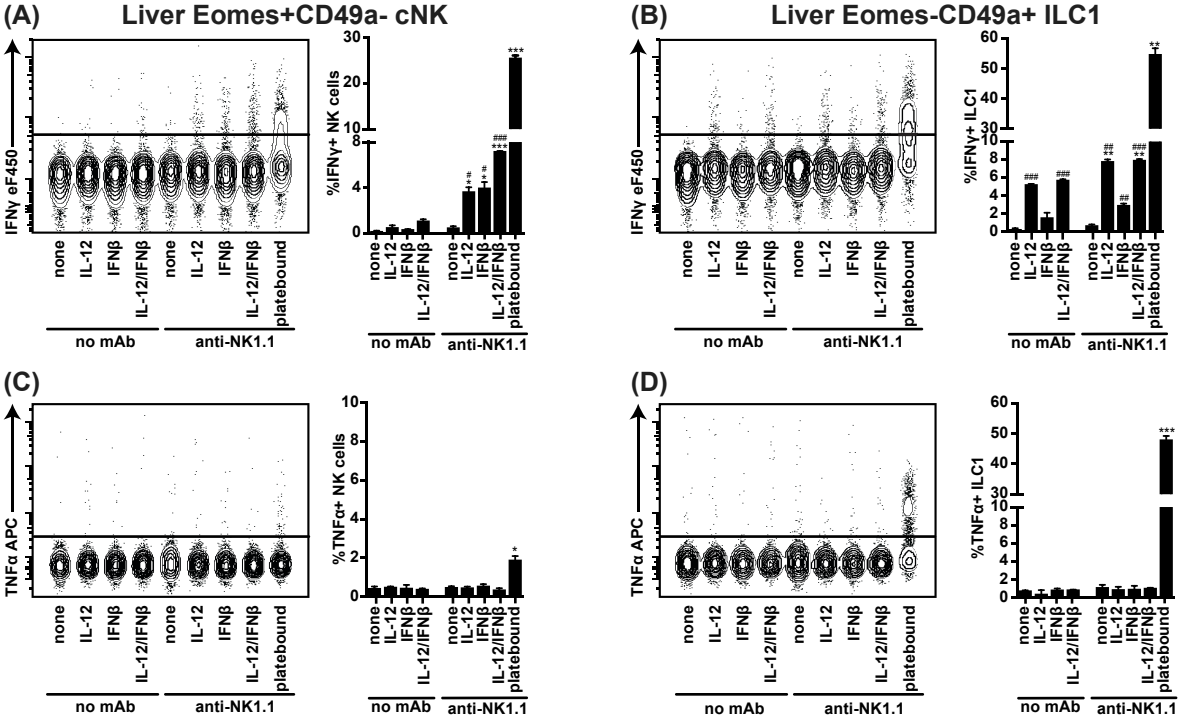
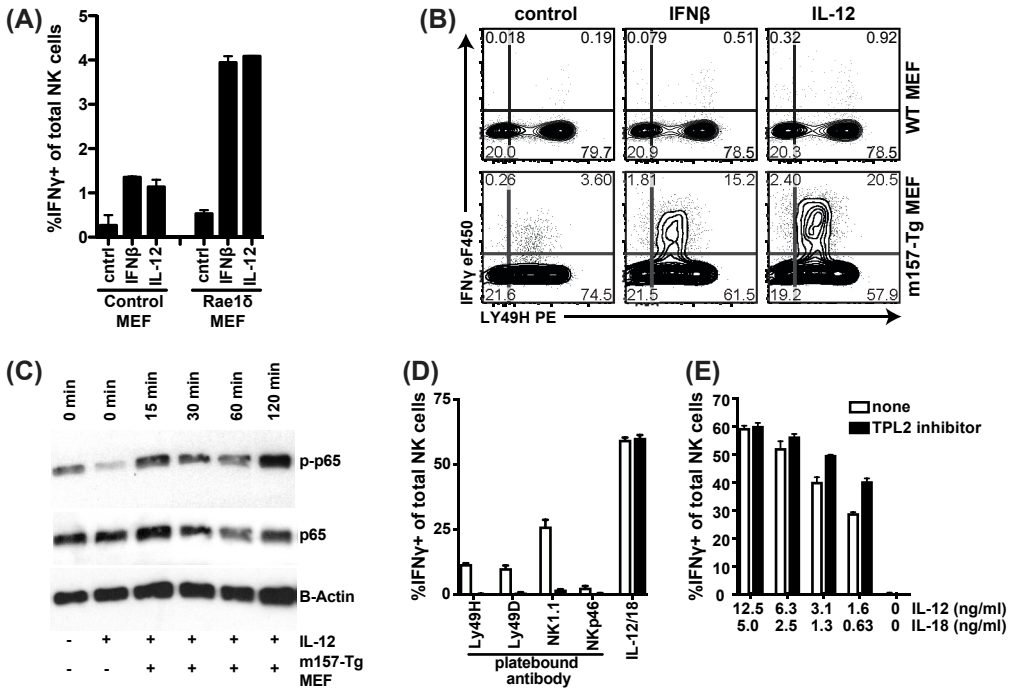
Piersma et al, Supplementary Figure 1



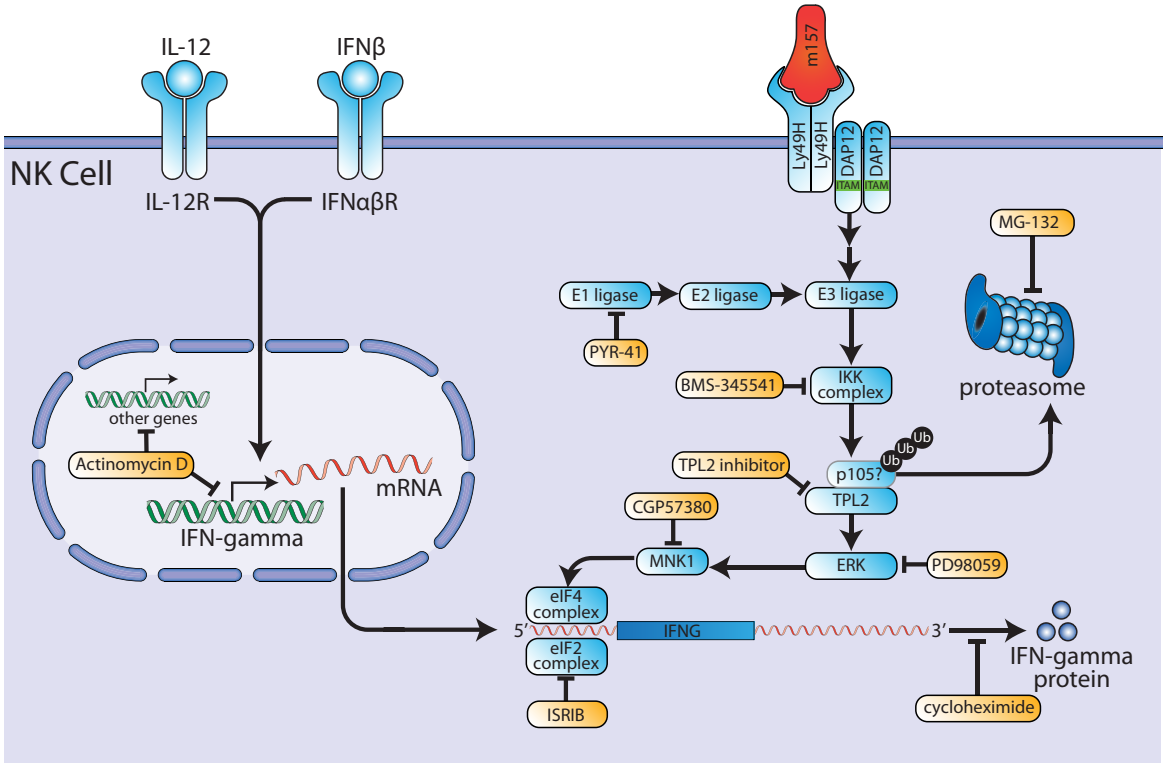
sFigure 1: cNK versus ILC1 have differential requirements for cytokine production.
Liver single cell suspensions from WT mice were stimulated in the presence of soluble or plate-bound antibody and the indicated cytokines. NKp46+Eomes+CD49a- cNK cells (**A, C**) or NKp46+Eomes-CD49a+ ILC1 (**B, D**) were analyzed for IFNγ (**A, B**) and TNFα production (**B, D**). The *-symbols indicate statistical comparison to same condition without antibody, the #-symbols indicate comparison to soluble antibody only or unstimulated within the same group. ### or ***p<0.001, ## or **p<0.01, # or *p<0.05, ns= not significant.

Piersma et al, Supplementary Figure 2



sFigure 2: Requirements for dual signaling in response to Rae1δ and m157-Tg stimulation. (**A**) Splenocytes from WT mice were mixed with Rae1δ or WT MEF in the presence of the indicated cytokines. IFNγ production by NK cells are shown. (**B**) Purified NK cells were stimulated with m157-Tg or WT MEF in the presence or absence of IFNβ or IL-12. (**C**) Purified NK cells were pre-stimulated with IL-12, after which the NK cells were stimulated with m157-Tg MEF for the indicated time. B-actin, phospho-p65 and were analyzed by western blot. (**D**) percentage of IFNγ+ NK cells from Figure 5G. (**E**) Splenocytes were stimulated with indicated concentration of IL-12 and IL-18 in the presence or absence of 9 μM TPL2 inhibitor. Percent of IFNγ producing NK cells are shown.

Piersma et al, supplemental Figure 3



sFigure 3: Model of activation receptor dependent IFNγ production. Cytokines such as IL-12 and IFN-I that are produced during virus infection induce transcription of Ifng. Upon recognition of a virus-infected cells through ligand-receptor interactions, such as m157-Ly49H NK cells signal through the Ubiquitin-IKK-TPL2-ERK-MNK1-eIF4 axis to initiate translation of Ifng mRNA into protein. Indicated in orange are the pharmacological inhibitors used in this study and their targets.