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SARS-CoV-2 ORF7a and Spike do not antagonize Tetherin

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BST-2 or Tetherin is a component of the innate immune system that has antiviral properties and is stimulated by interferons. Tetherin has been shown to provide non-specific inhibition of the release of enveloped virions from the surface of infected cells by targeting and tethering them to the plasma membrane. Several viruses have evolved Tetherin antagonists that allow them to be released from the cell surface including HIV, Influenza, Ebola virus, and SARS-CoV-1. In the case of SARS-CoV-1, it has been suggested that the ORF7a accessory protein is responsible for Tetherin antagonism. Because of its similarity to SARS-CoV-1 and its enveloped nature, it has been speculated that SARS-CoV-2 also contains an antagonist to Tetherin. The two main proponents for SARS-Cov-2 Tetherin antagonism are ORF7a and the Spike glycoprotein (S) found on the surface of the virus. Our goal was to determine if Tetherin has an inhibitory effect on viral release of viral like particles containing the SARS-CoV-2 S protein, and if ORF7a or SARS-CoV-2 S have an effect on the inhibitory function of Tetherin. Our data suggests that while Tetherin is capable of inhibiting VLPs containing the S protein, neither ORF7a nor S appear to antagonize Tetherin.