Supplementary figure 4. Screening of possible risk factors that could cause the up-regulation of CRACM1 in RA T cells. Given the role of cytokines in the pathogenesis of RA and the fact that the most common treatments for RA target these inflammatory mediators, we conducted a pilot screening to identify potential mechanisms involved in the up-regulation of CRACM1 in naive CD4\(^+\) T cells of patients with active RA. Naïve CD4\(^+\) T cells from healthy donors were cultured with the indicated doses of MTX, dexamethasone, IFN-γ and TNF-α. (a-d) After three days of incubation, CRACM1 protein levels were detected by western blotting after CRACM1 was precipitated from cell lysates using a CRACM1-specific antibody. The results are presented as the % increase in the CRACM1 expression over that of control cells cultured in plain media (means ± SEM, * p<0.05, ** p<0.001) (n=3-5). (e-h) Cell viability in the presence of MTX (e), dexamethasone (f), IFN-γ (g) and TNF-α (h) according to the commonly used cell-viability assay. The results are presented as the % increase in cell viability compared to the control cells (means ± SEM, * p<0.05, ** p<0.001) (n=3-5).