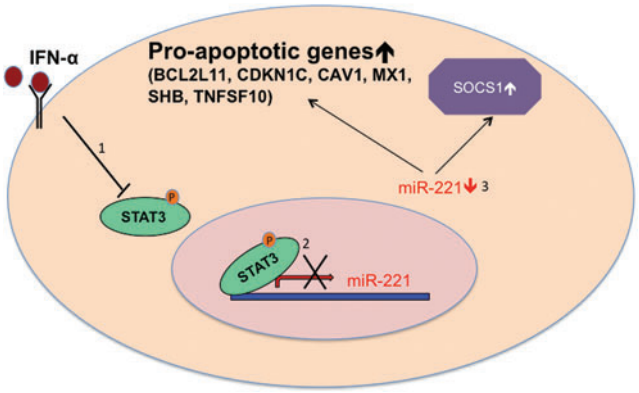

SUPPLEMENTARY FIG. S4. Model figure depicting our findings regarding IFN-α-induced miR-221 downregulation and its relevance in mDC functionality. STAT3 drives miR-221 expression and is therefore required for maintaining basal miR-221 levels. Binding of IFN-α to its receptor, IFNAR, results in STAT3 inhibition, which leads to miR-221 downregulation. miR-221 targets BCL2L11 (proapoptotic protein), CDKN1C (proapoptotic protein), and SOCS1 (negative regulator of JAK/STAT signaling), thus regulating apoptosis and IL-6/TNF-α production in mDCs. miR-221 indirectly controls the expression of various other proapoptotic proteins, CAV1, MX1, SHB, and TNFSF10 as well.