Zheng et al, Supplemental Data

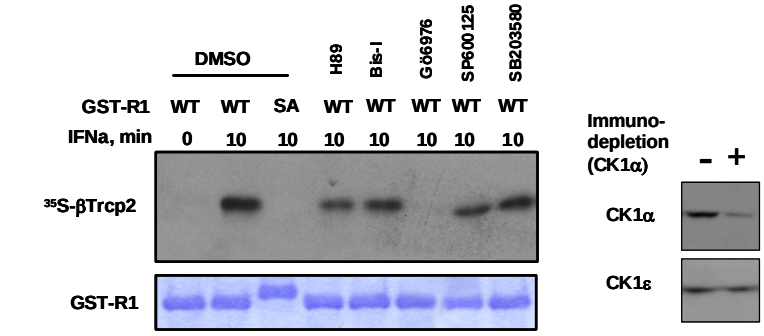

Figure S1: Binding of ^{35}S-β-Trcp2 to GST-IFNAR1 proteins (wild type or S535,539A mutant, "SA") upon their phosphorylation using CK1α-depleted lysates from 293T cells treated with IFNα as indicated. Efficacy of CK1α immunodepletion in the lysate from IFN-treated cells was verified by immunoblot using either anti-CK1α or anti-CK1ε antibodies (right panel). Levels of GST-IFNAR1 are analyzed by Coomassie Blue staining. The SA mutant migrates slower on SDS-PAGE due to the presence of four additional amino acids in the linker (1). Effects of various protein kinase inhibitors added to the reaction during the phosphorylation stage was also analyzed.

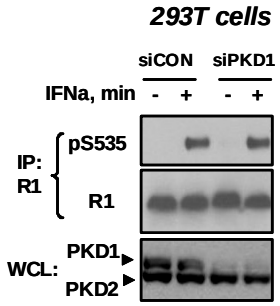

Figure S2: Phosphorylation of endogenous IFNAR1 in 293T cells transfected with siRNA against PKD1. Levels of PKD species were also analyzed by immunoblot in WCL.

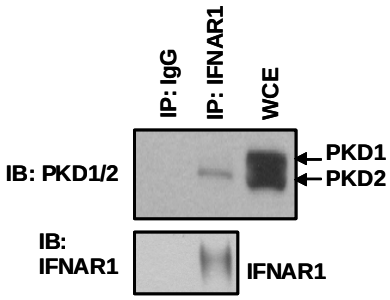

Figure S3: Co-immunoprecipitation of endogenous IFNAR1 and PKD2 from the lysates of 293T cells. Control reaction utilized an irrelevant monoclonal IgG antibody (anti-Flag).

Supplemental References

1. **Liu, J., W. C. HuangFu, K. G. Kumar, J. Qian, J. P. Casey, R. B. Hamanaka, C. Grigoriadou, R. Aldabe, J. A. Diehl, and S. Y. Fuchs.** 2009. Virus-induced unfolded protein response attenuates antiviral defenses via phosphorylation-dependent degradation of the type I interferon receptor. Cell Host Microbe **5:**72-83.