Editorial
Volume 9:1, 2021

Journal of Neurological Disorders

ISSN: 2329-6895 Open Access

Editorial on JAK-STAT pathway

Athanasios Zisakis
Department of Pathology and Neurosurgery, Red Cross Hospital, Greece

Abstract

The JAK-STAT pathway in cytokine receptor signalling can activate STATs, which can bind to DNA and allow the transcription of genes involved in immune cell division, survival, activation and recruitment. For example, STAT1 can enable the transcription of genes which inhibit cell division and stimulate inflammation

Keywords: Immune Cell Division • Cytokine • Genes • Inflammation • Cell Division

Editorial

The Janus kinase/signal transducers and activators of transcription (JAK/STAT) pathway is one of a handful of pleiotropic cascades used to transduce a multitude of signals for development and homeostasis in animals, from humans to flies. In mammals, the JAK/STAT pathway is the principal signaling mechanism for a wide array of cytokines and growth factors. JAK activation stimulates cell proliferation, differentiation, cell migration and apoptosis. These cellular events are critical to hematopoiesis, immune development, mammary gland development and lactation, adipogenesis, sexually dimorphic growth and other processes. Predictably, mutations that reduce JAK/STAT pathway activity affect these processes (reviewed by Igaz et al., 2001; O'Shea et al., 2002)

SOCS proteins are a family of at least eight members containing an SH2 domain and a SOCS box at the C-terminus (reviewed by Alexander, 2002). In addition, a small kinase inhibitory region located N-terminal to the SH2 domain has been identified for SOCS1 and SOCS3. The SOCS complete a simple negative feedback loop in the JAK/STAT circuitry: activated STATs stimulate transcription of the SOCS genes and the resulting SOCS proteins bind phosphorylated JAKs and their receptors to turn off the pathway. The SOCS can affect their negative regulation by three means. First, by binding phosphotyrosines on the receptors, SOCS physically block the recruitment of signal transducers, such as STATs, to the receptor. Second, SOCS proteins can bind directly to JAKs or to the receptors to specifically inhibit JAK kinase activity. Third, SOCS interact with the elongin BC complex and cullin 2, facilitating the ubiquitination of JAKs and, presumably, the receptors. Ubiquitination of these targets decreases their stability by targeting them for proteasomal degradation.

***Address for Correspondence:** Athanasios Zisakis, Department of Pathology and Neurosurgery, Red Cross Hospital, Greece, Tel: + 8129137235; E-mail: Athanasios@yahoo.com*

Received 10 January, 2021; **Accepted** 20 January, 2021; **Published** 27 January, 2021

Conclusions

JAK-STAT pathway has critical roles in the regulation of immune system, especially the fate of T helper cells. Th cells play a central role in direction of the immune responses. JAKs are associated with cytokine receptors, which are activated upon stimulation and they phosphorylate STAT proteins, enabling them to be transported to the nucleus. Several regulators, such as PTPs, SOCS and PIAS families have been described to modulate the function of the JAK-STAT pathway. Since any dysregulation in the JAK-STAT pathway and their regulators may lead to pathological consequences; therefore, signaling pathways are potential therapeutic approaches which targeting of them may lead to develop new strategies in the treatment of different diseases, particularly T cell mediated diseases. Targeting strategies may result in numerous benefits, for instance side effect and unwanted reactions may be diminished and bystander pathways remain intact. We hope that the severity and burden of the diseases can be alleviated and decreased by the development of new drugs and precise targeting of these proteins in pathological circumstances. Studies and experiments on the regulation of the JAK-STAT pathway are ongoing and other aspects of their functions need further elucidation.

How to cite this article: Zisakis, Athanasios. "Editorial on JAK-STAT pathway." J Neurol Disord 9 (2021):444.