Samina Salim, PhD
Associate Professor
Pharmacological and Pharmaceutical Sciences

Early Life Stress: Role of Oxido-Inflammatory Processes

Dr. Salim is a tenured Associate Professor at the College of Pharmacy, University of Houston, TX. Prior to this, she worked as a post-doctoral fellow at the Karolinska Institute, Stockholm, Sweden. Subsequently, she moved to the United States and advanced my research training in the field of Pharmacology and Neuroscience. As a scientist, she has extensive experience in basic neuroscience work focused on neurobehavioral studies, but my core interests are anchored in the real-life domains of health, social, and human phenomena that could be empirically studied and understood to improve mental health and general well-being. Pursuing this line of work and building on her basic neuroscience expertise, she has initiated large scale population-based self-report data collection work in refugee populations in the greater Houston area. Within this framework, she works extensively on biological, methodological, theoretical, and empirical anchoring of the constructs of social participation, social support, post-migratory living conditions, and trauma susceptibility and resiliency among refugees. Her work is presently focused in two areas: basic neuroscience work and the design of longitudinal studies of refugee health delineating the process of post-resettlement change in health in relation to social contexts. Through this work, she has initiated extensive collaborations with the civil society, national and local non-profit organizations.

Abstract: Adverse experiences during early life may contribute to psychiatric conditions later in life. In fact, young children who experience traumatic event(s) during early life, a sensitive developmental period, are considered highly vulnerable to psychiatric disorders in adult life. Interestingly, not all children who experience stressful events are equally at risk of developing later life psychiatric disorders. Some are resilient in spite of being exposed to the same risk factors, while others are susceptible. Importantly, the relationship between early life stress and later life psychiatric symptoms is not clear, and the mechanistic basis for resilience is also not known. Our recently published studies have revealed that rats when exposed to early life single prolonged stress (EL-SPS) at postnatal day (PND) 25, exhibited depression-like behavior, 9 weeks later, at PND90. Interestingly, two subsets of rats were identified within the PND90 group of rats, “susceptible” with depression-like behavior and “resilient”,

without depression-like behavior (Liu et al. 2017). These observations have prompted us to ask: ***What is the biochemical basis of resilience and susceptibility?*** To address this question, we have focused our attention on oxidative stress. It is well known that the biochemical integrity of the brain is vital for normal functioning of the central nervous system (CNS). One of the contributing factors of cerebral biochemical impairment is the process of oxidative stress, which occurs upon excessive free radical production due to insufficiency of the counteracting antioxidant response system. The brain with its high oxygen consumption and lipid-rich content is highly susceptible to oxidative stress. Therefore, oxidative stress-induced damage to the brain has a strong potential to negatively impact normal CNS functions. Relevant to this, our preliminary data demonstrates that the depression-susceptible phenotype is associated with high oxidative stress, low antioxidant status, defective redox-sensitive Nrf2 transcription factor signaling and hyperactivity of NF-KB transcription factor, selectively in the pre-frontal cortex (PFC) of the brain. The resistant phenotype displayed low oxidative stress, high antioxidant status, activated Nrf2 and inactivated NF-KB signaling. We postulate that early life stress induces oxidative stress due to insufficiency of detoxifying mechanisms in some, which creates an allostatic load in the brain (high oxidative stress, low antioxidant response, defective Nrf2 function, NF-KB-mediated inflammation). Clearly, if oxidative stress is mitigated, the allostatic load will be eliminated, and consequently the susceptibility also should disappear.