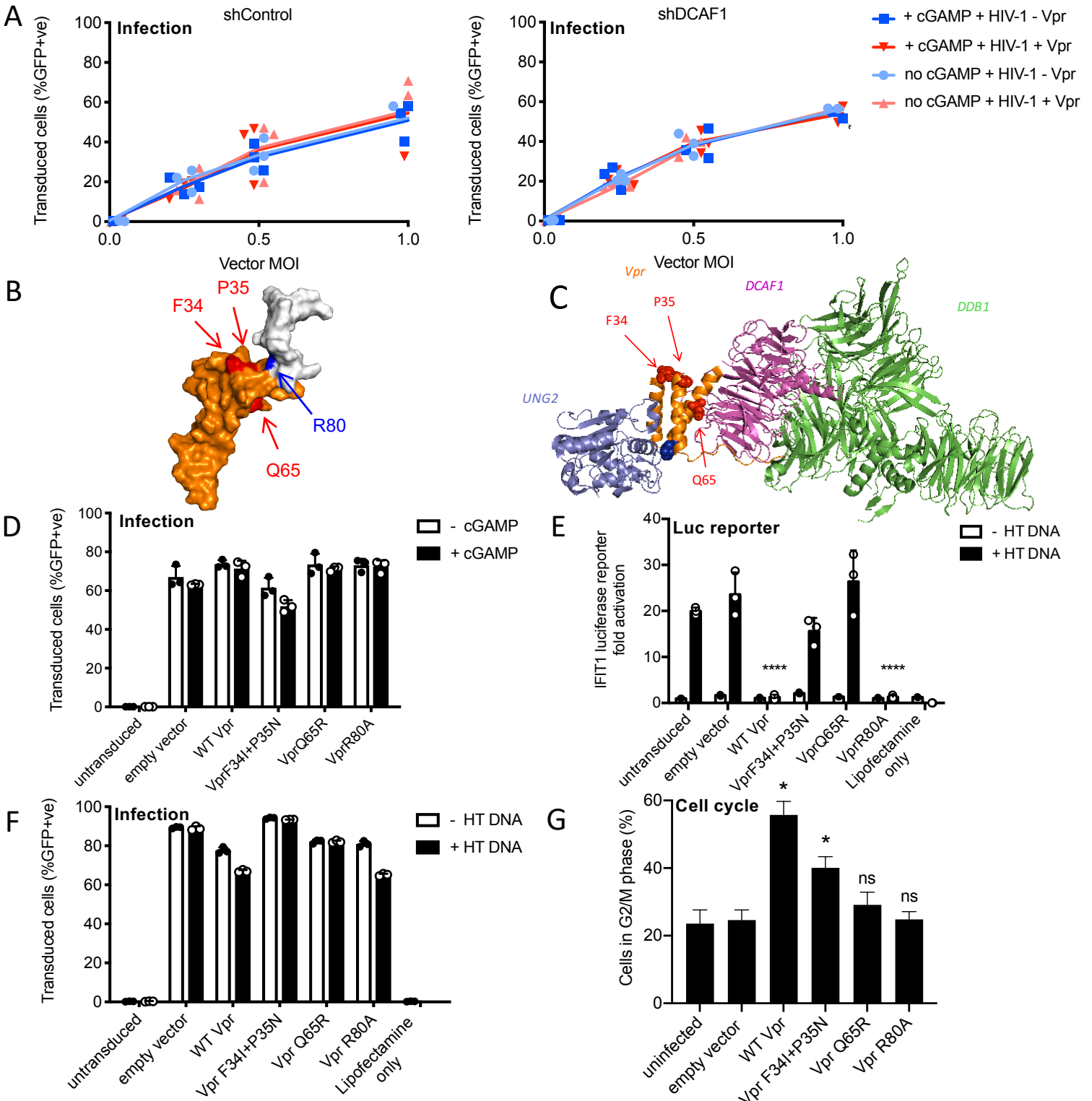

Figure 3 figure supplement 1 Vpr inhibition of innate immune activation is dependent on DCAF1 but independent of cell cycle arrest

(**A**) Percentage of THP-1 cells in Figure 3C transduced by the vector encoding Vpr and GFP, or empty vector encoding GFP alone, at the indicated MOI and treated with cGAMP (5 µg/ml) or left untreated. (**B**) NMR structure of full length Vpr showing position of Vpr mutants (PDB: 1M8L). White region (c-terminus) of Vpr shown in (B) is unresolved in the crystal structure (C). (**C**) Crystal structure of Vpr (orange) with its target protein UNG2 (blue) and cofactors DCAF1(pink) and DDB1 (green) showing position of Vpr mutations (PDB: 5JK7). (**D**) Percentage of THP-1 cells in Figure 3F transduced by the vector encoding WT, or mutant, Vpr and GFP (MOI 1), or empty vector encoding GFP alone (MOI 1), and treated with cGAMP (5 µg/ml), or left untreated as a control. (**E**) Fold induction of IFIT1-Luc after HT-DNA (5 µg/ml) transfection in cells expressing WT, or mutant, Vpr from a lentiviral vector (MOI 1), or empty vector (MOI 1), or in untransduced IFIT1-Luc reporter THP-1 cells. (**F**) Percentage of THP-1 cells in Figure S3E transduced with HIV-1 vector encoding WT, or mutant, Vpr and GFP (MOI 1), or empty vector encoding GFP alone (MOI 1), and transfected with HT-DNA (5 µg/ml) or left untransfected as a control. (**G**) Percentage of THP-1 cells in G2/M phase of cell cycle after transduction with an empty vector (MOI), or vector encoding WT Vpr, or mutant Vpr, (MOI 1) or left untransduced as a control. Mean+/- SEM n=2. Unless stated data are expressed as means ± SD (n = 3). Data is analysed using two-way ANOVA test. * ($p<0.05$), ** ($p<0.01$), *** ($p<0.001$), **** ($p<0.0001$) compared to empty vector. Data are representative of three (A), (D) or two (E-G) independent