

# Expression of SOCS1, SOCS2, and SOCS3 in growth hormone-stimulated skin fibroblasts from children with idiopathic short stature

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**Background/aim:** Possible etiologies of idiopathic short stature (ISS) include a range of conditions, some of which may be caused by defects in the modulation of the growth hormone (GH)-signaling pathway. The Janus kinase/signal transducer and activator of transcription pathway is regulated by several mechanisms, including negative feedback regulation by the suppressors of cytokine signaling (SOCS). However, the specific induction of SOCS transcript levels in fibroblasts from ISS patients has not been studied. **Methods:** We determined the transcript levels of the SOCS1-3 genes under basal conditions, and in the presence or absence of stimulation with rhGH for 24 h in skin fibroblast cultures obtained from patients with ISS and children with normal height. **Results:** Under basal conditions, ISS patients express higher SOCS2-3 transcript levels than control children. After incubation with recombinant human GH (rhGH), the transcript levels of SOCS2 increased significantly in ISS patients compa