IDENTIFICATION OF PSYCHOLOGICAL STRESS INITIATED MOLECULES IN CARDIOVASCULAR IMPAIRMENT PREVENTION

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Intense psychological stress initiates numerous molecular and biochemical alterations in the human body. Stress factors impact a wide variety of pathways to sustain/preserve homeostasis. However, the activation and/or inhibition of specific genes which control optimal gene expression and the biochemical pathways associated with these regulations require further in-depth investigations. The primary goal of our research was to identify novel protective molecules and pathways capable of enhancing adult organ regeneration and repair.
Plasma samples were collected from armed forces cadets who participated in higly controlled and supervised stress situations prior to and immediatly following the training session. First, we performed 2D gelelectrophoresis following silver stain detection. Candidates were further investigated by MALDI-TOF analysis. Secondly, we performed LC-MSMS analyses following trypsin digestion and SP3 protocol purification.
We identified more than 200 protein targets to be altered in the screens. Supported by findings of others, Apolipoprotein A-I and Alpha-1 antitrypsin were respectively detected as significant with both methods. Moreover, our results indicated a significant decrease in plasma SPARC/SPARCL1 levels following thirty minutes of psychological stress. As recent findings indicate, elevation of these proteins was associated with maladaptive right ventricular remodelling in mice. The relationship between SPARC/SPARCL1 and stress requires further investigations in humans.
In our research we identified numerous potential marker molecules associated with short term psychological stress response of the human body. We genuinely believe, future therapeutic utilization or inhibition of the detected molecules can equally prevent irreversible tissue damage and support organ regeneration and repair in adults.

Keywords: Psychological stress, Proteomics, Adaptation, Regeneration

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