Editorial

Molecular and Genetic Basis of Stress

A person's reaction to trauma depends on the traumatic situation itself, personality characteristics of the person exposed to trauma, and post-traumatic social environment. Stressor must be extreme event that is extremely dangerous or fatal nature, and which is outside normal human experience [1].

Studies investigating psychological consequences of military and civil trauma confirmed the correlation between the nature and intensity of trauma, previous traumatic experience, and psychological consequences. Stress causes the autonomic nervous system hyperactivity. If the stress is extreme or constant symptoms of hyperactivity, increased heart rate, increased respiration, sweating, muscle tension, insomnia and increased anxiety are becoming significant for the prolonging the symptoms of PTSD. Our cells are well adapted to exposure to a mild stress for a short time. In contrast there are potentially serious consequences of exposure to the prolonged stress[2].

Various damages arising from the war in Bosnia (1992 - 1995) are almost undetectable, and the consequences for the mental health of the population of Bosnia and Herzegovina are long and painful. It is estimated that in Bosnia and Herzegovina there are 1.75 million people who have some stress-related mental disorders, of which 1 million in the Federation.

PTSD may be represented by mutations that must be carried by many genes. There may even be epigenetic reasons for the disorder that have nothing to do with heritable mutations per se. Epigenetic means related to functional changes in the genome that can be regulated by external environmental events that do not involve alterations in the genetic code. One epigenetic mechanism is called "methylation," a molecular process that affects the activity of a large percentage of genes. Epigenetic investigations say that methylation may be involved in the development of stress regulation in early life[3].

A number of longitudinal studies have looked at independent variables spanning the entire course of the illness. A 10-year follow-up study in Holocaust survivors clearly demonstrated decline in stress hormone levels for those patients in whom PTSD developed or in whom PTSD was chronic. No such decline was shown for those survivors who did not present with PTSD. Indeed, they showed a relative increase in hormone levels [4].

Hippocampal volume has also been studied (using the same types of experiments as the twin studies described previously). Hippocampal volume can be quite variable. It can change as a result of environmental exposure, duration of certain illnesses, and even the age of the subject. As noted, shrunken hippocampal volume has been shown in PTSD patients, but most of these are younger cohorts. When one examines PTSD in older cohorts, the differences in volume evaporate, and no linkage with disease is observed [5].

Microarray analysis is a powerful way to look at global gene expression profiles in tissues which can identify relevant molecular processes. On that way we have enough tools on hand to find the genes responsible for the disorder. Studying such genes can lead to creating effective treatments, including designing medications that are not only specific to the disorder but also specific to an individual's experience with the disorder.

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