

---

# THE WINTER DILEMMA

---

Sebastian Contreras<sup>1†</sup>, Philipp Dönges<sup>1†</sup>, Joel Wagner<sup>1†</sup>, Simon Bauer<sup>1†</sup>, Sebastian B. Mohr<sup>1</sup>,  
Jonas Dehning<sup>1</sup>, Emil N. Iftekhar<sup>1</sup>, Mirjam Kretzschmar<sup>2</sup>, Michael Maes<sup>3</sup>, Kai Nagel<sup>4</sup>,  
André Calero Valdez<sup>5</sup>, and Viola Priesemann<sup>1,6\*</sup>

<sup>1</sup>Max Planck Institute for Dynamics and Self-Organization, Göttingen, Germany.

<sup>2</sup>University Medical Center Utrecht, Utrecht, The Netherlands.

<sup>3</sup>Karlsruhe Institute of Technology, Karlsruhe.

<sup>4</sup>Technische Universität Berlin, Berlin, Germany.

<sup>5</sup>RWTH Aachen University, Aachen, Germany.

<sup>6</sup>Institute for the Dynamics of Complex Systems, University of Göttingen, Göttingen, Germany.

\* Corresponding Author: Viola Priesemann (viola.priesemann@ds.mpg.de)

† These authors contributed equally

## Abstract

As summer in the northern hemisphere comes to an end, changes in daylight, temperature, and weather — and people’s reaction to them — will be the drivers of a disadvantageous seasonality of SARS-CoV-2. With the seasonal odds against us, stabilization of new COVID-19 cases and hospitalizations requires high immunity levels in the population or sufficient non-pharmaceutical interventions (NPIs). However, compliance with mandatory NPIs, vaccine uptake, and individual protective measures depend on individual opinions and decisions. This in turn depends on the individuals’ communication network, as well as access to and personal consumption of information, e.g about vaccine safety or current infection levels. Therefore, understanding how individual protection-seeking behavior affects disease spread is crucial to prepare for the upcoming winter and future challenges.

As summer in the northern hemisphere comes to an end, changes in daylight, temperature, and weather — and people’s reaction to them — will be the drivers of a disadvantageous seasonality of SARS-CoV-2. With the seasonal odds against us, stabilization of new COVID-19 cases and hospitalizations requires high immunity levels in the population or sufficient non-pharmaceutical interventions (NPIs). However, compliance with mandatory NPIs, vaccine uptake, and individual protective measures depend on individual opinions and decisions. This in turn depends on the individuals’ communication network, as well as access to and personal consumption of information, e.g about vaccine safety or current infection levels. Therefore, understanding how individual protection-seeking behavior affects disease spread is crucial to prepare for the upcoming winter and future challenges.

Protection-seeking behavior can be triggered, for instance, when COVID-19 incidence and ICU occupancy and thus personal risk is perceived to be high. Due to this inter-dependency between information and behavior, a

dilemma arises for the coming winter. On the one hand, maintaining NPIs to keep the reproduction number  $R$  low implies lower COVID-19 incidence — thus diminishing incentives to reduce contacts or get vaccinated; thereby, one risks a severe wave as soon as restrictions are lifted (especially considering waning immunity from those immunized before winter). On the other hand, relaxing restrictions more than current vaccination levels allow can lead to excess morbidity and mortality [1, 2].

To demonstrate the extent of this dilemma, we use a standard susceptible-exposed-infected-recovered (SEIR) model with explicit compartments for fatalities, intensive care units (ICUs), and vaccination (first time and booster vaccines), and also waning immunity, and seasonality. To account for behavioral changes that are induced by the perceived risk, we include a feedback-loop between information on ICU occupancy and the level of contacts, i.e., the reproduction number, and vaccination willingness (cf. Figs S1 and S2, and Supplementary Material). Explicitly, we assume that increases in ICU occupancy i) decrease the spreading rate of COVID-19, accounting for protection-seeking behavior and voluntary reduction of mobility [3, 4], and ii) increase vaccine acceptance among hesitant individuals [5]. We model these two loops to act on different timescales, as individuals can adapt their number of contacts and their intensity on a daily basis, while deciding to get vaccinated takes longer. Using state-of-the-art parameters and survey results reported in literature (Table S1, Supplementary Material), we analyze three scenarios of government-imposed NPIs over winter: 1) immediately lifting all NPIs, 2) maintaining mild NPIs, and 3) maintaining moderate NPIs to sustain low case numbers (cf. Fig1A). Note that the parameters are characteristic for a country with moderate (50–70 %) immunity among the population.

We find that assuming no restrictions throughout winter will trigger a steep increase in case numbers and hospitalization (Fig 1, black lines). As a consequence, individuals would voluntarily reduce their contacts and be more inclined to accept a vaccine offer (Fig 1D, E). However, this surge will increase morbidity and mortality because the effect of vaccination is not instantaneous. In contrast, countries with higher levels of immunity will likely not overwhelm their ICUs with a seasonal wave even if abolishing NPIs (cf. Fig. S3). In the opposite corner scenario —sustaining moderate NPIs and low case numbers— might lead to low COVID-19 incidence through winter but risks a rebound wave in spring (Fig 1, blue). This is because the low incidence over winter may imply i) low natural immunity, ii) lacking incentives for vaccination, and iii) lower chances of refreshing immune memory upon re-exposure to the virus [6]. The resulting low immunity levels (cf. Fig 1 E) can then fuel a high rebound wave. Similar rebound waves have been observed for other seasonal respiratory viruses [7, 8].

Altogether, the way we face this winter will impact long-term COVID-19 transmission dynamics and thus determine i) the morbidity and mortality burden to societies, ii) the probability of having an off-seasonal COVID-19 wave when lifting NPIs, iii) the magnitude of the self-regulation effect induced by the information-behavior feedback loop, and iv) whether and when we will reach appropriate immunity levels to transit from epidemicity to endemicity smoothly. Whether the solution to this winter dilemma is to accept high case numbers is not obvious; aside from higher mortality and morbidity (Fig 1F) we would accept higher probabilities of generating new SARS-CoV-2 variants and risk the long-term success of vaccination [9, 10]. An upcoming challenge for authorities is to find ways to engage individuals with vaccination programs without requiring high case numbers for that. Further research on this interaction between information and disease spread is highly needed to offer better preparedness for current and future global health infectious disease threats.

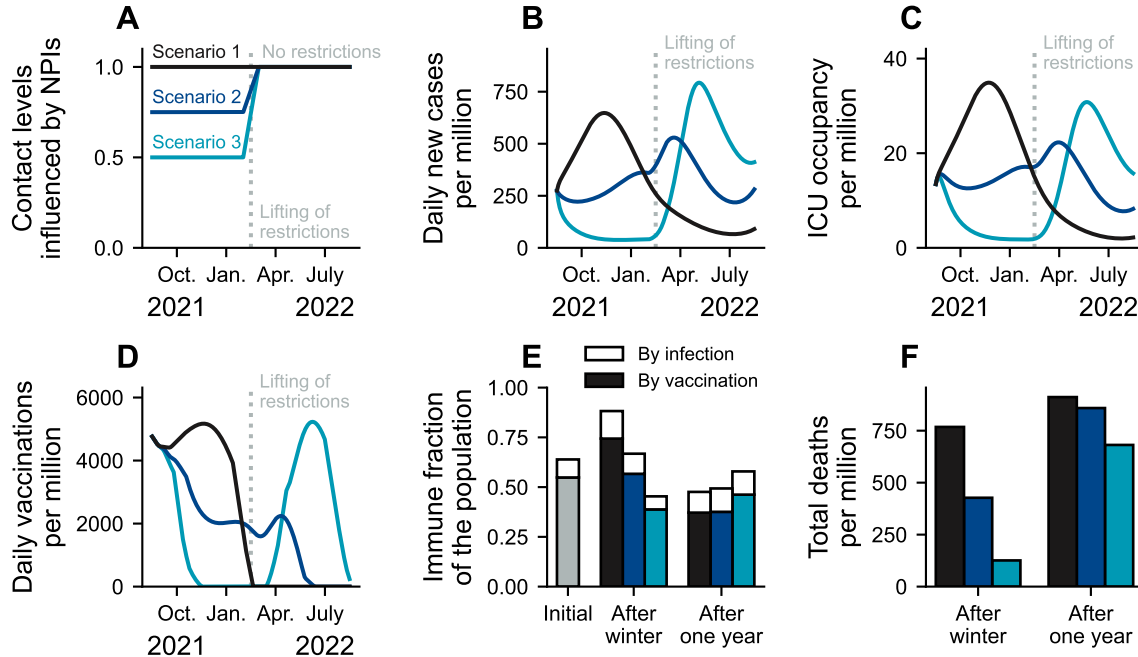


Figure 1: **COVID-19 restrictions planning through winter: a long-term dilemma.** The interplay of non-pharmaceutical interventions (NPI), that are sustained through winter 2021/2022, with people’s protection-seeking behavior will determine case numbers and ICU occupancy over winter and beyond. **A:** We explored three scenarios of mandatory NPI stringency in winter, gradually lifting all restrictions in March 2022. **B, C:** Scenario 1: having no restrictions causes a steep increase in case numbers and ICU occupancy that triggers protection-seeking behavior among the population. In this situation, the self-regulation of contacts, growing vaccine uptake, and higher rates of natural immunization would contribute to stabilizing case numbers (**D, E**), bearing, however, high mortality and morbidity in winter (**F**). Scenario 2: Maintaining mild restrictions would curb the overwhelming of ICUs while allowing for higher vaccine uptakes and natural immunity rates. Scenario 3: Maintaining moderate restrictions throughout winter will minimize COVID-19 cases and hospitalizations in winter, generating a shared perception of safety across the population. However, low vaccine uptake and rates of naturally acquired immunity through winter together with waned immunity will cause a severe rebound wave when restrictions are completely lifted in March (**D–F**).

## Acknowledgments

Authors with affiliation "1" received support from the Max-Planck-Society. SB and SBM were financially supported by the German Federal Ministry of Education and Research (BMBF) as part of the Network University Medicine (NUM), project egePan, funding code: 01KX2021.

## References (1–10 for the Main Text, 11–39 for the Supplementary Material)

- [1] Simon Bauer, Sebastian Contreras, Jonas Dehning, Matthias Linden, Emil Iftekhar, Sebastian B Mohr, Alvaro Olivera-Nappa, and Viola Priesemann. Relaxing restrictions at the pace of vaccination increases freedom and guards against further covid-19 waves. *PLoS computational biology*, 17(9):e1009288, 2021.
- [2] Sebastian Contreras and Viola Priesemann. Risking further COVID-19 waves despite vaccination. *The Lancet Infectious Diseases*, 2021.

- 
- [3] Pierre Nouvellet, Sangeeta Bhatia, Anne Cori, Kylie EC Ainslie, Marc Baguelin, Samir Bhatt, Adhiratha Boonyasiri, Nicholas F Brazeau, Lorenzo Cattarino, Laura V Cooper, et al. Reduction in mobility and covid-19 transmission. *Nature communications*, 12(1):1–9, 2021.
- [4] James N Druckman, Samara Klar, Yanna Krupnikov, Matthew Levendusky, and John Barry Ryan. Affective polarization, local contexts and public opinion in america. *Nature human behaviour*, 5(1):28–38, 2021.
- [5] Gul Deniz Salali and Mete Sefa Uysal. Effective incentives for increasing covid-19 vaccine uptake. *Psychological Medicine*, pages 1–6, 2021.
- [6] Eric L Brown and Heather T Essigmann. Original antigenic sin: the downside of immunological memory and implications for covid-19. *Mosphere*, 6(2):e00056–21, 2021.
- [7] Gabriela B Gomez, Cedric Mahé, and Sandra S Chaves. Uncertain effects of the pandemic on respiratory viruses. *Science*, 372(6546):1043–1044, 2021.
- [8] Ivan Sanz-Muñoz, Sonia Tamames-Gómez, Javier Castrodeza-Sanz, José María Eiros-Bouza, and Raul Ortiz de Lejarazu-Leonardo. Social distancing, lockdown and the wide use of mask; a magic solution or a double-edged sword for respiratory viruses epidemiology? *Vaccines*, 9(6):595, 2021.
- [9] Viola Priesemann, Rudi Balling, Melanie M Brinkmann, Sandra Ciesek, Thomas Czypionka, Isabella Eckerle, Giulia Giordano, Claudia Hanson, Zdenek Hel, Pirta Hotulainen, et al. An action plan for pan-european defence against new SARS-CoV-2 variants. *The Lancet*, 397(10273):469–470, 2021.
- [10] Emil Nafis Iftekhar, Viola Priesemann, Rudi Balling, Simon Bauer, Philippe Beutels, André Calero Valdez, Sarah Cuschieri, Thomas Czypionka, Uga Dumpis, Enrico Glaab, Eva Grill, Claudia Hanson, Pirta Hotulainen, Peter Klimek, Mirjam Kretzschmar, Tyll Krüger, Jenny Krutzinna, Nicola Low, Helena Machado, Carlos Martins, Martin McKee, Sebastian Bernd Mohr, Armin Nassehi, Matjaž Perc, Elena Petelos, Martyn Pickersgill, Barbara Prainsack, Joacim Rocklöv, Eva Schernhammer, Anthony Staines, Ewa Szczurek, Sotirios Tsiodras, Steven Van Gucht, and Peter Willeit. A look into the future of the COVID-19 pandemic in Europe: an expert consultation. *The Lancet Regional Health - Europe*, 8:100185, 2021.
- [11] Tomáš Gavenčiak, Joshua Teperowski Monrad, Gavin Leech, Mrinank Sharma, Sören Mindermann, Jan Marcus Brauner, Samir Bhatt, and Jan Kulveit. Seasonal variation in sars-cov-2 transmission in temperate climates. *medRxiv*, 2021.
- [12] Nicholas G. Davies, Sam Abbott, Rosanna C. Barnard, Christopher I. Jarvis, Adam J. Kucharski, James D. Munday, Carl A. B. Pearson, Timothy W. Russell, Damien C. Tully, Alex D. Washburne, Tom Wenseleers, Amy Gimma, William Waites, Kerry L. M. Wong, Kevin van Zandvoort, Justin D. Silverman, CMMID COVID-19 Working Group<sup>1</sup>‡, COVID-19 Genomics UK (COG-UK) Consortium<sup>‡</sup>, Karla Diaz-Ordaz, Ruth Keogh, Rosalind M. Eggo, Sebastian Funk, Mark Jit, Katherine E. Atkins, and W. John Edmunds. Estimated transmissibility and impact of SARS-CoV-2 lineage B.1.1.7 in England. *Science*, March 2021.
- [13] Karen Y Oróstica, Sebastian Contreras, Sebastian B Mohr, Jonas Dehning, Simon Bauer, David Medina-Ortiz, Emil N Iftekhar, Karen Mujica, Paulo C Covarrubias, Soledad Ulloa, et al. Mutational signatures and transmissibility of sars-cov-2 gamma and lambda variants. *arXiv preprint arXiv:2108.10018*, 2021.
- [14] Matan Levine-Tiefenbrun, Idan Yelin, Rachel Katz, Esmá Herzog, Ziv Golan, Licita Schreiber, Tamar Wolf, Varda Nadler, Amir Ben-Tov, Jacob Kuint, et al. Decreased SARS-CoV-2 viral load following vaccination. *medRxiv*, 2021.

- 
- [15] Smriti Mallapaty. Can COVID vaccines stop transmission? Scientists race to find answers. *Nature*, 2021.
- [16] Victoria Jane Hall, Sarah Foulkes, Ayoub Saei, Nick Andrews, Blanche Oguti, Andre Charlett, Edgar Wellington, Julia Stowe, Natalie Gillson, Ana Atti, Jasmin Islam, Ioannis Karagiannis, Katie Munro, Jameel Khawam, The SIREN Study Group, Meera A. Chand, Colin Brown, Mary E. Ramsay, Jamie Lopez Bernal, and Susan Hopkins. Effectiveness of BNT162b2 mRNA Vaccine Against Infection and COVID-19 Vaccine Coverage in Healthcare Workers in England, Multicentre Prospective Cohort Study (the SIREN Study). SSRN Scholarly Paper ID 3790399, Social Science Research Network, Rochester, NY, 2021.
- [17] Laith J Abu-Raddad, Hiam Chemaitelly, and Adeel A Butt. Effectiveness of the bnt162b2 covid-19 vaccine against the b. 1.1. 7 and b. 1.351 variants. *New England Journal of Medicine*, 2021.
- [18] Elisabeth Mahase. Covid-19: Where are we on vaccines and variants? *BMJ*, 372:n597, 2021.
- [19] Noa Dagan, Noam Barda, Eldad Kepten, Oren Miron, Shay Perchik, Mark A. Katz, Miguel A. Hernán, Marc Lipsitch, Ben Reis, and Ran D. Balicer. BNT162b2 mRNA COVID-19 Vaccine in a Nationwide Mass Vaccination Setting. *New England Journal of Medicine*, 2021.
- [20] Yinon M Bar-On, Yair Goldberg, Micha Mandel, Omri Bodenheimer, Laurence Freedman, Nir Kalkstein, Barak Mizrahi, Sharon Alroy-Preis, Nachman Ash, Ron Milo, et al. Protection of bnt162b2 vaccine booster against covid-19 in israel. *New England Journal of Medicine*, 2021.
- [21] Ross J Harris, Jennifer A Hall, Asad Zaidi, Nick J Andrews, J Kevin Dunbar, and Gavin Dabrera. Impact of vaccination on household transmission of SARS-COV-2 in england. *medRxiv*, 2021.
- [22] Iván Martínez-Baz, Ana Miqueleiz, Itziar Casado, Ana Navascués, Camino Trobajo-Sanmartín, Cristina Burgui, Marcela Guevara, Carmen Ezpeleta, Jesús Castilla, et al. Effectiveness of covid-19 vaccines in preventing sars-cov-2 infection and hospitalisation, navarre, spain, january to april 2021. *Eurosurveillance*, 26(21):2100438, 2021.
- [23] Yinon M Bar-On, Avi Flamholz, Rob Phillips, and Ron Milo. Science forum: SARS-CoV-2 (COVID-19) by the numbers. *Elife*, 9:e57309, 2020.
- [24] Ruiyun Li, Sen Pei, Bin Chen, Yimeng Song, Tao Zhang, Wan Yang, and Jeffrey Shaman. Substantial undocumented infection facilitates the rapid dissemination of novel coronavirus (SARS-CoV-2). *Science*, 368(6490):489–493, 2020.
- [25] Xi He, Eric H Y Lau, Peng Wu, Xilong Deng, Jian Wang, Xinxin Hao, Yiu Chung Lau, Jessica Y Wong, Yujuan Guan, Xinghua Tan, et al. Temporal dynamics in viral shedding and transmissibility of COVID-19. *Nature Medicine*, pages 1–4, 2020.
- [26] Feng Pan, Tianhe Ye, Peng Sun, Shan Gui, Bo Liang, Lingli Li, Dandan Zheng, Jiazheng Wang, Richard L Hesketh, Lian Yang, et al. Time course of lung changes on chest CT during recovery from 2019 novel coronavirus (COVID-19) pneumonia. *Radiology*, page 200370, 2020.
- [27] Yun Ling, Shui-Bao Xu, Yi-Xiao Lin, Di Tian, Zhao-Qin Zhu, Fa-Hui Dai, Fan Wu, Zhi-Gang Song, Wei Huang, Jun Chen, et al. Persistence and clearance of viral RNA in 2019 novel coronavirus disease rehabilitation patients. *Chinese medical journal*, 2020.
- [28] Andrew T Levin, William P. Hanage, Nana Owusu-Boaitey, Kensington B. Cochran, Seamus P. Walsh, and Gideon Meyerowitz-Katz. Assessing the age specificity of infection fatality rates for COVID-19: systematic review, meta-analysis, and public policy implications. *European Journal of Epidemiology*, 2020.

- [29] Matthias Linden, Sebastian B. Mohr, Jonas Dehning, Jan Mohring, Michael Meyer-Hermann, Iris Pigeot, Anita Schöbel, and Viola Priesemann. Case numbers beyond contact tracing capacity are endangering the containment of COVID-19 . *Dtsch Arztebl International*, 117(46):790–791, 2020.
- [30] Henrik Salje, Cécile Tran Kiem, Noémie Lefrancq, Noémie Courtejoie, Paolo Bosetti, Juliette Paireau, Alessio Andronico, Nathanaël Hozé, Jehanne Richet, Claire-Lise Dubost, et al. Estimating the burden of SARS-CoV-2 in France. *Science*, 369(6500):208–211, 2020.
- [31] Yair Goldberg, Micha Mandel, Yinon M Bar-On, Omri Bodenheimer, Laurence S Freedman, Eric Haas, Ron Milo, Sharon Alroy-Preis, Nachman Ash, and Amit Huppert. Waning immunity of the bnt162b2 vaccine: A nationwide study from israel. *medRxiv*, 2021.
- [32] Denis Sauré, Miguel O’Ryan, Juan Pablo Torres, Marcela Zuniga, Emilio Santelices, and Leonardo J Basso. Dynamic igg seropositivity after rollout of coronavac and bnt162b2 covid-19 vaccines in chile: a sentinel surveillance study. *The Lancet Infectious Diseases*, 2021.
- [33] Jackson S Turner, Wooseob Kim, Elizaveta Kalaidina, Charles W Goss, Adriana M Rauseo, Aaron J Schmitz, Lena Hansen, Alem Haile, Michael K Klebert, Iskra Pusic, et al. Sars-cov-2 infection induces long-lived bone marrow plasma cells in humans. *Nature*, pages 1–5, 2021.
- [34] Zijun Wang, Frauke Muecksch, Dennis Schaefer-Babajew, Shlomo Finkin, Charlotte Viant, Christian Gaebler, Hans-Heinrich Hoffmann, Christopher O Barnes, Melissa Cipolla, Victor Ramos, et al. Naturally enhanced neutralizing breadth against sars-cov-2 one year after infection. *Nature*, 595(7867):426–431, 2021.
- [35] Cornelia Betsch, Lothar Wieler, Michael Bosnjak, Michael Ramharter, Volker Stollorz, Saad Omer, Lars Korn, Philipp Sprengholz, Lisa Felgendreff, Sarah Eitze, et al. Germany covid-19 snapshot monitoring (cosmo germany): Monitoring knowledge, risk perceptions, preventive behaviours, and public trust in the current coronavirus outbreak in germany. 2020.
- [36] Olivier J Wouters, Kenneth C Shadlen, Maximilian Salcher-Konrad, Andrew J Pollard, Heidi J Larson, Yot Teerawattananon, and Mark Jit. Challenges in ensuring global access to COVID-19 vaccines: production, affordability, allocation, and deployment. *The Lancet*, 2021.
- [37] Rahul K Arora, Abel Joseph, Jordan Van Wyk, Simona Rocco, Austin Atmaja, Ewan May, Tingting Yan, Niklas Bobrovitz, Jonathan Chevrier, Matthew P Cheng, et al. Serotracker: a global sars-cov-2 seroprevalence dashboard. *The Lancet Infectious Diseases*, 21(4):e75–e76, 2021.
- [38] Hannah Ritchie, Edouard Mathieu, Lucas Rodés-Guirao, Cameron Appel, Charlie Giattino, Esteban Ortiz-Ospina, Joe Hasell, Bobbie Macdonald, Diana Beltekian, and Max Roser. Coronavirus pandemic (covid-19). *Our World in Data*, 2021. <https://ourworldindata.org/coronavirus>.
- [39] T Riffe, E Acosta, et al. Coverage-db: A database of covid-19 cases and deaths by age, 2020.

## Supplementary Material

### Data availability

Source code for data generation and analysis is available online on GitHub [https://github.com/Prieseemann-Group/covid19\\_winter\\_dilemma](https://github.com/Prieseemann-Group/covid19_winter_dilemma).

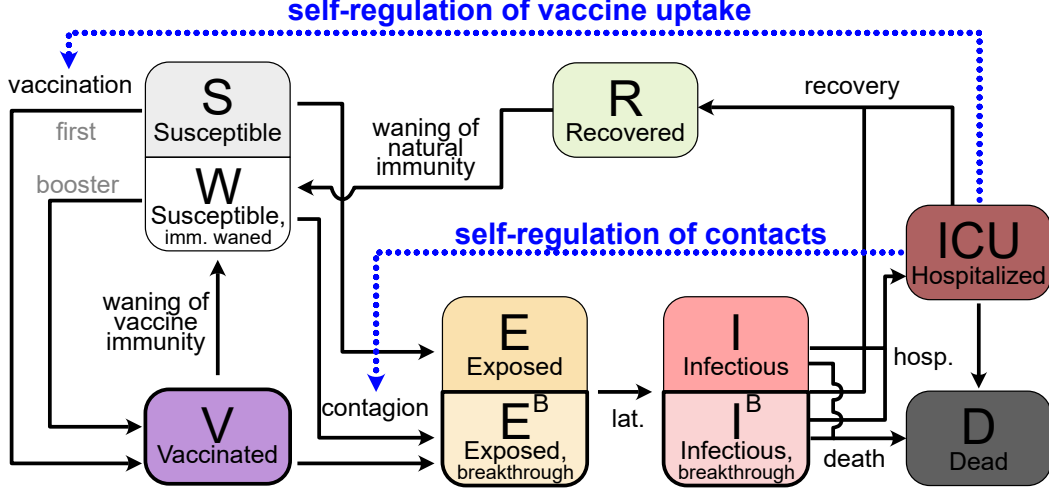
### S1 Model

We model the spreading dynamics of SARS-CoV-2 following a mean-field SEIRD-ICU deterministic formalism through a system of differential equations. Our model incorporates disease-spreading dynamics, ICU stays, and the roll-out of a single-dose vaccine, that is equivalent to the two doses of most COVID-19 vaccines. Both vaccine-induced and naturally-acquired immunity wane over time, but vaccine-induced immunity wanes faster. For a graphical representation of the infection and core dynamics, see Fig. S1. In our model, susceptible individuals can acquire the virus from infected individuals and subsequently progress to the exposed ( $S \rightarrow E$ ) and infectious ( $E \rightarrow I$ ) compartments. The waning immunity is modelled via a compartment ( $W$ ) which features no protection against infection but against severe course of the disease ( $W \rightarrow E^B$ ), and also differs from  $S$  in respect to the vaccination dynamics. We assume that vaccines offer no perfect sterile immunity and that a fraction of vaccinated people is infected upon contact with the infectious groups, i.e., we follow a leaky vaccination implementation. In contrast to unvaccinated individuals, vaccinated individuals and those with waned immunity move to different exposed ( $V, W \rightarrow E^B$ ) and infectious ( $E^B \rightarrow I^B$ ) compartments. These breakthrough infections still offer a certain protection against a severe course of the disease, i.e., have reduced probabilities to go to ICU or die. Individuals exposed to the virus ( $E, E^B$ ) progress from the exposed to the infectious compartments ( $I, I^B$ ) at a rate  $\rho$ . The infectious compartments have three different possible transitions: i) direct recovery ( $I, I^B \rightarrow R$ ) with rate  $\gamma$ , ii) progression to ICU ( $I, I^B \rightarrow \text{ICU}$ ) with rate  $\delta$  (reduced by  $(1-\kappa)$  for  $I^B$ ) or iii) direct death ( $I, I^B \rightarrow D$ ) with rate  $\theta$  (reduced by  $(1-\kappa)$  for  $I^B$ ). Individuals receiving ICU treatment recover either at a rate  $\gamma_{\text{ICU}}$  ( $\text{ICU} \rightarrow R$ ) or die at a rate  $\theta_{\text{ICU}}$  ( $\text{ICU} \rightarrow D$ ). Another important property of this model is the self-regulation of contacts and vaccine acceptance that influences the disease and vaccination dynamics based on the current and past ICU occupancy.

#### S1.1 Reproduction Number

Our model includes the effects of governmental non-pharmaceutical interventions, individuals self-regulating their contacts based on perceived risk, and seasonality. Each is represented by a multiplicative factor on the gross reproduction number  $R_t$ , i.e., the total number of offspring infections that a single case would generate in a fully susceptible population.

The base reproduction number (no self-regulation, no seasonality)  $R_t^{\text{base}}$  is chosen to represent one of the three scenarios modeled (see Fig. 1): The immediate lifting of all restrictions (high  $R_t^{\text{base}}$ ), moderate restrictions over winter (moderate  $R_t^{\text{base}}$ ) and strong restrictions over winter (low  $R_t^{\text{base}}$ ). It is assumed that in March next year all restrictions will be lifted in all scenarios. Easy-to-follow measures such as improved hygiene might still be kept in place which results in a small reduction of the natural reproduction number  $R_0$ . The abolishment of NPIs is modeled by a linear increase in  $R_t^{\text{base}}$  that lasts four weeks and is centered around 1.March. Independent of governmental decisions, each individual has the freedom to adapt his or her behavior in accordance with perceived risk, which plays out not only in regards to vaccination willingness but also in



Supplementary Figure S1: **Figure S1: Compartmental model with feedback loops between hospital occupancy and spreading rate and vaccination willingness.** Transition rates are listed in Table S1, but omitted in the figure for clarity purposes.

regards to contact regulation. An exponential term is thus multiplied onto the reproduction number and depends on the past ICU occupancy  $H_R$  (see sec. S1.1.1) and a sensitivity constant  $\alpha_R$ . At last, seasonality is modeled by a time dependent sinusoidal modulation factor  $\Gamma(t)$  that depends on the sensitivity  $\mu$  and the day with the highest effect on seasonality  $d_\mu$ , which for our purpose can be set to zero, corresponding to January 1 [11]. The full reproduction number is then given by

$$R_t(H_R, t) = R_t^{\text{base}} \exp(-\alpha_R H_R) \frac{\Gamma(t)}{\Gamma(360 - d_0)}, \quad (1)$$

$$\Gamma(t) = 1 + \mu \cos\left(2\pi \frac{t + d_0 - d_\mu}{360}\right) \quad (2)$$

where  $\frac{1}{\Gamma(360 - d_0)}$  is for normalization such that seasonality only decreases  $R_t$ , i.e. neglecting the behavior term  $R_t^{\text{base}}$  corresponds to the peak value in winter. For simplicity in our model one month has 30 days and a full year thus 360 days which does not affect the results on our time horizon.

### S1.1.1 Memory on perceived danger

Perceived danger for the individual, transmitted by e.g., mass media or affected acquaintances, depends on ICU not only at the present moment but also on the past. That way, self-regulation of contacts and vaccine uptake is a function of the past development of the ICU occupancy  $H$ . We assume that the memory of past ICU development is smooth, meaning that ICU occupancies long ago are remembered less and less as time passes. To incorporate this into our model we calculate the convolution of the ICU with a gamma distribution, effectively "weighting" the past development of ICU. That way, ICU occupancy a few days ago is "remembered" more and thus influences people's behavior at the present moment more than the ICU occupancy that lies further in the past. That way, the reproduction number becomes dependent on  $H_R(t)$  via

$$H_R(t) := \text{ICU} * \mathcal{G}_{p=1, b_R} = \int_{-\infty}^t dt' \text{ICU}(t') \mathcal{G}_{p=1, b_R}(-t' + t), \quad (3)$$



where the arguments of the gamma distribution are set to  $p = 1$  and  $b_R = 4$ .

Time memory for vaccination willingness is assumed to work in the same way but with different gamma distributions. First of all, there is a delay between the decision to be vaccinated and the onset of immunity. Secondly, vaccination willingness is assumed to depend on a longer time interval of the ICU occupancy. Combined, it translates into a gamma distribution that is shifted in time and looks flatter which is characterized by the parameters  $\tau_u, \tau_w$  and  $b_v = 14$ :

$$H_{u,w}(t) := \text{ICU} * \mathcal{G}_{p=1,b_v} = \int_{-\infty}^t dt' \text{ICU}(t') \mathcal{G}_{p=1,b_v}(-t' - \tau_{u,w} + t). \quad (4)$$

The subscripts  $_u$  and  $_w$  indicate first and booster doses respectively. The parameter  $\tau_u$  is larger than  $\tau_w$  because we include the delay of around 6 weeks for most vaccines that need two doses. Booster doses are usually only a single dose so  $\tau_w$  is just the delay between administration of the dose and onset of immunity which we assume to be 2 weeks. For the initial conditions of  $H$  and  $H_R$  we set  $\text{ICU}(t < 0) = \text{ICU}(t = 0)$  in the past. This simplification affects the results only negligibly for a short initial time.

### S1.1.2 Waning Immunity

Our model includes two types of immunity: immunity as a result of vaccination and immunity as a result of natural infection. In both cases immunity wanes over time although it is believed that natural immunity lasts longer and thus has a lower waning rate. On average, vaccine-induced immunity wanes after  $(\Omega_v^{\text{base}})^{-1}$  months and naturally-acquired immunity after  $(\Omega_n^{\text{base}})^{-1}$  months. Furthermore, we assume that immunized individuals can refresh their immune memory upon contact with the virus which translates into infection level- and  $R_t$ -dependent waning rates  $\Omega_{v,n}(I_{\text{eff}}, R_t)$ , where the effective incidence  $I_{\text{eff}}$  corresponds to the total size of the infectious pools  $I$  and  $I^B$  but acknowledges reduced virulence of breakthrough infections (see sec. S1.3. Furthermore an influx of  $\Psi$  was added to account for infections from abroad. In the limit of high infection levels the waning rate should converge to zero and in the limit ( $I_{\text{eff}} \rightarrow 0$ ), where no refreshing happens, it should be at its base value  $\Omega_{v,n}^{\text{base}}$ . Using a logistic function that meets these requirements and decreases linearly for low infection levels, we can express the waning immunity as a function of  $I_{\text{eff}}$  and the reproduction number  $R_t$ :

$$\Omega_v(I_{\text{eff}}, R_t) = 2\Omega_v^{\text{base}} \left( 1 - \frac{1}{1 + \exp(-c_v R_t I_{\text{eff}})} \right), \quad (5)$$

$$\Omega_n(I_{\text{eff}}, R_t) = 2\Omega_n^{\text{base}} \left( 1 - \frac{1}{1 + \exp(-c_n R_t I_{\text{eff}})} \right). \quad (6)$$

If the "effective refreshing rate" is  $I_{\text{eff}} R_t = \frac{1}{c_{v,n}}$  we get the approximation  $\Omega_{v,n} \approx \Omega^{\text{base}}/2$  so we can get an estimate for  $c_{v,n}$ : An incidence of  $I_{\text{eff}} = \frac{1}{R_t c_{v,n}}$  corresponds to the case when the rate of waning immunity is halved, meaning that every second individual had his immunity refreshed in a given time frame. To find  $c_{v,n}$  we consider the incidence necessary such that, in this given time frame, half of the population was infected. Using that a typical infection lasts  $\mathcal{O}(\frac{1}{\gamma+\delta+\theta})$  days, the incidence at which after a certain amount of time  $T$  half the population was infected is  $I = \frac{M}{2} \frac{1}{\gamma+\delta+\theta} \frac{1}{T}$ . Because every individual on average refreshes the

immunity of  $R_t$  individuals we divide by  $R_t$  and set this equal to  $I_{\text{eff}} = \frac{1}{R_t c}$ . The time frame should be the waning immunity time frame  $T = (\Omega_{v,n}^{\text{base}})^{-1}$ . Thus, we can obtain an estimate for  $c_{v,n}$  as

$$c_{v,n} \approx \frac{2}{M} \frac{(\gamma + \delta + \theta)}{\Omega_{v,n}^{\text{base}}}. \quad (7)$$

## S1.2 Model Equations

The combined contributions of the infection-spreading and vaccination dynamics are represented by the set of equations below. The time evolution of our model is then completely determined by the initial conditions of the system. The first order transition rates between compartments are given by the probability for an individual to undergo this transition divided by the average transition time e.g., the recovery rate  $\gamma$  is the probability that an individual recovers from the disease divided by how the time span of the recovery process. Note that in principle  $\gamma$  should be different for the  $I$  and  $I^B$  compartment, as the probability to recover is larger for breakthrough infections. We neglect this difference as it is negligible within the margin of error since the probability to recover is close to 1 in both cases.

$$\frac{dS}{dt} = - \underbrace{\gamma R_t(H_R) \frac{S}{M} I_{\text{eff}}}_{\text{unvaccinated infections}} - \underbrace{\phi(H_u) M}_{\text{first vaccinations}} \quad (8)$$

$$\frac{dV}{dt} = - \underbrace{(1 - \eta) \gamma R_t(H_R) \frac{V}{M} I_{\text{eff}}}_{\text{breakthrough infections}} + \underbrace{(\phi(H_u) + \varphi(H_w)) M}_{\text{vaccinations}} - \underbrace{\Omega_v V}_{\text{waning vaccine immunity}} \quad (9)$$

$$\frac{dW}{dt} = - \underbrace{\gamma R_t(H_R) \frac{W}{M} I_{\text{eff}}}_{\text{waned infections}} - \underbrace{\varphi(H_w) M}_{\text{booster vaccinations}} + \underbrace{\Omega_v V + \Omega_n R}_{\text{waning immunity}} \quad (10)$$

$$\frac{dE}{dt} = \underbrace{\gamma R_t(H_R) \frac{S}{M} I_{\text{eff}}}_{\text{unvaccinated exposed}} - \underbrace{\rho E}_{\text{end of latency}} \quad (11)$$

$$\frac{dE^B}{dt} = \underbrace{\gamma R_t(H_R) \frac{(1 - \eta) V + W}{M} I_{\text{eff}}}_{\text{vaccinated and waned exposed}} - \underbrace{\rho E^B}_{\text{end of latency}} \quad (12)$$

$$\frac{dI}{dt} = \underbrace{\rho E}_{\text{start of infectiousness}} - \underbrace{(\gamma + \delta + \theta) I}_{\rightarrow \text{ICU, death and recovery}} \quad (13)$$

$$\frac{dI^B}{dt} = \underbrace{\rho E^B}_{\text{start of infectiousness}} - \underbrace{(\gamma + (\delta + \theta)(1 - \kappa)) I^B}_{\rightarrow \text{ICU, death (reduced) and recovery}} \quad (14)$$

$$\frac{d\text{ICU}}{dt} = \underbrace{\delta (I + (1 - \kappa) I^B)}_{\text{transition to ICU}} - \underbrace{(\gamma_{\text{ICU}} + \theta_{\text{ICU}}) \text{ICU}}_{\text{recovery from ICU}} \quad (15)$$

$$\frac{dD}{dt} = \underbrace{\theta (I + (1 - \kappa) I^B)}_{\text{death without ICU}} + \underbrace{\theta_{\text{ICU}} \text{ICU}}_{\text{death in ICU}} \quad (16)$$

$$\frac{dR}{dt} = \underbrace{\gamma (I + I^B)}_{\text{direct recovery}} + \underbrace{\gamma_{\text{ICU}} \text{ICU}}_{\text{recovery from ICU}} - \underbrace{\Omega_n R}_{\text{waning natural immunity}} \quad (17)$$

$$\frac{du^{\text{current}}}{dt} = \underbrace{M\phi(H_u)}_{\text{current first vaccinations}} \quad (18)$$

$$\frac{dw^{\text{current}}}{dt} = \underbrace{M\varphi(H_w)}_{\text{current booster vaccinations}} \quad (19)$$

$$I_{\text{eff}} = \underbrace{(I + \sigma I^B + \Psi)}_{\text{effective incidence}} \quad (20)$$

 Table S1: **Model parameters.** The range column either describes the range of values used in the various scenarios.

Parameter	Meaning	Value (default)	Range	Units	Source
$R_t^{\text{base}}$	Max. Reproduction number (gross)	{2.0, 3.5, 5.0}	0–5	–	[1, 12, 13]
$\eta$	Vaccine eff. against transmission	0.75	0.6–0.85	–	[14–17]
$\kappa_{\text{obs}}$	Observed vaccine eff. against severe disease	0.95	0.75–0.98	–	[17–20]
$\kappa$	Vaccine eff. against severe disease	0.8	–	Eq. 22	
$\sigma$	Relative virulence of vaccinated to unvaccinated individuals	0.5	0.5 – 1	–	[21, 22]
$\tau_u, \tau_w$	Memory time of the ICU capacity and delay to immunization	2, 6	–	weeks	Assumed
$\rho$	Latency rate	0.25	–	day <sup>-1</sup>	[23, 24]
$\gamma$	Recovery rate	0.1	0.088 – 0.1	day <sup>-1</sup>	[25–27]
$\gamma_{\text{ICU}}$	Recovery rate from ICU	0.13	0.08 – 0.2	day <sup>-1</sup>	[1, 28–30]
$\delta$	Av. hospitalization rate $I \rightarrow \text{ICU}$	0.0019	$10^{-5} - 0.007$	day <sup>-1</sup>	[1, 28–30]
$\theta$	Av. death rate	$5.4 \cdot 10^{-4}$	$2 \cdot 10^{-6} - 0.005$	day <sup>-1</sup>	[1, 28–30]
$\theta_{\text{ICU}}$	Av. ICU death rate	0.0975	0.088 – 0.100	day <sup>-1</sup>	[1, 28–30]
$\alpha$	Sensitivity of the population to ICU occupancy	–	–	day <sup>-1</sup>	Estimated
$\Omega_v^{\text{base}}$	Waning imm. rate (base, vaccination)	$\frac{2/3}{360}$	–	day <sup>-1</sup>	[31, 32]
$\Omega_n^{\text{base}}$	Waning imm. rate (base, natural)	$\frac{1}{360}$	–	day <sup>-1</sup>	[33, 34]
$\mu$	Sensitivity to seasonality	0.267	0.141–0.365	–	[11]
$d_\mu$	Day with the strongest effect on seasonality	0	–	day	[11]
$d_0$	Day when the time series starts	240	–	day	[11]
$\phi_0, \varphi_0$	Administration rate (first-time and refreshing doses resp.)	0.0025	–	day <sup>-1</sup>	[1]
$\chi_0, \chi_1$	Fraction of the population refusing vaccine (first and booster resp.).	0.1, 0.2	–	–	[35]
$u_{\text{base}}$	Base acceptance of first dose	0.5	–	–	[36]
$\Psi$	Influx of infections	1	–	People/day	assumed

Table S2: Model variables.

Variable	Meaning	Units	Explanation
$M$	Population size	People	Default value: 1000000
$S$	Susceptible pool	People	Non-infected people that may acquire the virus.
$V$	Vaccinated pool	People	Non-infected, vaccinated people. Less likely to be infected or develop severe symptoms
$W$	Waned immunity pool	People	Non-infected people whose immunity (vaccine-induced or natural) has already waned, thus may acquire the virus.
$E$	Exposed pool	People	People exposed to the virus.
$E^B$	Exposed pool (break-through infection)	People	People exposed to the virus (breakthrough infection).
$I$	Infectious pool	People	Infectious people.
$I^B$	Infectious pool (break-through infection)	People	Infectious people (breakthrough infection).
ICU	Hospitalized (total)	People	Hospitalized people.
$R$	Recovered (total)	People	Recovered people (naturally or after requiring intensive care).
$H$	Av. ICU occupancy	People	Auxiliary variable measuring the average ICU occupancy.
$u^{\text{current}}, w^{\text{current}}$	Vaccinated individuals, independent of the compartment	People	Integral over the vaccination rates
$R_t$	Reproduction number (gross)	—	Eq. 1
$N$	New infections (Total)	cases day <sup>-1</sup>	$N = \gamma R_t (\text{ICU}) \frac{I_{\text{eff}}}{M} (S + W + (1 - \eta) V)$ .
$\kappa$	Effective vaccine efficacy against severe course	cases day <sup>-1</sup>	$\kappa = 1 - \frac{1 - \kappa_{\text{obs}}}{1 - \eta}$
$\Gamma$	Seasonal var. of SARS-CoV-2 infectiousness	—	Eq. 2.
$c_v, c_n$	Inverse incidence at which waning immunity is halved	People	Eq. 7
$\phi(t), \varphi(t)$	Administration rate of first-time and refreshing vaccine doses (resp.)	doses/day	Eq. 23, 26

### S1.2.1 Initial conditions

Our initial conditions are chosen to represent the current situation in different countries as best as possible. Despite different population sizes of the countries we look at, the population size in our model is set to  $M = 10^6$  individuals. Thus, the difference between the countries is solely described by the initial distribution of that population over the model compartments. Let  $x$  be the vector collecting the variables of all different pools:

$$x = [S, V, W, E, E^B, I, I^B, ICU, R, D, u^{\text{current}}, w^{\text{current}}, H] \quad (21)$$

In that way,  $\sum_{i \leq 10} x_i = M$  because  $u^{\text{current}}$  and  $w^{\text{current}}$  are counted independent of their compartment and  $H$  is a measure for past ICU. The base reproduction number  $R_t^{\text{base}}$  is initially set corresponding to one of our three different scenarios and then increased linearly to 5 in March 2022, which corresponds to the natural reproduction number of the virus reduced only slightly by e.g., improved hygiene that is kept in place. To find reasonable estimates for the initial conditions of each compartment we take data for the total number of administered vaccines and total cases on 1. September. To estimate the size of  $W$  we consider the exponential decay of  $V$  and  $R$ . The exponential decay constant  $\Omega_{v,n}^{\text{base}}$  relates to the half life  $T^{1/2}$  of the decaying process via  $T_{v,n}^{1/2} = \frac{\ln 2}{\Omega_{v,n}^{\text{base}}}$ . Using the half life we can estimate how many people vaccinated or recovered until a certain point in time would have moved to the  $W$  compartment. We neglect first order corrections regarding individuals who have been vaccinated as well as infected and were thus counted twice in the data. We calculate the initial conditions for the exposed and infected compartments by first estimating  $E + E^B$  as  $\frac{1}{\rho}$  times the daily new cases and  $I + I^B$  as  $\frac{1}{\gamma + \delta + \theta}$  times the daily new cases. To find the fraction of breakthrough infections among all infected individuals we calculate their fraction as  $\frac{(V+R)(1-\eta)}{S+(V+R)(1-\eta)}$  and build up the compartments  $E, E^B, I$  and  $I^B$  accordingly. An under-reporting factor is multiplied onto  $R$  and to the fraction of  $W$  that comes from  $R$ . It is estimated from seroprevalence studies [37] and the total reported case number, dividing the two numbers.  $u^{\text{current}}$  is the total number of vaccinations administered and  $w^{\text{current}}$  is set to zero. For our base model the initial conditions are listed in Tab.S3.

Table S3: **Initial conditions for Germany.**

$S$	$V$	$W$	$E$	$E^B$	$I$	$I^B$	ICU	$R$	D	$u^{\text{current}}$	$w^{\text{current}}$
317850	548356	39337	731	368	1785	897	13	90663	0	603900	0

### S1.3 Vaccination effects

Our model includes the effect of vaccination, where vaccines are for simplicity administered with a single-dosage delivery scheme. There is some evidence that the vaccines partially prevent the infection with and transmission of the disease [15, 16]. Our model incorporates both the effectiveness against infection and against severe course of the disease following a 'leaky' scheme, i.e., vaccinated individuals have smaller chances to be infected by a factor of  $(1 - \eta)$ , and those with a breakthrough infection or waned immunity have a lower probability of going to ICU by a factor of  $(1 - \kappa)$  than unvaccinated individuals, where  $\kappa$  can be obtained from

$$(1 - \eta)(1 - \kappa) = (1 - \kappa_{\text{obs}}), \quad (22)$$

with  $\kappa_{\text{obs}}$  denoting the full protection from severe disease as observed in studies. Furthermore, we assume breakthrough infections carry a lower viral load and are thus less infectious by a factor of  $\sigma$  [21]. The infection rate depends on the sum of the infected compartment, the breakthrough infected compartment with a lower viral load and an external influx. It can be expressed via the effective incidence  $I_{\text{eff}} = (I + \sigma I^B + \Psi)$ . All parameters and values are listed in Table S1. Note that that these parameters are to be understood as averages across vaccine types.

## S1.4 Vaccine uptake

Vaccine dynamics is an important aspect of our model because it has a strong influence on infection dynamics due to reduced transmissibility. Incorporating willingness to be vaccinated into our model requires to make a decision on how vaccines are administered. Vaccine uptake is described by two different functions, one for susceptible individuals ( $\phi$ ) and one for individuals whose immunity has waned ( $\varphi$ ). The idea is to vaccinate only if willingness for vaccine uptake is larger than the fraction of already vaccinated. For Fig. 1 we use a step-wise approach for this transition, described in S1.4.1. We compare it with a ramping approach described in S1.4.2. A comparison between the outcomes of the two methods is shown in Fig. S2.

### S1.4.1 Step-wise approach

In this approach, we use functions that represent the willingness to be vaccinated in dependence of the ICU occupancy. If the group of individuals who are willing to be vaccinated with a first dose ( $u^{\text{willing}}$ ) is larger than the group of already vaccinated ( $u^{\text{current}}$ ), vaccinations are carried out at a rate proportional to the difference of the two, or at a maximum administration rate  $\phi_0$ , depending on which one is lower:

$$\phi(H_u) = \begin{cases} \min \left\{ \phi_0, u^{\text{willing}}(H_u) - \frac{1}{M} u^{\text{current}} \right\} & \text{if } u^{\text{willing}}(H_u) \geq \frac{1}{M} u^{\text{current}}, \\ 0 & \text{else.} \end{cases} \quad (23)$$

$H$  is a function dependent on the past development of the ICU occupancy as discussed in S1.1.1 . The fraction of people who are willing to be vaccinated for a first time can shift between a minimum and a maximum value ( $u_{\text{base}}$  and  $u_{\text{max}} = 1 - \chi_0$ ), representing the general observed acceptance for the first dose and people who are strictly opposed to vaccines or cannot be immunized because of age or other preconditions respectively. Willingness to be vaccinated depends on perceived danger, which the ICU occupancy is a suitable measure for. The willingness is then represented by

$$u^{\text{willing}} = u_{\text{base}} + (u_{\text{max}} - u_{\text{base}})(1 - \exp(-\alpha_u H_u)) . \quad (24)$$

Willingness to accept booster doses is modeled in a similar way, without a base willingness:

$$w^{\text{willing}} = (1 - \chi_1)(1 - \exp(-\alpha_w H_w)) . \quad (25)$$

If the number of people willing to be vaccinated with a booster dose is larger than the number of people that already received one, vaccinations are carried out from the waned compartment  $W$  to compartment  $V$  at a rate

$$\varphi(H_w) = \begin{cases} \min \left\{ \varphi_0, w^{\text{willing}}(H_w) - \frac{1}{M} w^{\text{current}} \right\} & \text{if } w^{\text{willing}}(H_w) \geq \frac{1}{M} w^{\text{current}}, \\ 0 & \text{else.} \end{cases} \quad (26)$$

### S1.4.2 Ramping approach

In our first step-wise approach vaccinations are only carried out if more people are willing to be vaccinated than there are currently vaccinated. This hard transition might not be realistic because it can be assumed that in a real world scenario the transition would be smooth, leading to vaccinations being carried out over a longer time frame and not abrupt. We can twist our first approach to incorporate this effect by replacing the step function  $\phi(H_u)$  by

$$\phi(H_u) = \begin{cases} 0 & \text{if } u^{\text{willing}}(H_u) \leq \frac{1}{M} u^{\text{current}} - \epsilon \\ \phi_0 & \text{if } u^{\text{willing}}(H_u) \geq \frac{1}{M} u^{\text{current}} + \epsilon \\ \frac{\phi_0}{2\epsilon} \left( u^{\text{willing}}(H_u) - \frac{1}{M} u^{\text{current}} + \epsilon \right) & \text{else.} \end{cases} \quad (27)$$

For booster doses, the replacement  $\phi \leftrightarrow \varphi$  and  $u \leftrightarrow w$  has to be made. This corresponds to a linear increase with a slope dependent on  $\epsilon$ , where vaccinations are started to be carried out when vaccination willingness is larger than the current vaccinated minus  $\epsilon$ . That way,  $\epsilon$  is a measure for the smoothness of the transition between the state where vaccinations are carried out and the state where they are not.

### S1.4.3 Tracking of vaccinated individuals

In our first two approaches, vaccination rates between the susceptible ( $S$ ) and waned ( $W$ ) compartment depend on the difference between willingness for vaccination uptake and the currently vaccinated. Thus, it is necessary to keep track of how many people received a first and booster dose respectively. Implementing this is achieved by integrating over the vaccination rates. It translates into two additional differential equations:

$$\frac{d}{dt} u^{\text{current}} = M\phi(H) \quad \text{and} \quad \frac{d}{dt} w^{\text{current}} = M\varphi(H). \quad (28)$$

The initial conditions for  $u^{\text{current}}$  and  $w^{\text{current}}$  are chosen according to the initial conditions of  $V$  and  $W$ .

### S1.5 Assessment of the sensitivity to ICU occupancy

The opinion dynamics in our model depend on the parameters  $\alpha_R$ ,  $\alpha_u$  and  $\alpha_w$ . To get an estimate for their magnitude we look at estimated incidences that cause a change in the stability of the system due to change in behavior. The effective reproduction number  $R_t^{\text{eff}}$  is defined as the reproduction number times the fraction of the population that is susceptible:

$$R_t^{\text{eff}} \approx \frac{S+W}{M} R_t(H_R) + \frac{V}{M} (1-\eta) R_t(H_R). \quad (29)$$

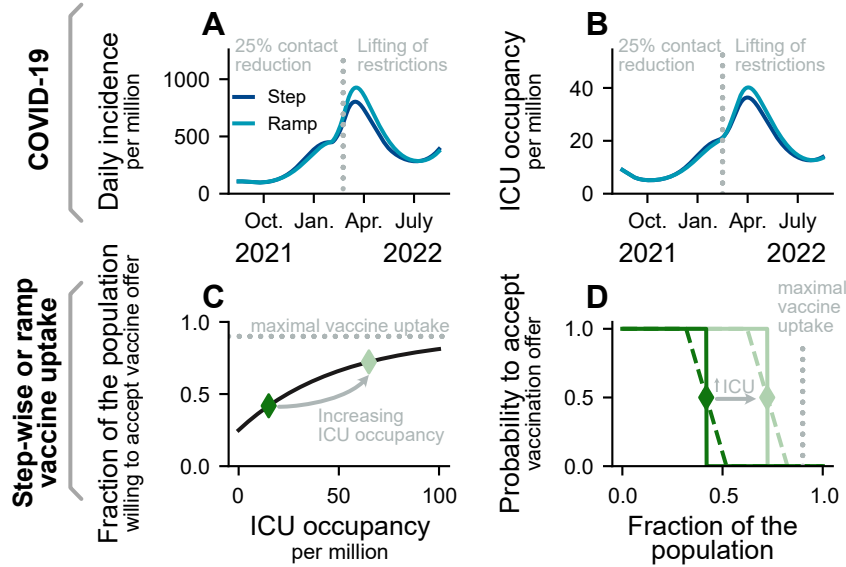
If  $R_t^{\text{eff}} = 1$  the system is at an equilibrium which means that the incidence is constant. Imposing equilibrium conditions at time  $t = t_{\text{eq}}$ , we can obtain an equation ruling the balance between all stabilizing and destabilizing contributions:

$$\exp(-\alpha_R H_{R,\text{eq}}) R_t^{\text{base}} \Gamma(t_{\text{eq}}) \left( \frac{S_{\text{eq}} + W_{\text{eq}}}{M} + \frac{V_{\text{eq}}}{M} (1-\eta) \right) \stackrel{!}{=} 1 \quad (30)$$

Thus, we can estimate  $\alpha_R$  as:

$$\alpha_R \approx \frac{1}{H_{R,\text{eq}}} \ln \left( R_t^{\text{base}} \Gamma(t_{\text{eq}}) \frac{S_{\text{eq}} + W_{\text{eq}} + (1 - \eta)V_{\text{eq}}}{M} \right). \quad (31)$$

Solving for  $\alpha_R$  we need to assume at which ICU occupancy the behavioral changes are strong enough to lead to a tipping of the system. This method is usable to obtain the right orders of magnitude. Setting  $R_t^{\text{base}} = 4$  and  $\Gamma(t_{\text{eq}}) = 1$  corresponds to an equilibrium situation in winter with mild restrictions. Estimating the vulnerable fraction of the population at that point to be a half, we can estimate  $\alpha_R \approx \frac{\ln 2}{H_{R,\text{eq}}}$ . Assuming that under such conditions the behavioral changes only lead to a tipping of the system at full ICUs ( $\approx 70$  per million for Germany [1]) we get the estimate  $\alpha_R \approx 0.1$ . For  $\alpha_u$  and  $\alpha_w$  we assume that the decision to get vaccinated does not require as much self discipline as contact reduction and estimate them as twice and three times larger than  $\alpha_R$  respectively:  $\alpha_u \approx 0.2$ ,  $\alpha_w \approx 0.3$ .



Supplementary Figure S2: **Modeling choices for the opinion-epidemic feedback loop, modulating vaccine uptake and contacts, affects case development in comparable pandemic situations.** **A, B:** Assuming that individuals react protectively to the information they receive from the pandemic, self-regulation of contacts and vaccine uptake could stabilize disease spread. The model approach to this affects disease spread. **C, D:** Assuming that vaccine uptake and willingness are decoupled, we can represent vaccine uptake from an on-off perspective happening when vaccine willingness (partially modulated by ICU occupancy) is higher than the current uptake. In this setting, vaccination can be a step function or a ramp centered where vaccine uptake meets the vaccine willingness.

## S2 Other countries

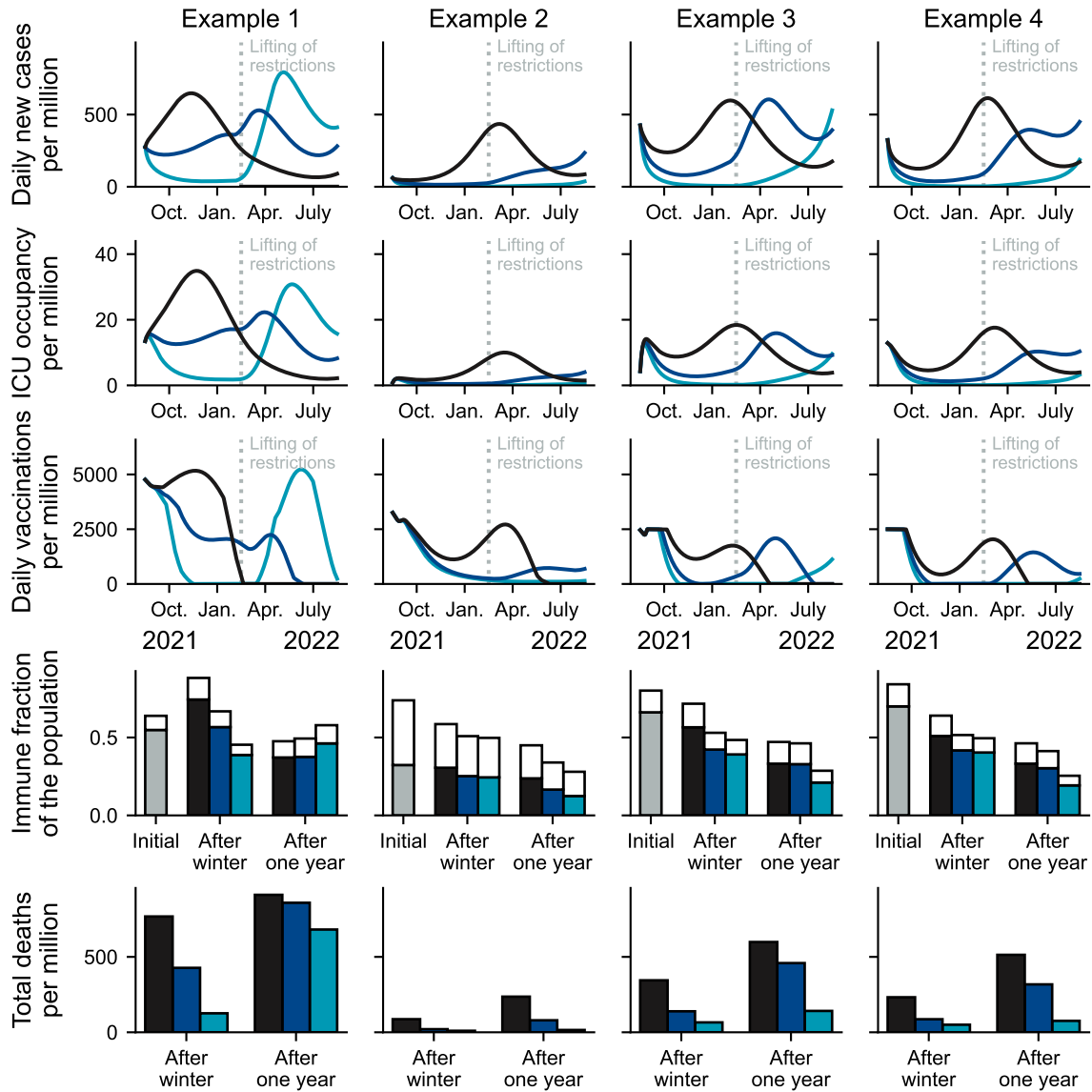
Our results were obtained using initial conditions that reflect the current situation in Germany. By simulating scenarios that are realistic for other countries we can check our model for sensitivity in regards to initial conditions. Of special interest are countries with different vaccination rates and that previously had differing numbers of COVID-19 cases and thus a different structure with regards to the  $S$ ,  $V$ ,  $R$  and  $W$  compartments. For this comparison we chose the countries Denmark, Portugal and the Czech Republic. Initial conditions were calculated with data as described in Sec. S1.2.1 and using data from [38]. Aside from the different initial conditions we incorporated different transition rates  $\delta$ ,  $\theta$  and  $\theta_{\text{ICU}}$  that represent different age structures



within the countries [39]. Using the age dependent rates reported in [1] we assume that the transition rates for each age group is the same among all countries and calculated the overall transition rate as  $\bar{k} = \sum_i w_i k_i$  where  $i$  counts different age groups,  $w_i = \frac{M_i}{M}$  is the fraction of each age group of the total population and  $k_i$  is the transition rate of age group  $i$ . All country specific parameters can be found in Tab. S4. Fig. S3 shows the comparison between the countries.

Table S4: **Country-dependent parameters for Fig. S3.**

Example	Inspiring Country initial conditions	$\frac{V}{M}$ [%] [38]	$\frac{R}{M}$ [%] [37]	Under-reporting factor	$\delta$	$\theta$	$\theta_{ICU}$
1	Germany	54.8	9.6	2.4	0.0019	$5.4 \cdot 10^{-4}$	0.0976
2	Czech Republic	32.3	41.5	3.3	0.0017	$4.1 \cdot 10^{-4}$	0.0979
3	Denmark	66.2	13.9	2.9	0.0017	$4.3 \cdot 10^{-4}$	0.0979
4	Portugal	70.0	14.2	1.7	0.0019	$5.2 \cdot 10^{-4}$	0.0976



Supplementary Figure S3: **Differences between the levels of immunity and their nature will determine the extent of the winter dilemma across countries.** Considering the same scenarios of Fig. 1 in the main text (0, 25% or 50% of restrictions in winter), here we study the effect of different initial conditions in immunity inspired by four countries (from left to right, Germany, Czech Republic, Denmark, and Portugal). We also adapted the probability of requiring intensive care and the infection fatality ratio to capture population demographics in these examples. Across scenarios, we see that countries with higher initial immunity will have less severe waves. However, considering waning immunity and low vaccination rates, their preparedness for 2022 winter wave would be lower. While these results can hold in the short-term, we expect them to be affected by differences in i) the definition of intensive care and the hospital resources, ii) population’s risk perception and sensitivity to ICU occupancy, compliance to NPIs, age-stratified vaccine uptake, and degree of solidarity, and iv) contact structure and intensity. Thus, the above highlights the need for research in this direction.