Type 1 IFNs in human *versus* mouse

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In their comments on the excellent work by Nguyen *et al.*[1], O'Shea and Visconti[2] discuss mouse *versus* human differences in intracellular cytokine signaling and raise questions about the clinical relevance of the reported findings. They state that "it will be important to assess in human viral infections whether type 1 IFNs induce or inhibit IFN-γ production and T_H1 differentiation. Given the differences between mice and humans, it is difficult to predict what happens."

In a recent study, we have shown that IFN-β administration to human volunteers depressed the secretion of IFN-γ and various other cytokines of the T_H1 network by a factor of ten in PBMCs stimulated *ex vivo* by concanavalin A or OKT3[3]. This effect lasted for about 24 hours after administration and was steadily reproducible over five weeks of treatment that was given once or three times per week. These observations are in line with the findings of Nguyen *et al.,* and could possibly explain the effectiveness of type 1 IFNs in treating multiple sclerosis. It suggests that, despite differences in intracellular STAT1 and STAT4 signaling, humans and mice may not react differently, given their response to type 1 IFNs. This should be an invitation for mice specialists to read clinical articles.

1. Nguyen, K. B. *et al. Nature Immunol.* **1**, 70–76 (2000).
2. O'Shea, J. J. & Visconti, R. *Nature Immunol.* **1**, 17–19 (2000).
3. Rothuizen, L.E. *et al. J. Neuroimmunol.* **99**, 31–41 (1999).