

Expanded View Figures

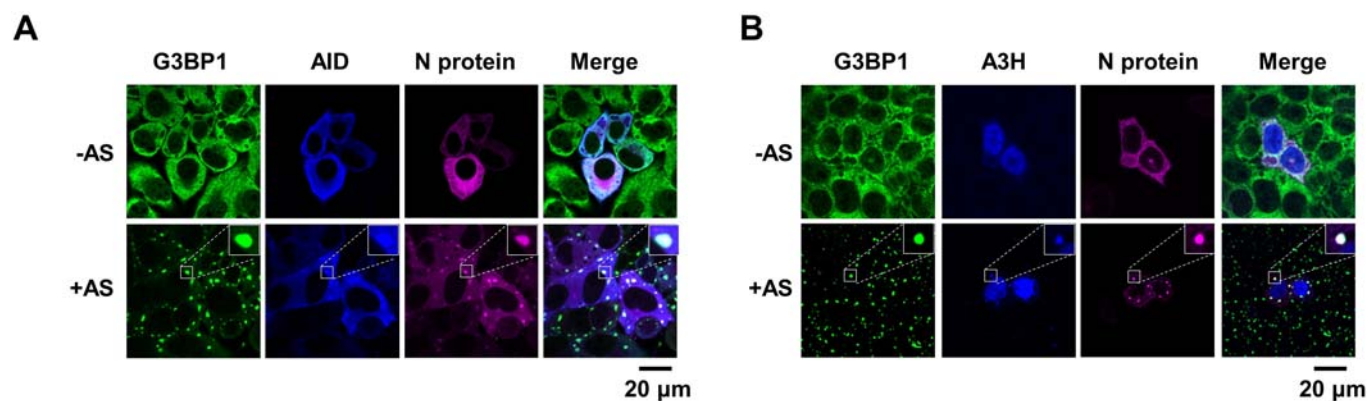
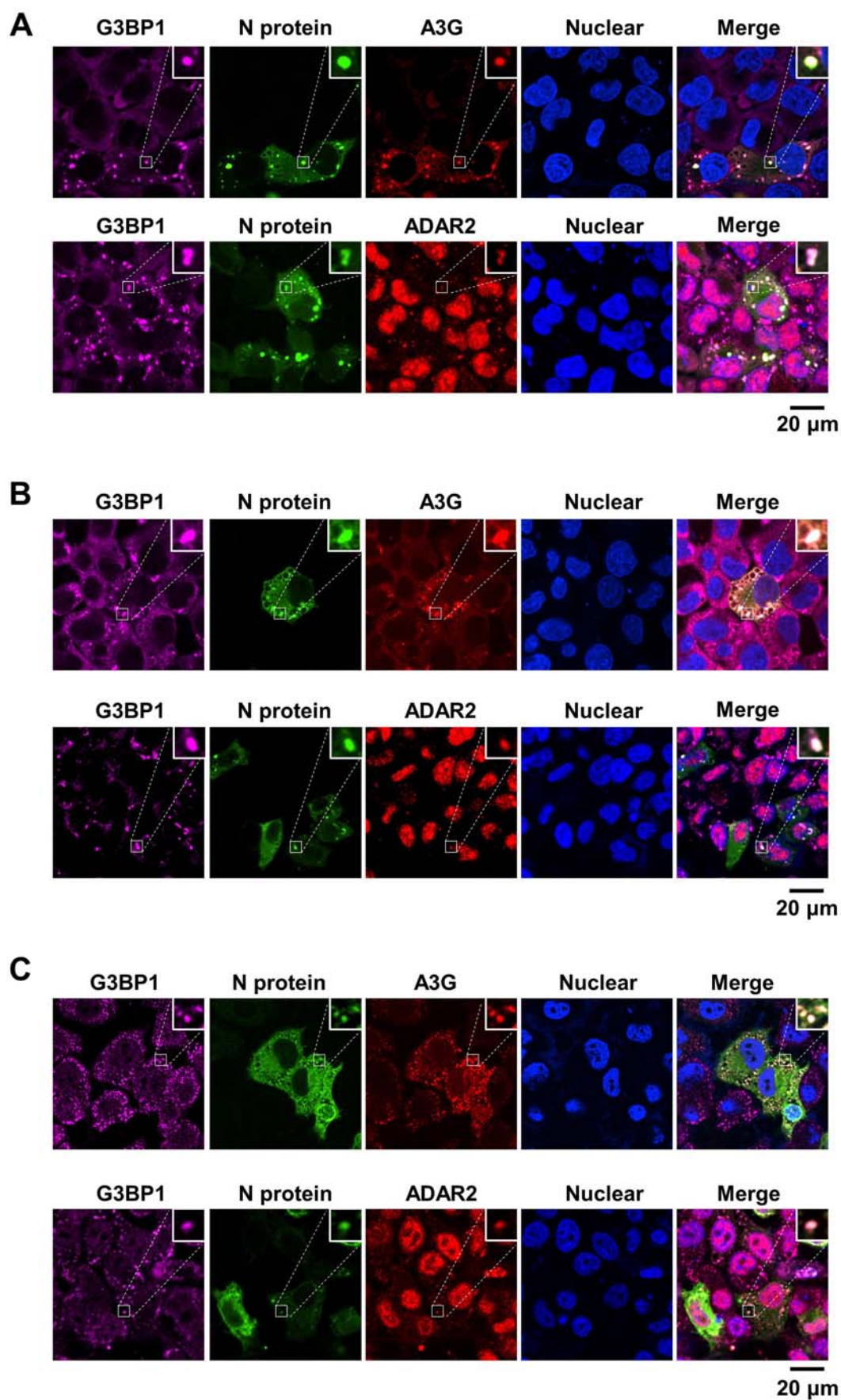


Figure EV1. Co-localization of N Protein with AID and A3H in SGs.

(A) AID co-localizes with N protein in SGs in HeLa cells. HeLa cells were transfected with the N gene and AID, then treated with AS for 45 min to induce SG formation, followed by immunostaining for N protein, AID and G3BP1. Scale bar: 20 μ m. (B) A3H co-localizes with N protein in SGs in HeLa cells. HeLa cells were transfected with the N gene and A3H, then treated with AS for 45 min to induce SG formation, followed by immunostaining for N protein, A3H and G3BP1. Scale bar: 20 μ m.



◀ Figure EV2. Specific co-localization of N protein with host deaminases under diverse stress conditions.

(A–C) A3G or ADAR2 co-localizes with N protein in SGs in response to various stressors. HeLa cells were transfected with the N gene and A3G or ADAR2, and treated with polyI:C for 7 h (A), DTT for 1 h (B) or sorbitol for 1 h (C) to induce SG formation, followed by immunostaining for N protein, A3G or ADAR2 and G3BP1. Scale bar: 20 μ m.

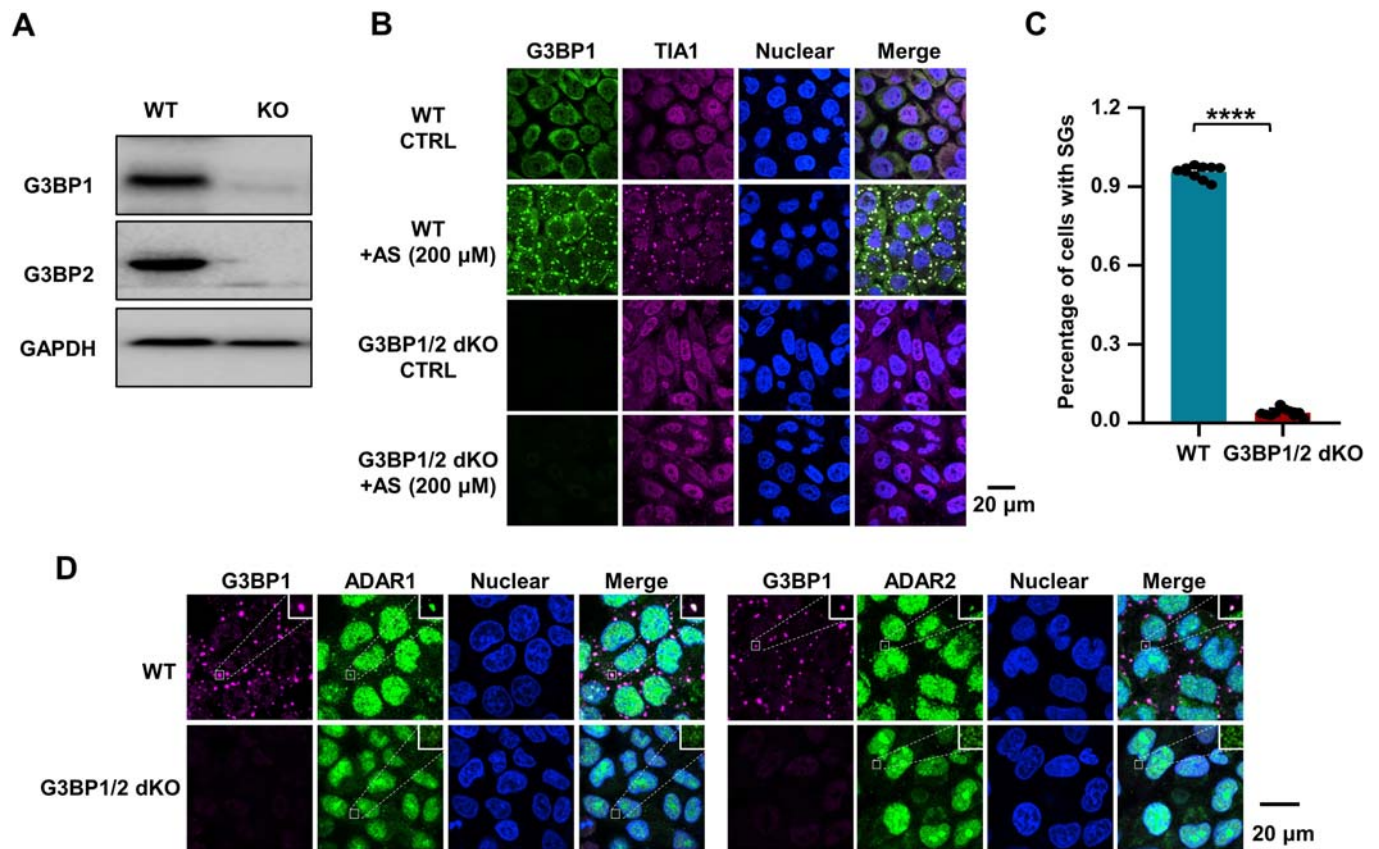
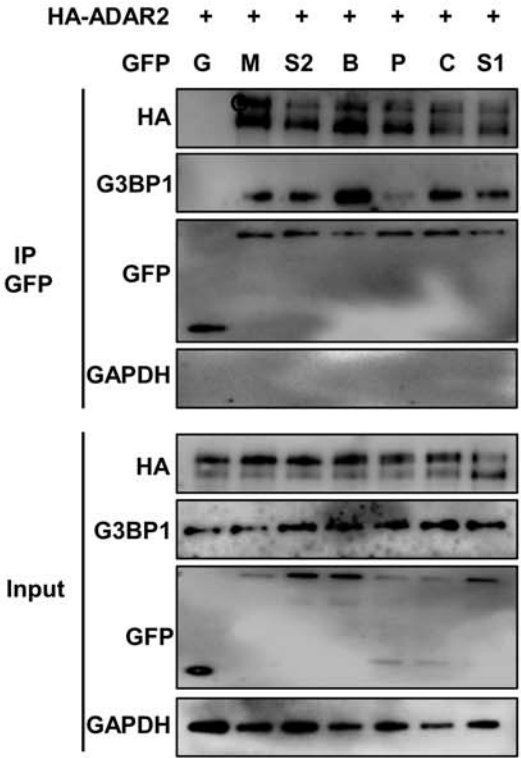


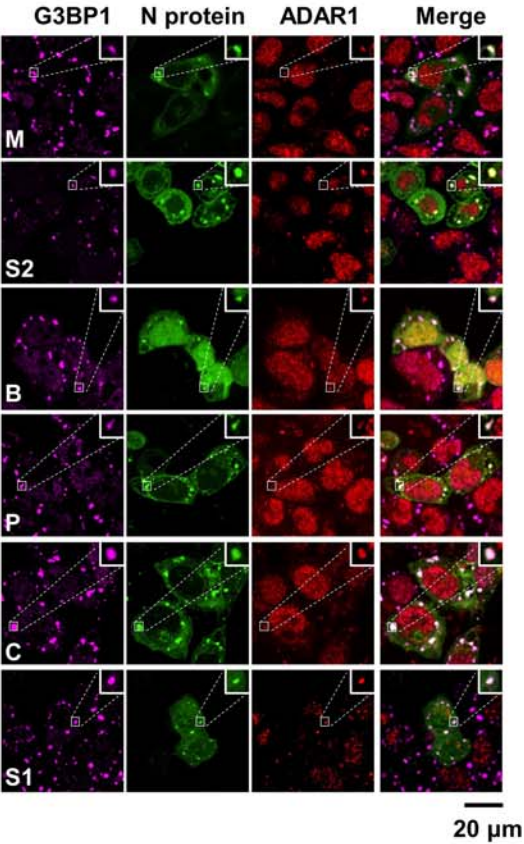
Figure EV3. The lack of G3BP1/2 disrupts the formation of N protein-deaminase complex-containing RNA condensates.

(A) CRISPR-Cas9-mediated G3BP1/2 knockout in HeLa cells. (B) G3BP1/2 dKO HeLa cells fail to form SGs under AS-induced stress. HeLa cells with or without G3BP1/2 dKO were treated with AS for 45 min to induce SG formation, followed by immunostaining for the endogenous G3BP1 (GFP) and endogenous TIA1 (red). Scale bar: 20 μm. (C) Quantification of SGs, expressed as the percentage of cells containing SGs in fixed HeLa cells with or without G3BP1/2 dKO. Data are shown as means $\pm$ SEM ($n = 10$ independent images). Statistical analysis was performed with a two-tailed unpaired t-test. **** $P < 0.0001$. (D) Depletion of G3BP1/2 abolishes the localization of ADAR1/2 in SGs under stress. HeLa cells with or without G3BP1/2 dKO were treated with AS for 45 min to induce SG formation, followed by immunostaining for endogenous G3BP1 and endogenous ADAR1 or ADAR2. Scale bar: 20 μm. Source data are available online for this figure.

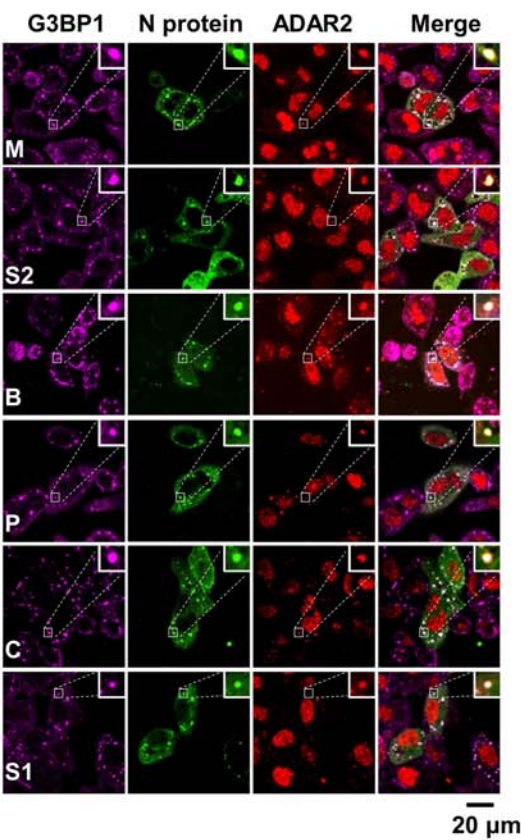
A



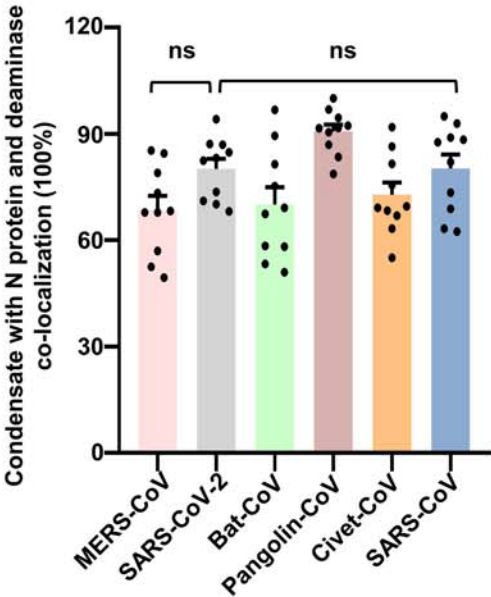
B



C



D



**Figure EV4. Functional characteristics of coronavirus N protein.**

(A) Interaction assay between N protein and ADAR2. HeLa cells were co-transfected with plasmids encoding GFP-tagged N proteins of MERS-CoV, SARS-CoV-2, bat-CoV, civet-CoV, pangolin-CoV and SARS-CoV or GFP control, and HA-tagged ADAR2. Cell lysates were immunoprecipitated with an anti-GFP antibody, and the expressed proteins were analyzed by western blotting. (B) N proteins from MERS-CoV, SARS-CoV-2, bat-CoV, civet-CoV, pangolin-CoV and SARS-CoV exhibit the ability to co-localize with ADAR1 in SGs in HeLa cells. HeLa cells transfected with various N proteins were treated with AS for 45 min to induce SG formation, followed by immunostaining for N protein, ADAR1 and G3BP1. Scale bar: 20 μ m. (C) N proteins from MERS-CoV, SARS-CoV-2, bat-CoV, civet-CoV, pangolin-CoV and SARS-CoV exhibit the ability to co-localize with ADAR2 in SGs in HeLa cells. HeLa cells transfected with various N proteins were treated with AS for 45 min to induce SG formation, followed by immunostaining for N protein, ADAR2 and G3BP1. Scale bar: 20 μ m. (D) Quantification of condensates with co-localization of N protein and deaminase in fixed HeLa cells. Data are shown as mean $\pm$ SEM ($n = 10$ independent images). Statistical analysis was performed with a one-way ANOVA test. ns > 0.05, ns, not significant.