Why Lockdowns Are Futile and Face Masks Effective in Slowing Down COVID-19-Like Pandemic — Quantitative Analysis with Compartmental Model

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Received: 23.02.2022 & Accepted: 29.04.2022

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There exist a number of complex and often nonlinear phenomena in physical and biophysical systems that can be efficiently approached by systems of differential equations. Even though the deterministic character of the solutions is typically not adequate to describe the actual behaviour of given systems, this approach is very well suited to describe the influence of the well-defined factors on the evolution of the systems described by properly chosen dynamical models. For example, a compartmental model with three groups of people (*susceptible, infected*, and *recovered*) is able to capture some of the general principles related to the dynamics of a pandemic in a biophysical system such as the human population. Here, motivated by the ongoing COVID-19 pandemic, with the help of a proper generalisation of the simple model, we analyse influence and efficacy of commonly invoked counter-pandemic actions — lockdowns and mandatory face masks — in reducing the number of fatalities. To reach this goal, our model takes into account the number of hospitalised persons and the fraction of those hospitalised who need special treatment in intensive care units. We show that even if there is an optimal time for introduced lockdowns to be effective, it is impossible to reach in practice due to the limited capacity of the health system. The calculations indicate that wearing face masks decreases the number of hospitalised people and the total death toll. Half of the population appropriately wearing masks, even the home-made ones (with an efficacy of only about 60%), would halve the peak value of those needing intensive medical treatment. Our study indicates a slightly greater effectiveness of masks worn by healthy people, which is related to the fact that ill people do not protect themselves.

topics: COVID-19, lockdown, face masks, SIR-like model

1. Introduction

The dynamical systems, in which different classical or quantum states change in time, are usually formulated in terms of differential equations, sometimes called maps [1]. The examples range from classical and quantum physics, through chemistry, to biophysical and biological problems. In the latter context, the different states are called compartments. The system's evolution is usually described by non-linear equations and needs not to be deterministic. Here we propose to use a compartmental model to study the dynamics of the COVID-19 pandemic. Our main aim is to quantitatively analyse the role of face masks and lockdowns in combating the pandemic.

The surprisingly rapid development of the COVID-19 pandemic caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection at the beginning of 2020 has spread fears about and, in some regions, caused [2] the breakdown of the medical systems. Under these circumstances, the governments of many countries have adopted different measures and policies of

non-medical character to slow down the spread of pandemic [3] — with only some being scientifically motivated [4–6]. As the main route of spread of the pandemic has been found to be via air [7–10], the main measures included: quarantining people with fever symptoms, wearing face masks in public places[†1] [11], restricting direct social contacts, and lockdowns or even curfews in some countries [12]. Many of these actions, e.g. lockdowns, albeit, as discussed later, are effective in temporal slowing down the epidemic, have a negative influence on the economy and cause significant social costs. However, despite general recommendations [13, 14], the non-medical measures are often contested [15]. For instance, the direct observation performed by one of us some time ago in Warsaw, capital city of Poland, found that many individuals were hesitant to wear masks[†2], with some of them being not convinced about their usefulness.

Obviously, vaccinations against the SARS-CoV-2 virus constitute the main tool to combat the COVID-19 pandemic. However, the vaccine hesitancy observed in many countries [16–18] provides a barrier to herd immunity. Also, the waning immunity after both natural infection and vaccination [19], as well as novel pathogen mutations[†3] [20] make uncertain the future development of the COVID-19 pandemic. In such a situation, non-medical measures will remain an important tool in the fight against COVID-19.

To reach our goal, we have modified the standard model of epidemic dynamics [21], commonly known as the SIR model — short for the names of the main compartments: susceptible (S), infected (I), and recovered (R). The modifications take into account the hospitalised persons (H) and those who need intensive treatment (T) in our baseline model (see Sect. 2). In the next step, we additionally divide the population into masked (M) and unmasked (U) (see Sect. 5). The novel compartments allow quantitative analyses of the role of face masks in preventing the collapse of the national health system with a limited number of beds and ventilators in intensive care units.

The SIR model [22–24] and its modifications [25–30] have often been used to quantitatively analyse the dynamics of the ongoing pandemic. The accuracy of the outcome depends on the question asked [31, 32] and the availability of the proper input parameters. It is generally believed that a simple SIR model is unable to quantitatively capture the dynamics of the pandemic [26]. However, it is a very good model to study the changes in the dynamics due to some external actions affecting its parameters [29], as is the case in the present work.

We note that other possible states in the pandemic, like exposed, asymptomatic, subclinical, etc., and complex social (e.g. age structure) and cultural or political aspects would make the model more realistic. For instance, as exposed in the Hong Kong example, despite nearly universal mask-wearing, an outbreak of the pandemic has been observed, as many infections took place in households [33]. We refrain in the present paper from taking these important factors into account.

As a first problem, we study the optimal time to introduce the lockdown. We quantify it by the threshold fraction of individuals (T_{thr}) needing intensive medical treatment. Interestingly, we found a first-order phase transition of total mortality for the critical value of T_{thr}, beyond which the final death toll is the same independently of the duration of lockdowns. In sum, our analysis suggests that lockdowns can be effective only in the short run and that any too late or too early lockdown is counterproductive, especially in light of severe economic consequences. Regarding face masks, we have studied how their usage and effectiveness correlate with the fraction of the population needing hospitalization or treatment in intensive care units (ICU). In accordance with the early review [34], we conclude that even simple cloth masks are likely life-saving measures in a resource-limited environment. Namely, we found that half of the population appropriately wearing masks, even the home-made ones (with an efficacy of only about 60%), would halve the peak value of those needing intensive medical treatment. Interestingly, our study indicates a slightly greater effectiveness of masks worn by healthy people, which is related to the fact that ill people do not protect themselves.

Some aspects related to face masks and their effect on the pandemic have been studied earlier [28, 35–37]. They include social, economic and health issues related to diminishing the infection rate and changes in the epidemic dynamics. The agent-based model to examine the effectiveness of wearing masks has been developed [36], while the dynamics of the COVID-19 pandemic of individual agents were simulated by the generalised SIR-like model. The outcome of the studies is that social distancing alone is not effective in slowing down

[†1] All information regarding the actual situation in Poland can be found on the governmental pages. The actual measures are described in [11].

[†2] The direct observations performed by one of us (H.K.) in various randomly chosen public places in Warsaw (capital city of Poland) between 1 December 2020 and 31 January 2021 on the probe of total 6583 individuals found that 4230 of them wore masks, while the rest i.e. 2353 did not. This means that roughly 35.7% of population is not convinced about masks efficacy. In smaller cities this percentage may be even larger. It has to be stressed that Poland does not belong to the countries where wearing masks is an accepted cultural norm.

[†3] The early studies of the omicron variant are not convergent [20] with some of them (mainly South African) indicating that it is less severe but others (UK, Denmark) suggest that this variant leads to similar as delta hospitalisation rates of infected, recovered and vaccinated. However, the proportion of positively tested and hospitalised children seem to be larger then earlier, which might be due to the fact that many have not yet been vaccinated.

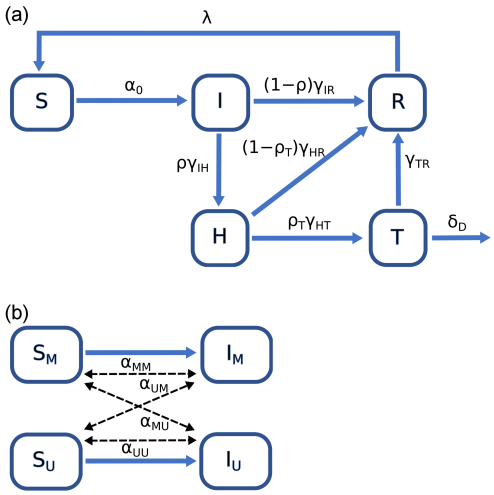

Fig. 1. (a) Schematic representation of the standard SIR model enriched by the H and T compartments denoting infected individuals who are hospitalised H and the part of those hospitalised who need medical treatment T in ICU. (b) The modification of the first two compartments of our model if the population is divided into those masked (M) and unmasked (U). We also show here various infection rates. Note that thick solid arrows indicate the flow of individuals between compartments, while dashed arrows indicate all possible contacts between susceptible and infected.

the disease. However, in conjunction with a high degree of compliance in the use of masks, regardless of whether the wearer displays symptoms, slows the spread of infection. Interestingly, the study finds [38] that used face masks are unsuitable to replace nasopharyngeal swabs in COVID-19 diagnosis.

Previous studies on the efficacy of wearing face masks mainly concentrated on their protective role for an individual person. The present work, on the contrary, focuses on the entire population, using the estimated effectiveness of face masks in individuals to learn about the pandemic evolution in the whole population. Our study adds significant quantitative arguments in favour of obligatory face masks in public spaces as an important preventive measure. It shows a slowdown in the spread of COVID-19-like disease and a reduction in the number of people requiring intensive medical treatment and also in the total death toll, in the community with a high fraction of the population wearing properly fit, albeit not necessarily the most effective masks.

The rest of the paper is organised as follows. In the next section (Sect. 2), we present our baseline model with six compartments. In Sect. 3, we discuss the values of parameters used in the actual calculations. The dynamics of the COVID-19-like epidemic, which follows from the baseline model, are briefly presented in Sect. 4, with particular emphasis on the effectiveness of lockdowns depending on their effectiveness and duration. Section 5 introduces face coverage into the model. The effect of the efficacy of face masks and the fraction of the population wearing them on the number of hospitalised and mortality is studied in Sect. 6. The discussion in the context of dental practices is the subject of Appendix A. We end up with the concluding Sect. 7. The paper is supplemented by a simple discussion of the reproduction number, which is presented in Appendix B.3.

2. The baseline model

Our main goal in this work is to generalize the standard SIR model of epidemic [21] in two directions. First, we take into account the number of hospitalised (H) persons and the number of those patients who need intensive care treatment (T) and evaluate the effect of lockdown very often administered by governments. The second direction is related to the effect of usage of the face masks on the dynamics of the epidemic. In this section, we first formulate the model with additional compartments but without the effect of face masks. After the precise formulation of the baseline model, we discuss its general properties. In particular, we pay special attention to the effect of the lockdown on shortening the pandemic.

We denote by $S(t)$ all individuals who at time t are susceptible and may contract a disease through contact with a member of an already infected group $I(t)$. To track those who need intensive care treatment, we allow for the compartment of hospitalised $H(t)$ and those who need special treatment, like ventilation. The latter compartment is called $T(t)$ and represents a number of individuals who at time t occupy beds in intensive care units (ICUs). Some of the individuals from this group, which we denote by $D(t)$, will die (at rate δ_D), and some will recover with the γ_{TR} rate. The group of recovered is denoted by $R(t)$. The interrelation between the considered compartments is illustrated on the graph shown in Fig. 1a. We assume that not all infected need hospitalisation. Some of those infected are asymptomatic (and, at the same time, very difficult to identify), and some of those identified will recover after a few days of stay at home. Indeed, the results of paper [39] suggest that the number of detected people with infection may be one to two orders of magnitude lower than the actual number of infected. This uncertainty is the main reason why we do not introduce the compartments of asymptomatic or pre-symptomatic infected individuals [28, 29]. We denote by ρ the fraction of those infected who need hospitalisation. Among hospitalised, there is a ρ_T fraction needing intensive care treatment, and they enter the compartment T at the γ_{HT} rate. Some of the individuals from this compartment will recover, and others will unfortunately die. In principle, the rates of these two events may differ depending on age and comorbidity, but

we will not tweak the model any further and will keep it as simple as possible. Thus we do not introduce other compartments. We do not consider the asymptomatic or pre-asymptomatic compartments, as it is difficult to reliably estimate the precise number of patients belonging to those classes [40], as well as determine their true transmission capability. The careful reader may wonder why we are using the multiplicative parameters here, as it is the product that counts in the calculation. There are two reasons. Firstly, we do not want to increase the number of compartments, e.g. those who will recover without hospitalisation after becoming ill and those who will recover without intensive treatment only after hospitalisation. Secondly, such notation provides a clear physical interpretation.

We underline that extracting the compartment $T(t)$ out of $H(t)$ is of critical importance in the process of decision-making in order to slow down the pandemic development. This is because the number of accessible beds in ICUs is the parameter limiting the maximum health service efficiency. In the case of the COVID-19 pandemic, it is related to the number of respirators V, which cannot be easily expanded. In contrast, the number of beds in hospitals is virtually limited by the country's health/economy capacity. In principle, field hospitals can be opened in large objects like stadiums if the economy allows it.

In the following, we normalise the number of individuals in every compartment by the total population N but use the same upper case symbols to denote the (normalised) compartments. The model does not take into account newborns and those whose death is not related to the considered epidemic, so the population is constant ($N = \text{const}$). Thus after the normalisation the conservation of population is given by $S(t) + I(t) + R(t) + H(t) + T(t) + D(t) = 1$.

The equations describing the dynamics of the continuous version of the model read

$$\frac{\mathrm{d}S(t)}{\mathrm{d}t} = -\alpha_0 S(t) I(t) + \lambda R(t), \tag{1}$$

$$\frac{\mathrm{d}I(t)}{\mathrm{d}t} = \alpha_0 S(t) I(t) - (1-\rho)\gamma_{IR} I(t) - \rho\gamma_{IH} I(t), \tag{2}$$

$$\frac{\mathrm{d}R(t)}{\mathrm{d}t} = (1-\rho)\gamma_{IR} I(t) + (1-\rho_T)\gamma_{HR} H(t) + \gamma_{TR} T(t) - \lambda R(t), \tag{3}$$

$$\frac{\mathrm{d}H(t)}{\mathrm{d}t} = \rho\gamma_{IH} I(t) - (1-\rho_T)\gamma_{HR} H(t) - \rho_T \gamma_{HT} H(t), \tag{4}$$

$$\frac{\mathrm{d}T(t)}{\mathrm{d}t} = \rho_T \gamma_{HT} H(t) - \gamma_{TR} T(t) - \delta_D T(t), \tag{5}$$

$$\frac{\mathrm{d}D(t)}{\mathrm{d}t} = \delta_D T(t). \tag{6}$$

The most important assumption our model shares with the standard one is homogeneous mixing, which means that all uninfected people face the same risk of contacting those already infected. Generally, this might not be true, as the chance of getting infected while working at home is smaller than in a crowded place. We relax another important assumption of the original SIR model, which is related to the total immunity of persons who enter the R compartment. In the above model, the parameter λ is responsible for possible waning immunity after disease or vaccination. This is an important parameter, as the extent of protection against asymptomatic infection and the duration of vaccine-induced humoral and cellular immunity [41] are indeed still unknown [42]. One possible scenario is that COVID-19 may become an endemic disease [41].

The symbols γ_{IH} and γ_{HT} denote, respectively, the rate at which the group of infected persons is being hospitalised and the rate at which those who need special treatment enter the T compartment. Assuming that each person in the ICU needs a respirator for, say, τ_V days, one notes that, on average, the number of individuals in the T compartment should not exceed V/τ_V, where V is the number of available respirators. If it does, the medical system will have too low a capacity to treat all those who need ventilation[†4] [43], and this will result in additional fatalities. The parameter λ is related to the period of full immunity after the COVID-19 symptoms or after the vaccination. The ongoing observations report [44] that only 8.4% of all COVID-19 cases occurred in fully vaccinated individuals, and relatively few of these patients required hospitalization. The infections in July 2021 in England were driven mainly by the Delta variant and mostly concerned younger unvaccinated individuals [45]. The same is true as far as the Omicron variant is concerned.

3. Model parameters

The estimation of the parameters entering the model requires significant inputs from clinical observations of the disease and statistical, geographic, demographic and other data. For the presented calculations, we assume values close to those used in related previous studies [28, 29, 46]. As noted earlier, our main aim is to study the effect of the usage of face masks and some general trends in the dynamics of pandemic under this non-pharmaceutical personal prevention measure. Thus we shall use a simple set of parameters — for most purposes,

[†4]For comparison at the beginning of COVID-19 epidemic in Poland there were in total 27 respirators per 10000 people. According to the official information the number of respirators available in 2021 exclusively for COVID-19 patients is about 8 per 100000 people [43].

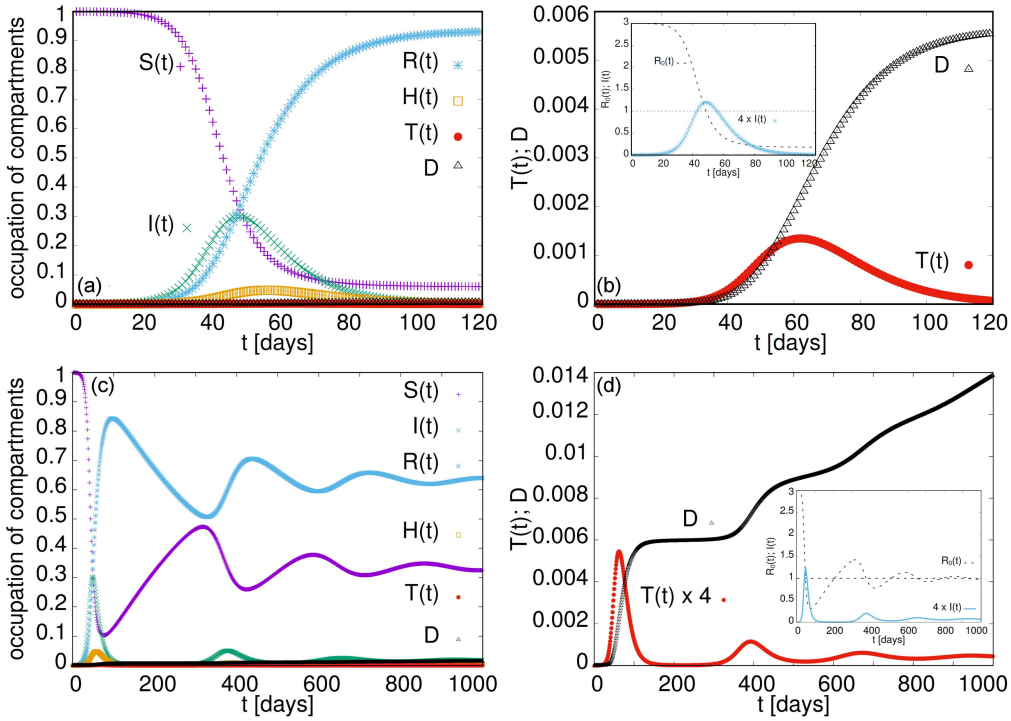

Fig. 2. The dynamics of the pandemic over 120 days (panels (a) and (b)) from its beginning with the initial value of infected $I(0) = 5000/N_{PL}$. We use $\alpha_0 = 0.3$, and all recovery and death rates equal 0.1 and $\lambda = 0$. Panels (c) and (d) show the evolution of the epidemic over 1000 days for $\lambda = (1/365)$ day^{-1} corresponding to the average duration of immunity of about one year. After about 3 years, the epidemic becomes an endemic disease with a roughly constant number of people in each compartment and a linear in time total death toll.

we take $\alpha_0 = 0.3$, and all rates related to the recovery or death are assumed to be the same and equal 0.1. Throughout the paper, all parameters describing the dynamics of the pandemic are expressed in [days]$^{-1}$. So, for example, the recovery rate 0.1 above means that, on average, the illness lasts 10 days. The rate $\gamma = 0.1$/day translates to the time to recovery $= 1/\gamma = 10$ days. To take into account the fact that some infected are asymptomatic and others recover after a few days of stay at home, we have introduced a parameter ρ, which denotes the fraction of infected who needs hospitalisation and ρ_T is a fraction of hospitalised needing intensive treatment. The clinical observations indicate [30] that $\rho \approx 0.2$ and $\rho_T \approx 0.06$. As a side remark — these rates are dimensionless numbers.

Now, with the vaccination available in many countries for all willing to accept it[†5] [16], the important question is how will the epidemic evolve with waning immunity. In order not to increase the number of parameters and compartments, we have not explicitly introduced the group of vaccinated people. The existing preliminary studies [47] seem to indicate that the period of full immunity after infection lasts around 9 months. There exist very preliminary estimates of vaccine efficacy [48], which is still under debate [49]. Most practitioners [50] expect the need to repeat the vaccination after some time. It is a matter of future studies and observations to find out if booster vaccination will make immunity persistent or if COVID-19 will become an endemic disease. Due to these uncertainties, we keep the parameter λ as a free one and consider scenarios with various values of λ^{-1}. We consider $\lambda^{-1} = 365$ days as a representative value for waning immunity after the infection/vaccination.

[†5]Contrary to the early days of vaccination campaign, the vaccines are easily accessible now in Poland. The actual problem being that a number of people remain hesitant to vaccination. This observation agrees with preliminary studies reported recently [16]. Around 44% in France and 66% respondents in Italy say they would accept a COVID-19 vaccine.

4. Simple predictions of the baseline model

From (2) and (1), assuming $\lambda = 0$ and denoting $\bar{\gamma} = (1-\rho)\gamma_{IR} + \rho\gamma_{IH}$, one finds the relation

$$\frac{\mathrm{d}I}{\mathrm{d}S} = -1 + \frac{\bar{\gamma}}{\alpha_0 S}, \tag{7}$$

which after integration leads to

$$I(t) + S(t) - \frac{\bar{\gamma}}{\alpha_0}\ln(S(t)) = I(0) + S(0) - \frac{\bar{\gamma}}{\alpha_0}\ln(S(0)) = \text{const}, \tag{8}$$

the relation, which our baseline model shares with the original SIR model [21]. The parameter $\frac{\alpha_0}{\bar{\gamma}}$ coincides with the basic reproduction number $\mathcal{R}_0$ (see Appendixes). The other important parameter related to the vaccinations is the so called herd immunity threshold; for simple SIR-like models it is defined as $1 - \frac{1}{\mathcal{R}_0}$. For our standard set of parameters $\mathcal{R}_0 = \alpha_0/\bar{\gamma} \approx 3$, one finds the herd immunity at the level $\approx 67\%$, which is still well above the vaccine acceptance ratio in many countries. It has to be added that the herd immunity of the heterogeneous model [51] is expected to be lower and of about 43%. However, new virus mutations and vaccine efficacy may lead to higher values of herd immunity, and this lower number may not be correct.

To get more feeling about the dynamics predicted by the baseline model, we have assumed the following (standard) set of parameters: $\gamma_{IR} = \gamma_{IH} = \gamma_{HR} = \gamma_{TR} = \gamma_{HT} = 0.1$, $\rho = 0.2$, $\rho_T = 0.06$, and initial values $I(0)$=5000/N_{PL}, $S(0) = 1 - I(0)$. The value 5000 corresponds to the number of infected at the beginning of September 2021 in Poland.

In Fig. 2a and b, we show the short time behaviour of the pandemic for $\lambda = 0$ and in Fig. 2c and d, the long time behaviour assuming $\lambda = 1/365$ day^{-1}.

Figure 2a shows the relative occupation of each compartment, and $\lambda = 0$ means that after recovery the person is immune to the disease. With the assumed parameters, the maximal number of infected appears after about 62 days from the outbreak of epidemic and reaches 30% of the population, with about 5% needing hospitalisation and 0.15% requiring intensive medical treatment. Probably no country in the world could survive such an epidemic, as these are huge ratios, which, e.g., for Poland (with the total population $N = N_{PL}$ with $N_{PL} = 38 \times 10^6$) translate into the following numbers of about 11.4 million infected, 1.9 million hospitalised, and 0.57 million in ICUs. Figure 2b shows the number of patients needing intensive medical treatment and the total number of deaths. Note that the mortality reaches nearly 0.6% of the population. In the inset of Fig. 2b we show the time dependence of the basic reproduction number. It diminishes from the initial value of 3, while the number of infected grows until R_0 takes on a value equal to 1. Only after that point $I(t)$ starts to decrease in agreement with the general discussion in Appendixes. The number of infected rises exponentially over the first couple of weeks after the outbreak of the pandemic, reaches the maximum, and goes down, albeit at a slower rate, until it essentially dies out after more than 120 days of pandemic duration. There is a well visible delay between maximums of $I(t)$, $H(t)$ and $T(t)$ with the shift of about 10 days ($\approx 1/\bar{\gamma}$).

Figure 2c and d show the results for the epidemic with waning immunity assuming $\lambda = \frac{1}{365}$ day^{-1}. This corresponds to the average duration of immunity of about one year. The evolution of the epidemic over the first 1000 days is shown. Without any interventions, the pandemic shows damped oscillatory behaviour and reaches the essentially steady state in all compartments, except that related to $D(t)$, which grows roughly linearly with time. This means that some people will always die after contracting the disease. This is easy to understand from (6), which for truly constant $T(t) = T_0 = \text{const} \neq 0$ gives $D(t) = \delta_D T_0 t$ — the function linearly growing with time. As seen in Fig. 2, the disease enters an endemic state (see Appendixes) about three years after the outbreak of the first wave. Unfortunately, the death toll over this period of time reaches more than 1.4% of the population and continues to grow with time. No government can accept such a scenario, and that is the main reason for introducing different measures to slow down the pandemic, make the health system sustainable and decrease mortality. Let us remark that in order to plot occupations of different compartments on the same plot, we often need some of them to multiply or divide by a numerical factor, as indicated.

There are two important principles that policy-makers all around the world take into account when handling and trying to slow down the COVID-19 pandemic. The first one is that the number of people needing intensive medical care (compartment $T(t)$) at a given instance of time cannot exceed the available number of beds or respirators in ICUs, which determines the health system's capacity to avoid triage. The second principle is to minimize the number of deaths. Even with vaccination but without herd immunity or with waning immunity, the most popular actions undertaken by policy-makers are temporal lockdowns and other restrictions.

To study the effectiveness of lockdowns quantitatively, we introduce novel parameters in the model. The decision-makers typically observe the number of hospitalised persons and/or the number of those who need intensive care treatment. If the corresponding parameter, say $T(t)$, exceeds the threshold value T_{thr}, the lockdown is introduced, and this stops the pandemic by rapidly diminishing the infection rate α_0 to a new value $\alpha_L = \alpha_0/w$, where w is the effectiveness of the lockdown, duration of which is denoted by τ. The interesting questions are: when the lockdown has to be introduced, and for how long in order to be most effective?

To study this, we take our standard set of parameters and introduce — like in [29] — the lockdown at time t_L when the ratio of people needing intensive treatment exceeds the assumed threshold value $T(t_L) \geq T_{\text{thr}}$. If the lockdown's effectiveness is w and duration is τ, this means that the infection ratio changes with time as

$$\alpha(t) = \begin{cases} \alpha_0, & t < t_L, \\ \alpha_0/w, & t_L \leq t \leq t_L + \tau, \\ \alpha_0, & t > t_L + \tau. \end{cases} \tag{9}$$

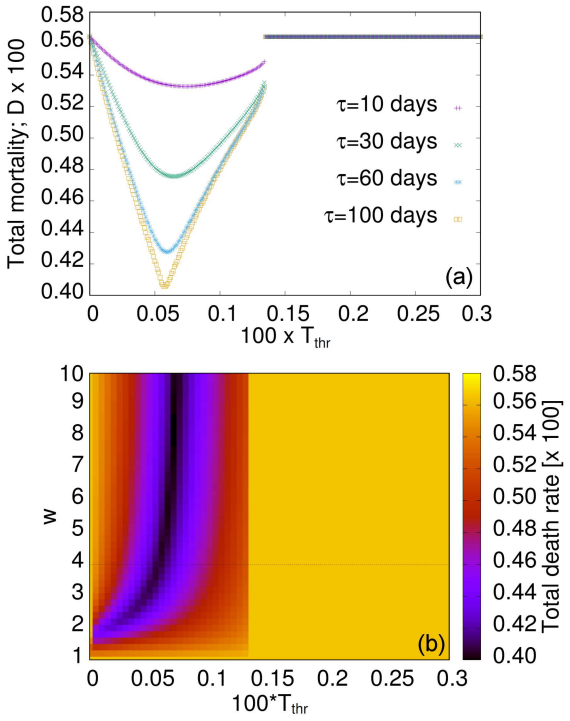

Fig. 3. (a) The effect of lockdowns (measured by total mortality D of varying length τ characterised by $w = 4$ vs the threshold value $T_{\rm thr}$ of its introduction. Panel (b) shows the phase portrait on the plain $(T_{\rm thr}, w)$. Here the total death rate is plotted as a function of the threshold number of patients needing intensive care when the lockdown is introduced $T_{\rm thr}$ and the effectiveness of lockdown w.

In Fig. 3a we show the total mortality at the end of epidemic vs the threshold $T_{\rm thr}$ for $w = 4$ and a few values of the lockdowns' duration τ. We use our standard set of parameters with 5000 people infected at the start of the epidemic simulation. There are two features worth underlying. First is the existence of a clear minimum of D, position of which slightly depends on the lockdown's duration. For $\tau = 100$ days, the minimum appears for $T_{\rm thr} \approx 0.00057$ and it moves to $T_{\rm thr} = 0.00064$ for $\tau = 30$ days. The increase of the lockdown's duration decreases total mortality, unless the lockdown is introduced too late (here for $T_{\rm thr} \geq 0.0013$). For $\tau = 100$ days, the minimal value of $D \approx 0.00405$, while for $\tau = 60$ days, $D_{\rm min} \approx 0.00475$. The second feature is the first-order phase transition of $D(T_{\rm thr})$ observed for the critical value of the threshold parameter, which for a given set of parameters appears for $T_{\rm thr}^{cr} \approx 0.00134677$. As seen in Fig. 3, this value does not depend on the duration of the lockdown (Fig. 3a) or its effectiveness w (Fig. 3b) but strongly depends on the infection rate α_0 (not shown). Interestingly, beyond this critical value of the threshold, the final outcome of the pandemic is the same independently of the duration of lockdowns. This means that any late lockdown is counterproductive as it leads to the same number of fatalities. Similarly, earlier lockdowns are also not very effective. We shall discuss this in the following.

Figure 3b shows the phase diagram on the plane $(T_{\rm thr}, w)$ for the same set of epidemic parameters as in Fig. 3a and duration $\tau = 100$ days of the lockdown. For each w, there exists an optimal threshold value when the final D is the lowest. With increasing w, it slowly moves to higher values of threshold. The dashed line at $w = 4$ corresponds to w assumed in Fig. 3a. For $T_{\rm thr} > T_{\rm thr}^{cr}$, the death toll does not depend either on $T_{\rm thr}$ or w and approaches its maximal value of $D \approx 0.00564$. It means that nearly 0.6% of the population dies if no action is taken. The existence of the optimal threshold seems to be good news for policy-makers. The trouble is to guess the optimal value in practice, when the modelling may not be accurate, e.g. due to a lack of appropriate values of parameters. Another problem is related to the capacity of the medical system. The total number of beds in hospitals in Poland equals 213 000, those available to COVID-19 patients nearly 30 000 and the number of respirators for COVID-19 about 3000 [11]. This means that the realistic value of the threshold parameter for Poland, as measured by the plain number of respirators, is $T_{\rm thr} \approx 7.9 \times 10^{-5}$ — nearly an order of magnitude below the number $\approx 6 \times 10^{-4}$ for the introduction of an optimal lockdown, as can be seen in Fig. 3a. This shows that lockdowns are effective only as short term means to slow down the epidemic.

To get more detailed information on these issues, let us assume for a while that the parameters above describe a real COVID-19-like pandemic with the initial value of basic reproduction number of $R_0 = 3$. This is a realistic value for the second wave of COVID-19 [11]. To judge the feasibility of lockdowns and the dynamics of the epidemic under such conditions, we use the same set of parameters and calculate the time evolution of the epidemic for $w = 4$ and a few values of $T_{\rm thr}$.

Figure 4a illustrates the time dependence of the T compartment for a few values of $T_{\rm thr}$ and shows the mortality $D(t)$. The duration of the lockdown is 100 days, and its effectiveness is $w = 4$. In a scenario without a lockdown, the model predicts a single wave with relatively large peak values of individuals needing intensive treatment (≈ 0.00135) and a large death toll reaching $\approx 0.56\%$ of the population. Introducing the lockdown when $T_{\rm thr} = 0.0006$, i.e., it is close to the optimal value, diminishes the peak in $T(t)$ and results in diminishing of D by about one third. Interestingly, the lockdowns introduced at earlier stages of epidemic with $T_{\rm thr} = 0.0004$, 0.0003, 0.0002 or $T_{\rm thr} = 0.0001$ result in second, third, etc., waves. The final number of fatalities is smaller than without any action but slightly larger than or approaching that obtained for optimal $T_{\rm thr}$. However, as discussed earlier, the maximal values of $T(t)$ exceed by orders of magnitude the capacity of the health system. Importantly, after the

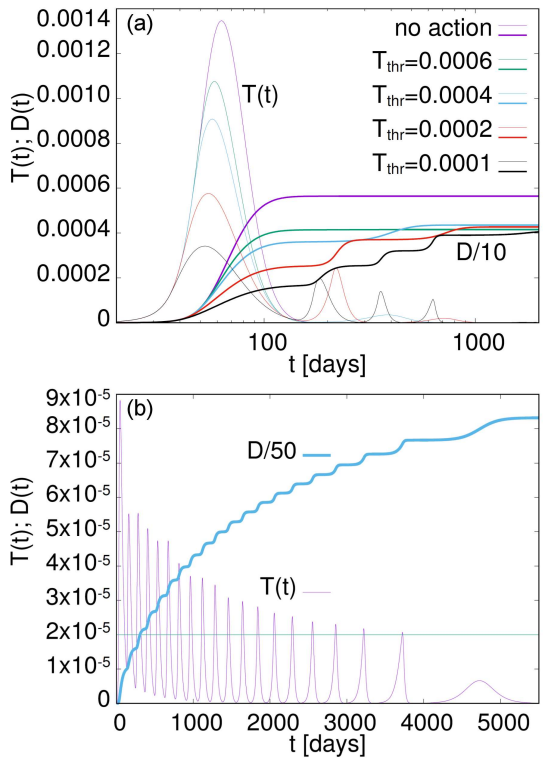

Fig. 4. (a) The compartment $T(t)$ (thin lines) and the mortality $D(t)/10$ (thick lines of the same colour) are plotted as functions of time from the start of an epidemic for a number of threshold values of $T_{\rm thr}$ when the 100 days long lockdown period of effectiveness w was introduced. (b) The example of the same dependence as in (a) for the realistic value of the threshold chosen in such a way that the maximum in $T(t)$ during the first epidemic wave does not exceed the assumed capacity of the system. The total mortality of this 15 years long epidemic is about 26% lower than without any action and, moreover, the health system is properly functioning all the time.

lockdown is introduced, the number of people needing treatment still grows for some period of time and attains a value of more than four times the assumed lockdown threshold. This is related to the number of infected $I(t_l)$ at the time of lockdown start and the delay in the appearance of peaks in other compartments, as discussed earlier. That is why the lockdown has to be introduced well before the health system's capacity is reached.

In Fig. 4b we show the results for the threshold $T_{\rm thr} = 0.00002$, which is well below the capacity of the health system in Poland, and observe the dynamics of the epidemic over a very long period of time. It is assumed that 100 days long lockdowns with effectiveness $w = 4$ are introduced each time the number of individuals needing intensive medical care reaches the threshold value. As can be seen, this will result in the appearance of many epidemic waves with lower peaks of $T(t)$ in consecutive waves, until the epidemic finally dies out after 20 waves or over about 16 years. The final outcome of this scenario in terms of total number of fatalities is desirable from a social point of view but is very costly economically. The result is only valid for the assumed full immunity of recovered individuals, i.e., for $\lambda = 0$.

Needless to say, quantitatively similar data are obtained by using different indicators for the introduction of lockdown. The above results are in accord with previous studies, in which authors coupled the pandemic with the economy [29]. We have assumed the homogeneous mixing model. As argued earlier, the heterogeneity of the population significantly affects the outcomes of the model [51]. The authors of that paper analysed the effects of heterogeneity and underlined that preventive measures may not be effective if they are too strong.

The lockdowns are damaging to the economy, education and social contacts. There exist, however, less demanding and not so costly ways to slow down the epidemic and decrease mortality. One of them is the obligatory usage of face masks in public spaces by as large as possible part of the population. One can imagine that usage of face masks will decrease the effective value of infection rate α_0. Instead of arbitrarily rescaling α_0 to take into account such restrictions, in the next section we extend the above baseline model by explicitly including the fraction of people wearing masks. The strength of such a strategy in building the model of pandemic dynamics is that the effectiveness of masks is a rigid and known parameter. Also, the statistics of people wearing masks can be obtained via direct measurements. This, in turn, leads to a quantitative evaluation of the masks' effectiveness and the fraction of the population wearing them in the pandemic dynamics.

5. The generalised model with use of face masks

To take the effect of face masks into account, in this section we divide each of the previously mentioned compartments into two. One contains those individuals who wear and the second those who do not wear masks. We denote them as, respectively, S_M, I_M and S_U, I_U, etc. We also introduce four different parameters α_{ij}, with $i, j = M, U$ denoting the corresponding infection rates. Thus if both of the encountering individuals wear masks, the infection rate is denoted by α_{MM}. If only the individual from compartment S_M (I_M) wears a mask, the corresponding rate is denoted by α_{MU} ($\alpha_{\rm UM}$). The last entry of the matrix α_{ij}, namely α_{UU}, denotes the rate of disease transfer when both individuals do not wear masks. By definition, this rate of infection equals the previously used α_0, so $\alpha_{UU} = \alpha_0$. Figure 1b shows the schematic division of the S and I compartments and the corresponding model parameters.

In the literature [28], the infection matrix α_{ij} is sometimes expressed in terms of the bare infection rate α_0, the masks' effectiveness ϵ_i denoting the so-called inward effectiveness describing the protection of the susceptible (but not infected) person against catching the disease, and ϵ_o — the outward effectiveness. In the last case, the mask protects (others) against the transmission of the disease (i.e., it is worn by the infected individual). The relation between the two sets of parameters is given by $\alpha_{UU} = \alpha_0$, $\alpha_{MM} = \alpha_0(1-\epsilon_i)(1-\epsilon_o)$, $\alpha_{MU} = \alpha_0(1-\epsilon_i)$ and finally $\alpha_{UM} = \alpha_0(1-\epsilon_o)$. In general, ϵ_i and ϵ_o need not take on the same value.

Many other factors, in addition to wearing masks, decide about the spread of the disease and affect the α_0 rate. These other factors, like lockdowns discussed earlier, will not be directly taken into account in this section. We want to analyse the effects of masks on slowing the epidemic down and delaying the possible collapse of the health system. We keep track of the M and U groups in all compartments, as it is very likely that persons wearing masks before infection will do the same after recovery and, appropriately, at the rate λ will move back to the S_M compartment. It is understood that in the following equations, each compartment is divided into M and U parts, so, e.g., $R = R_M + R_U$, etc., even though we use a compact notation whenever possible. The masks affect the infection rates only, as explained above. All other rates, like the recovery rates γ_{IR}^M and γ_{IR}^U for the infected population from I_M, I_U to R_M, R_U compartments, may also slightly depend on whether the person wears a mask or not, as the study on the animal model suggests [52]. However, we assume these rates to be independent of the M and U. In this section we also assume $\lambda = 0$.

With the new notation, our compact equations describing the dynamics of the model with masks read

$$\frac{\mathrm{d}S_M(t)}{\mathrm{d}t} = -\alpha_{MM}S_M(t)I_M(t) - \alpha_{MU}S_M(t)I_U(t) + \lambda R_M(t), \tag{10}$$

$$\frac{\mathrm{d}S_U(t)}{\mathrm{d}t} = -\alpha_{UM}S_U(t)I_M(t) - \alpha_{UU}S_U(t)I_U(t) + \lambda R_U(t), \tag{11}$$

$$\frac{\mathrm{d}I_M(t)}{\mathrm{d}t} = \alpha_{MM}S_M(t)I_M(t) + \alpha_{MU}S_M(t)I_U(t) - (1-\rho)\gamma_{IR}I_M(t) - \rho\gamma_{IH}I_M(t), \tag{12}$$

$$\frac{\mathrm{d}I_U(t)}{\mathrm{d}t} = \alpha_{UM}S_U(t)I_M(t) + \alpha_{UU}S_U(t)I_U(t) - \rho\gamma_{IH}I_U(t), \tag{13}$$

$$\frac{\mathrm{d}R_i(t)}{\mathrm{d}t} = (1-\rho)\gamma_{IR}I_i(t) + (1-\rho_T)\gamma_{HR}H_i(t) + \gamma_{TR}T_i(t) - \lambda R_i(t), \tag{14}$$

$$\frac{\mathrm{d}H_i(t)}{\mathrm{d}t} = \rho\gamma_{IH}I_i(t) - (1-\rho_T)\gamma_{HR}H_i(t) - \rho_T\gamma_{HT}H_i(t), \tag{15}$$

$$\frac{\mathrm{d}T_i(t)}{\mathrm{d}t} = \rho_T\gamma_{HT}H_i(t) - \gamma_{TR}T_i(t) - \delta_D T_i(t), \tag{16}$$

$$\frac{\mathrm{d}D_i(t)}{\mathrm{d}t} = \delta_D T_i(t). \tag{17}$$

The subscript i in (14)–(17) takes on two values, $i \in \{M, U\}$, and serves as a bookkeeping parameter marking individuals who originally belonged to M or U compartments.

6. Results: full model with masks

We shall now concentrate on the role of face masks. To get quantitative results, one needs to know what fraction of the population wears masks and what is the inward and outward effectiveness of masks. There exist partial studies related to the masks' effectiveness [53, 54]. The inward effectiveness varies from more than a few % up to about 75% for homemade cotton masks and from above 70% up to above 95% for surgical and N95-like masks. The outward effectiveness of all types of masks was essentially lower [28]. Albeit, as discussed in Sect. 1, there are some controversies about masks use and their role in flattening the pandemic curves, most researchers and practitioners seem to agree that statistically, the usage of face masks is an important non-pharmaceutical prevention instrument. Moreover, the obligatory wearing of face masks in public places is arguably the simplest preventive measure to implement. Also, as our studies indicate, it is an efficient policy, provided the masks are properly used. The existing studies related to dental practices, where the most popular personal protection equipment (PPE) is simply a face mask worn by dentists, discussed in Appendix A, seem to additionally support this point of view.

In the following, we shall study the role of this PPE on the dynamic of pandemics. As both the fraction x of population (properly) wearing the masks and the masks' effectiveness are accessible, but in practice not well known model parameters, we study the dynamics of the epidemic and the total death rate as functions of x, ε_i and ε_o. Before showing the numerical outcomes of the model, let us note that the relative ratio of people in, e.g., S_M and S_U compartments is not constant and changes as the epidemic evolves. The point is that individuals from both these compartments move at various rates to other compartments. As found in Appendixes, the ratio S_M/S_U changes and its time dependence is governed by the inward effectiveness of masks

$$\frac{S_M(t)}{S_M(0)} = \left(\frac{S_U(t)}{S_U(0)}\right)^{1-\epsilon_i}. \tag{18}$$

This result is valid for a model with $\lambda = 0$ and arbitrary values of other parameters. No similarly general analytic dependence can be found for other compartments. An important remark is here in order. While the ratio of masked to unmasked in S compartments does depend on time, as explained above, the sum of masked from all compartments is constant. We have $S_M + I_M + R_M + H_M + T_M + D_M = x$ at all times. The same is obviously true for the fraction of the unmasked population which equals $(1 - x)$.

In Fig. 5a we show the dependence of the maximum value of ill people needing intensive therapy $T_{\max} = \max_t T(t)$ on the effectiveness of masks $\epsilon_i = \epsilon_o = \epsilon$. Figure 5b shows a similar dependence of the total death rate D towards the end of the analysed outbreak. Various curves correspond to varying fraction x of people wearing face masks. In these calculations we used our standard set of parameters with $\lambda = 0$ and the initial values $I_U(0) = (1 - x)\,Inf$, $I_M(0) = x\,Inf$, where Inf is the initial relative number of infected individuals $Inf = 5000/N_{Pl}$. The remaining fraction of population is at the beginning of epidemic in the susceptible S_{ini} compartment with x denoting the fraction of those wearing masks since the first day, i.e., $S_M(0) = x\,S_{ini}$ and $S_U(0) = (1 - x)\,S_{ini}$.

Note that wearing the face masks has generally a very positive effect by diminishing the peak value $T_{\max}$ of those needing intensive treatment. It may even contribute to sustainability of the health system if the fraction x is large enough. In the case of Poland the threshold value measured by the number of available respirators in ICUs is in Fig. 5a marked by the thick solid curve. Our model predicts that 90% of the population wearing the standard cotton masks with effectiveness around 0.6 or higher would contribute to diminishing $T_{\max}$ below the threshold value and thus prevent the breakdown of the health system. Having said this, we want to stress that the model is rather simple and does not take many effects into account, like the possibility

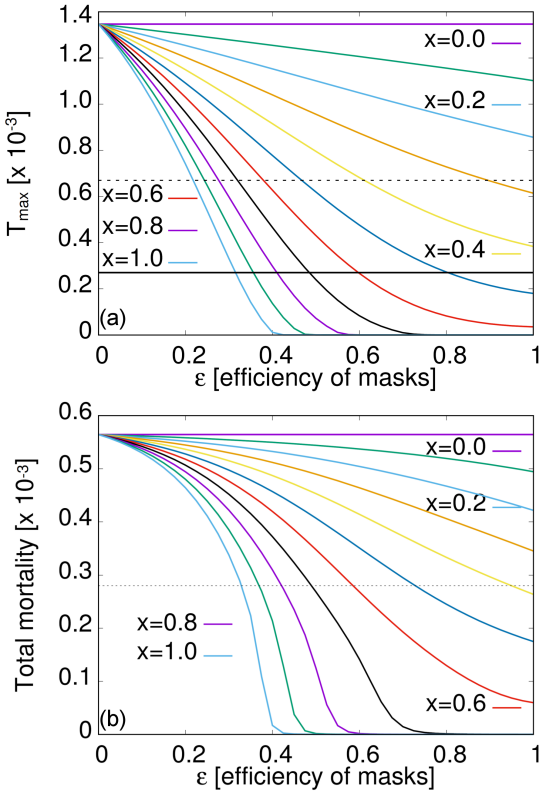

Fig. 5. The peak value of the number of persons who need intensive treatment while hospitalised (a) and total mortality rate vs masks' effectiveness $\varepsilon_i = \varepsilon_o = \varepsilon$ (b) for different fractions x of the population wearing masks. The lower solid line in panel (a) at 0.00027 signals the breakdown of the Polish health system as $T_{\max}(t)$ the relative number in ICU exceeds the capacity of the system measured in terms of available respirators. We used the parameters $\alpha_0 = 0.3$, all recovery and death rates equal 0.1, $\lambda = 0$, and the initial number of infected $Inf = 5000/N_{PL}$, where N_{PL} equals the population of Poland. In both panels, dashed lines denote half of the corresponding maximal value.

of infections at home, when nobody wears masks and the presence of one asymptomatically ill family member may contribute to larger than expected disease spreading.

Figure 6 shows similar data as in Fig. 5, but now vs fraction x of susceptible population wearing masks. Comparing both Figs. 5 and 6 one notes a similar positive effect of x and ϵ on the maximal value of those who need intensive therapy $T_{\max}$. Slightly bigger differences are observed in panels showing total death rate. In both Figs. 5 and 6 we have assumed the inward and outward effectiveness to be the same. One can note that $x > 0.7$ of population wearing the masks (with effectiveness of about 60%) would decrease $T_{\max}$ below its value 2.7×10^{-4} which was a threshold value of Polish health care system [46] at the beginning of 2020. The basic reproduction number decreases below the

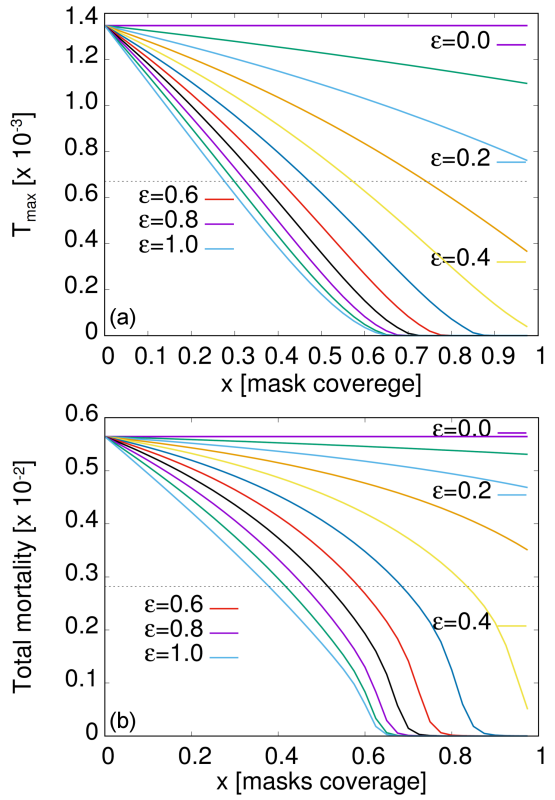

Fig. 6. (a) The maximal number of persons who need intensive treatment while hospitalised vs the fraction x of susceptible individuals wearing the masks. (b) The total mortality vs fraction of masked individuals. We used the parameters $\alpha_0 = 0.3$, all recovery and death rates equal 0.1 and $\lambda = 0$.

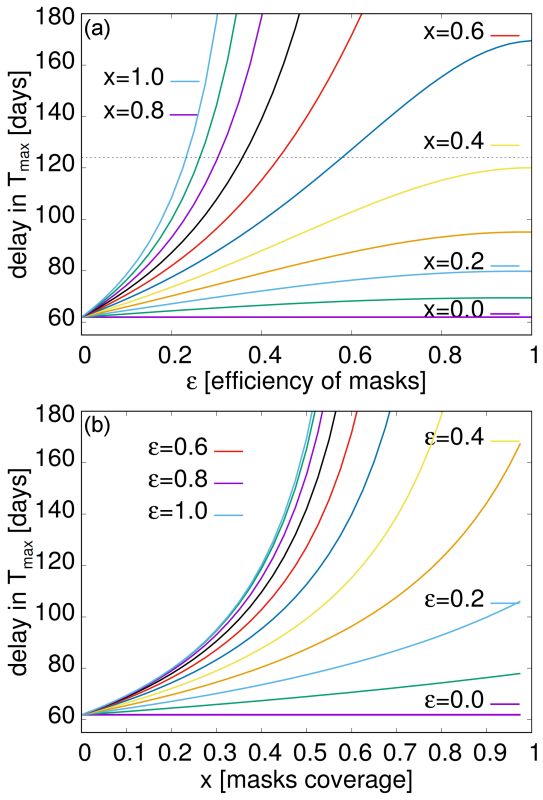

Fig. 7. (a) The time elapses from the beginning of pandemic outbreak when $T(t)$ takes on maximal value vs effectiveness of masks for various fraction of the population wearing the masks. The dashed line denotes the doubling of the period when the maximum occurs in comparison to no masks situation. Panel (b) shows the x dependence of the same delay time for a number of effectiveness $\epsilon = \epsilon_i = \epsilon_o$.

threshold value 1 for about 80% of population wearing masks with effectiveness 0.7 as follows from the expression (B37).

Another important social factor is the delay of the peak in the number of individuals requiring special treatment. For the assumed values of parameters the peak in $T(t)$ appears after about 62 days from the beginning of the epidemic if no action is undertaken. Assuming that wearing face masks is the only preventive action one finds that depending on the masks' effectiveness (assuming $\epsilon_i = \epsilon_o$) that this time may increase up to 150 days if half of the population would wear masks with effectiveness of about 0.7 as shown in Fig. 7a. For this set of parameters roughly the same delay may be obtained if about 60% of the population wear the face masks with effectiveness of about 0.5 (Fig. 7b). Comparison of two panels in Fig. 7 allows to conclude that dependence of the delay in $T_{\max}$ on fraction (x) of population wearing the masks for various masks' effectiveness is qualitatively very similar to the dependence on the masks' effectiveness for different x.

With two groups of susceptible and infected people wearing the masks, it is interesting to know more details related to the effect of inward vs outward effectiveness on the pandemic. Who should wear masks: infected, susceptible or both? The simple and certainly correct answer is both. However, it is important to know if healthy, uninfected people wearing masks contribute to the slow-down of the pandemic as much as those infected? Would it be enough for only infected people to wear the masks, and in this way protect others? To answer such questions, we calculate the ratio of patients requiring intensive treatment by paying attention to who wears the masks. In Fig. 8, we show the dependence of $T_{\max}$ on ϵ_i calculated for $\epsilon_o = 0$ (solid lines) and on ϵ_o for $\epsilon_i = 0$ (symbols). To gain an even better grip of the issue we calculate $T_{\max}$ for $x = 0.2$ (magenta), $x = 0.5$ (green) and $x = 0.8$ (blue).

Behaviour that is interesting and, at a first glance, unexpected is shown in Fig. 8a. It is a weaker dependence of $T_{\max}$ on ϵ_o in comparison to ϵ_i. In other words, the efficacy of masks worn by healthy (susceptible) individuals is larger than the corresponding efficacy of masks worn by infected (i.e., ill) individuals despite the same effectiveness ϵ. The absolute value of the effect decreases with increasing the fraction x of the masked population. Similar

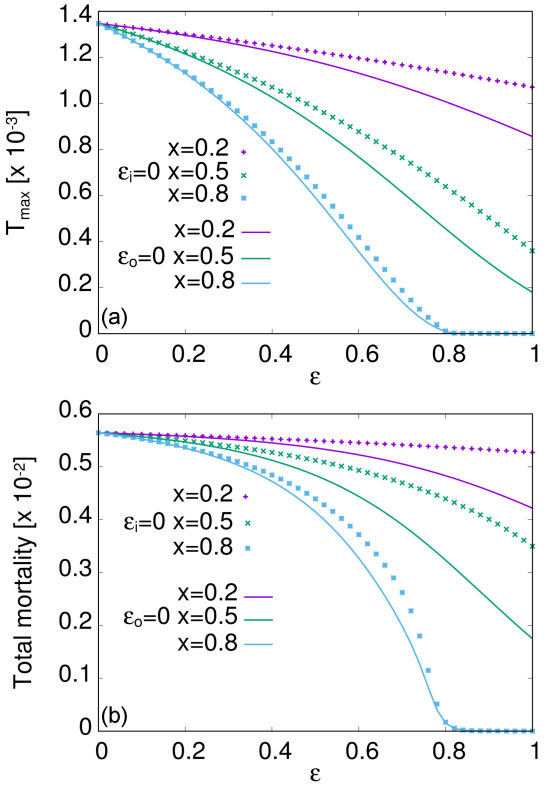

Fig. 8. (a) The maximal value of $T(t)$ vs effectiveness of masks for $x = 0.2$ (magenta, pluses), 0.5 (green, crosses) and 0.8 (blue, stars). The continuous line show the dependence of $T_{\max}$ on ϵ_i obtained for $\epsilon_o = 0$, while lines with symbols show the dependence on ϵ_o for $\epsilon_i = 0$. (b) Similar dependence of the fatalities rate on $\epsilon_{o(i)}$.

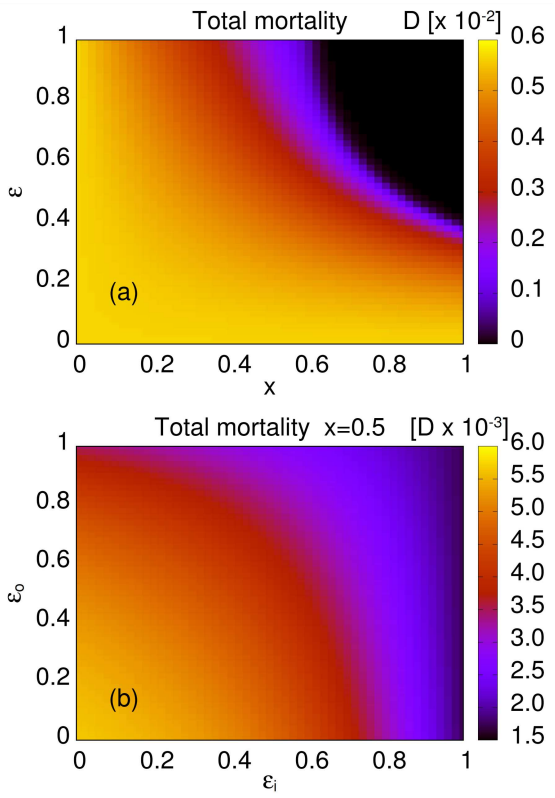

Fig. 9. The total mortality D vs masks' coverage x and their effectiveness ($\epsilon = \epsilon_i = \epsilon_o$) is shown in panel (a). Panel (b) illustrates dependence of mortality on inward ϵ_i and outward ϵ_o effectiveness of masks for $x = 0.5$.

behaviour is observed in the dependence of total death rate shown in Fig. 8b. The detailed dependence is slightly different, with slightly weaker changes of total death rate $D_{\max}$ at low masks' effectiveness and its faster decrease at higher effectiveness and larger masks' coverage. To gain quantitative information, we define the following (x and ϵ dependent) ratios

$$R_T = \frac{T_1 - T_2}{T_1 + T_2}, \tag{19}$$

$$R_D = \frac{D_1 - D_2}{D_1 + D_2}, \tag{20}$$

where $T_2 = T_{\max}(\epsilon_i, \epsilon_o{=}0)$, $T_1 = T_{\max}(\epsilon_i{=}0, \epsilon_o)$, $D_2 = D_{\max}(\epsilon_i, \epsilon_o{=}0)$, $D_1 = D_{\max}(\epsilon_i{=}0, \epsilon_o)$. These ratios may be used to quantify the efficacy of inward or outward protecting masks. Assuming the respective effectiveness $\epsilon = 0.7$, we find $R_T \approx 4.2\%$ for $x = 0.2$, $R_T \approx 10.9\%$ for $x = 0.5$, and $R_T \approx 15.2\%$ for $x = 0.8$. The ratio R_D takes on the following approximate values 3.4%, 9.21% and 13.7% for $x = 0.2,\ 0.5$ and 0.8, respectively. The above results indicate the very important role of masks worn by healthy people in a susceptible group. The asymmetry and the greater importance of masks worn by susceptible is related to the fact that masks worn by infected do not prevent them from the illness, as they are already ill.

From this we conclude that for the population as a whole it is very important that as large fraction of the population as possible wears masks. Figure 8 clearly shows that even with vanishing outward masks' effectiveness, the increase of inward effectiveness strongly reduces $T_{\max}$. We thus propose another piece of advice to the general public (especially to those whose are healthy and argue that they do not infect others): *wear masks primarily to protect yourself and by this contribute to stopping the spread of the disease.*

In view of the preceding discussion, it is important to look at the phase portraits. With three, essentially free, parameters: masks' coverage x, inward ϵ_i and outward ϵ_o masks' effectiveness, we propose to look at the total mortality $D_{\max}$ dependence on the (x, ϵ) plane with $\epsilon_i = \epsilon_o = \epsilon$, and at the same characteristics on the plane (ϵ_i, ϵ_o) assuming $x = 0.5$. The masks' coverage of $x = 0.5$ is chosen for illustration purposes. However, a qualitatively similar behaviour is obtained for other values of x.

In the colour-coded Fig. 9, the mortality rate is shown depending on the masks' coverage x and their effectiveness, assuming $\epsilon_i = \epsilon_o$ (panel a). There is

a slight asymmetry between the masks' coverage and the effectiveness. The more significant asymmetry is visible in Fig. 9b. As discussed in connection with Fig. 8 we observe that, regardless of the masks' effectiveness, masks worn by healthy (susceptible) individuals have a greater effect in diminishing total mortality in comparison to masks worn by infected individuals. This is because, in the latter case, the masks do not protect the wearers who remain ill until recovery. Figure 9 summarises the results of using face masks and shows that the masks' coverage is as important, or even slightly more important, than their effectiveness. Both infected and susceptible should wear masks, albeit asymmetry in the efficacy of outward and inward protecting masks of the same effectiveness does exist.

7. Conclusions

We have formulated a compartmental model of the spread of the disease and adapted it to study some aspects of the dynamics of the COVID-19-like epidemic. The compartments have been chosen in such a way to make them useful for our goal. The resulting model is non-linear and features some interesting behaviour, like the scaling law (see (25)) and phase transition (seen in Fig. 3 and discussed in Sect. 4). These features of the model, albeit interesting in themselves, are outside the scope of the study in this paper.

We have applied the generalised compartmental model to study the effects of lockdowns and one of the PPE (namely the usage of face masks) on the dynamics of COVID-19-like epidemic. Analysis of the baseline model without masking resulted in the observation that lockdowns are very important tools to slow down the evolution of the epidemic, but have a rather weak effect on total mortality. Their primary role is to make the health systems sustainable. Meanwhile, wearing face masks in public places is effective in protecting susceptible individuals. We have been mainly interested in the question of whether the masks could contribute to the sustainability of the health care system given its limited resources. The general conclusion is very encouraging, namely, a large enough fraction of the population wearing even home-made cloth masks may flatten the curve and delay the appearance of the maximum of those requiring treatment in ICU. Moreover, this PPE significantly contributes to diminishing the total mortality rate.

Taking into account the hesitation about the vaccines observed across Europe, our study shows that considering the simple prevention mechanism is an important way of slowing down the pandemic and saving a large number of people. This is true despite the ongoing vaccination campaign. The identified novel mutations of SARS-CoV-2 viruses in conjunction with vaccine hesitancy are responsible for the outbreaks of new epidemic waves. The face masks thus remain even for vaccinated individuals, an important PPE against the disease.

Appendix A: Discussion — lessons from dental practices

Among different medical professions not directly involved in the COVID-19 treatment, dentists arguably belong to the group most exposed to the SARS-CoV2 virus [55]. This is related to the fact that part of the treatment is using dental bur or scaler, which creates aerosols. The airborne spread of viruses is one of their most important transmission routes [7, 8]. It is followed by direct contact with infected but asymptomatic patients. However, the airborne, contact and contaminated surface spread, all are of great importance in dental practice due to the specificity of the dental procedures, i.e., close contact with the patient, the droplets produced during the treatment and unavoidable contact with saliva droplets or even blood that may contain high concentrations of viruses [56].

The French [57] and USA [58] studies indicate that the number of infected dentists is below the average, which is rather surprising in view of their much higher than average exposure. Taking into account that the main protective equipment of dentists are face masks, this seems to additionally support our results. The studies related to the USA [58] concluded that "This indicates that the current infection control recommendations may be sufficient to prevent infection in dental settings". A similar conclusion has been reached in the French [57] case — the authors of this paper showed that "oral health-care professionals were surprisingly not at higher risk of COVID-19 than the general population". The early studies among the dentists in Poland [59] seem to indicate that around 0.5% of them contracted the disease. This number is higher than the average for the Polish population at the same time, which was around 0.2%. The said report describes the number of infected dentists as marginal, probably taking into account a much higher risk of this profession. This seems to indicate that security-wise the standard practices adopted in dental clinics provide good enough protection measures.

However, in view of high exposure to viral transmission, some researchers call for a new protocol to protect dental healthcare workers and students [60]. The encouraging fact is that the knowledge related to COVID-19 disease is good among dental professionals [61]. On the other hand, the hesitancy about vaccination is relatively large among dental students. As a recent study reveals [62], about 45% of dental students are hesitant to receive the vaccine. The same study shows that 23% of medical students hesitate to vaccinate. Very similar numbers were reported in another study [63]. Both studies seem to indicate the necessity of educational curricula development in dental studies in order to increase the knowledge about vaccines, their safety and effectiveness.

Importantly, early work [64] on the impact of periodontal disease on hospitalisation and mortality during the COVID-19 pandemic did not find compelling evidence to link these two facts. The connection between COVID-19 and periodontal disease has been analysed in [65]. The authors propose a mechanism that may be responsible for the similarities between COVID-19-induced cytokine storm and cytokine expression profile involved in periodontitis, and argue that oral hygiene and periodontal treatment are mandatory in the COVID era.

Appendix B: Analytical results

B.1. Numerical solution of the system of differential equations

To solve the differential system of equations representing both the baseline and the model with masks taken into account, we applied the Runge–Kutta method [66] of the fourth-order.

In the studies of epidemiological models, it is important to predict the immediate future of the epidemic based on its current state. Such a role plays a basic reproduction number representing an average number of infections generated at time t by each infected person. Its calculation is presented in the forthcoming subsection. Another important notion is the disease-free equilibrium (DFE) state. For the model written schematically as

$$\frac{\mathrm{d}\boldsymbol{y}}{\mathrm{d}t} = \boldsymbol{f}(\boldsymbol{y}), \tag{21}$$

with boldface characters denoting vectors, this state is defined by $\boldsymbol{f}(\boldsymbol{y^*}) = 0$. Our model contains three compartments of infected (I, H, T) and exhibits DFE for $\boldsymbol{y^*} \equiv \{S^*, I^*, R^*, H^*, T^*\}^{\mathrm{T}} = \{1, 0, 0, 0, 0\}^{\mathrm{T}}$, with T denoting transpose operation. The model with $\lambda = 0$ does not allow for an endemic equilibrium point [67], characterised by $I^* \neq 0$. Meanwhile, as discussed in Sect. 4, the endemic state is expected for $\lambda \neq 0$.

B.2. Time dependence of M and U populations

It is instructive to rewrite (10)–(13) of the general model with the α_{ij} parameters expressed in terms of inward and outward masks' effectiveness. With $\lambda = 0$, one finds

$$\frac{dS_M}{\mathrm{d}t} = -\alpha_0(1-\epsilon_i)\Big[(1-\epsilon_o)I_M + I_U\Big]\, S_M, \tag{22}$$

$$\frac{\mathrm{d}S_U}{\mathrm{d}t} = -\alpha_0\Big[(1-\epsilon_o)I_M + I_U\Big] S_U. \tag{23}$$

Dividing both (22) and (23), one obtains

$$\frac{\mathrm{d}S_M}{S_M} = (1-\epsilon_i)\frac{\mathrm{d}S_U}{S_U}, \tag{24}$$

which can be integrated from the initial value $S_M(0)$ to the final (at time t) $S_M(t)$ to get

$$\frac{S_M(t)}{S_M(0)} = \left(\frac{S_U(t)}{S_U(0)}\right)^{1-\epsilon_i}. \tag{25}$$

The scaling relation (25) depends on the initial values and inward masks' effectiveness only. However, it is valid for arbitrary fraction x of masked and for arbitrary value of outward effectiveness ϵ_o. It provides the universal signature of the model. The numerical results (not shown here) agree exactly with the above scaling law.

B.3. Reproduction number

The basic reproduction number, usually denoted by $\mathcal{R}_0$, is a very useful parameter deciding about the evolution of an epidemic. The growth of the number of infections is signalled by $\mathcal{R}_0 > 1$, while $\mathcal{R}_O < 1$ signals the decline. The basic reproduction number is a threshold parameter in models with disease-free equilibrium (DFE). A general method of its calculation, which we shall follow, is well suited for compartmental models, and has been presented in [68, 69]. The idea is to write (21) in the form

$$\frac{\mathrm{d}y_i}{\mathrm{d}t} = \mathcal{F}_i(\boldsymbol{y}) - \mathcal{V}_i(\boldsymbol{y}), \tag{26}$$

with $\mathcal{F}_i$ describing the rates of appearance of new infections (in infected compartments), while $\mathcal{V}_i$ denotes the transitions between other infected compartments [68, 69]. The reproduction number $\mathcal{R}_0$ of the DFE is given by

$$\mathcal{R}_0 = \rho(FV^{-1}), \tag{27}$$

where ρ denotes the spectral radius and the matrices F and V denote the next generation matrices with matrix elements

$$F_{ij} = \left(\frac{\partial \mathcal{F}_i(\boldsymbol{y})}{\partial y_j}\right)_{\boldsymbol{y}=\boldsymbol{y^*}} \tag{28}$$

and

$$V_{ij} = \left(\frac{\partial \mathcal{V}_i(\boldsymbol{y})}{\partial y_j}\right)_{\boldsymbol{y}=\boldsymbol{y}^*}, \tag{29}$$

and the indices i and j run over compartments describing new infections [69]. With three infected compartments I, H, T in the general model with masked compartments, one finds for the DFE

$$F = \begin{pmatrix} \alpha_0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \tag{30}$$

and

$$V = \begin{pmatrix} (1-\rho)\,\gamma_{IR} + \rho\,\gamma_{IH} & 0 & 0 \\ -\rho\,\gamma_{IH} & (1-\rho_T)\,\gamma_{HR} + \rho_T\,\gamma_{HT} & 0 \\ 0 & -\rho_T\gamma_{HT} & \gamma_{TR} + \delta_T \end{pmatrix}. \tag{31}$$

This leads to the basic reproduction number

$$\mathcal{R}_0 = \frac{\alpha_0}{(1-\rho)\gamma_{IR} + \rho\gamma_{IH}}, \tag{32}$$

the result a slightly more general than that of simple SIR model. To derive it on epidemiological basis, it is enough to rewrite the equation for the change of $I(t)$ in the form

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \left[\frac{\alpha_0 S}{(1-\rho)\gamma_{IR} + \rho\gamma_{IH}} - 1\right] \times \left[(1-\rho)\gamma_{IR} + \rho\gamma_{IH}\right] I(t), \tag{33}$$

and note that the number of infected grows with time if the term $[\frac{\alpha_0 S}{(1-\rho)\gamma_{IR}+\rho\gamma_{IH}} - 1]$ is positive and dies out if it is negative. Thus the first term in parentheses, i.e., $\frac{\alpha_0 S}{(1-\rho)\gamma_{IR}+\rho\gamma_{IH}}$ plays a role of basic reproduction number. At time $t = 0$, one has $I \ll S$, so $S \approx 1$ and the above parameter reduces to $\mathcal{R}_0$.

For the general model with masks the DFE for $\lambda = 0$ is given by the set

$$(S_M^*, S_I^*, I_M^*, I_U^*, R_M^*, R_U^*, H_M^*, H_U^*, T_M^*, T_U^*) = (x, 1-x, 0, 0, 0, 0, 0, 0, 0, 0). \tag{34}$$

There are six infected $(I_M^*, I_U^*, H_M^*, H_U^*, T_M^*, T_U^*)$ compartments. The matrices F and V thus become 6×6. They read

$$F = \begin{pmatrix} x\alpha_{MM} & x\alpha_{MU} & 0 & 0 & 0 & 0 \\ (1-x)\alpha_{UM} & (1-x)\alpha_{UU} & 0 & 0 & 0 & 0 \\ \rho\gamma_{IH} & 0 & 0 & 0 & 0 & 0 \\ 0 & \rho\gamma_{IH} & 0 & 0 & 0 & 0 \\ 0 & 0 & \rho_T\gamma_{HT} & 0 & 0 & 0 \\ 0 & 0 & 0 & \rho_T\gamma_{HT} & 0 & 0 \end{pmatrix}, \tag{35}$$

and V is diagonal matrix with elements: $(1-\rho)\gamma_{IR} + \rho\gamma_{IH}$, $(1-\rho)\gamma_{IR} + \rho\gamma_{IH}$, $(1-\rho_T)\gamma_{HR} + \rho_T\gamma_{HT}$, $(1-\rho_T)\gamma_{HR} + \rho_T\gamma_{HT}$, $\gamma_{TR} + \delta_D$ and $\gamma_{TR} + \delta_D$.

Multiplying matrix F by the inverse of V and calculating the spectral radius $\rho(M)$, which for a quadratic matrix $M = FV^{-1}$ is its largest eigenvalue, one finds the reproduction number

$$\mathcal{R}_0^M = \frac{1}{2}\frac{x\alpha_{MM} + (1-x)\alpha_{UU} + \sqrt{(x\alpha_{MM} - (1-x)\alpha_{UU})^2 + 4x(1-x)\alpha_{MU}\alpha_{UM}}}{(1-\rho)\gamma_{IR} + \rho\gamma_{IH}}. \tag{36}$$

The actual value of $\mathcal{R}_\prime^{\mathcal{M}}$ depends on the fraction of masked, the contact rates and other (clinical) parameters. Introducing inward and outward effectiveness of masks, as in Sect. 5, one finds

$$\mathcal{R}_0^M(x, \epsilon_i, \epsilon_o) = \frac{\alpha_0}{(1-\rho)\gamma_{IR} + \rho\gamma_{IH}}\left[(1-x) + x(1-\epsilon_i)(1-\epsilon_o)\right]. \tag{37}$$

In agreement with expectations, this expression immediately reduces to the result (32) when $x = 0$ or $\epsilon_i = \epsilon_o = 0$.

Acknowledgments

The work of JMW-M and KIW has been partially supported by the Authors' institutions. MMW is supported by the Foundation for Polish Science through the International Research Agendas program co-financed by the European Union within the Smart Growth Operational Programme.

References

[1] M.R. Roussel, *Nonlinear Dynamics. A Hands-on Introductory Survey*, Morgan & Claypool Publishers, 2019.

[2] D. Sridhar, D. Gurdasani, *Science* **371**, 230 (2021).

[3] D. Adam, *Nature* **580**, 316 (2020).

[4] S. Flaxman, S. Mishra, A. Gandy et al., *Nature* **584**, 287 (2020).

[5] K. Soltesz, F. Gustafsson, T. Timpka et al., *Nature* **588**, E26 (2020).

[6] Y. Yin, J. Gao, B.F. Jones, D. Wang, *Science* **371**, 128 (2021).

[7] L. Morawska, Junji Cao, *Environ. Int.* **139**, 105730 (2020).

[8] L. Morawska, J.W. Tang, W. Bahneth et al., *Environ. Int.* **142**, 105832 (2020).

[9] T. Dbouk, D. Drikakis, *Phys. Fluids* **32**, 063303 (2020).

[10] C.C. Wang, K.A. Prather, J. Sznitman, J.L. Jimenez, S.S. Lakdawala, Z. Tufekci, L.C. Marr, *Science* **373**, 981 (2021).

[11] Coronavirus: Information and Recommendations, Temporary Limitations, www.gov.pl.

[12] B. Csutak, P. Polcz, G. Szederkenyi, arXiv:2105.04423 (2021).

[13] Centers for Disease Control and Prevention.

[14] World Health Organization, Mask Use in the Context of COVID-19: Interim Guidance, 1 December 2020.

[15] J. Wong, E. Claypool, *Qual. Soc. Work.* **20**, 206 (2021).

[16] L.W. Heyerdahl, M. Vray, B. Lana et al., *Vaccine* **40**, 1191 (2022).

[17] M. Sallam, *Vaccines (Basel)* **9**, 160 (2021).

[18] S.R. Davis, R.D. Ampon, L.M. Poulos, G.B. Marks, B.G. Toelle, H.K. Reddel, "Willingness to Receive Vaccination Against COVID-19: Results from a Large Nationally Representative Australian Population Survey", *medRxiv* preprint (2021).

[19] K. A. Lythgoe, M. Hall, L. Ferretti et al., *Science* **372**, eabg0821 (2021).

[20] H. Ledford, *Nature* **600**, 577 (2021).

[21] W.O. Kermack, A.G. McKendrick, *Proc. Royal Soc. London A* **115**, 700 (1927).

[22] M. Kreck, E. Scholz, arXiv:2104.00786v2 (2021).

[23] A.M. Carvalho, S. Gonalves, *Physica A* **566**, 125659 (2021).

[24] G.A. Muoz-Fernndez, J.M. Seoane, J.B. Seoane-Seplveda, *Chaos Solit. Fractals* **144**, 110682 (2021).

[25] Z. Liao, P. Lan, Z. Liao, Y. Zhang, S. Liu, *Sci. Rep.* **10**, 22454 (2020).

[26] A. Bodini, S. Pasquali, A. Pievatolo, F. Ruggeri, arXiv:2102.07566v3 (2021).

[27] V. Ram, L.P. Schaposnik, *Sci. Rep.* **11**, 15194 (2021).

[28] S.E. Eikenberry, M. Mancuso, E. Iboi, K. Eikenberry, Y. Kuang, E. Kostelich, A.B. Gumel, *Infect. Dis. Model.* **5**, 248 (2020).

[29] M. Aucouturier, H.J. Herrmann, *Int. J. Mod. Phys. C* **32**, 2150150 (2021).

[30] M. Cruz-Aponte, J. Caraballo-Cueto, arXiv:2006.01363v2 (2020).

[31] S. Moein, N. Nickaeen, A. Roointan, N. Borhani, Z. Heidary, S.H. Javanmard, J. Ghaisari, Y. Gheisari, *Sci. Rep.* **11**, 4725 (2021).

[32] N.N. Chung, L.Y. Chew, *Sci. Rep.* **11**, 10122 (2021).

[33] M. Martín-Sánchez, W.W. Lim, A. Yeung et al., *J. Infect.* **83**, 92 (2021).

[34] S. Esposito, N. Principi, C.C. Leung, G.B. Migliori, *Eur. Respir. J.* **55**, 2001260 (2020).

[35] C.J. Worby, H.-H. Chang, *Nat. Commun.* **11**, 4049 (2020).

[36] A. Catching, S. Capponi, M.T. Yeh, S.E. Bianco, R. Andino, *Sci. Rep.* **11**, 15998 (2021).

[37] K.M.A. Kabir, T. Risa, J. Tanimoto, *Sci. Rep.* **11**, 12621 (2021).

[38] A. Smolinska, D.S. Jessop, K.L. Pappan et al., *Sci. Rep.* **11**, 13476 (2021).

[39] S. Tiwari, C.P. Vyasarayani, A. Chatterjee, *Sci. Rep.* **11**, 8106 (2021).

[40] A.L. Rasmussen, S.V. Popescu, *Science* **371**, 1206 (2021).

[41] A. Telenti, A. Arvin, L. Corey et al., *Nature* **596**, 495 (2021).

[42] S.V. Subramanian, A. Kumar, *Eur. J. Epidemiol.* **36**, 1237 (2021).

[43] A. Dąbek, Rekordowa liczba zajętych respiratorów w Polsce. Tak źle nie było od początku pandemii, 2021 (in Polish).

[44] J.M. Musser, P.A. Christensen, R.J. Olsen et al., *Am. J. Pathol.* **192**, 320 (2022).

[45] P. Elliott, D. Haw, H. Wang et al., "REACT-1 Round 13 Final Report: Exponential Growth, High Prevalence of SARS-CoV-2 and Vaccine Effectiveness Associated with Delta Variant in England During May to July 2021", preprint, 2021.

[46] Z. Burda, *Entropy* **22**, 1236 (2020).

[47] J.C. Law, M. Girard, G.Y.C. Chao et al., *J. Immunol.* **208**, 429 (2022).

[48] S. Amit, G. Regev-Yochay, A. Afek, Y. Kreiss, E. Leshem, *Lancet* **397**, 875 (2021).

[49] N. Doria-Rose, M.S. Suthar, M. Makowski, *N. Engl. J. Med.* **384**, 23 (2021).

[50] C. Tien, "How Long Will COVID-19 Vaccine-Induced Immunity Last?", 2022.

[51] T. Britton, F. Ball, P. Trapman, *Science* **369**, 846 (2020).

[52] J.F.-W. Chan, S. Yuan, A.J. Zhang et al., *Clin. Infect. Dis.* **71**, 2139 (2020).

[53] N.J. Rowan, R.A. Moral, *Sci. Total Environ.* **772**, 145530 (2021).

[54] A.P. Sunjaya, L. Morawska, *Disaster Med. Public Health Prep.* **14**, e42 (2020).

[55] G. Spagnuolo, D. De Vito, S. Rengo, M. Tatullo, *Int. J. Environ. Res. Public Health* **17**, 2094 (2020).

[56] J. Cleveland, S.K. Gray, J. Harte, V. Robison, A. Moorman, B. Gooch, *J. Am. Dent. Assoc.* **147**, 729 (2016).

[57] S. Jungo, N. Moreau, M.E. Mazevet, A.-L. Ejeil, M.B. Duplan, B. Salmon, V. Smail-Faugeron, *PLoS ONE* **16**, e0246586 (2021).

[58] C.G. Estrich, M. Mikkelsen, R. Morrissey, M.L. Geisinger, E. Ioannidou, M. Vujicic, M. W.B. Araujo, *J. Am. Dent. Assoc.* **151**, 815 (2020).

[59] Naczelna Izba Lekarska, Raport z Badania Ankietowego pt. "COVID-19 a Prowadzenie Praktyk i Podmiotów w Stomatologii", 2020, (in Polish).

[60] C. Maulani, S. Lelyati, C. Masulili, E.I. Auerkari, *AIP Conf. Proc.* **2344**, 030002 (2021).

[61] P. Banerjee, S.K. Pandey, B.S. Munde, G.D. Nagargoje, S. Mohani, M.A. Shinde, *J. Pharm. Bioallied Sci.* **13**, 162 (2021).

[62] A.K. Kelekar, V.C. Lucia, N.M. Afonso, A.K. Mascarenhas, *J. Am. Dent. Assoc.* **152**, 596 (2021).

[63] A.K. Mascarenhas, V.C. Lucia, A. Kelekar, N.M. Afonso, *J. Dent. Educ.* **85**, 1504 (2021).

[64] H. Larvin, S. Wilmott, J. Wu, J. Kang, *Front. Med.* **23**, 604980 (2020).

[65] H. Utomo, I. Komang, E. Wijaksana, C. Prahasanti, *Dent. Hypotheses* **12**, 28 (2021).

[66] M.M. Woolfson M.S. Woolfson, *Mathematics for Physics*, Ch. 19, Oxford University Press, Oxford 2007.

[67] C.M. Batistela, D.P.F. Correa, M. Bueno, J.R.C. Piqueira, *Chaos Solit. Fractals* **142**, 110388 (2021).

[68] P. van den Driessche, J. Watmough, *Math. Biosci.* **180**, 29 (2002).

[69] P. van den Driessche, *Infect. Dis. Model.* **2**, 288 (2017).