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综述

SOCS3与BCR-ABL阴性的骨髓增殖性疾病

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摘要
一系列细胞因子通过JAK/STAT通路诱导细胞因子信号转导抑制因子(SOCS)基因的表达，SOCS蛋白又负反馈调节细胞因子信号转导通路， 形成细胞因子信号转导反馈调节环。在BCR-ABL阴性的骨髓增殖性疾病的发病机制中，JAK2V617F点突变的发现是一个重大的突破。JAK2V617F点突变可导致SOCS3基因表达的增高，但通过某种机制逃逸了SOCS3的负向调控作用。

关键词

细胞因子信号转导抑制因子；细胞因子信号转导通路；JAK/STAT通路；骨髓增殖性疾病

分类号

SOCS3 and BCR-ABL fusion gene negative myeloproliferative diseases

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Abstract
Several cytokines induce the expression of the suppressor of cytokine signaling （SOCS）gene through JAK/STAT pathway, while SOCS protein negatively regulates signal transduction of cytokine pathway, and a negative feedback loop of cytokine signal transduction is thus completed. In BCR-ABL fusion gene negative myeloproliferative diseases，the discovery of JAK2V617F point mutation is an important landmark. Recently，it was demonstrated that JAK2V617F point mutation accompanied with highexpression of SOCS3. However, it is not clear why JAK2V617F mutant could escape the negative regulation by suppressor of cytokine signaling 3 in some of MPD patients.

Key words suppressor of cytokine signaling-3 (SOCS3)；signal transduction pathway；JAK/STAT pathway；myeloproliferative diseases

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