



Microscopic dynamics modeling unravels the role of asymptomatic virus carriers in SARS-CoV-2 epidemics at the interplay between biological and social factors



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ABSTRACT

The recent experience of SARS-CoV-2 epidemics spreading revealed the importance of passive forms of infection transmissions. Apart from the virus survival outside the host, the latent infection transmissions caused by asymptomatic and presymptomatic hosts represent major challenges for controlling the epidemics. In this regard, social mixing and various biological factors play their subtle, but often critical, role. For example, a life-threatening condition may result in the infection contracted from an asymptomatic virus carrier. Here, we use a new recently developed microscopic agent-based modelling framework to shed light on the role of asymptomatic hosts and unravel the interplay between the biological and social factors of these nonlinear stochastic processes at high temporal resolution. The model accounts for each human actor's susceptibility and the virus survival time, as well as traceability along the infection path. These properties enable an efficient dissection of the infection events caused by asymptomatic carriers from those which involve symptomatic hosts before they develop symptoms and become removed to a controlled environment. Consequently, we assess how their relative proportions in the overall infection curve vary with changing model parameters. Our results reveal that these proportions largely depend on biological factors in the process, specifically, the virus transmissibility and the critical threshold for developing symptoms, which can be affected by the virus pathogenicity. Meanwhile, social participation activity is crucial for the overall infection level, further modulated by the virus transmissibility.

1. Introduction

Epidemics spreading of new coronavirus in population is a collective social phenomenon. It arises from individual actors' behaviour that can get infected and spread the infectious agents via multiplexity of contacts. In this regards, the human participation activity conditioned by daily mobility patterns has been recognised as a primary driving force for the epidemics spreading [1–3]. Contrary to many social processes, the epidemic spreading has a vital biological component, which is prominent at the elementary interactions scale [4,5]. In this respect, the recent developments with the SARS-CoV-2 epidemics have revealed several new features that were not previously recognised in virus spreading, see recent update in Ref. [6]. These are sizeable effects of the passive modes of the infection transmission [8–12], which can be related to the virus

biology and strongly individual susceptibility of the human hosts to this particular virus [13–16]. Specifically, these latent infection transmissions rely on the virus long survival time outside the human host [14, 17], which enables an indirect transmission to a new host. On the other hand, a considerable amount of asymptomatic hosts can remain unidentified [18–21]. Recent empirical studies of COVID-19 epidemics reveal the silent transmissions occurring in different social or familiar circles [22–24]. Efforts are made to evaluate the contributions of pre-symptomatic and asymptomatic cases and their implications to control the epidemics [25,26], in particular, by combining mathematical modelling with available biomedical data for a given population. For example, in an outstanding study [26], the authors take into account data on the seroprevalence for SARS-CoV-2 antibodies in populations on nine different locations in conjunction with compartmental epidemics

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modelling to project “the temporal evolution and credible intervals of the effective reproduction number and the symptomatic, asymptomatic and recovered populations”. Noteworthy, these results revealed a symptomatic fraction that remains below 20% in all locations. Here, we address these issues using the microscopic agent-based modelling to reveal how different outcomes at the population level can emerge in the interplay of high-resolution social dynamics and relevant biological factors.

Since the very beginning of the SARS-CoV-2 epidemics, it has been revealed that a wide spectrum of symptoms may occur, from very mild or none, on one side, to severe symptoms and life-threatening pneumonia requiring ICU treatment and possible fatal outcome [14]. Apart from potential virus mutations over time, e.g., changed transmissibility and pathogenicity [11,27,28], the observed individual susceptibility of human actors to the virus may range from a specific genetic origin to diverse factors related to the individual’s health condition [13,29,30]. The symptoms develop over a short period between 2 and 7 days. Meanwhile, the asymptomatic cases spontaneously recover within a period of one to two weeks, changing infectiousness over time [24,31]. The evidence of the temporal variation of infectiousness before and after the symptoms as well as in asymptomatic carriers are subject of investigations and debated among researchers [32,33]. Even though their viral load varies differently in time [32,34], both symptomatic and asymptomatic hosts are the virus carriers and can spread the infection, cf. Fig. 1. Specifically, the circulating viruses produced by asymptomatic hosts can infect a susceptible individual, who, depending on the susceptibility, may or may not develop symptoms. Similarly, the viruses produced by presymptomatic carriers can lead to asymptomatic as well as symptomatic cases. Moreover, some measurements suggest that the viral contents are proportional to the severity of symptoms, and can vary over the infectious time [34]. Early estimates were that the proportion of asymptomatic cases could be as large as 80% of all infected [14]. A recent meta-analysis of the available data suggests a wide variation in the estimates from 20% up to 60% of asymptomatic carriers [20,21].

Given the occurrence of a sizable amount of asymptomatic virus carriers, it is challenging to estimate the right parameters and predictions of the epidemics from the immediate data analysis [26,35,36]. Thus, it constitutes a considerable problem for efficient combat with the virus spreading [19,37–39]. The problem is increasingly more scientifically interesting in the third-wave epidemics because the virus circulates among a large fraction of the population. Hence, a better understanding of the factors that determine the proportion of the asymptomatic virus carriers in a given social environment, and their impact on the overall infection growth is vital for managing the disease outbreak. In this work, we tackle these problems using the microscopic dynamics approach within the agent-based modelling framework developed in Ref. [1].

Complementary to the standard mean-field models with continuous-time dynamics of interdependent equations for groups [40,41], the microscopic agent-based modelling of SARS-CoV-2 epidemics gains an increasing attention [1,42–47]. As remarkable examples, we mention the modelling frameworks that were developed to simulate the epidemic outcomes by including accurate patterns of mobility at a particular geographic location [44] or statistical data of a given population [47]. This modelling approach provides us with intrinsic mechanisms of the epidemic processes, and the information on how it develops from the elementary-interaction level to the global-scale outcome. Moreover, it allows for considering individual features of the actors, their mobility and location of the interactions and participation in coupled stochastic processes [1,42–46] and intra-host viral dynamics [47]. We have recently developed an agent-based model for the latent infection

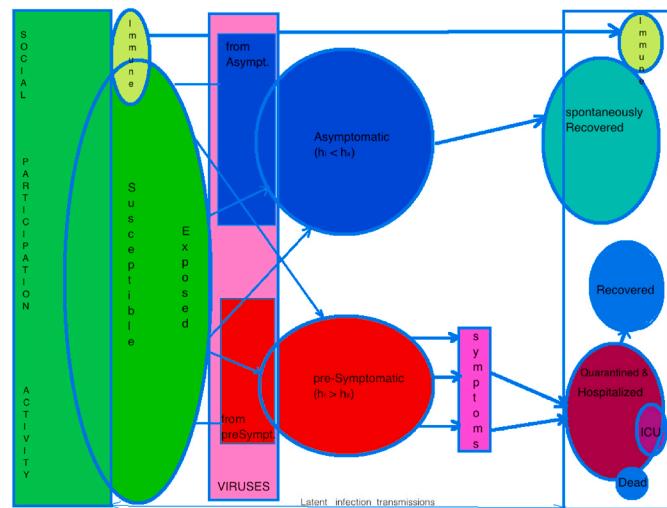


Fig. 1. Schematic view of the processes: new susceptible individuals become exposed to active viruses. An infected individual is “asymptomatic”, if its susceptibility is low $h_i < h_x$, or “presymptomatic” if its $h_i \geq h_x$, and develops symptoms over time; subsequently, it becomes “quarantined” or “hospitalised” and thus removed to a controlled environment. Whereas, “asymptomatic” carriers over time become “spontaneously recovered”. The active circulating viruses consist of a group produced “by asymptomatic” and “by presymptomatic” hosts. Thus, they can infect either the “asymptomatic” or “presymptomatic” group, depending on the agent’s susceptibility. Groups shown in the right rectangle do not contribute to the studied latent transmissions.

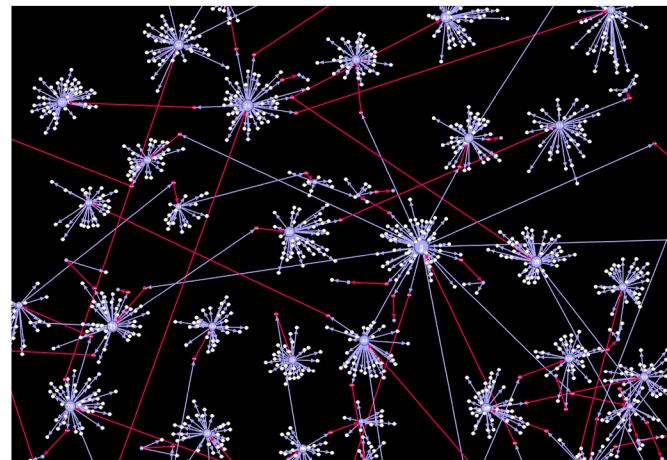


Fig. 2. The close-up bipartite network of Host (large, blue) and Virus (small, white) nodes. The directed blue edges connect the Host with the viruses that it produced. Infection paths along the directed edges between successive Hosts via Virus nodes are shown in red.

transmissions [1], where the human actors possess individual susceptibility to the virus. It thus allows us to differentiate between highly susceptible individuals, who can develop symptoms from those who are less susceptible and may become asymptotically infected. The new host’s susceptibility accordingly modulates the probability of getting infected and the probability of that host to produce a new generation of viruses. Moreover, the model enables us to consider a finite survival time of the virus outside the host and different exposure times of each human

actor. The process is visualised as a growing bipartite graphs, see Fig. 2, with host nodes who are producing the virus nodes (infected spots) during their infectious time and proportionally to the degree of infectiousness. A more detailed description of the model is given in the next section, see also [1] for further details. This graphic representation of the process enables us to identify an infection path leading to each case. Therefore, in this modelling approach, each infection event where an actor encounters the virus is marked by the new host's features and the host that produces the active virus.

In this work, we extend the developed modelling framework for latent infections such that we keep the information about the preceding host's susceptibility when the virus is transmitted to a new host. In this way, each infection case can be distinguished as either coming from a sensitive, i.e., potentially symptomatic case before it gets hospitalised or quarantined, or otherwise from a low-susceptibility host being asymptomatic. The *threshold susceptibility* h_x , which distinguishes the highly susceptible agents from those that are potentially asymptomatic, is a varying parameter in our model. The values of this parameter depend on the health and genetic factors of a given population and on the pathogenicity of the virus in question, possibly assessable from some empirical data [13,18,19,21]. We note that these stochastic processes involving symptomatic and asymptomatic hosts are strongly entangled at the level of interactions between the agents and viruses as the most elementary constituents of the process, cf. Fig. 1. Therefore, they can be suitably differentiated only at the microscopic interaction scale within individual-based modelling. Precisely, by tracing every infection event, we can differentiate the virus original host and, thus, determine its contribution to the growth of the infectious curve over time. With the extensive simulations, we demonstrate how the asymptomatic host's contribution to the infection growth varies with the social participation activity and the biological factors that determine the threshold susceptibility and transmission rate. Our simulations confirm that the overall infection level critically depends on social participation activity. Meanwhile, the biological factors are primarily responsible for the respective proportions of the asymptomatic and presymptomatic hosts in the overall infection curve.

2. Latent infection transmissions: model details

We adopt the model for latent infection transmissions developed in Ref. [1], specifically, introducing the differentiation between an asymptomatic infected case from those who can develop symptoms, as schematically shown in Fig. 1. As in the original work [1], the visualisation of the process by an evolving bipartite graph enables us to identify the infection path leading to each agent. It consists of infected individuals (Host nodes), who can produce viral spots (Virus nodes) during their infectious time, cf. Fig. 2. The *social participation* activity drives the dynamics; as a good proxy, we use an empirical time series s_t inferred from social networks [48]. In analogy to the time series of the human mobility within cities [44,49], this time series represents a cumulative activity of an open social group in an area with circulating viruses. Thus, it does not assume any prior relationships among participants (see also the Discussion section).

Features that influence the dynamics are several individual characteristics of the human actors $H\{\dots\}$ and viruses $V\{\dots\}$ summarised as follows

$$H\{i; h_i, T_h^i, r_h^i; state\}; V\{j; T_v; g_j, O_h^{i \rightarrow j}; state\}. \quad (1)$$

Specifically, apart from a unique identity, i , each human actor has its

individual *susceptibility* to the virus, h_i , which critically determines its role in the process; in particular, it modifies the agent's likelihood of infection in contact with the virus and also its productivity of new viruses after infection has occurred, as explained below. For each created agent, it is taken from a uniform random distribution in the range $h_i \in [0, 1]$. Furthermore, each agent has a characteristic exposition to the viruses specified by its *exposure time* T_h^i measured in hours; we take a uniform random number $T_h^i \in [1, T_h]$, where $T_h = 24$ hours. As mentioned in the Introduction, when a highly susceptible agent $h_i \geq h_x$ is infected, it can develop symptoms over a certain number of days, $r_h^i \in [2, 7]$, characteristic to that agent; consequently, it stops contributing to the latent transmissions by moving to a controlled environment (hospital or quarantine) [1]. Meanwhile, individuals with the susceptibility below a threshold $h_i < h_x$ would not develop symptoms by contracting the viruses; they will stay asymptotically infected and eventually will spontaneously recover after r_s days and removed from the process. In the model, r_s is a parameter equal to all asymptomatic agents. The infectious time of each host is counted from the moment the infection occurred. Both the asymptomatic and presymptomatic hosts can produce viruses throughout their infectious time, although in different amounts [6,7]. In the model, the amount of viruses is proportional to the host's susceptibility, which remains unchanged throughout its infectious time [1]. On the other hand, each virus node is characterised by a unique identity j , survival time T_v , and its generation g_j (the number of the hosts along the infection path) [1]. Besides, for this work, each Virus node has a new property, $O_h^{i \rightarrow j}$, which is given by the host's susceptibility that produced that virus. At present, we do not consider different variants of the virus; thus, a unique survival time T_v applies as a parameter. Here, we also do not consider potential mutations along the infection path. In Ref. [1], by keeping track of the number of hops of the virus (virus generation) since the original infection case, its potential mutations can eventually moderate the transmission rate. For the present work, we keep the mutation factor fixed, i.e., $g = 1$. Other values can also be analysed with the developed framework once reliable data on mutation patterns become available [28]. During the process, each virus's state changes from "active" when its host produces the virus to "expire" once its survival time exceeds the value T_v . Similarly, the state of each human actor changes from "susceptible" to "exposed (for active viruses)" to "active carrier" when infected. Further differentiation is caused by its susceptibility corresponds to a transition path in Fig. 1 towards either "spontaneously recovered" or "hospitalised or quarantined", see section III for simulation details.

As in the original model, in each event the basic transmission rate λ_0 is modulated by the individual susceptibility of the agent i encountering the virus at the moment t , see Eq. (2). In addition, the probability λ_t^i also varies in time due to the fluctuations in the global viral load $V(t)$. Specifically,

$$\lambda_t^i = \lambda_0(\Phi_t + 1)h_i g, \quad (2)$$

where the global feedback factor $\Phi_t \equiv dV(t)/dH_a(t)$ follows the temporal fluctuations of the viral load with respect to the current number of active carriers $H_a(t)$. Note that the upper limit of the virus production rate at time t corresponds to a hypothetical situation where each active carrier has the maximum susceptibility $h_i = 1$ producing a new virus at every time step. Hence, the temporal feedback in the fluctuating transmission rate of Eq. (2) accounts for the actual *heterogeneity* of the virus carriers. The potential origin of the hosts' heterogeneity mentioned in the Discussion can further modulate the transition rate.

3. Microscopic dynamics and sampled quantities

As shown in Fig. 1, the process is driven by the empirical time series $\{s_t\}$ representing the temporal fluctuations of the social activity level. Thus, this time series's resolution and length define the time step and duration of simulations, respectively. Moreover, its intensity determines the number of agents created during the simulations as $N_t = \sum_t s_t$. Specifically, at an *hourly resolution* it brings s_t new susceptible agents, which become exposed to active viruses. Note that the viruses can survive outside the hosts, such that the currently active viruses are those produced by all active carriers (asymptomatic as well as presymptomatic) within the past $T_v = 4$ hours, corresponding to the considered virus survival time. Each agent remains exposed for a period corresponding to its exposure time T_h^i , during which it can get infected with the probability given by Eq. (2). If infected, the agent is removed from the exposed agent's list and appears in one of the infected agent's groups, i.e., asymptomatic (if its susceptibility is below the threshold h_x), or presymptomatic, if $h_i \geq h_x$. During their respective infectious times, the agents in both groups produce new viruses with a pace that is modulated by the agent's susceptibility. After developing symptoms within an individual time interval of $r_h^i \in [2, 7]$ days, each symptomatic agent is hospitalised or quarantined and removed from the process. Whereas, each asymptomatic agent stays in the process until its spontaneous recovery after r_s days (equal to all agents). A detailed program flow is given in the Appendix.

As explained above, in the model, we keep information about the origin of each virus. Hence, in each new infection event, the number of infected n_{ta} increases by one if the virus originates from an asymptomatic host, and, in the case of a presymptomatic host, n_{ts} is increased. In each case the total number of infected agents per time step, $n_t = n_{ta} + n_{ts}$ increases, however, the relative proportions of n_{ta} and n_{ts} can vary, depending on several parameters, as we show in the following. As it is schematically indicated in Fig. 1, these two processes are strongly interlinked at the microscopic scale. Particularly, the infection *by presymptomatic* hosts can end up as an asymptomatic as well as a presymptomatic case, depending on the susceptibility of the new exposed agent. Similarly, an infection *by asymptomatic* hosts can result in either an asymptomatic or symptomatic case; but each symptomatic case may eventually end up in the intensive care unit with an uncertain outcome, depending on its susceptibility [13,50]. Given the difficulty in detecting asymptomatic virus carriers in real life, understanding the intrinsic mechanisms along this potential line of events is of great importance.

This microscopic modelling framework allows us to keep full control of the process, which results in different time-varying quantities, as shown in Fig. 3. Specifically, for the time period spanning eight weeks and resolution of 1 h, at each time step $i = 1, 2, \dots, t$ new agents are imported, and their individual properties h_i and T_h^i are fixed. Then at each time step, we determine the number of currently exposed agents e_t , the number of agents infected from viruses by asymptomatic hosts, n_{ta} , and by-presymptomatic hosts, n_{ts} . By respecting the individual hospitalisation time for each presymptomatic host and spontaneous recovery time for all asymptomatic ones, as well as the virus survival time, we compute the number of active carriers $H_a(t)$ and active viruses $V(t)$. Having these quantities at hand, we determine the actual transmission rate λ_t^i via eq. (2), at each infection event. Its temporal profile can express the impact of the basic transmission rate λ_0 , as shown in Fig. 3 bottom panel, as well as a potential self-tuning during the process, e.g., by altered social activity or virus mutations [1].

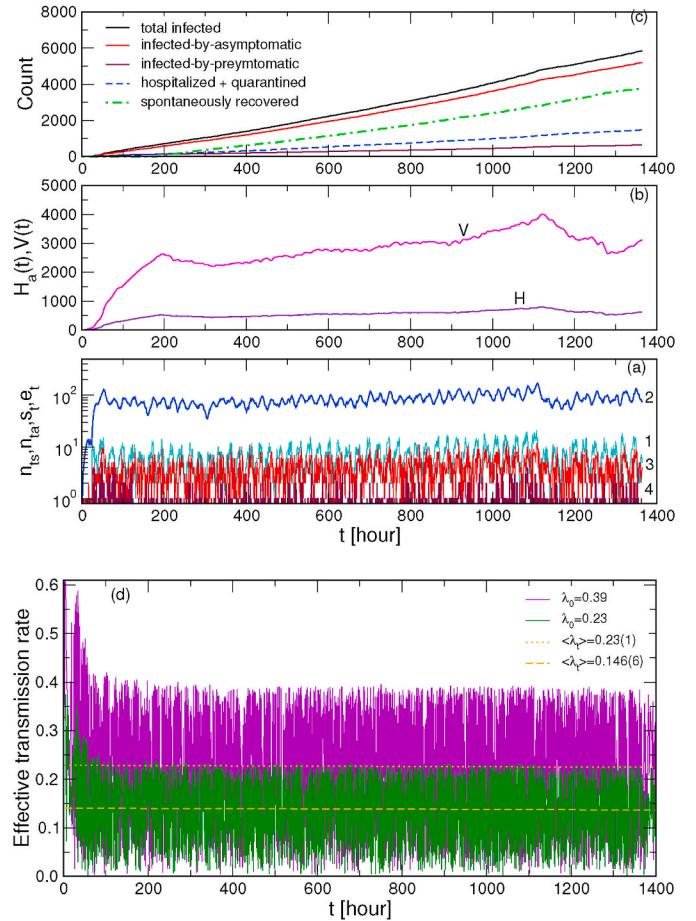


Fig. 3. Temporal fluctuations of the number of agents: (a) 1-socially active or susceptible s_t , 2-exposed agents e_t , 3-infected by asymptomatic n_{ta} and 4-by presymptomatic n_{ts} hosts. (b) H-The number of active carriers $H_a(t)$ and V-active viruses $V(t)$. (c) The corresponding total number of infected agents I_t (infectious curve), the proportions of infected by asymptomatic and by presymptomatic hosts, and the total number of spontaneously recovered, and hospitalised and quarantined over time. In the simulations, we fixed $T_v = 4$ h, maximum exposure time $T_h = 24$ h, the spontaneous recovery time $r_s = 7$ days, and the threshold value $h_x = 0.8$ and $\lambda_0 = 0.23$. (d) The actual values of the transmission rate λ_t^i occurring in the sequence of infection events within the corresponding time frame, for two values of the basic rate λ_0 indicated in the legend. The number of agents created is 9040; among them, 5823 became infected and produced 1307183 virus nodes during the simulation.

In the following, we simulate the infection process by varying the relevant parameters, i.e., the transmission rate λ_0 , the threshold susceptibility h_x , and the recovery time r_s of agents and the social activity level. These simulations enable us to assess their impact on the proportion of infections caused by asymptomatic and presymptomatic virus carriers.

4. Proportion of cases infected by asymptomatic carriers for varied parameters

As shown above, the cases *infected by asymptomatic* hosts can be differentiated at the microscopic dynamics scale from the cases *infected*

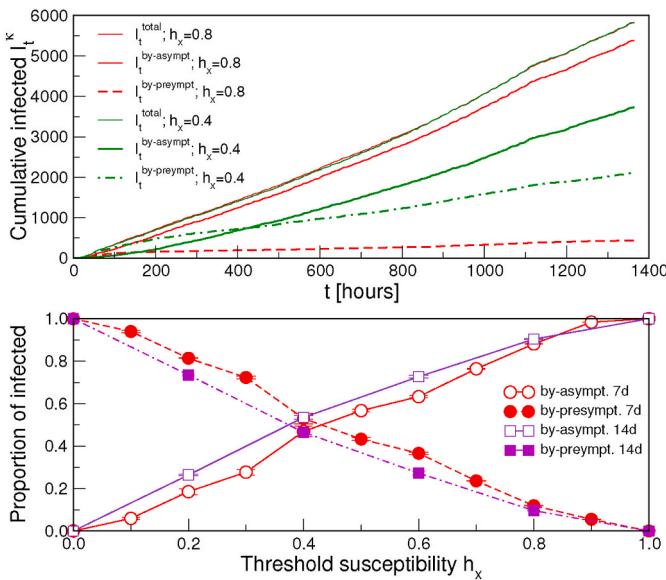


Fig. 4. Top panel: Infection curve I_t^{tot} versus time (thin full lines) and the respective proportions of the cumulative number of infected by asymptomatic hosts (thick full lines) and by presymptomatic hosts (broken lines) for the threshold susceptibility $h_x = 0.8$ and $h_x = 0.4$. The recovery time of asymptomatic cases is $r_s = 14$ days, other parameters are fixed ($\lambda_0 = 0.23$, $T_h = 24$ h, $T_v = 4$ h). Bottom panel: For varied threshold susceptibility h_x , the emergent proportions of infected by asymptomatic (open symbols) and by presymptomatic (filled symbols) plotted against h_x ; two sets of curves correspond to $r_s = 14$ and 7 days, as indicated in the legend.

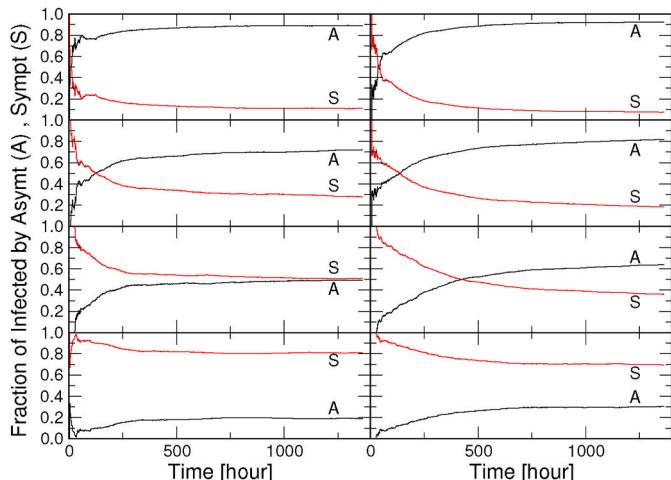


Fig. 5. Time evolution of the proportions of the infections by the asymptomatic (A) and presymptomatic (S) cases for varied threshold susceptibility $h_x = 0.8$, 0.6, 0.4, and 0.2 (top to bottom panels). The spontaneous recovery time of asymptomatic cases is $r_s = 7$ days (left column) and 14 days (right column).

by presymptomatic hosts. Consequently, their relative contributions to the growth of the infectious curve can be systematically estimated. Here, we focus on how these proportions vary in time (always starting from one symptomatic infected case), and how they depend on relevant parameters. Firstly, for a given social activity time series and fixed

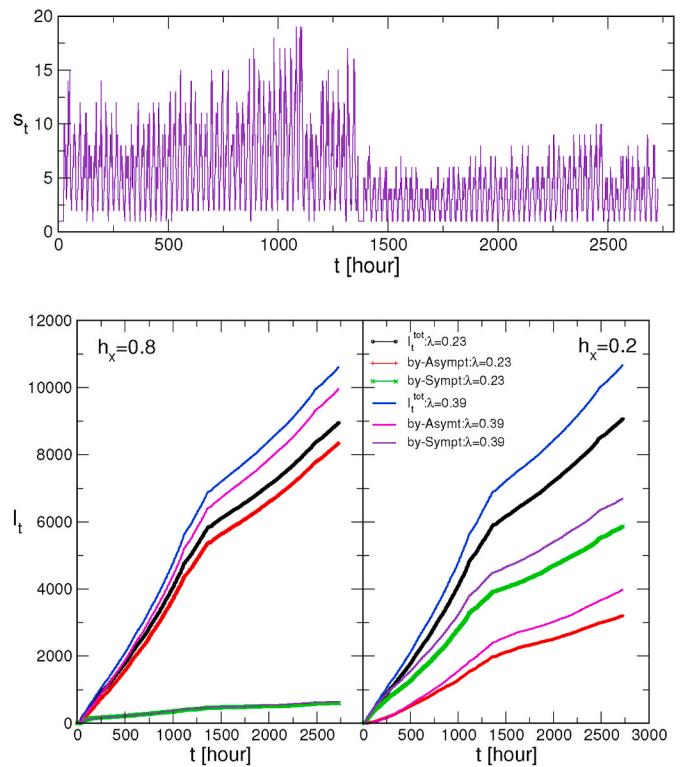


Fig. 6. Top panel: Varied-intensity social participation activity time series s_t . Bottom panels: For two threshold susceptibilities h_x , shown in each panel, the total infection curve I_t and the corresponding proportions of infected by asymptomatic and by presymptomatic carriers for two different values of the basic transmission rate $\lambda_0 = 0.23$ and 0.39 . The number of agents created during the simulations is 13905; the number of infected agents 10674 and viruses 802254 occurred for $h_x = 0.2$, meanwhile 10611 agents and 2679841 virus nodes appeared when $h_x = 0.8$.

maximum exposure times of agents $T_h = 24$ hours, we consider different values of the threshold susceptibility h_x and the recovery time of asymptomatic hosts r_s . Specifically, for the same driving time series s_t as in Fig. 3, we show in Fig. 4 that the relative proportions of the cases infected by asymptomatic and by presymptomatic hosts strongly depend on the recognised threshold susceptibility. Meanwhile, the total number of infected remains statistically similar, being chiefly conditioned by the social participation activity. For example, for the threshold $h_x = 0.8$, corresponding to 80% asymptomatic cases, the fraction of infected by asymptomatic carriers levels up at 94% of all cases, whereas the remaining 6% cases are infected by presymptomatic virus carriers. The corresponding temporal evolution of these fractions is shown in the top-right panel of Fig. 5, starting from a single symptomatic case. On the other hand, by assuming that asymptomatic infections comprise of 40% of all cases, i.e., the threshold susceptibility $h_x = 0.4$, the total number of infected by presymptomatic carriers is initially higher, but the events attributed to infections by asymptomatic carriers win at later times. Eventually, for even lower values of the threshold, the number of infected by asymptomatic hosts remain below the number of infected by presymptomatic. The exact proportions evolve over time, as shown in Fig. 5, depending not only on the threshold value but also on the infectious time r_s of the participants. The outcomes (averaged over the last 682 evolution steps) are shown in the bottom panel of Fig. 4 for the

whole range of threshold values and two infectious periods of the asymptomatic carriers, $r_s = 7$ and 14 days, respectively.

Next, we examine how these proportions depend on the social participation level and the basic transmission rate λ_0 . For this purpose, we extend the duration of the process. We use the same driving signal for the first eight weeks and then the signal with the reduced intensity but the same fractal structure for the following eight weeks, as shown in the top panel of Fig. 6. The simulation results for the infectious curves are shown in two bottom panels in Fig. 6. As this figure shows, the reduced social participation activity leads to the gradually slower growth of the total infection curve, in agreement with the findings in Ref. [1] supporting the idea of social lock-down measures. Here, we are interested in how these variations in the social activity level combined with the transmission rate can affect the relative proportions of the infected by asymptomatic and by presymptomatic carriers. Specifically, we consider two cases of the threshold susceptibility, $h_x = 0.8$ and $h_x = 0.2$, and two basic transition rates; the value $\lambda_0 = 0.23$, according to early estimates of the average rate [6,42], and 70% increased transmission rate $\lambda_0 = 0.39$, suggested by news regarding recent mutations; the results are displayed in Fig. 6. These results reveal that, for a low basic transmission rate, even though the social activity level strongly influences the total number of cases, the *relative proportions* of these cases infected by asymptomatic and by presymptomatic carriers remain virtually unaffected. However, the increased basic transmission rate increases both the total number of infected and alters the proportions of the infected by asymptomatic and by presymptomatic carriers. Moreover, these proportions are dramatically different when the susceptibility threshold is high, e.g., 0.8, compared to the case when it is as low as 0.2. Particularly, in the first case, a practically entire increase of the infection curve for the increased transmission rate can be attributed to the infections by asymptomatic carriers. On the other hand, the situation is not symmetrical when the number of asymptomatic comprises 20% of all infected. In this case, we find that the proportions of infected by presymptomatic carriers exceed the proportion of infected by asymptomatic by an amount, which increases with the increased basic transmission rate.

5. Discussion and conclusions

We have studied the microscopic dynamics modelling of SARS-CoV-2 epidemics by building on the modelling framework developed in Ref. [1]. By keeping information about the host that produces a virus implicated in an infection event, we have been able to disentangle the cases attributed to asymptomatic from those caused by presymptomatic virus carriers. With the extensive simulations that comprise up to 16 weeks of the evolution time with the hourly resolution, we have demonstrated how the corresponding proportions of the infection curve vary in time and depend on the implicated bio-social factors. Dealing with a highly nonlinear stochastic process, we note that changing a parameter that affects the events at the microscopic interaction scale may lead to an altered course of events and a different final outcome. At the same time, our results revealed certain regularities regarding the groups of social and biological factors. Specifically:

- The overall infection level critically depends on social participation activity. Hence, the increase of the infection curve can be forcefully controlled, e.g., by temporally reducing the social activity level, having the other factors fixed;
- For a given total infection, the relative ratio of the cases infected by asymptomatic carriers to the cases infected by presymptomatic carriers crucially depends on several biological factors. These are, the

threshold susceptibility (depending on the virus pathogenicity and human genetic and other health factors of the implicated actors), and the virus transmissivity;

- The interplay between social and biological factors can be altered, increasing the proportion of cases attributed to the asymptomatic carriers, when the virus transmissivity considerably increases. For example, the considered situation where the basic transmission rate is increased by 70% is motivated by recently debated potential mutations of SARS-CoV-2, see Refs. [27,28].

In this modelling framework, we consider high-resolution temporal fluctuations of a community's cumulative social participation but without a specified spatial dimension. For different challenges of modelling COVID-19 epidemics, see the agent-based [42–47] and compartmental dynamics models [51,52]. Our model focuses on the individual features of agents and viruses that impact the infection process at the elementary interaction scale, while accounting for the global-scale outcomes, and in this sense, it is unique in the current literature. The model can be adequately extended to consider the virus mutations during the entire process [1]. Such mutations can lead to markedly altered pathogenicity or transmissivity, resulting in the simultaneous presence of different strains. At the agents' level, a time-dependent infectiousness profile during the agent's infectious time can be included. Moreover, individual or group's attitudes towards respecting non-medical prevention measures can be implemented at different levels, i.e., by modifying the basic transmission rate or affecting each infection event.

In conclusion, our microscopic dynamics modelling of the SARS-CoV-2 epidemics reveals the interplay between different biological and social factors of this nonlinear process, which shapes the increase of the infectious curve and the proportions attributed to asymptomatic and presymptomatic virus carriers. Given social participation activity under control, our results shed light on the intrinsic action mechanisms of the key biological factors. In particular, these are the critical threshold susceptibility to the virus and the increased virus transmissivity, which lead to the increased proportions of the infections by asymptomatic carriers. Thus, our theoretical study suggests that the role of asymptomatic and presymptomatic carriers in the SARS-CoV-2 epidemics can be revealed by assessing these biological factors. Apart from the health-related and genetic features of a given population, these factors depend on the predominant virus type and mutations. At a community level, assuming that pertinent empirical data on the virus transmissivity and pathogenicity can be available, these findings should assist in better estimates of the impact of the hidden asymptomatic carriers, and consequently, in the design of the appropriately improved measures.

Declaration of competing interest

Authors declare no conflict of interest.

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Appendix. Program flow

Algorithm 1 Latent Infection Transmissions:
Asymptomatic & Presymptomatic carriers

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1: INPUT:  $s_t$  time series and parameters  $T_v, T_h, \lambda_0, h_x, r_s$ ;
2: Define object arrays (nodes)  $H\{i; h_i, T_h^i, r_h^i, state\}$ ,
 $V\{j; T_v; g_j, O_h^{i \rightarrow j}, state\}$ ,  $Edge(id, src, dst, time)$ ;
3: Start empty lists  $H_{exposed}$ ,  $H_{active}$ ,  $V_{active}$ ; add first
infected  $H$  node to  $H_{active}$  list; set its  $t^1 = 0$ ,  $h^1 = 1$ ;
4: Start input time series  $s_t$  (defines time  $t$ ); reset counters;
5: while ( $cin >> t >> s_t$ ) do
6:   in each time step  $t$ :
7:     for all nodes on  $H_{active}$  list do
8:       with prob.  $\propto$  the node's susceptibility  $h^i$  create a
new  $V$  node; its time is  $t$ , its  $O_h^{i \rightarrow j}$  inferred from the
creator  $H$  susceptibility, its state is "active"; put it
to  $V_{active}$  list; create an Edge from  $H \rightarrow V$ , specify
its time as  $t$ ;
9:     end for
10:    Update the transmission probability  $\lambda_t^i$ ;
11:    for all  $1 \leq i \leq s_t$  do
12:      Create a new  $H$  node and set its state as "susceptible",
susceptibility  $h^i$  as  $rand \in [0, 1]$ , exposure time
 $T_h^i$  as  $rand \in [1, T_h]$ . Add it to  $H_{exposed}$  list;
13:    end for
14:    for all nodes on  $H_{exposed}$  list do
15:      with prob.  $\lambda_t^i$  connect the  $H$  node to a random
 $V$  node on  $V_{active}$  list; change its state to "infected
by asymptomatic", if the connected  $V$  node carries
 $O_h^{i \rightarrow j} < h_x$ , else as "infected by presymptomatic";
add to the  $H_{active}$  list; create the Edge from  $V \rightarrow H$ ;
mark its time as  $t$ ;
16:    end for
17:    Revise the lists  $V_{active}$ ,  $H_{exposed}$  and  $H_{active}$  considering
 $\Delta t$  between the current time  $t$  and the node's
time:
18:    for all nodes in  $V_{active}$  list do
19:      if  $\Delta t > T_v$  then
20:        (virus survival time exceeded): remove from the
list;
21:      end if
22:    end for
23:    for all nodes in  $H_{exposed}$  list do
24:      if the status changed to "infected (by...)" then
25:        (infected): remove from the list;
26:      else
27:        if  $\Delta t > T_h^i$  then
28:          (its exposure time exceeded): remove from the
list;
29:        end if
30:      end if
31:    end for
32:    for all nodes in  $H_{active}$  list do
33:      if  $\Delta t > r_s$  days then
34:        (recovered): remove from the active carriers list;
35:      else
36:        if the node's  $h^i > h_x$  then
37:          take  $r_h^i$  as  $rand \in [2, 7]$  days;
38:          if  $\Delta t > r_h^i$  then
39:            (hospitalized, quarantined): remove from the
list;
40:          else
41:            keep on  $H_{active}$  list;
42:          end if
43:        end if
44:      end if
45:    end for
46:    Sampling temporal quantities of interest;
47:  end while
48: Sampling network and statistical quantities of interest;
49: END
```

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