



Fig. S11. Cocktail of ASK-related antibodies substantially blocks SARS-CoV-2 entry.

a and b, KREMEN1, ASGR1 or ACE2 expressed HEK293E cells were incubated with SARS-CoV-2 S-ECD-hFc ($10\mu\text{g/ml}$ final concentration) in the presence of anti-ASGR1 S23 antibody (**a**) or anti-KREMEN1 K33 antibody (**b**). S-ECD binding relative to control antibody treatment were measured by flow cytometry ($n=2$). **c**, S23 and K33 ($5\mu\text{g/mL}$ final concentration) do not affect SARS-CoV-2 S-driven entry into ACE2 dependent NCI-H1650 and Huh-7 cells. Luciferase activity was measured 48 hr post-infection ($n=3$). **d**, NCI-H661 and NCI-H1944 were infected with S-pseudotyped SARS-CoV-2 in the presence of Ab-414 and K33 ($5\mu\text{g/mL}$ final concentration) separately or combined. Luciferase activities were measured 60 hr post-infection (mean \pm SEM, $n=3$). **e**, Immuno-fluorescence staining showed the expression of ACE2, ASGR1 and KREMEN1 in normal human lung samples (Bar = $100\mu\text{m}$). **f**, Human lung organoids were infected with (right) or without (left) rVSV-GFP/SARS-CoV-2 S chimeric virions (SARS-CoV-2/GFP). GFP⁺ cells were measured by flow cytometry 24hr post infection. Data in a-d are present as mean \pm SEM, unpaired two-tailed Student's *t*-tests, * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.