Keywords: Autism Spectrum Disorder, Structural MRI, Psychiatric Comorbidities, Neuromorphometry

Psychological Trauma-Induced Calprotectin Potentiates Pro-Inflammatory T-Lymphocyte Differentiation and Memory Formation

Cassandra Moshfegh[1], Safwan Elkhatib[1], Gabrielle Watson[1], and Adam Case[1]

[1]University of Nebraska Medical Center

Background: Post-traumatic stress disorder (PTSD) is a debilitating psychological disorder that increases the risk of inflammatory diseases by >3 fold, but the mechanisms remain elusive. Using a preclinical model of PTSD, we identified elevated calprotectin (+216%, p=0.0009), a pro-inflammatory protein, in the circulation of mice after psychological trauma. Calprotectin correlated with T-lymphocyte mitochondrial superoxide levels and inflammatory cytokine expression. Therefore, we hypothesized that psychological trauma leads to pro-inflammatory T-lymphocytes through calprotectin-mediated signaling, which predisposes inflammatory diseases.
Methods: We examined the effects of calprotectin on T-lymphocytes ex vivo through exogenous supplementation. After treatment, we investigated inflammatory, metabolic, and redox parameters.
Results: Intriguingly, calprotectin did not alter mitochondrial superoxide, metabolic state, or pro-inflammatory cytokine expression when directly supplemented to T-lymphocytes. However, when calprotectin was supplemented to dendritic cells (antigen presenting cells) prior to co-culture with T-lymphocytes, we observed an approximate 20% increase in mitochondrial superoxide in T-lymphocytes (similar to in vivo after psychological trauma) compared to no calprotectin (p=0.02). Furthermore, T-lymphocytes showed elevations in the secreted pro-inflammatory cytokines interleukin 17A (14%, p=0.1495), interleukin 15 (24%, p=0.004), and interleukin 23 (8%, p=0.0069). These cytokines promote memory T-lymphocyte formation, as well as exacerbate inflammatory diseases increased with PTSD. Using calprotectin knock-out mice, we will soon test the causality of this protein to inflammation in our preclinical model of PTSD.
Conclusions: These data suggest that calprotectin promotes a pro-inflammatory and memory T-lymphocyte phenotype through antigen presenting cells. Given this, calprotectin may serve as a new therapeutic target or biomarker for inflammatory sequelae following PTSD.
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Keywords: T Cells, PTSD - Posttraumatic Stress Disorder, Inflammation, Calprotectin, Mitochondrial Superoxide

Psychological and Physical Morbidity of Sexual Assault Among Adult Women Students

Lauren Gullett[1], Nicole A. Short[1], Grace Burud[1], Megan Lechner[2], Kathy Bell[3], Jenny Black[4], Jennie Buchanan[5], Rhiannon Reese[6], Gordon Reed[7], Catherine Rossi[8], Patricia Nouhan[9], Carolyn Phillips[10], Sandra L. Martin[1], Israel Liberzon[11], Sheila A.M. Rauch[12], Kenneth A. Bollen[1], and Samuel McLean[1]

[1]Institute for Trauma Recovery, University of North Carolina School of Medicine, [2]Memorial Hospital, [3]Tulsa Forensic Nursing Services, [4]Austin SAFE, [5]Denver Health, [6]Crisis Center of Birmingham, [7]Christiana Care, [8]Cone Health, [9]Wayne State University, [10]DC Sane, [11]University of Michigan, [12]Emory University

Background: The high risk of sexual assault (SA) faced by female college students is the subject of increasing national attention, but few prospective studies have enrolled college women sexual assault survivors in the immediate aftermath of SA and assessed adverse mental and physical health outcomes over time.
Methods: Adult women students <25 years old (M=20.8) presenting for emergency care in the immediate aftermath of SA were enrolled. Outcome assessments included evaluation for substantial posttraumatic stress (PTSD Checklist-5>33), anxious and depressive (PROMIS>60) symptoms and clinically significant new or worsening pain (CSNWP; Δ>2 on a NRS) six weeks and six months after SA.
Results: Study participants (n=151, 71% White, 14% Black, 27% Latina) experienced a high burden of adverse mental and physical health outcomes after SA. Clinically significant PTS symptoms six weeks (79/151(52.3%)) and six months (61/151(40.4%)) after SA were common, as were at least moderately severe depressive symptoms at six weeks and six months (68/126(45.0%) and 53/115(46.1%), respectively), and anxiety symptoms at six weeks (80/125(53.0%)) and six months (74/116(49.0%)). CSNWP six weeks (88/151(58.3%)) and six months (68/151(45.0%)) after SA were also common.
Conclusions: Adverse physical and mental health outcomes are common among women students who are sexually assaulted. Further studies are needed which evaluate longitudinal health, academic, and career outcomes of women students experiencing sexual assault.
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Psychopathy Mediates the Link Between Drug Abuse and Violence for NGRI Acquittees

Nicholas Thomson[1], Carla Galusha[2], Elizabeth Wheeler[3], Lindsay Ingram[4], and **Jasmin Vassileva**[5]

[1]Virginia Commonwealth University, School of Medicine, [2]Eastern State Hospital, [3]Bay Forensic Psychology, [4]Oregon State Hospital, [5]Virginia Commonwealth University

Background: Substance misuse have been long associated with an increased risk of violent and aggressive behavior. However, not all substance users are violent. Thus, it is important to understand what vulnerabilities increase the risk of substance-related violence, especially in high-risk populations, such as those with serious mental illness. A clinical construct that may be important to understanding the link between alcohol and drug abuse and violence is psychopathy. This study tested if psychopathy mediated the link between (1) alcohol abuse and (2) drug abuse and violent crime and