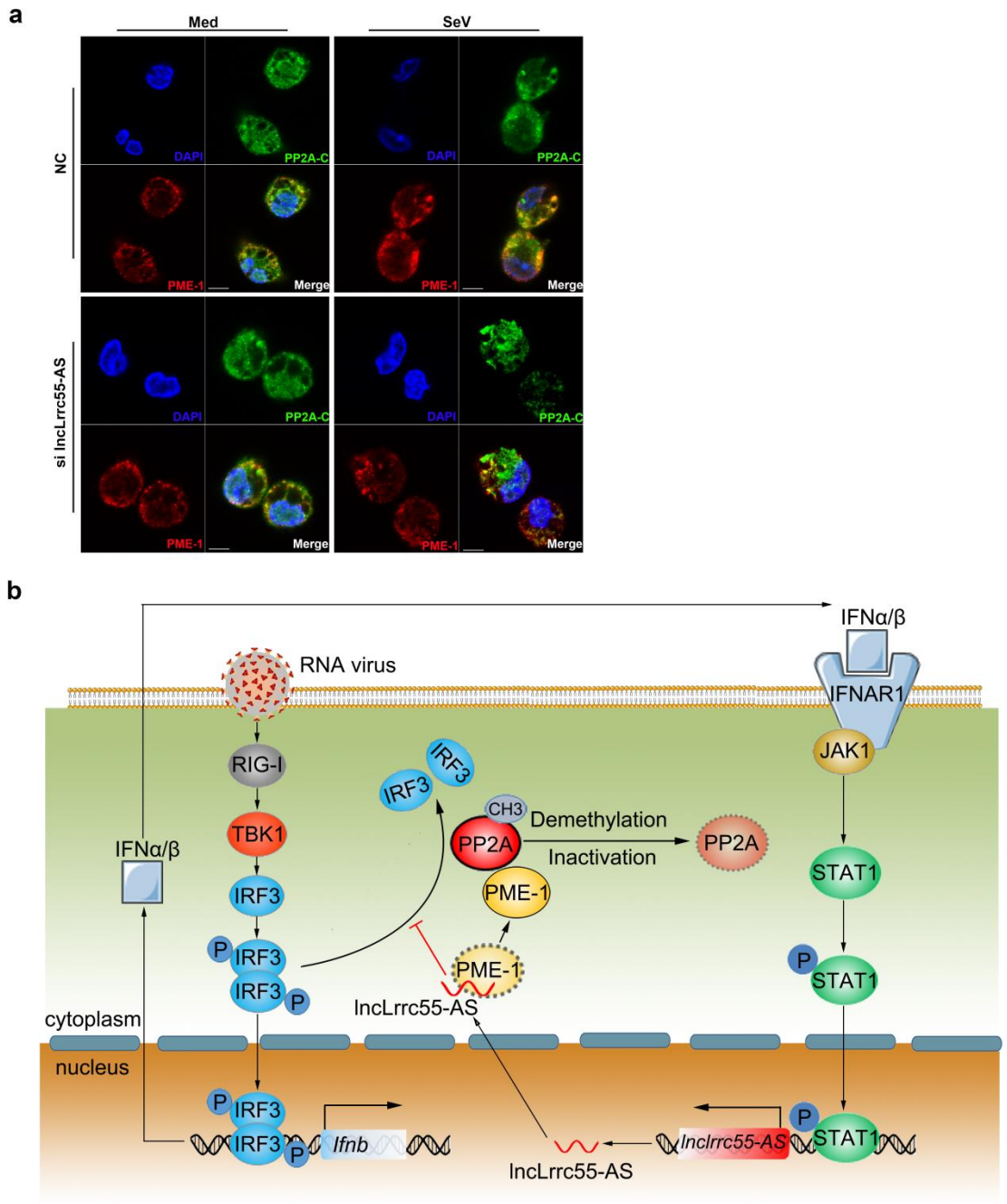

Supplementary information, Fig. S10 lncLrrc55-AS promotes the interaction of PME-1 and PP2A. **a** IF detection of the endogenous PME-1 and PP2A-C in NC or lncLrrc55-AS-silenced peritoneal macrophages infected with or without SeV for 12 hours. DNA (blue) was stained by DAPI and PP2A-C (green) and PME-1(red) were detected by antibodies. Scale bars, 10 μm. Data are representative of three independent

experiments with similar results. **b** Working model of type I interferon-inducible lncLrrc55-AS feedback promotes type I interferon production in antiviral immunity by binding methylesterase PME-1 to strengthen IRF3 signaling. lncLrrc55-AS is upregulated by IFN-JAK-STAT signal pathway in response to viral infection or other innate stumuli. The cytoplasmic lncLrrc55-AS directly binds to methylesterase PME-1 and promotes PME1 to catalyze demethylation and inactivation of PP2A, thus inhibiting PP2A-mediated dephosphorylation of IRF3. As a consequence, lncLrrc55-AS strengthens IRF3 phosphorylation and empowers IRF3-initiated induction of type I IFN in antiviral innate immunity.