variable) and BDI total score, anxiety and anger subscales as individual predictors, adjusting for potential confounding factors.

Results: Forty-one subjects were≤ 50 years of age, 41 were female and 46 were non-white. Systolic and diastolic blood pressure and heart rate significantly increased in response to mental stress (48 ± 23 and 30 ± 13 (mmHg), and 28 ± 18 (bpm); $p<0.001$ for each), but this increase was not related to psychosocial risk factors. Neither psychosocial risk factors were associated with baseline CAix. In unadjusted analysis, BDI total score, trait anger and trait anxiety were all significantly associated with an increase in CAIx with mental stress. After adjustment for demographic factors, CAD risk factors, CAD severity, and hemodynamic changes induced by mental stress, each 1-point increase in the BDI total score was associated with 0.34 units increase in mental stress-induced change in CAIx (95% CI: 0.10 – 0.57, $p=0.005$). The association did not persist for trait anger and anxiety, however. None of the psychological factors were related to changes in CAIx induced by physical stress.

Conclusion: Higher depressive, but not anxiety or anger symptoms are associated with an increase in arterial stiffness during mental stress. This finding may provide a mechanistic link for the association between depression and adverse cardiovascular outcomes.

Abstract 1275

WE ARE WATCHING YOU: TYPE D PERSONALITY IS ASSOCIATED WITH EXAGGERATED CARDIOVASCULAR STRESS REACTIVITY BUT ONLY UNDER HIGH SOCIAL EVALUATIVE THREAT

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The Type D personality has been associated with a range of negative health outcomes including cardiovascular disease. A potential mechanism is large magnitude cardiovascular reactivity to stress. However, the studies on reactivity to acute psychological stress in Type D individuals have reported equivocal findings, potentially due to the varying social aspects of the stressor employed. The present study, examined whether cardiovascular reactivity in Type D and Non-Type D undergraduate students differed according to an asocial (31 Type D , 30 Non-Type D: 52% female) or social (35 Type D, 34 Non-Type D: 55% female) version of the stress task. Type D personality was assessed using the DS14 questionnaire, with participant's blood pressure and heart rate recorded at rest and during a 15-minute stress protocol comprising the Stroop and a mental arithmetic task. With adjustment for age, cardiovascular fitness, perceived stressfulness (which differed between the groups) and baseline levels (which did not differ between groups), there were significant group x condition interactions for systolic ($p = .010$) and diastolic ($p = .029$) blood pressure, and heart rate ($p = .033$) reactivity. Under the social condition, Type D individuals exhibited significantly greater systolic blood pressure ($p = .010$), and heart rate ($p = .009$) reactivity, with no group differences under the asocial condition. Diastolic blood pressure reactivity did not significantly vary according to Type D status within either condition. Interestingly, Type D individuals' responses were somewhat lower than non-Type D individuals for all reactivity measures in the asocial condition, although this was not significant. This study highlights that Type D individuals only exhibit exaggerated haemodynamic reactions under conditions of high social evaluative threat. This suggests that the possible mechanism underlying the association between Type D personality and increased cardiovascular disease risk is via stress responses in highly social situations.

Thursday, March 13 from 1:00 to 2:15 pm

Symposium 1329

Aging in the Context of Chronic Disease: Premature, Accelerated, Normal, or Successful?

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General increases in life expectancy and significant advances in diagnostic precision, treatment, and supportive care have modified the morbidity and mortality landscape of the United States. More individuals are living with, and in some cases, living longer with human immunodeficiency virus (HIV), major depressive disorder (MDD), schizophrenia, and some cancers. While this shift in chronic disease epidemiology is encouraging, concomitant increases in morbidity burdens are observed. Clinical and population based studies have documented excess risk for age-associated morbidities and downward trends in the average ages of functional impairments such as frailty, cognitive decline, and compromised independence. Clinicians and researchers have begun to explore whether certain chronic diseases, therapies, and behavioral/lifestyle factors might singularly or synergistically perturb biological, physical, psychological, and social trajectories of aging across the lifespan. A critical issue is whether the converging data suggest the emergence of premature or accelerated aging phenotypes in the context of chronic disease. To address this question, four preeminent clinician-scientists will present their empirical data and scholarly perspectives drawn from long-standing programs of research in HIV, MDD, schizophrenia, and pediatric cancers. Each presentation will highlight plausible biobehavioral mechanisms and opportunities to promote healthy lifespans and successful aging in the context of chronic diseases.

Individual Abstract Number: 1782

HIV DISEASE AS A MODEL FOR THE STUDY OF AGING

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HIV infected adults who are effectively treated with antiretroviral therapy (ART) have an unexplained excess risk for several age-associated complications, including cardiovascular disease, osteopenia, cancer, liver dysfunction, renal dysfunction, and neurocognitive disease. Many of these complications are predicted by immune dysfunction and inflammation, both of which persist during effective ART. Indeed, the immune system during ART shares some similarities with that seen in the very old ("immunosenescence"). In the context of other risk factors associated with healthy aging—particularly social isolation, poverty substance abuse, and polypharmacy—there are growing concerns that the multimorbidity of HIV disease will lead to early onset of age-associated functional impairments (e.g., frailty). A