Multi-pathogens spreading dynamics in an individual-based model accounting for home isolation and heterologous effects

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Introduction

The spread of an infectious disease is a complex biological process influenced by a variety of factors, including the co-circulation of other infectious agents. When pathogens invade the same site of infection, competitive or co-operative interactions may occur, respectively reducing or increasing the chance of a successful host invasion. Based on hypotheses reported in the literature, we show through a simulation study how heterologous effects in combination with home isolation can affect the spread of COVID-19 and influenza, and of two COVID-19 strains.

Data and Methods

To describe the spread of two co-circulating pathogens z_1, z_2 in a closed population, we extended the framework of the effective contact process [1]. Briefly, contact interactions are first considered between pairs of individuals, e.g., i, j, and infection events result from such contacts according to a time-varying probability:

$$p_{i,j}^{z_1}(t) = q_{i,j}^{z_1} \nu_i^{z_1}(t) \rho_j^{z_1,z_2}(t, t_{v_j}^{z_1,z_2}, t_j^{z_1,z_2}) \qquad (1)$$

where:

- $q_{i,j}^{z_1}$ is the *transmission potential* for pathogen z_1.
- $\nu_i^{z_1}$ is the *infectiousness measure*.
- $\rho_j^{z_1,z_2}$ is the *interaction term* which reflects the effect of previous exposures ($t_j^{z_1,z_2}$ and $t_{v_j}^{z_1,z_2}$).

Simulation model We developed an individual-based model in a population with two-levels of mixing, assuming that symptomatic carriers experiencing a COVID-19 infection restrict their contacts to local interactions at symptom onset. Household network and contact rates are informed using data for Belgium, and disease-specific parameters set to represent influenza and COVID-19.

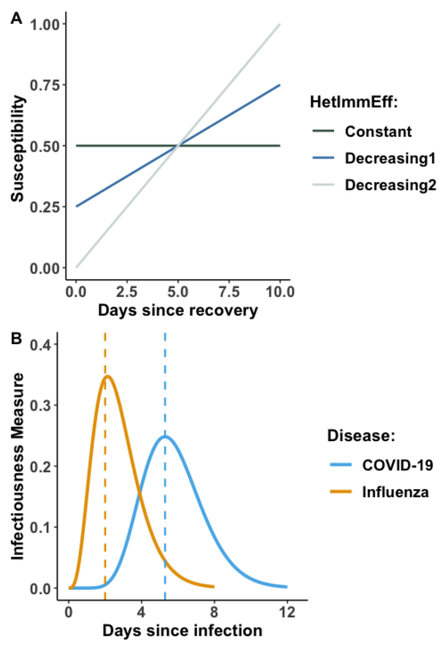

Figure 1: Effect of an enhanced innate response on susceptibility (A), and infectiousness measures (continuous lines) and incubation periods (dashed lines) for COVID-19 and influenza (B).

Results

Simulations show that home isolation because of a COVID-19 symptomatic infection affects the co-circulation of influenza (Figure 2 - A). When additionally assuming that an antigen-independent response drives heterologous effects [2], COVID-19 outbreaks are less severe if the disease is introduced at the same time as influenza. The invasion probability and the severity of an influenza outbreak strongly reduce when the pathogen is seeded later than COVID-19 (Figure 2 - B).

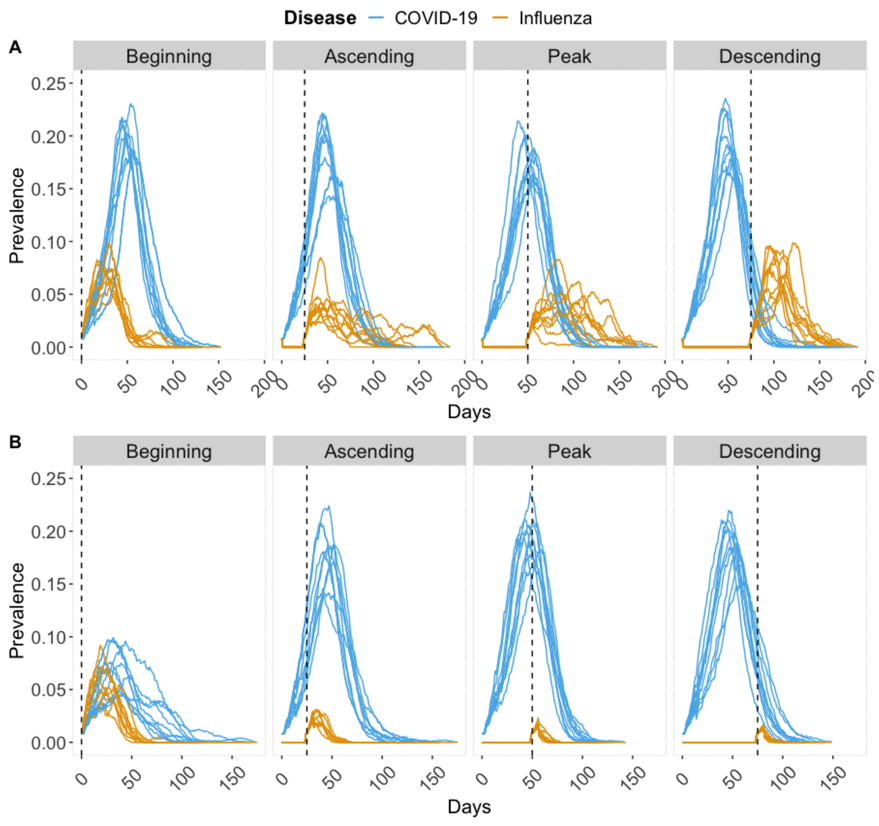

Figure 2: Incidence curves for the co-circulation of COVID-19 and influenza when considering home isolation (A), and home isolation and an enhance immune response (B).

Moreover, we implemented an increased susceptibility to COVID-19 after a previous influenza infection [3]. We assumed the susceptibility to linearly decay from the value 2 to 1 after 30 days, while still considering a competitive interaction for influenza after COVID-19. As a result, COVID-19 gains an advantage from an earlier influenza introduction, or when the infection is introduced after influenza (Figure 3). Limited influenza outbreaks are observed only when this disease is seeded before or at the same time of COVID-19.

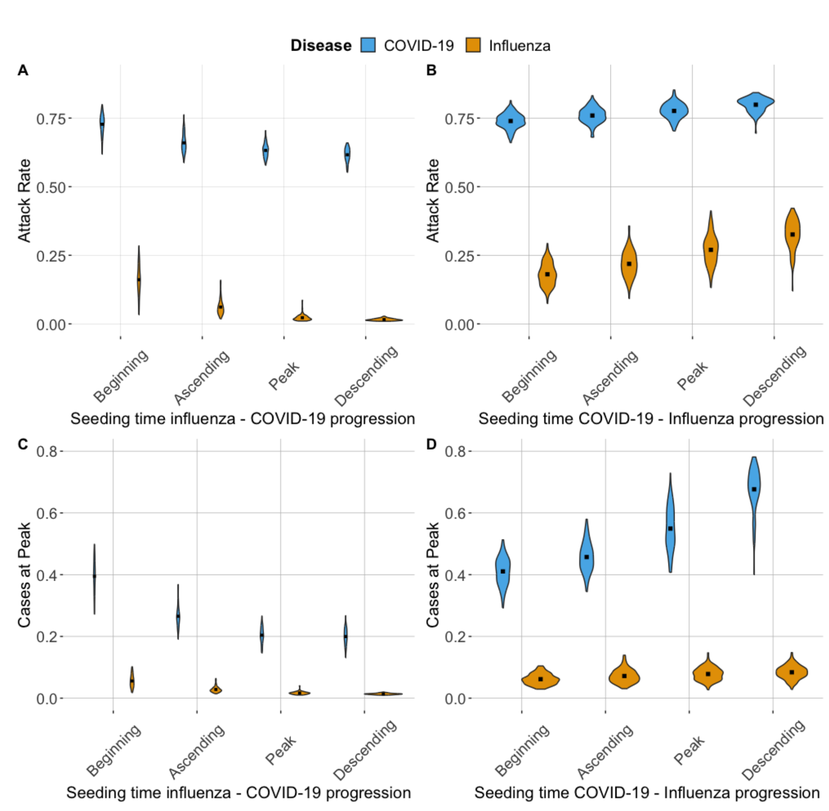

Figure 3: Attack rate (A,B) and cases at peak (C,D) when considering home isolation, increased and decreased susceptibility after an influenza and COVID-19 infection respectively, and different seeding times.

We further considered two COVID-19 strains, i.e. Omicron and Delta, informing the antigen-specific heterologous effects using vaccine effectiveness estimates against Omicron symptomatic infections [4]. While vaccination decreases the overall attack rate (Figure 4 - A), a similar number of cases at peak is observed for an increase in coverage (Figure 4 - B).

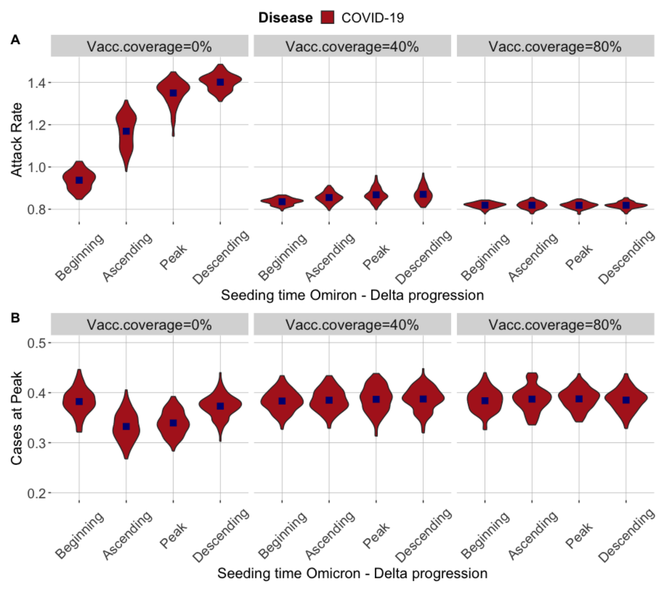

Figure 4: Attack rate (A) and number of cases at peak (B) for a different vaccination coverage when cases for both strains are jointly considered.

Summary

The co-circulation of different pathogens influences their spreading dynamics. We tested a few hypotheses representing immunological mechanisms that could affect the spread of influenza and COVID-19, and of two COVID-19 strains, computing a substantial impact on the spread of such diseases. This work highlights the need of collecting data from which the interactions among different pathogens can be quantified.

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References

[1] Torneri, A., et al. (2022). eLife, 11, e75593. https://doi.org/10.7554/eLife.75593 [2] Chan, K. F., et al. (2018). The Journal of infectious diseases, 218(3), 406–417. https://doi.org/10.1093/infdis/jiy184 [3] Kim, S.Y., et al. (2021). Sci Rep 11, 21568. https://doi.org/10.1038/s41598-021-00428-x. [4] Chemaitelly, H., et al. (2022). Nat Commun 13, 3082. https://doi.org/10.1038/s41467-022-30895-3

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