Le Yin et le Yang de la voie de la kynurénine

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The 'Yin' and the 'Yang' of the kynurenine pathway: excitotoxicity and neuroprotection imbalance in stress-induced disorders

Abstract

The amino-acid tryptophan (TRY) is converted into kynurenine (KYN) and subsequent metabolites by the tryptophan/catabolites (TRY/CAT) pathway (kynurenine pathway). 'Excito-toxic' and 'neuro-protective' metabolites are produced, which modulate the glutamatergic neurotransmission. The TRY/CAT pathway is activated by hypothalamic-pituitary-adrenal endocrine induction during stress by corticoids hormones, and the excitotoxic branch of the TRY/CAT pathway is activated by proinflammatory cytokines. During stress and major depressive disorders, it is generally accepted that inflammation induces an imbalance toward the excitotoxic branch of the TRY/CAT pathway, causing changes in brain connectivity in corticolimbic structures and therefore psychocognitive abnormalities. In neurodegenerative diseases, the activation of the oxidative branch of the TRY/CAT pathway has been frequently reported. We propose a comprehensive survey of the TRY/CAT pathway (kynurenine pathway) abnormalities in stress and
inflammation-induced MDD and neurodegenerative diseases. As TRY/CAT pathway is a common feature of stress, inflammation, affective disorders, and neurodegenerative diseases, we discuss the status of the TRY/CAT pathway as a possible link among chronic stress, inflammation, depressive disorders and neurodegenerative diseases. This review does not claim to be exhaustive, but in a pharmacological perspective, it will be proposed that modulation of the excitotoxicity/neuroprotection balance is a valuable strategy for new and more effective treatments of mood disorders.

CONTACT :
Dr. Pascal Barone : pascal.barone@univ-tours.fr

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