

# Stress and the immune response m3

Psychology



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Introduction Inflammatory responses are normally initiated by injury or trauma. As the process starts, there are certain chemical factors that are released. These chemical factors are in turn responsible for cellular changes and vascular changes that are in turn translated as inflammation. These compounds are known to originate mainly from plasma, mast cells, the lining of the blood cells which is gladdened with endothelial cells, damaged cell tissues platelets and white blood cells. During inflammation, one of the chemicals that are released is histamine. The cells release this chemical and it in turn triggers a rise in vascular permeability as well as vasodilatation.

#### PTSD and the complement system

PTSD normally associates with hyper activation of the classical pathway of the complement. The over-activation of the terminal pathway is a characteristic of PTSD. The complement alternative pathway is also hypo activated (Allison, 2008). Thus, components related to the inflammatory process are also involved in the pathogenesis of the phenomenon that is PTSD. The processes involved with and associated with PTSD is the common thing among the PTSD patients. These methods deal with the various mentioned components (Ryan, 2012).

#### Inflammatory responses and the brain

Neuroendocrinology responses initiated by stress explains the interactions between the CNS and the immune system. Neuroendocrine responses initiated by stress and the nervous system are mediated by hypothalamic CRF. Inflammatory responses affect mood because of the interconnection between CNS and the immune system. Prolonged inflammatory responses result in a bad mood. Factors that might cause an inflammatory process to be pronged include the nature of the injury causing the inflammation

(Friedman, 2007).

### Conclusion

An extremely stressful situation might lead to the dysregulation of cortisol. This occurrence can lead to the perpetuation of feelings of depression. Thus, PTSD can prolong the inflammation process.

### References

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