

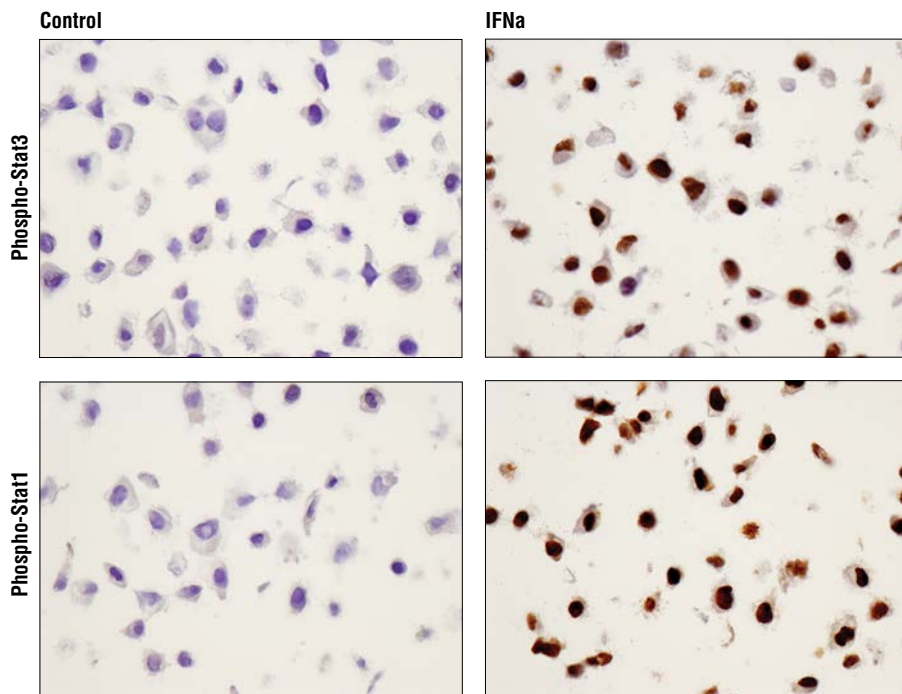
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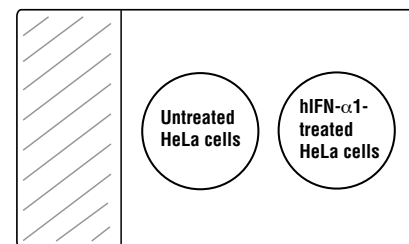
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Immunohistochemical analysis of paraffin-embedded HeLa cell pellets, control (left) or IFN $\alpha$ -treated (right), using Phospho-Stat3 (Tyr705) (D3A7) XP<sup>®</sup> Rabbit mAb #9145 (top) and Phospho-Stat1 (Tyr701) (58D6) Rabbit mAb #9167 (bottom).

**Background:** Stat proteins serve as transcription factors in growth and survival pathways stimulated by growth factor and cytokine activation of receptor proteins. Receptor activation promotes tyrosine phosphorylation of Stat proteins, resulting in Stat dimerization and translocation to the nucleus where they regulate expression of numerous proteins that control cell growth, survival, differentiation and pathogen resistance (1). Stat1 is essential in IFN- $\alpha$  and IFN- $\gamma$  stimulated pathways and is abnormally activated in many tumors (2,3). Both Stat1 $\alpha$  (91 kDa) and Stat1 $\beta$  (84 kDa) isoforms are activated by IFN- $\alpha$  but only Stat1 $\alpha$  responds to IFN- $\gamma$ . Phosphorylation of Stat1 at Tyr701 induces Stat1 dimerization, nuclear translocation and DNA binding (4). Transcription factor Stat3 possesses oncogenic potential and anti-apoptotic activities; a number of human tumors display constitutively activated Stat3 (5,6). Activation of Stat3 follows phosphorylation at Tyr705, resulting in dimerization, nuclear translocation and DNA binding (7). Expression of Stat3 $\alpha$  (86 kDa) and Stat3 $\beta$  (79 kDa) isoforms correlates with cell type, ligand and cell maturation stage (8).

**Description:** Each control slide contains formalin fixed, paraffin-embedded HeLa cells, untreated, treated with Human Interferon- $\alpha$ 1 (hIFN- $\alpha$ 1) #8927 that serve as a control for Phospho-Stat1 (Tyr701) and Phospho-Stat3 (Tyr705) immunostaining. Western blot analysis was performed on extracts derived from the same cells to verify the efficacy of the hIFN- $\alpha$ 1 treatment.



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