SMGroup

SM Journal of Depression Research and Treatment

Letter to the Editor

Common Pathways between Depression and Infection: The Mediating Role of Pro-inflammatory Cytokines

Ilias I Vlachos*

Department of Psychiatry, Athens University, Greece

Article Information

Received date: Apr 27, 2015
Accepted date: Oct 22, 2015
Published date: Oct 27, 2015

***Corresponding author**

Ilias IVlachos, Department of Psychiatry, Athens University, Greece, Tel: 0030-2130-226571; Email: vlachos.ilias@gmail.com

Article DOI 10.36876/smjdrt.1008

Letter to the Editor

A promising area of research on depression is its interactions with the immune system, taking place within the field of psychoneuroimmunology. Indications of brain-immune system interactions exist at different levels of organizations: in animal studies, stressful events can disorganize the immune response and increase susceptibility or even mortality to experimentally induced tumours [1]. In humans, immune function may be distorted by chronic or acute emotional conditions, triggered by stressful life events. Major life changes (e.g. divorce) have been found to correlate with immune system deficiencies [2].

The brain communicates with the immune system via the Autonomous Nervous System (ANS) and the neuroendocrine secretion of the pituitary gland as expressed by the Hypothalamus-Pitutary-Adrenal (HPA)-axis. Major Depressive Disorder (MDD) seems to stimulate the HPA axis and trigger immune responses simulating sickness behavior by the activation of common inflammatory responses termed as Inflammatory Responses System (IRS) [3].

Sickness behavior is a behavioral complex which becomes typically induced by acute infections or tissue injury in different mammalian species. The characteristic pattern of behavior consists of malaise, hyperalgesia, fever, lack of motivation and vitality, disinterest to social interaction with the environment , sleepiness , reduced motor activity, reduced interest in exploring the environment, in sexual encounters, anhedonia, weight loss, fatigue, lack of concentration and anxiety.Sickness behavior seems to be induced by the activation of Pro-Inflammatory Cytokines (PICs), mainlyIL-1, TNF-a και IL-6 [4]. The Central Nervous System (CNS) receives (CNS) neuronal and chemical signals of the peripheral inflammatory response via the vagal nerve, activated by the PICs. The PICs deactivate energy consuming processes like motor, neurocognitive and reproductive activity. In this way, metabolic energy becomes withdrawn from the brain and the peripheral organs and gets redirected to confront the adverse effects of the invasive pathogens [5]. Immune system responses heavily depend on calories and increase their consumption during the activation of sickness behavior. Hence, motor interception (via somnolence, fatigue and malaise) contributes to the required energy conservation.

There are plenty of indications that clinical depression is an immuno-inflammatory disorder characterized among other parameters by increased levels of PICs as well as of acute phase proteins, including C-reactive protein and haptoglobin [6]. Characteristic symptoms of major depressive disorder, which resemble sickness behavior, include appetite and weight loss, fatigue, lethargy, sleep disturbances , reduced motor activity and lack of concentration (fever is not included). Moreover, “vegetative” symptoms of depression like appetite and weight loss, psychomotor retardation were found to correlate with inflammatory biomarkers (increased levels of haptoglobin) [7]. Increased levels of C-reactive protein were further found to be of prognostic value not only for the severity of the current depressive episode but of relapsing depression as well [8]. It is also known that PICs mediate in rendering behavior more vulnerable to stressful life events as in the case of maternal deprivation [9].

Taking into consideration that the interactions between depression and inflammation are bidirectional, as current reviews suggest [10], it is of interest to further investigate their common pathways of induced pro-inflammatory cytokines in order for this complex phenomenon to be elucidated.

References

1. Nakamura T, Walker AK, Sominsky L, Allen T, Rosengren S, Hodgson DM. Maternal separation in early life impairs tumor immunity in adulthood in the F344 rat. Stress. 2011; 14: 335-343.

2. Glaser R, Sheridan J, Malarkey WB, MacCallum RC, Kiecolt-Glaser JK. Chronic stress modulates the immune response to a pneumococcal pneumonia vaccine. Psychosom Med. 2000; 62: 804-807.

OPEN ACCESS

ISSN: 2573-3389

3. Maes M. A review on the acute phase response in major depression. Rev Neurosci. 1993; 4: 407-416.

4. Dantzer R, O'Connor JC, Freund GG, Johnson RW, Kelley KW. From inflammation to sickness and depression: when the immune system subjugates the brain. Nat Rev Neurosci. 2008; 9: 46-56.

5. Peters A. The energy request of inflammation. Endocrinology. 2006; 147: 4550-4552.

6. Leonard B, Maes M. Mechanistic explanations how cell-mediated immune activation, inflammation and oxidative and nitrosative stress pathways and their sequels and concomitants play a role in the pathophysiology of unipolar depression. NeurosciBiobehav Rev. 2012; 36: 764–785.

7. Maes M, Berk M, Goehler L, Song C, Anderson G, Gałecki P, et al. Depression and sickness behavior are Janus-faced responses to shared inflammatory pathways. BMC Med. 2012; 10: 66.

8. Liukkonen T, Silvennoinen-Kassinen S, Jokelainen J, Räsänen P, Leinonen M, Meyer-Rochow VB, et al. The association between C-reactive protein levels and depression: Results from the northern Finland 1966 birth cohort study. Biol Psychiatry. 2006; 60: 825-830.

9. Hennessy MB, Paik KD, Caraway JD, Schiml PA, Deak T. Proinflammatory activity and the sensitization of depressive-like behavior during maternal separation. Behav Neurosci. 2011; 125: 426-433.

10. Kasten E. Can Infection Give You the Blues? Mind. Scientific American. 2015; 26: 46-49.

Citation: Vlachos II. Common Pathways between Depression and Infection: The Mediating Role of Pro-inflammatory Cytokines. SM J Depress Res Treat. 2015; 1(2): 1008.
https://dx.doi.org/10.36876/smjdrt.1008