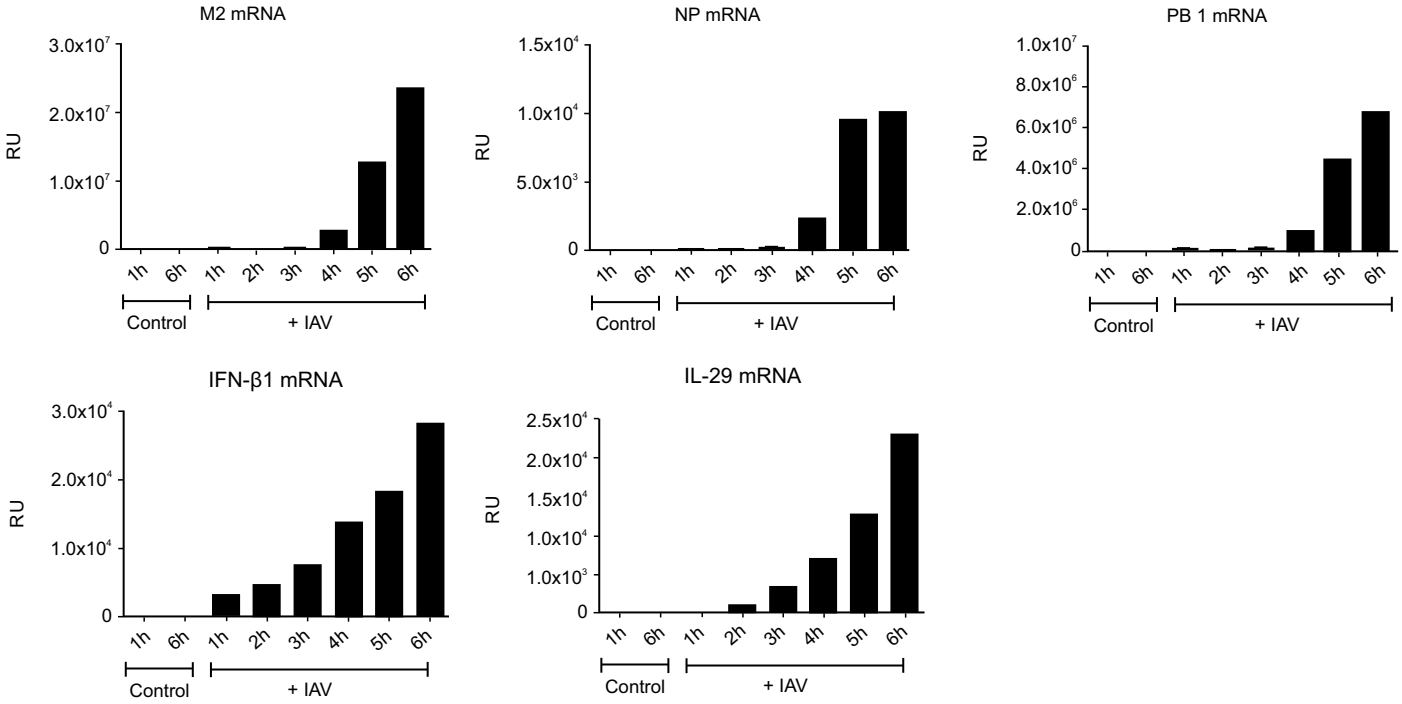
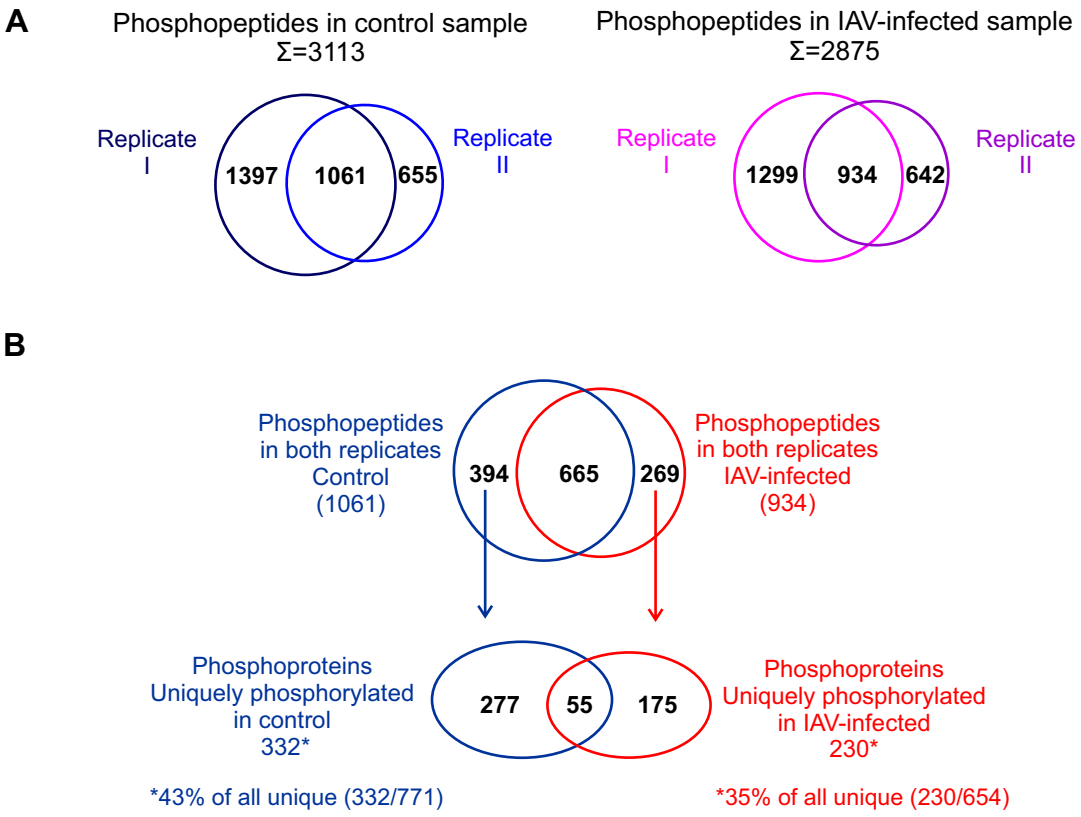
SUPPLEMENTARY FIGURES (S1-S5 Figs)

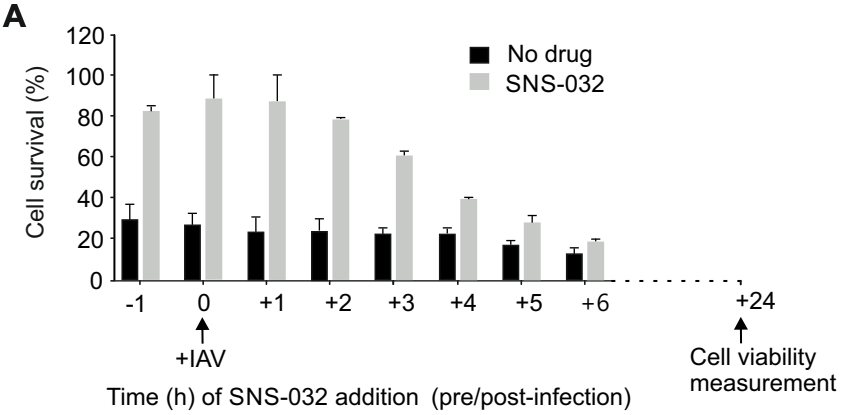


S1 Fig. Early antiviral response and viral replication in influenza A virus infected macrophages. Primary human macrophages from three different donors were infected with influenza A virus for the indicated times or left uninfected (control). The donor samples were pooled and the mRNA expression of influenza A virus genes *M2*, *NP*, and *PB1* and human interferon genes *IFN-β1* and *IL-29* were measured with quantitative RT-PCR. Comparable data were obtained from two separate experiments.



S2 Fig. Comparison of the phosphopeptide identifications in untreated (control) and IAV infected samples in the two biological replicates. (A) In all, 1061 (34%) and 934 (32%) phosphopeptides were identified in both replicates in control and IAV infected samples, respectively. Results are in accordance with our previous publications (Öhman et al. Phosphoproteomics combined with quantitative 14-3-3-affinity capture identifies SIRT1 and RAI as novel regulators of cytosolic double-stranded RNA recognition pathway. *Mol Cell Proteomics*. 2014; 13(10): 2604-17) and (Öhman et al. Phosphoproteome characterization reveals that Sendai virus infection activates mTOR signaling in human epithelial cells. *Proteomics*. 2015; 15: 2087-2097). (B) The phosphopeptides that were identified in both biological replicates were compared between the two conditions. In all, 507 different proteins had unique phosphorylations, in infeced or control conditions, present in both biological replicates.

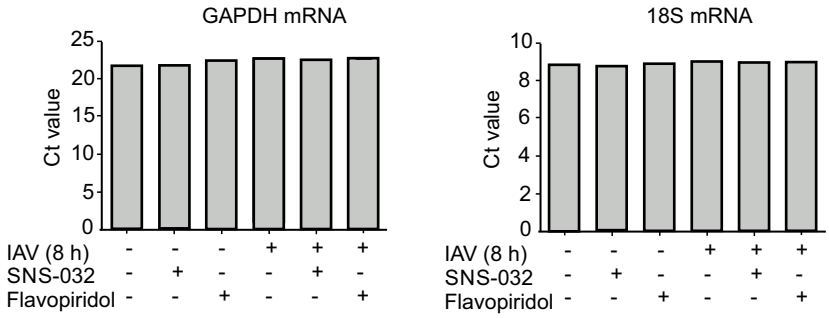
A



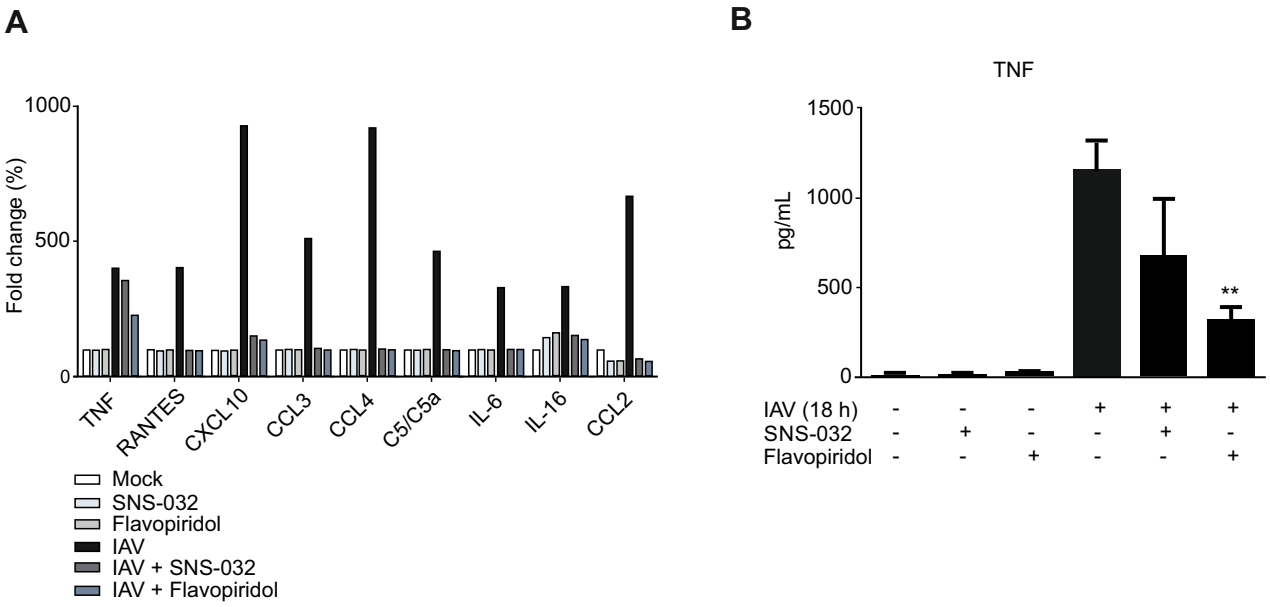
B

	Ec_{50} (nM)	Cc_{50} (nM)	SI
SNS-032	40	> 200	> 500
Flavopiridol	200	> 200	> 100

S3 Fig. (A) SNS-032 rescues IAV infected macrophages if administrated in the early time points of infection. Cells were treated with 0.3 μM SNS-032 at the indicated time points before or after infection with IAV, or left untreated. The cell viability was measured 24 h post infection. Data is presented as mean and SD of n = 3 biological replicates. (B) Macrophages were treated with different concentrations of flavopiridol or SNS-032 one hour before infection with IAV (MOI 0.01). The cell viability was measured at 24 h post infection. EC_{50}; half maximum effective concentration, CC_{50}; half maximum cytotoxic concentration, SI; selectivity index.



S4 Fig. Selected CDK inhibitors do not interfere with the general gene transcription. Primary human macrophages from three different donors were pretreated with CDK inhibitors (0.3 μM SNS-032 or flavopiridol) one hour before infection. The donor samples were pooled after 8 h of influenza A virus or mock infection and the levels of internal controls, the human housekeeping gene *GAPDH* and *18S* ribosomal RNA, were measured.



S5 Fig. Selected CDK inhibitors decrease the secretion of pro-inflammatory cytokines and chemokines in influenza A virus infected macrophages. (A) Human primary macrophages derived from three different donors were left untreated or pretreated with SNS-032 or flavopiridol (0.3 μM) one hour before mock- or IAV infection. At 20 h post-infection, the growth medium was collected and the samples from the donors were pooled. The secretion of cytokines was measured with a cytokine array proteome profiler. The changes in concentrations were calculated and compared to the mock sample. (B) Human primary macrophages (from three different donors) were left untreated or pretreated with SNS-032 or flavopiridol (0.3 μM) 1 h before mock- or influenza A virus infection. The growth medium was collected 18 h post-infection and the concentration of TNF was measured with ELISA. The concentration was compared to the infected and untreated sample. Data were analyzed with Student's t test, n = 3, **$p < 0.01$.