The Mechanics of Pandemics: Empowering Boltzmann creep for the prediction of COVID-19 fatality trends

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ABSTRACT: Ever since Boltzmann's landmark paper from 1874, integro-differential equations for viscoelasticity have been a cornerstone for reliable quantification of the time-dependent response of solid mechanical systems: their creep strains depend on the *accumulated effect of all load events having taken place before*. This naturally includes rock mechanics, both as regards its narrower meaning of rock creep testing, and its broader sense of creeping tunnel support systems such as shotcrete shells in the context of the New Austrian Tunneling Method. When the COVID-19 pandemic evidenced often sobering performance of traditional epidemiological models [where the system response only depends on the current state of the system, and not on the entire history], the question arose whether the Boltzmann integro-differential equations may be as efficient for "virus loads", as they have proved to be for classical mechanical loads. Evaluating infection and fatality data from more than 100 countries, this question was affirmatively answered. Here we go one step further, and discuss recently obtained results for "*aging* pandemics" in analogy to aging creep experienced in shotcrete systems where the material maturation due to the hydration reaction evolves at a similar pace as the creep strains do. We observe exponentially decaying fatality rates throughout the first thousand days of the COVID-19 pandemic, with a characteristic time of around 180 days in the case of Austria.

Keywords: SARS-CoV-2, COVID-19, concrete creep, viscoelasticity, integro-differential equations, optimization.

1 INTRODUCTION

Ever since the landmark paper of (Boltzmann 1874), integro-differential equations for viscoelasticity have been a cornerstone for reliable quantification of the time-dependent response of solid mechanical systems, with the following basic concept: the creep strains at a particular point in time depend on the *accumulated effect of all load events having taken place until this time point,* i.e. on the entire load *history.* In other words, viscoelastic materials have a "memory". This naturally includes rock mechanics, both as regards its narrower meaning of rock creep testing (Wang et al, 2016), and its broader sense in terms of creeping tunnel support measures, from creep tests on cement paste (Irfan-Ul-Hassan et al, 2016), to shotcrete shells (Scharf et al, 2022; Ullah et al, 2010; Ullah et al, 2012) in the context of the New Austrian Tunneling Method (Rabcewicz, 1965). When the COVID-19 pandemic evidenced often sobering performance of traditional epidemiological models [where the system response only depends on the current state of the system, and not on its entire history (Kermack and McKendrick, 1927; Fanelli & Piazza, 2020)], the question arose whether the Boltzmann integro-differential equations may be as efficient for "virus loads", as they have proved to be for classical mechanical loads. Evaluating infection and fatality data from more than 100

countries, territories, and US states, this question was indeed affirmatively answered (Ukaj et al, 2021, Scheiner et al, 2020). Here we go a step further, and discuss recently obtained results (Ukaj et al, 2023) for "*aging* pandemics" in analogy to aging creep experienced in shotcrete systems where the material maturation due to the hydration reaction evolves at a similar pace as the creep strains do. In this case, it turned out beneficial to formulate Boltzmann's accumulation or superposition principle in rate form (Scheiner and Hellmich, 2009): corresponding creep rate functions associated to a particular maturation degree are again convoluted over the entire load history, but they are only valid for one single point in time, namely then when the aforementioned maturation degree has been attained.

The remainder of the current paper is organized as follows: Section 2 introduces the theoretical framework for an aging fatality model, and Section 3 presents the underlying data recorded over the course of the COVID-19 pandemic. Numerical results are presented in Section 4, while respective conclusions are drawn in Section 5.

2 DEVELOPMENT OF AN AGING FATALITY MODEL

Following up on the proposition made in (Ukaj et al. 2021), we consider the COVID-19-related overall fatality trend as the cumulation of the fatalities developing due to the single, daily infection increments occurring (a few days up to a few weeks) prior to a certain point in time in the overall fatality trend. This notion is fully consistent with Boltzmann's famous "elastic after-effect" principle (Boltzmann 1874), the basis for hereditary mechanics; hence, the subscript "her" will be used to indicate this approach, see, e.g., Eq. (1). Furthermore, we here account for the possibility that parameters governing the corresponding kernel functions, called fatality functions in (Ukaj et al. 2021), may change over time; this change coming as the result of e.g. improved treatment options or virus mutations during the considered epidemic event. From a mechanics of materials viewpoint, such changes in the kernel function relate to the phenomenon of "aging creep", which necessitates modification of the traditional form of Boltzmann's superposition principle. In more detail, following our creep law for aging concrete (Scheiner & Hellmich, 2009), where not one, but a series (time integral) of convolution integrals associated to different aging states, were employed, and where each of these convolution integrals gave access, not to the total creep strains, but to the *creep strain rates*, we formulate an integro-differential equation for the fatality rate at time t, $\dot{F}_{\mathrm{her}} = \mathrm{d}F_{\mathrm{her}}/\mathrm{d}t$, with F_{her} being the number of fatalities predicted by the hereditary mechanics-based model, reading mathematically as

$$\dot{F}_{\mathrm{her}}(t) = \int_{-\infty}^{t} \dot{J}_{\mathrm{f}}(\zeta - \tau)\, \dot{C}(\tau)\, \mathrm{d}\zeta \,, \tag{1}$$

In Eq. (1), $\dot{C}$ is the rate of infection numbers becoming effective at time τ, ζ is the integration (time) variable, and $\dot{J}_{\mathrm{f}}$ is the rate of the fatality function, the analogue to the creep function in classical hereditary mechanics. Clearly, the fatality function needs to be defined such that it aligns with the actual transition from infections into corresponding fatalities. In the following, we consider to that end a very simple option, discussed in more detail in Ukaj et al (2023): a certain fraction of the infections recorded at time τ translates into fatalities after a time delay in step-wise fashion. Hence, the respective rate of such as fatality function reads as

$$\dot{J}_{\mathrm{f}}(\zeta - \tau) = f_{\mathrm{f}}(\zeta)\, \delta(\zeta - \tau - T_{\mathrm{f}}) \,. \tag{2}$$

In Eq. (2), f_{f} denotes the fatality fraction at time ζ, $\delta(\zeta - \tau - T_{\mathrm{f}})$ denotes the Dirac delta function, with $\delta = 0$ if $\zeta \neq \tau + T_{\mathrm{f}}$, and $\delta = \infty$ if $\zeta = \tau + T_{\mathrm{f}}$, and T_{f} denotes the characteristic time of fatal illness; implying that

$$\int_{-\infty}^{\infty} \delta(\zeta - \tau - T_\mathrm{f})\,\mathrm{d}\zeta = 1\,. \tag{3}$$

Hence, considering Eq. (2) in Eq. (1) leads to

$$\dot{F}_\mathrm{her}(t) = f_\mathrm{f}(t)\,\dot{C}(t - T_\mathrm{f})\,. \tag{4}$$

Next, we need to find reasonable approximations for the fatality and infection rates. To that end, we consider

$$\dot{F}_\mathrm{her}(t) = \frac{\Delta F_\mathrm{her}(t)}{\Delta t} \quad \text{and} \quad \dot{C}(t) = \frac{\Delta C(t)}{\Delta t}\,. \tag{5}$$

Typically, $\Delta t = 1$ d, implying that ΔF_her and ΔC are the daily fatality and infection increments. Inserting Eq. (5) in Eq. (4), and integrating the resulting equation with respect to Δt yields

$$\Delta F_\mathrm{her}(t) = f_\mathrm{f}(t)\,\Delta C(t - T_\mathrm{f})\,. \tag{6}$$

Since recorded infections are not available continuously, but at discrete time points (i.e., at each day), Eq. (6) is employed accordingly,

$$\Delta F_\mathrm{her}(t_n) = f_\mathrm{f}(t_n)\,\Delta C(t_n - T_\mathrm{f}), \tag{7}$$

where $t_n = n\,\Delta t, n = 1 \ldots N_t$, N_t being the number of considered points in time, $N_t = 1000$ days, and (as already mentioned above) $\Delta t = 1$ d. At a specific point in time, t_m, the total number of fatalities thus reads as

$$F_\mathrm{her}(t_m) = \sum_{n=1}^{m} \Delta F_\mathrm{her}(t_n) = \sum_{n=1}^{m} f_\mathrm{f}(t_n)\,\Delta C(t_n - T_\mathrm{f})\,. \tag{8}$$

3 CONSIDERED DATA

In this contribution, we focus on the data acquired in Austria for the first 1000 days of the pandemic (i.e., from February 25, 2020 until November 20, 2022), considering as data source the reference website Worldometer (Worldometer 2023). In particular, the following data is needed for model calibration and evaluation: daily infection numbers (i.e., the daily reported coronavirus-positive test results), ΔC, and the daily number of people that deceased due to COVID-19, ΔF. In order to filter out data collection irregularities, the trends of the daily infection and fatality numbers were subject to smoothing (considering for that purpose the 7-day moving average).

4 MODEL APPLICATION AND NUMERICAL RESULTS

The model presented in Section 2 has already been evaluated in quite similar form in (Scheiner et al. 2020 and Ukaj et al. 2021), revealing that for a pandemic model relating recorded infection numbers with the corresponding fatality trends, consideration of the time delay between infections and fatalities is crucial. Notably, in both works, (piecewise) constant model parameters were considered. However, with COVID-19 pandemic developing over a time span over some two to three years, it is easy to see that the effects of COVID-19 have decreased in severity, due to mutations of the virus, treatment options, and the buildup of immunity and resistance due to previous infections and

vaccinations. In this contribution, these effects are accounted for, in terms of varying model parameters.

In particular, the model presented in Section 2 has been evaluated in two different ways. Firstly, we have back-calculated combinations of the two model parameters governing Eqs. (7) and (8), based on which the recorded fatality trend is precisely related, via Eqs. (7) and (8), to the recorded infection trend. To that end, we rearrange Eq. (7) as follows:

$$f_{\mathrm{f}}(t_n) = \frac{\Delta F(t_n)}{\Delta C(t_n - T_{\mathrm{f}})}. \tag{9}$$

Varying the characteristic time of fatal illness, T_{f}, between 0 and 50 days, the corresponding fatality fraction, f_{f}, can be straightforwardly computed, see Figure 1.

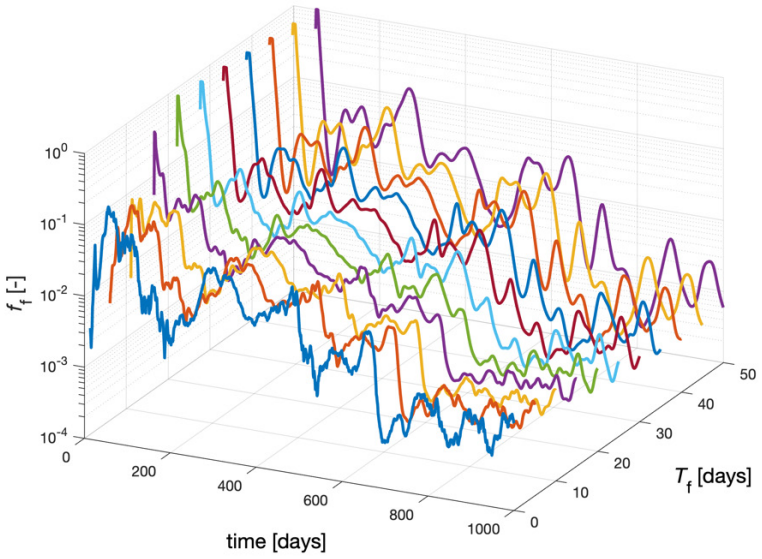

Figure 1. Developments of the fatality fraction f_{f} computed by means of Eq. (9), considering different values of the characteristic time of fatal illness, T_{f}. Note that, for the sake of better readability, physically meaningless values of f_{f} are cut off at 1.

Secondly, we infer from the results of the first study that parameter f_{f} decays over time, and that this decay can be approximated by an exponential function, defined via parameters a and b,

$$f_{\mathrm{f}}(t) = a \exp(-b\, t)\,. \tag{10}$$

Inserting Eq. (10) into Eq. (8) allows for finding the combination of parameters a and b which yields the minimum error between model predictions, F_{her}, and observations, F. Thereby, the computed errors, and consequently the optimal parameter combinations depend on T_{f} as well; hence, it is varied again between 0 and 50. Mathematically, this task can be written as follows:

$$\min(E) = \min\left(\frac{1}{N}\sum_{n=1}^{N_t}|F(t_n) - F_{\mathrm{her}}(t_n)|\right) \rightarrow a_{\mathrm{opt}}(T_f), b_{\mathrm{opt}}(T_f)\,. \tag{11}$$

In Eq. (11), E denotes the average absolute error between the recorded fatalities F and the model-predicted ones, F_{her}, according to Eq. (8), considering for that purpose the aforementioned 1000 first days of the pandemic; hence, $N_t = 1000$ days. Figure 2 shows the numerical results of this optimization task. In particular, Figure 2(a) clearly indicates that the minimum error is obtained when

setting $T_f = 28$ days. Figure 2(b), in turn, is concerned with the question, whether this optimization task yields a unique pair of parameters at all (considering $T_f = 28$ days). The unambiguous peak of E^{-1} confirms the true "optimality" of the parameter pair $a_{opt} = 0.12186$ and $b_{opt} = 0.0055901$ d^{-1} (with the related error amounting to 439.9245). Finally, Figure 3 shows the satisfying agreement between F and F_{her}, and corroborates that a simple exponential function is able to describe the development of the parameters governing a pandemic model reasonably well.

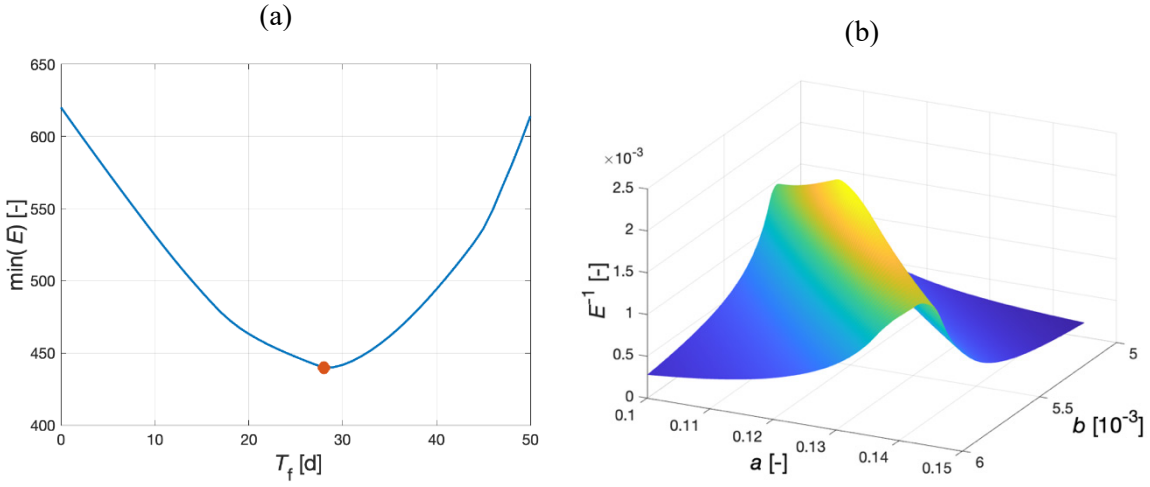

Figure 2. (a) Minimum error obtained through evaluation of Eq. (11) for T_f ranging from 0 to 50, with the minimum error indicated at $T_f = 28$ days; (b) The inverse of the mean error, according to Eq. (11), for the considered ranges of parameters a and b, with $a_{opt} = 0.1286$ and $b_{opt} = 0.0055901$ d^{-1} turning out as optimal parameters.

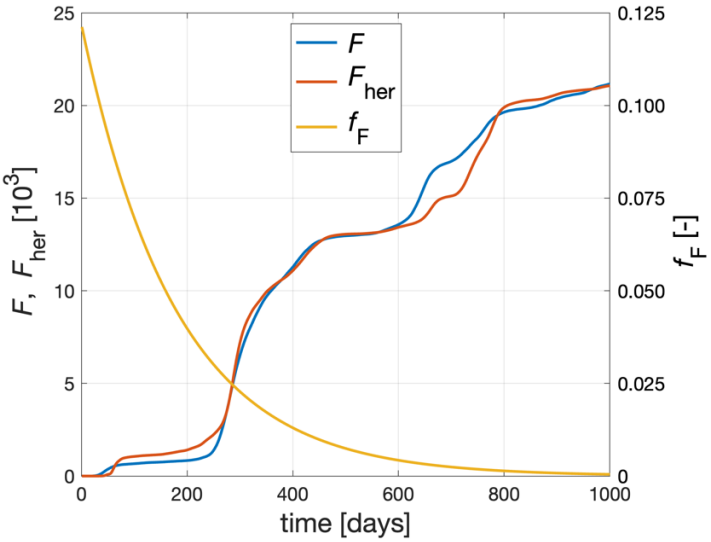

Figure 3. Comparison of recorded and model-predicted fatalities, together with the corresponding trend of the fatality fraction f_F.

5 SUMMARY AND CONCLUSIONS

This contribution presented an aging pandemic model, inspired by Boltzmann's famous superposition principle, relating recorded infection trends to the corresponding fatality trends. A numerical study based on the COVID-19 data recorded in Austria corroborates the soundness of this

approach, which may motivate research programs in the field of coupled integro-differential equations as a new avenue in mathematical biology, thereby overcoming limitations of the currently overwhelmingly popular SIR-models in state-of-the-art epidemiology (Fanelli & Piazza 2020; Kermack & McKendrick 1927). As a first step in this direction, aging pandemic modeling can be expanded from Austria to a global scale – similar to our hereditary epidemiology investigations reported in (Ukaj et al. 2021) for the non-aging case. Furthermore, nature and format of the fatality rate function deserves further scrutiny. Such activities are currently going on (Ukaj et al, 2023).

In conclusion, this study shows that old engineering fields, such as geomechanics or concrete engineering, and their proven theoretical concepts, such as the superposition principle or aging, may provide unexpected blueprints for new modeling endeavors driven by comparably young fields, such as computational epidemiology. Hence, in order to frame the insights gained by the present study in the most general way, reinventing the wheel may not be necessary to address the pending questions of our time, and maintaining an inter- and multidisciplinary perspective indeed promises routes to smart and swift solutions of intricate problems, which otherwise would not be accessible.

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