**Background**
- The early part of the SARS-CoV-2 pandemic coincided with circulation of other respiratory viruses, such as influenza, and the second wave is expected to coincide with the upcoming influenza season.
- Since SARS-CoV-2 is co-circulating with other respiratory viruses, viral coinfections are likely to occur. However, in the initial phase of the pandemic, clinicians noted fewer SARS-CoV-2 coinfections than were expected.
- We use mathematical models to investigate the mechanism for the lack of coinfections and to investigate what that might mean for a second wave.

**SARS-CoV-2 coinfections**
- In this model, the viruses interact via competition for the resource of cells.
- Viruses with higher growth rates infect cells faster than viruses with lower growth rate, leaving the slower virus with no cells to infect.

**Role of growth rate**
- Virus growth rates: IAV (5.14), RSV (13.6), hRV (11.9)
- Virus growth rates: PIV (9.07), hMPV (3.99), SARS (1.80)
- Viruses with higher growth rates infect cells faster than viruses with lower growth rate, leaving the slower virus with no cells to infect.

**Sequential infections**
- Even if the second infection is initiated after SARS-CoV-2, it can still suppress SARS-CoV-2.

**In-host coinfection model**
- In this model, the viruses interact via competition for the resource of cells.
- Target cells: \( \frac{dI}{dt} = -\beta_I T V - \beta_T V \)
- Infected cells: \( \frac{dI}{dt} = \delta I - \delta I \)
- Influenza virus: \( \frac{dV}{dt} = p I - c V \)
- Role of growth rate
  - Virus growth rates: IAV (5.14), RSV (13.6), hRV (11.9)
  - Virus growth rates: PIV (9.07), hMPV (3.99), SARS (1.80)
- Viruses with higher growth rates infect cells faster than viruses with lower growth rate, leaving the slower virus with no cells to infect.

**Epidemiological model**
- If SARS-CoV-2 is so easily suppressed by other respiratory viruses, what might be the implications for a second wave? We developed an epidemiological model where one virus prevents coinfection with the other.

**SARS-CoV-2 coinfections and implications**
- We find a double peak in the hospitalizations; the first caused by influenza patients, the second caused by SARS-CoV-2 patients.

**Hospitalization**
- We used partial rank correlation coefficient to determine which parameters are most important for determining the course of the epidemic.
- Aside from the hospitalization rates \( h_1 \) and \( h_2 \) total number of hospitalized patients is most strongly determined by the infection rate of influenza while the timing between peaks is determined by both infection rates and the hospitalization rate of influenza.

**Parameter sensitivity**
- Within the host, coinfecting viruses compete for the resource of cells.
- SARS-CoV-2 appears to have a lower within host growth rate than other respiratory viruses and tends to be suppressed during coinfections.
- At the population level, co-circulation of influenza and SARS-CoV-2 leads to a temporary suppression of SARS-CoV-2 infections. Once people have recovered from influenza, SARS-CoV-2 reappears.
- This results in a double peak in the number of infected people — the first peak caused by influenza, the second by SARS-CoV-2.