



POSTER PRESENTATION

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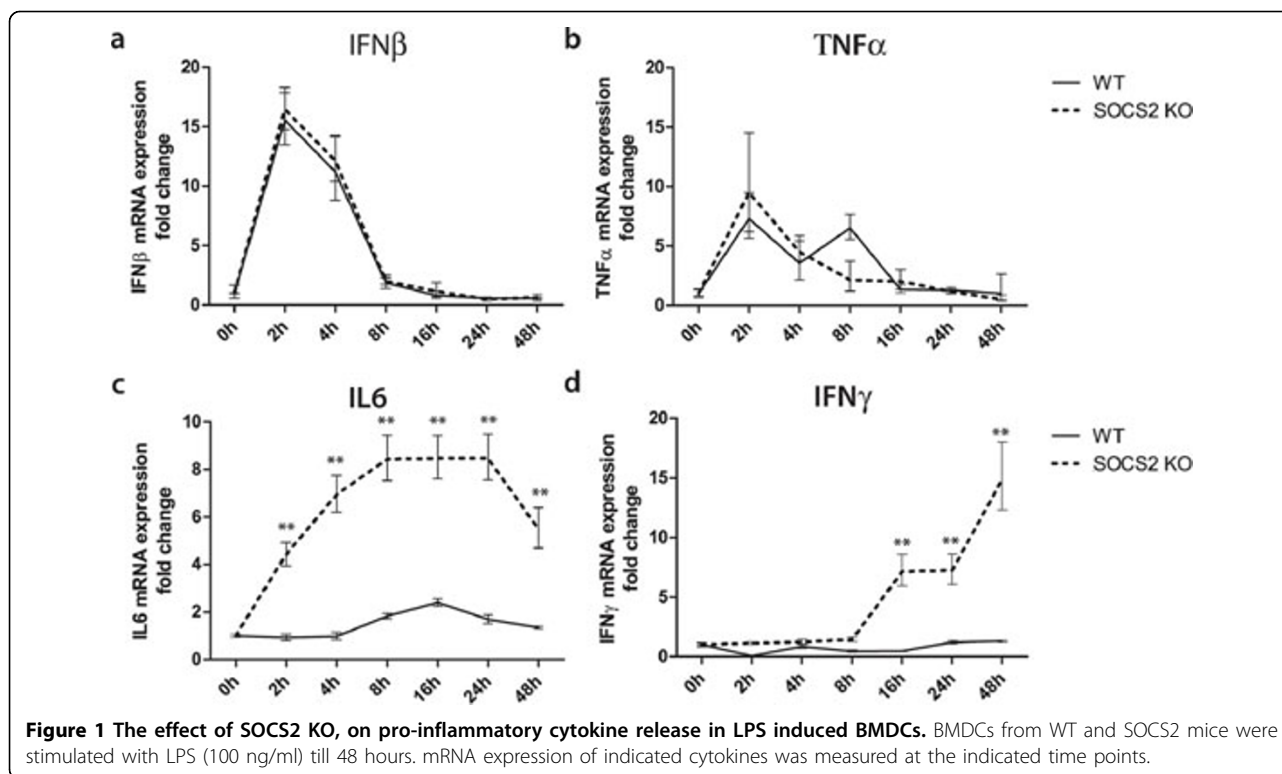
SOCS2 expression in bone marrow derived dendritic cells is a positive regulator of T cell activation

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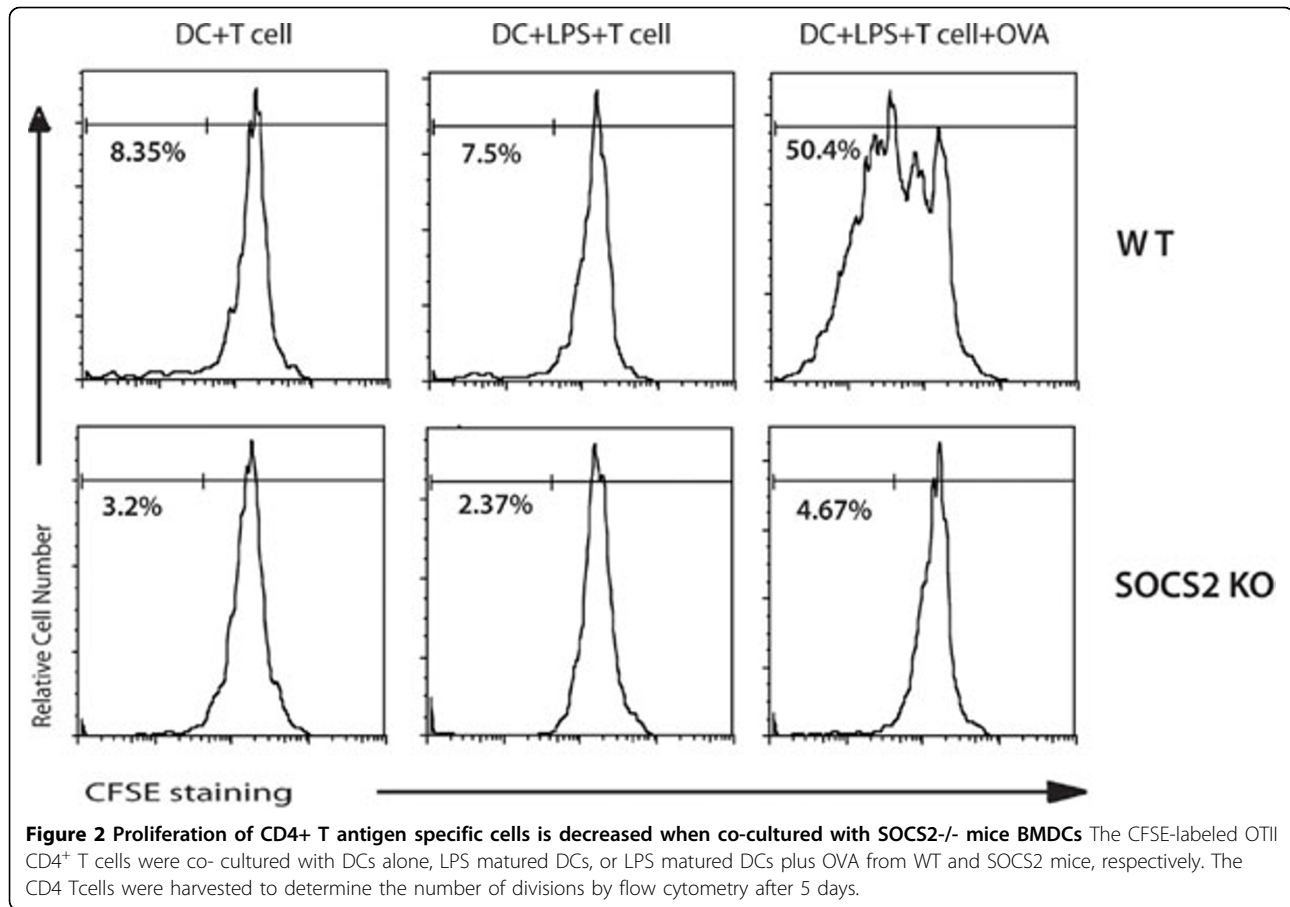
Background

After a completed T cell response the activation of DCs needs to be terminated to avoid harmful inflammation or autoimmune disease. Besides the negative regulation of

JAK/STAT signaling pathway on growth hormone and prolactin for suppressor of cytokine signaling (SOCS) 2 [1], murine SOCS2^{-/-} DCs are recently found to be hyper-responsive to microbial stimuli and refractory to the inhi-



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bitory actions of the anti-inflammatory mediator LXA4 [2]. Thus, we investigate the role of SOCS2 in DC antigen presentation.

Materials and methods

Mice: SOCS2 deficient mice and transgenic OT-II mice.
Mouse bone marrow-derived dendritic cells (BMDCs): Mouse bone marrow cells were incubated 7 days with 20 ng/ml GM-CSF and 20 ng/ml IL-4 to create mouse BMDCs.
Quantitative RT-PCR: Real-time PCR was used to measure pro-inflammatory cytokines gene expression.
CFSE proliferation assay: BMDCs were incubated with 100 ng/ml LPS and 50 ng/ml OVA323-339 peptide. The next day CD90⁺ splenocytes from OT-II mice were labeled with CFSE and added to the BMDC culture for 5 days. Then cells were stained for CD4 and read by FACS.

Results

Increased production of pro-inflammatory cytokines by SOCS2^{-/-} BMDCs in response to LPS (see Figure 1)

SOCS^{-/-} BMDCs have a decreased capacity to activate naïve CD4⁺ T cells (see Figure 2)

Conclusions

SOCS2 is complex regulator of DC effector functionality, with an overall positive regulatory function on T cell activation.

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