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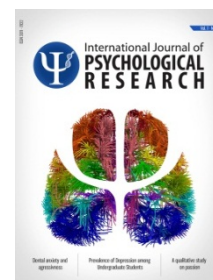
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Cognition and Inflammation: “The role of cytokines in cognitive performance”

Cognición e inflamación:
“El papel de las citoquinas en el
desempeño cognitivo”



Editorial

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The interactions between the immune system and nervous system have been the focus of

research in recent years. Perhaps due to the discovery and acknowledgement of the multiple functions that cytokines carry out in the central nervous system (CNS), going from mediators of neuropathologic processes to the degeneration and repair within the CNS.

Cytokines are a group of proteins of a low molecular weight that are produced generally during immune response, and they are classified into families. The main are chemokines, interferons, the tumor necrosis factor ones (TNF) and the interleukins (IL) (Arisi 2014).

Cytokines are secreted in many types of cells, in which are the endothelium, the lymphocytes, astrocytes, and neurons are included (Adler & Rogers 2005).

Generally, cytokines act as polypeptides hormones promoting the homeostasis in the CNS and the peripheral nervous system (PNS); thus, they constitute a central nucleus to delineate etiological aspects in those autoimmune diseases, viral, and neurodegenerative that evolve with variable cognitive impairments, and for which the cytokines expression in the CNS suggests a critical role in the stable cognitive performance (Hopkins & Rothwell 1995).

Nowadays, there is the idea that pathophysiological processes that underlay cytokines modulation, might constitute a therapeutic target in some neuropsychiatric diseases; in fact, some cytokines could have a pleiotropic action (causing multiple effects depending on the target cell), upon which they could regulate not only an inflammatory response, but also the migration, proliferation and cellular differentiation in the cerebral neurocyto-architecture, thus, impacting the expression of cognitive processes, such as memory formation and learning (Arisi 2014).

One of the most striking experimental approaches for the cytokines and cognition relationship has been focused in the psychological stress phenomenon and the beginning of an inflammatory response through the limbic-hypothalamus-pituitary-adrenal axis (HPA), and in a direct manner a change in the level of cortisol in the suprarenal cortex, altering the performance of the mineralocorticoids and glucocorticoids receptors (Slavish, Graham-Engeland, Smyth, & Engeland, 2014); under this condition, the HPA axis activation along with the hypothalamus, the amygdala and the hippocampus, elicit the emotional response activating the sympathetic nervous system (SNS)

In this context, an inflammatory response and the changes deriving from interleukins and chemokines via leucocytes could affect the synaptic transmission and the neuromodulation (Pilger, et al. 2014; Slavish, et al. 2014; von Känel, Kudielka, Hanebuth, Preckel, & Fischer, 2005).

These findings have contributed to the development of several researches for understanding how the levels of proinflammatory chemokines could explain the etiopathogenesis of CNS diseases and constitute a diagnostic and therapeutic target of relevance in neurodegenerative and neuropsychiatric diseases. In the same way, the analysis of cognitive performance has been associated to expression and production of interleukins (IL-1B IL-6), (McAfoose & Baune 2009; Simpson, et al. 2013); nonetheless, it is still necessary to replicate the results in different populations and with sensible and specific instruments that allow to engross its association.

On the other hand, the results obtained in terms of the association of personality structure and inflammation patterns are promising; the researches about inflammation and personality have mainly been focused in middle-aged adult and elderly population. If well there is Little known about the existence of a

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pattern of inflammatory processes in personality of youth populations, the most consistent findings reported until now have been observed with Interleukin-6 (IL-6, stimulating the secretion of ACTH and increasing the levels of cortisol) and the factor C, related to the conscientiousness traits (using the Big Five as a model of personality) (Luchetti, Barkley, Stephan, Terracciano, & Sutin, 2014); Allowing to determine the vulnerability of inflammatory processes from a bidirectional way in a dimensional perspective of traits (high and low levels of conscientiousness traits).

IL-6 has also been one of the most frequent markers associated to a cognitive impairment in the last decade; however, the results are still controversial; on one hand, the study conducted by Dr. Economos and helpers, which included a sample of 1224 participants found high levels of IL-6 associated to cognitive impairment and aside from other vascular risk factors (Economos, et al. 2013); Metti's and collaborators' prospective study with a cohort of 1323 participants, suggests that not necessarily IL-6 among other cytokines that express in the brain, is a putative marker of cognitive impairment. However, they could indicate an decrease in cognitive performance (Metti, et al. 2014).

Perhaps, to determine whether an inflammatory response which mediates the neuromodulation, neurogenesis and plasticity is constitutive in the evolution and latency of cognitive impairment requires to improve the experimental way by controlling variables such as:

- To improve the specificity and sensibility of neuropsychological tests that allow to identify the impairment levels and cognitive variables associated to changes in cognitive performance.
- To increase and refine the experimentations in animal models incorporating cognitive function protocols along with controlled designs of cytokine blockage.
- To introduce ancestry informative markers (AIM) that allow to obtain more information regarding the genetic differences between parental populations

(Parra, 2012), concluding the changes in the allelic frequencies and their relation to the levels of cytokines present in cognitive phenotype.

- To implement the use of cognitive and neurocognitive endophenotypes in psychiatry and psychopathology might add to the molecular associations deriving from the changes in cytokine levels that express in the brain, which exhibit a particular neurobiological correlate. This aspect would contribute in a significant way to the etiopathogenic of mental disorders that grow with inflammatory

processes (Cuartas Arias 2011; Cuartas Arias, et al. 2011).

- To improve the sample size and implement prospective studies that allows discriminating the changes in the cytokine levels, age and cognitive function

Finally, the control socio- demographic variables, for instance, socioeconomic status, alcohol consumption, drugs and tobacco abuse among others, could bound the reach and findings of research related to how the cytokine levels constitute a neuroregulating dynamics in cognitive performance.

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