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Probabilistic Mechanics of Glass Suggests a Model for COVID-19 Epidemics

[1]Gabriele Pisano, [1]Antonio Bonati and [1,2]Gianni Royer Carfagni

[1]Institute for Construction Technologies, National Research Council, Italy
[2]Department of Engineering and Architecture, University of Parma, Italy

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Corresponding Author:
Gabriele Pisano
Institute for Construction Technologies, National Research Council, Italy
Email: pisano@itc.cnr.it

Abstract: The presented statistical model allows us to identify the degree of development of an epidemic starting from the observation of the number of deaths in the invested region, categorized by the age of the victims. Recognition of which cases are associated with the disease is not necessary, as this results from the comparison with data on deaths in pre-epidemic conditions. The treatment, which has analogies with consolidated models in the stochastic mechanics of brittle materials, allows associating parameters such as the level of epidemic, the probability of developing a pathology, and the age of the victims, with other notions well-known to structural engineers, such as the stress, the dependence from fracture-mechanics of macroscopic strength on material defects, the size-effect. Strong simplifying hypotheses are made; therefore, new comparisons are needed with the actual data, often not organized and difficult to find. However, for the most populated Italian regions, the model shows a good agreement with the theoretical predictions, allowing a quantitative estimate of the epidemic level and the risk assessment based on age. Further studies will show whether the knowledge in material science may be conveniently borrowed in the field of epidemiology and vice-versa, to achieve mutual interdisciplinary progress.

Keywords: Probabilistic Mechanics, Glass, COVID-19, Mathematical Epidemiology, Weibull Statistics

Introduction

The purpose of this article is to show how well-studied models in mechanics, in particular in the stochastic fracture mechanics of brittle materials such as glass, can be extended to a very different area of speculation to define, on a statistical basis, the effects of epidemics. The comparison with the mechanical model, governed by similar equations, will allow the establishment of an intuitive correspondence between concepts well known to engineers, such as the stress state in a body, with other quantities, like the level of the epidemic. Of course, this represents just a first attempt, because further studies are certainly needed to demonstrate whether the models in the field of materials science may be conveniently borrowed, *mutatis mutandis*, in the field of epidemiology and viceversa, thereby obtaining mutual interdisciplinary progress.

Mathematical models have been used for almost a hundred years in the broad field of epidemiology. The most famous is certainly the SIR model, first formulated by Kermack and McKendrick (1927). Over the years, the basic concepts of the SIR model have been developed, with additions and changes, in more complex approaches (Beretta and Takeuchi, 1995; Shulgin *et al*., 1998; Bjørnstad *et al*., 2002; McCluskey, 2010). Whereas the SIR model and all its derivations define mathematical laws that describe how the virus is transmitted from an infected person to a healthy one, our approach is different. It starts from the manipulation of the number of total deaths, sorted by age, during a given period of observation in a certain territory, and through the comparison with the corresponding data in the same territory in pre-epidemic conditions, it provides a measure of the effects of the epidemic. The definition of an index of the epidemic, variable with time, could allow developing, *a posteriori*, a kinetic theory of the epidemic on a statistical basis, taking into account also the effects of the adopted countermeasures (locking down of the territory) and/or the availability of more effective cures, including a vaccine.

A statistical theory for the strength of epidemics based on this rationale was presented by Pisano and Royer Carfagni (2020) and applied to interpret the development of the first wave of the spread of COVID-19 outbreaks in Italy in 2020. Although the theory is very general, this case was

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reputed of interest because Italy was the first country in Europe to be heavily affected by the contagion, at a time when very little was known about it and, consequently, effective countermeasures could not be taken immediately. The proposed theory made it possible to follow the free circulation of the epidemic in the national territory during the unconstrained phase, categorizing and quantifying the subsequent modification of the scenarios, as an effect of the lockdown, the improvement of medical treatments, and the increase in availability in the intensive care units. Our purpose, here, is to show and discuss how this theory has a surprising interpretation that comes, in particular, from the probabilistic approach that is used to define, on a statistical basis, the strength of glass for structural applications. Hence, this article is aimed primarily not at epidemiologists but at engineers. It can be considered just as a curiosity, or a divertissement, but we prefer to think that it can stimulate new ideas that arise from multidisciplinarity and the transfer of knowledge.

Indeed, the followed procedure is inspired by stochastic mechanics in material science. In practice, albeit tentatively, we try to translate to another field of speculation basic concepts such as the macroscopic resistance of the material and its micro-mechanical interpretation, dictated by the probability that the body contains a crack that is critical for the applied state of stress, according to the laws of fracture mechanics. We have tried to grossly translate these concepts in the medical field so that the applied stress becomes the level of epidemic, the defect is now the pathology that can be developed in the individual, and the laws of fracture mechanics are the criterion that associates with every level of the epidemic the critical pathology that can lead to death. The life of the individual, appropriately re-scaled to take into account factors such as the genetics of the population, the quality of life and environment, the efficiency of the health system, and the gender, are associated, in the mechanical interpretation, with the dimensions of the body being studied. The larger the body, the greater the possibility of finding cracks of size critical for the applied stress; likewise, the longer the life, the greater the probability of developing pathologies critical for the level of the epidemic. This is a phenomenon usually referred to as the size effect in the language of material science.

To be able to deal with the question mathematically, we start from hypotheses that, although moving away from reality, give a qualitative view, hopefully sufficiently approximated. The representation will certainly be coarse at this time, but if it is simple, it will be possible to conveniently apply the calculation and to check quantitatively, or at least qualitatively, if the results obtained correspond to the statistical data and, therefore, to verify the validity of the starting hypotheses, paving the way to new results. Our approach has been somehow inspired by the rationale used to simplify a complex scenario in the pioneering studies by Vito Volterra in mathematical biology. Volterra first analyzed the special cases of only two species that contend for the same nutrition (Volterra, 1926) and, successively, other two species, one of which grows because it finds unlimited nourishment, while the second would become extinct due to lack of nourishment, but still lives at the expense of the former one (Volterra, 1962). Here, the basic concepts of fracture and stochastic mechanics, and in particular the "weakest-link-in-the-chain" rationale, are applied concerning the very special case in which only one parameter represents the level of the epidemic and another parameter denotes the level of pathology developable by individuals. This is done on a statistical basis, to define another quantity that can measure the effects of the epidemic. A generalization of such a concept will be certainly possible, with potential extensions to other branches of medicine. The effects of an epidemic are often measured by counting the number of deaths (Housworth and Langmuir, 1974; De Brito *et al*., 2017; Mavalankar *et al*., 2008), but if we let ourselves be guided by the methods of the mechanics of materials, we are led much further than where qualitative reasoning and empirical laws, based on data manipulation, could bring us, thanks to the formulation of precise equations and the definition of a single index, which accounts for both the spread of the epidemic and the capability of treating the consequent diseases.

Our findings do not contradict the results of the observations; indeed, they seem in perfect agreement with the results obtained by processing the statistical data relating to the deaths that were recorded during the COVID-19 waves that occurred in Italy in 2020. Concerning the data manipulated (Pisano and Royer Carfagni, 2020), here the analysis is extended to the second wave concerning the most populated regions of Italy, regions that are very different in terms of expectancy of life and level of the epidemic.

Probabilistic Mechanics of Glass

The model here described is inspired by the stochastic mechanics of brittle materials, based on general balance laws dictated by classical fracture mechanics. In particular, it is based on the "weakest-link-in-the-chain" rationale, proposed by Weibull (1939) to statistically interpret the variability in the population of macroscopic strengths observed in brittle materials like glass and ceramics.

The original Weibull theory did not provide a formal correlation between the statistical theory of the strength of materials and the balance laws specific to fracture mechanics, expressed by a specific fracture criterion. Freudenthal (1968) inferred the connection between the depths of an existing flaw in the body, schematized as micro-cracks, and the statistical distribution of macroscopic strengths; however, the relationship between the orientation of micro-cracks and applied stress through a fracture criterion was not explicitly considered. This was

done later on by Batdorf and Crose (1974). According to this model, the effect of the distribution of the sizes of the existing flaws is assumed in terms of a critical component of stress normal to the crack axis, and the probability that the orientation of the crack is such that the fracture criterion is satisfied is explicitly taken into account. Another model was formulated by Evans (1978), according to which an elemental strength distribution is established from test data. Then, through considerations about the crack orientations, the shear, and the normal stresses acting at the crack, the elemental distribution is correlated through a fracture criterion to different stress states.

Starting from the works by Freudenthal (1968) and Batdorf and Crose (1974), a micro-macro approach has been recently proposed by two of the authors (Pisano and Royer Carfagni, 2017), to associate the failure stresses with the distribution of crack sizes. Differently from the model by Batdorf and Crose, the construction of the statistical model for glass strength does not start from the distribution of cracks in terms of critical stresses, but in terms of depths: This allows for correlating the variations in the distribution of the macroscopic strengths with the crack scenarios.

The Micro-Macro Approach

The macroscopic strength of glass elements is governed by the presence of micro-defects laying on their surfaces. Such defects can be modeled as thumbnail micro-cracks, whose plane is orthogonal to the glass surface. To statistically represent the population of defects, following a consolidated theory (Freudenthal, 1968), one can consider (Pisano and Royer Carfagni, 2017) that the area A of the glass surface is ideally divided into Representative Area Elements (RAE), referred to as ΔA. The size of the RAE is compatible with the typical size of the cracks, in the sense that, while observing the actual defects commonly present on the surface of commercial glass, ΔA represents the element of the coarser mesh that can be drawn on the surface, in such a way that at most one crack is included in each element. Inspecting the glass surface with a microscope, it is possible, at least in principle, to measure in each RAE the size δ of the micro-crack there located. One can thus calculate the corresponding statistics, i.e., the probability of finding in one ΔA a crack of size δ. We assume that the RAE is much smaller than the area of the glass element ($\Delta A << A$) but, at the same time, it is much larger than the size of the crack it hosts ($\Delta A >> \delta$), so the stress concentration produced by the crack of one RAE has a negligible effect on the cracks belonging to the neighboring RAEs. In this respect, the effects of the stress on each crack are similar to those that would occur if the crack were in an infinite medium.

Glass breakage occurs when one dominant crack grows unboundedly in the stressed body: The larger the crack size δ, the lower the level of the action necessary to produce failure. In the theory of epidemiology, as it will be explained in the section "The Weibull theory in epidemiology", the parameter δ plays the role of the level of severity of the existing pathology developed by individuals, which is associated with the possibility of leading to death.

It is logical to assume that while the bulk of the distribution occurs for cracks of fairly small size, there is a small number of cracks of size much higher than the average value, which leads to a very long right-hand-side tail. This can be interpreted by a power law distribution of the Pareto type, i.e.:

$$p_{\Delta A}(\delta) = C\delta^{-\alpha} \tag{1}$$

where C is a normalization constant and $\alpha > 1$ is the scaling parameter. In general, the constant C may depend upon the time interval Δt ($C_{\Delta t} = f(\Delta t)$) during which the stress is applied, because of a subtle phenomenon usually referred to as subcritical crack propagation or static fatigue (Wiederhorn and Bolz, 1970), according to which cracks can slowly grow omothetically over time in a way proportional to the applied stress.

Since the Pareto distribution diverges for $\delta \to 0$, the normalization constant $C_{\Delta t}$ is found by imposing that there is a minimum crack size $\delta_{min,\Delta t}$, possibly dependent on Δt, representing the size of physiological defects in glass, naturally present from industrial manufacturing. Consequently, $C_{\Delta t}$ assumes the form:

$$\int_{\delta_{\min,\Delta t}}^{\infty} p_{\Delta A}(\delta)\,d\delta = \int_{\delta_{\min,\Delta t}}^{\infty} C_{\Delta t}\delta^{-\alpha}\,d\delta = 1 \Rightarrow C_{\Delta t} = \frac{\alpha-1}{\left(\delta_{\min,\Delta t}\right)^{-\alpha+1}} \tag{2}$$

Thus, the probability density function becomes:

$$p_{\Delta A}(\delta) = \frac{\alpha-1}{\delta_{\min,\Delta t}}\left(\frac{\delta}{\delta_{\min,\Delta t}}\right)^{-\alpha} \tag{3}$$

from which the cumulative probability function is derived in the form:

$$P_{\Delta A}^{\geq}(\delta) = \Delta A\left(\frac{\delta}{\eta}\right)^{1-\alpha}, \text{with}\,\eta = \frac{\delta_{\min,\Delta t}}{(\Delta A)^{1/(\alpha-1)}} \tag{4}$$

It is interesting to remark that the strength of glass is governed by large cracks so what is important is the right-hand-side tail of the distribution. Consequently, $\delta_{min,\Delta t}$ may be considered as a material parameter, not necessarily associated directly with the minimum (physiological) size of the flaw hosted in the RAE, whose importance consists in the fact that, in the expression (4), it analytically characterizes the statistics of large cracks in the asymptotic limit $\delta \to \infty$.

The probability of finding in the RAE a crack $\leq \delta$ is equal to $P_{\Delta A}^{\leq}(\delta) = 1 - P_{\Delta A}^{\geq}(\delta)$. Since no crack larger than δ is found in the whole area A if and only if none of the RAEs ΔA

contains a crack greater than δ, the probability $P_A^{\leq}(\delta)$ of finding a crack $\leq \delta$ in A is equal to the product of the probabilities that the crack size is $\leq \delta$ in all the RAEs that form A, whose number is $A/\Delta A$. By taking the limit for $\Delta A/A \rightarrow 0$ and recalling that the probability $P_A^{\leq}(\delta)$ of finding a crack $\geq \delta$ is $P_A^{\geq}(\delta) = 1 - P_A^{\leq}(\delta)$, one finally obtains:

$$P_{\Delta A}^{\geq}(\delta) = 1 - \exp\left[-A\left(\frac{\delta}{\eta}\right)^{1-\alpha}\right] \tag{5}$$

This is the classical two-parameter Weibull distribution (Weibull, 1939). The area A of glass enters in this expression and provides a phenomenon referred to as size-effect in the mechanics of materials: the higher the size A of the glass element, the higher the probability of finding a crack larger than δ. As it will be discussed later in the section "The Weibull theory in epidemiology", the counterpart of the size of the material body in the Weibull theory of epidemiology is the nominal life of the individual. The longer the nominal life, the higher the probability of developing a severe pathology.

Under bending, the tensile stress varies linearly in the thickness of the plate, but since the crack depth δ is in general much smaller than the thickness, we can repute that the opening stress remains almost constant in the whole crack. It is usually assumed that cracks in brittle materials open in mode I. According to Linear Elastic Fracture Mechanics (LEFM), the crack opens when the critical value K_{Ic}, which is characteristic of the material, is attained by the Stress Intensity Factor (SIF) $K_I = \sigma_{\perp} Y \delta^{1/2}$ in mode I, where $\sigma_{\perp}$ is the component of stress at the right angle with the crack plane and Y is the geometric factor that takes into account the crack shape. This is equal to $Y = 2.24/\pi$ for a semicircular thumbnail crack of radius δ. Hence, collapse occurs when:

$$K_{Ic} = Y \sigma_{\perp} \delta^{1/2} \tag{6}$$

This expression is identical, apart from the exponent of δ, to the death criterion that will be adopted in the section "The Weibull theory in epidemiology" for the theory of epidemiology.

Assume now that the glass surface A is stressed by a uniform equibiaxial state of stress (σ, σ). Such a condition is approximately verified when the glass plate is tested in the co-axial double-ring apparatus (Pisano and Royer Carfagni, 2016) and it represents the most severe condition, since only in this case, the probability that the maximum principal stress is at a right angle with the critical crack is 100%. Thus, in this condition $\sigma_{\perp} = \sigma$. Hence, for any given σ, the probability of failure equals the probability of finding a crack of size greater than $\delta = (K_{Ic}/(Y\sigma))^2$. Then, substituting Eq. (6) into (5) one obtains:

$$P_A^F(\sigma) = 1 - \exp\left[-A\left(\frac{\sigma}{\eta_0}\right)^m\right], \text{with } m = 2(\alpha - 1), \text{and } \eta_0 = \frac{K_{Ic}}{\eta^{1/2} Y} \tag{7}$$

where m and η_0 are the shape and scale parameters, respectively. This is again a two-parameter Weibull distribution.

Remarkably, since glass failure occurs when just one crack reaches its critical condition, the RAEs play the role of the rings of the chain: the weakest ring is the one that breaks in a chain under tension (Weibull, 1951) and breakage of one ring provokes failure of the whole chain under tension. Hence, following the weakest-link-in-the-chain rationale, kept fixed the size of the rings, the longer the chain, the higher the probability of finding a weak link (size effect).

The Weibull Theory in Epidemiology

A statistical model is now presented that, starting from the definition of nominal life, assesses the probability of finding a pathology of assigned level in the lifetime. Death is correlated with the development of a pathology that is critical for the level of the epidemic. An intuitive interpretation of the model can be obtained in the language of fracture mechanics since it is governed by the same equations that interpret the strength of brittle materials on a statistical base.

Real and Nominal Life and Probability of Developing a Pathology

The life of a person may be considered as a chain composed of lifetime segments, representative of the reference lifetime scale. One may think, for example, that each solar year, or any submultiple of it, represents a lifetime segment, i.e., one of the rings that add up to one another during a lifetime. However, this view may be too simplistic.

Alvin Toffler (1928-2016), an American writer, once said: "It is mathematically demonstrable that the concept of time is closely related to age: time passes faster for old people". This quote introduces the idea that the usual time unit, for example, the solar year, cannot represent the reference scale to measure the aging process throughout the whole human life. In other words, if one thinks of a linear process, where life is the mere sum of nominal life segments, each of which can be associated with the same physiological increase of "damage" in the human body, then the number of segments contained in one solar year of age should be higher for an old person than for a young person.

Let ΔA_n denote the reference nominal life segment. The real age A_r, expressed in several real time-segments ΔA_r, can be related to nominal age A_n, equal to the number of ΔA_n. In general terms, this is done through a re-scaling law of the type:

$$A_n = F(A_r) \tag{8}$$

The analysis of the data corresponding to the Italian COVID-19 epidemics has suggested, as will be demonstrated in the following, to consider the scaling law described in the following.

Setting $\Delta A_r = 1$ *solar year*, the nominal life A_n is expressed in several nominal years ΔA_n according to a function $F(A_r)$ of the form:

$$A_n = A_r + \left\langle A_r - 45 \right\rangle_+^{\gamma_1} + \left\langle A_r - 70 \right\rangle_+^{\gamma_2} \tag{9}$$

with $\gamma_1, \gamma_2 > 0$, where A_r is the real age now expressed in solar years and $\langle\cdot\rangle_+$ denotes the positive part function, which takes as input any real number and outputs the same number if it is nonnegative and 0 if it is negative. This means that there are two step-changes in life, the first one at 45 solar years and the second one at 70 solar years of age, beyond which, roughly speaking, "each year counts more". In general, the re-scaling law $F(A_r)$ depends upon the race of the population, the genetic heritage, the quality of environment and life, and the efficiency of the national health system and may be different between men and women.

Elizabeth Barrett Browning (1806-1861), an English poetess of the Victorian era, once said: "Parity of years, a woman is always younger than a man". We may re-interpret this phrase by saying that, parity of solar years of age, the nominal age for a woman is lower than for a man. However, we will not make such a distinction here and we will assume that such a rescaling law holds, on average, in the considered ensemble, represented by the population of an Italian region or province.

Let us suppose that the level of pathology that a person can develop can be synthetically measured by only one parameter δ. Observe that, in this simple version of the model, pathologies are not classified by typology and δ represents a general measure of the level of any pathology that can affect people. The model could be easily extended to consider different-in-type pathologies by assigning different Pareto functions to different types of pathologies. We assume that the probability to develop δ, in a given time interval Δt of observation, depends upon the number of reference life-segment $A_n/\Delta A_n$ and that serious pathologies (high δ) are less probable to be achieved than mild pathologies (low δ). One assumes that a universal statistical law *`a la* Pareto does exist, of the type:

$$p_{\Delta A_n}(\delta) = C_{\Delta t}\delta^{-\alpha}, with\, \alpha > 1 \tag{10}$$

which is the counterpart of Eq. (1) and represents the probability density function to develop the pathology of level δ in the time interval Δt, concerning the single life-segment ΔA_n. Following this rationale, ΔA_n represents a normalization factor for the natural human degradation due to age. This expression should have a universal value, valid for every person, whereas the distinction in terms of race, genetics, quality of environment and life, and gender, has been already taken into account through the re-scaling (8). Recall that the Pareto distribution is heavy-tailed. Hence, it respects the desired qualitative property, i.e., the probability decreases with the severity of the pathology δ.

The normalization constant $C_{\Delta t}$ is given by Eq. (2). Now, $\delta_{min,\Delta t}$ represents the physiological pathology level that is always achieved in the nominal life-segment, irrespective of any other factors, possibly dependent upon the duration of the observation Δt. This may represent the physiological minimal aging for the human body since no person can have an infinite life, in which no disease is developed.

By following the same rationale adopted in the section "Probabilistic mechanics of glass", one obtains the probability of developing a pathology greater or equal then δ in ΔA_n in the form:

$$P_{\Delta A_n}^{\geq}(\delta) = \left(\frac{\delta}{\delta_{\min,\Delta t}}\right)^{1-\alpha} = \Delta A_n \left(\frac{\delta/\delta_{\min,\Delta t}}{\eta}\right)^{1-\alpha} \tag{11}$$

where:

$$\eta = \frac{1}{\left(\Delta A_n\right)^{1/(\alpha-1)}} \tag{12}$$

Observe that $\delta_{min,\Delta t}$ carries the dependence upon the time of observation Δt, but this is not emphasized in the sequel, to simplify the notation.

The probability of finding a pathology of a level lower than δ is $P_{\Delta An}^{\leq}(\delta) = 1 - P_{\Delta An}^{\geq}(\delta)$. In the whole nominal life A_n, composed by $A_n/\Delta A_n$ nominal reference segments, the probability of finding a pathology of a level lower than δ coincides with the product of the probabilities of finding a level lower than δ in any one of the constituent ΔA_n. Therefore, one can write:

$$P_{\Delta A_n}^{\leq}(\delta) = \left[1 - \Delta A_n \left(\frac{\delta/\delta_{\min,\Delta t}}{\eta}\right)^{1-\alpha}\right]^{A_n/\Delta A_n} = \left[1 - A_n \frac{\Delta A_n}{A_n}\left(\frac{\delta/\delta_{\min}}{\eta}\right)^{1-\alpha}\right]^{A_n/\Delta A_n} \tag{13}$$

Such an expression can be simplified under the hypothesis that the ratio between ΔA_n and A_n is much lower than one. Assuming that such ratio tends to zero and observing that $\lim_{U'\to 0}\left[1 + aU'\right]^{1/U'} = \exp[a]$, one obtains:

$$P_{A_n}^{\geq}(\delta) = 1 - P_{A_n}^{\leq}(\delta) = 1 - \exp\left[-A_n\left(\frac{\delta/\delta_{\min}}{\eta}\right)^{1-\alpha}\right] \tag{14}$$

This is a two-parameter Weibull distribution (Weibull, 1951) carrying a size effect in terms of nominal age A_n, whose significance will be discussed through the mechanical interpretation, set forth in the section "Analogies with the statistical model for brittle materials' strength".

Probability of Death as a Function of Age and Level of Epidemic

Another assumption of paramount importance is that the "force" of the epidemic can be measured by only one parameter σ. This is again a great simplification, but it is not difficult to consider the contemporary action of more than one type of epidemic, by introducing other parameters and/or the uneven distribution of the epidemic in the territory, by considering small sub regions where it is presumably constants and then summing the effects. In any case, reference to one uniform level of the epidemic is useful to illustrate the method.

Death occurs when a function representing the combination of the level of epidemic and the level of pathology reaches a critical value *of* K_{cr}. A high level of epidemic provokes death at a low level of pathology and vice-versa. We conjecture that there is a death criterion represented by a function G (·,·,·), such that death occurs when:

$$\mathcal{G}\left(\delta,\sigma,A_n\right)-K_{cr}=0 \tag{15}$$

This is the most general case, where we consider a further dependence upon A_n, to take into account that the effect of an epidemic might be, e.g., more aggressive on the elderly than on young people. Given the nominal age A_n, this criterion associates any level of epidemic σ with the corresponding critical level of pathology δ/δ_{min}, i.e., the pathology beyond which death occurs, for a given level of the epidemic. Such a criterion implicitly defines the function:

$$\delta/\delta_{\min}=g\left(\sigma,A_n\right)\ such\ that\ \mathcal{G}\left(g\left(\sigma,A_n\right),\sigma,A_n\right)-K_{cr}=0 \tag{16}$$

In general, it is logical that for any nominal age A_n, the function $g(A_n)$ is *monotone decreasing*, because the higher the level of epidemic, the lower the corresponding critical pathology. Moreover, requiring that when $\sigma \to 0$ it takes a pathology $(\delta/\delta_{min})_{cr} \to \infty$ to cause death, one has the further condition:

$$\lim_{\sigma\to 0}\frac{1}{g\left(\sigma,A_n\right)}=0 \tag{17}$$

Because of the aforementioned properties of $g(A_n)$, given the level of epidemic σ, at the nominal age A_n, the probability of death coincides with a probability of developing a pathology whose level is higher than or equal to $\delta/\delta_{min} = g(\sigma, A_n)$. Substituting in (14), one obtains:

$$P_{A_n}^{D}\left(\sigma\right)=P_{A_n}^{\geq}\left(g\left(\sigma,A_n\right)\right)=1-\exp\left[-A_n\left(\frac{\eta}{g\left(\sigma,A_n\right)}\right)^{\alpha-1}\right] \tag{18}$$

In the simplest case, as it will be done in the section "the case of the COVID-19 epidemics in Italy", one can assume that $g(\sigma, A_n)$ does not depend on A_n and that the monotonicity property, together with the limit (17), can be interpreted by a power law function. Therefore, the criterion (15) can be re-written in the form:

$$K_{cr}-Y\sigma\left(\frac{\delta}{\delta_{\min}}\right)^{1/\beta}=0,\Rightarrow\frac{\delta}{\delta_{\min}}=\left(\frac{K_{cr}}{Y\sigma}\right)^{\beta} \tag{19}$$

with $\beta > 0$. Observe that, all other things being equal, the higher is β, the higher the critical pathology for the same level of epidemic σ. Therefore, the parameter β represents our capability of treating the disease caused by the epidemic, which may change during the critical phase thanks to the availability of more and more effective treatments. With no changes in the ability at curing the disease (δ) and in the level of the epidemic (σ), the effect of the epidemic increases with Y, which can represent the spread of the epidemic. Here, it is important to outline that, since $\delta/\delta_{min} \geq 1$, the product $(Y\sigma)/K_{cr} < 1$.

Hence, (18) can be re-written in the simplified form:

$$P_{A_n}^{D}\left(\sigma\right)=1-\exp\left[-A_n\left(\frac{\sigma}{K_{cr}/Y\eta^{1/\beta}}\right)^{(\alpha-1)\beta}\right]=1-\exp\left[-A_n\left(\frac{\sigma}{\eta_0}\right)^{m}\right] \tag{20}$$

where we have defined $m = (\alpha-1)\beta$ and $\eta_0 = K_{cr}/(Y\eta^{1/\beta})$. Again, η_0 carries the dependence upon the time of observation Δt through η, as per (12).

The probability of death can be expressed in terms of real-life A_r by using the re-scaling law (8). Substituting in (20), one obtains:

$$P_{A_n}^{D}\left(\sigma\right)=1-\exp\left[-F\left(A_r\right)\left(\frac{\sigma}{\eta_0}\right)^{m}\right] \tag{21}$$

Remarkably, both (18) and (20) again represent two-parameter Weibull distributions (Weibull, 1951), for which m and η_0 represent the scale and the shape parameters, respectively. Statistical laws of this type are commonly encountered in materials science.

The present approach could also cover the case in which the level of epidemic σ varies within the observed territory, or the epidemic is more lethal in a restricted category of population, but this is not done here.

Analogies with the Statistical Model for Brittle Materials' Strength

There are remarkable affinities, schematically shown in Fig. 1, between the model just presented and the statistical models used to describe, starting from a micro-mechanical motivation, the population of macroscopic strengths in the glass.

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First, glass breakage occurs when one dominant crack grows unboundedly in the stressed body: The larger the crack size δ, the higher the attitude of the body to break under applied loads. Likewise, in the epidemiological model, the parameter δ plays the role of the level of severity of the pathology developed by individuals, which is naturally associated with the possibility of leading to death. It is logical to assume that while the bulk of the distribution occurs for fairly small sizes, there is a small number of cracks of size much higher than the average value, which leads to a very long right-hand-side tail. This can be interpreted by the power law distribution of the Pareto type (1). The same assumption is made for the level of severity of pathology. Most of the pathologies can be considered mild, but there exist some pathologies that are much more severe than others. Hence, the probability density function for the crack size δ in ΔA (1) has the same form (10), where the nominal lifetime segment ΔAn is substituted by the RAE ΔA. It is of interest to outline that, both the normalization constants of the Pareto functions may depend upon the time interval Δt, which represents the load duration in the case of glass strength, and the observation time in the theory of epidemiology.

The area A of the glass surface enters in the Weibull expression for the failure probability (7) and provides a phenomenon referred to as size-effect in the mechanics of materials: the higher the size A of the glass element, the higher the probability of finding a crack larger than δ. In the theory of epidemiology, the counterpart of the size of the material body is the nominal life of the individual. The longer the nominal life, the higher the probability of developing a severe pathology. Hence, the nominal-life segments represent the rings in the chain of life, and the size effect in terms of nominal age A_n is a characteristic of the Weibull distribution (18), describing the probability of death at A_n under an epidemic of level σ.

The expressions for the critical conditions, i.e., the one for the critical SIF in mode I (6) and the death criterion (19), are identical, apart from the exponent β of δ: In LEFM $\beta = 2$, whereas in the section "Probability of death as a function of age and level of epidemic" the parameter β was not assigned, to let this parameter represent another degree of freedom, associated with our capability at treating the disease. In any case, the mechanical interpretation of the level of epidemic σ is that it represents the stress acting in the material, i.e., the cause that provokes the critical growth of cracks and, hence, the breakage of the body. This correspondence highlights the relationship between the assumed level of epidemic and the critical level of pathology.

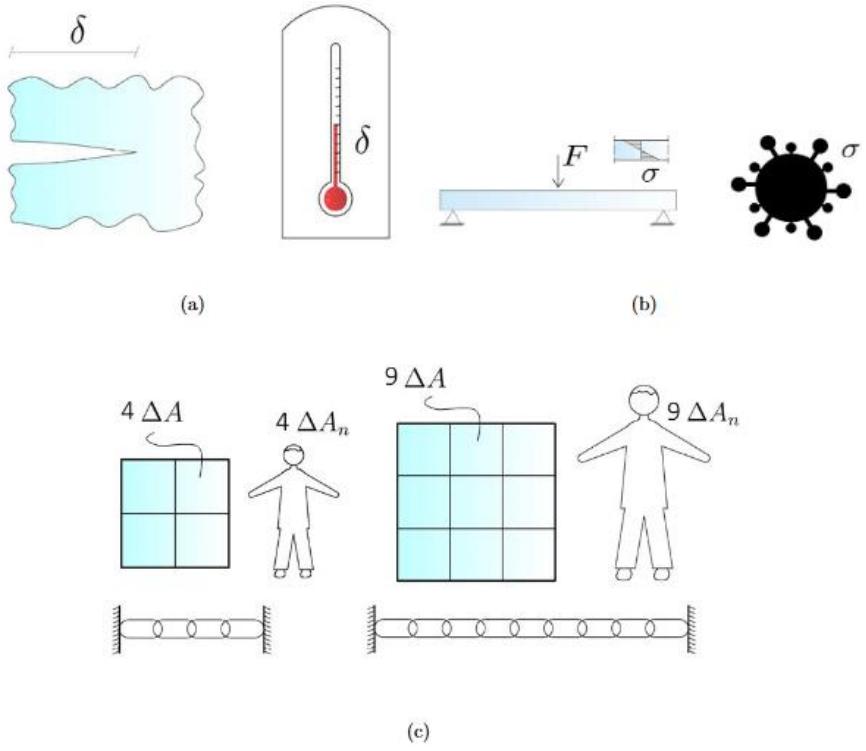

Fig. 1: Analogies between the statistical model for brittle materials' strength and the effect of an epidemic. (a) The depth of the cracks δ governs glass strength, while the level of pathology δ governs the risk of death; (b) σ is the effect of the external action in the model for glass strength while it is the intensity of the action of the epidemic in the theory of epidemiology; (c) the size effect is represented by the size of the glass plate in mechanics and by the length of life in epidemiology. A glass plate composed of 4 ΔA and a person of nominal age 4 ΔA_n are represented as chains of 4 links, while a glass plate composed of 9 ΔA and a person of nominal age 9 ΔA_n as chains of 9 links. The resistance of the links is of stochastic nature

However, the state of stress may not be uniform equibiaxial in the glass plate. This case has similarities with the condition in which the epidemic is not uniformly spread throughout the body. Moreover, one should consider that the effect of the epidemic on each individual may depend on other factors. In the mechanical analogy, for example, the crack opening stress $\sigma_\perp$ in (6) is the normal component of stress in the crack plane: the state of stress is represented by a tensor field and the orientation of the crack for the principal direction of tensile stress plays a dominant role. In a more refined study, also the level of the epidemic could acquire more structure, perhaps representable with a tensorial field, but such consideration can only be conjectural at this time. In any case, it may be useful to recall how the case of varying stress is treated in the mechanics of brittle materials, with the only aim to provide an example of what might be the consequences in a statistical model of epidemics.

To this aim, observe that Eq. (6) establishes a one-to-one correspondence between the stress $\sigma_\perp = \sigma_{cr}$ and the size of the crack $\delta_{cr} = [K_{Ic}/(\sigma_{cr}Y)]^2$ that is critical for that stress. Hence, the probability $P_{\Delta A}^{\leq}\left(\sigma_{cr}\right)$ of finding in ΔA a critical stress $\leq \sigma_{cr}$ is equal to the probability of finding a crack of size equal to or higher than δ_{cr}. Substituting in (11) after setting $A_n = A$, one obtains:

$$P_{\Delta A}^{\leq}\left(\sigma_{cr}\right) = P_{\Delta A}^{\geq}\left(\left(\frac{K_{Ic}}{\sigma_{cr}Y}\right)^2\right) = \Delta A\left(\sigma_\perp \frac{Y\sqrt{\eta}}{K_{Ic}}\right)^{2(\alpha-1)} \tag{22}$$

The probability of finding in the RAE a critical stress comprised between σ_{cr} and:

$$\sigma_{cr} + d\sigma_{cr} \; is \; \frac{d}{d\sigma_{cr}} P_{\Delta A}^{\leq}\left(\sigma_{cr}\right) d\sigma_{cr}$$

The number of RAEs composing the area A is again $A/\Delta A$ but, since the stress is variable, it is necessary to distinguish each element ΔA_i, $i = 1...A/\Delta A$, on basis of the state of stress acting there. Let $\Omega_{\Delta Ai}(\sigma_{cr})$ represent the angle containing the normals to all the possible crack planes for which the normal component of stress, in ΔA_i, is higher than the critical value σ_{cr} (Batdorf and Crose, 1974), with $0 \leq \Omega_{\Delta A}(\sigma_{cr}) \leq \pi$. Assuming that there is the same probability for any crack orientation, the probability of failure for ΔA_i in the interval $(\sigma_{cr}, \sigma_{cr}+d\sigma_{cr})$ reads:

$$dP_{\Delta A_i}^F = \frac{\Omega_{\Delta A_i}\left(\sigma_{cr}\right)}{\pi} \frac{dP_{\Delta A}\left(\sigma_{cr}\right)}{d\sigma_{cr}} d\sigma_{cr} \tag{23}$$

Consequently, the failure probability of ΔA_i is given by:

$$P_{\Delta A_i}^F = \int_0^\infty \frac{\Omega_{\Delta A_i}\left(\sigma_{cr}\right)}{\pi} \frac{dP_{\Delta A}\left(\sigma_{cr}\right)}{d\sigma_{cr}} d\sigma_{cr} \tag{24}$$

and the probability of survival is $P_{\Delta A_i}^S = 1 - P_{\Delta A_i}^F$. At the level of the whole area A, the survival probability is equal to the product of the survival probabilities of the elements, that is:

$$P_A^S = \prod_{i=0}^{A/\Delta A}\left[1 - \int_0^\infty \frac{\Omega_{\Delta A_i}\left(\sigma_{cr}\right)}{\pi} \frac{dP_{\Delta A}\left(\sigma_{cr}\right)}{d\sigma_{cr}} d\sigma_{cr}\right] \tag{25}$$

In this expression, the variability of the state of stress in the body is taken into account by the different values that $\Omega_{\Delta Ai}(\sigma_{cr})$ may assume in each ΔA_i.

Equation (25) can be specialized and simplified once the state of stress is known. If this is uniform, equibiaxial, and equal to σ, one has that $\Omega_{\Delta Ai}(\sigma_{cr}) = \pi$ when $\sigma_{cr} \leq \sigma$ and $\Omega_{\Delta Ai}(\sigma_{cr}) = 0$ when $\sigma_{cr} > \sigma$. Following the procedure illustrated in Section 3.1 of (Pisano and Royer Carfagni, 2017), one obtains again (7). In the most general case, reasoning as in (Pisano and Royer Carfagni, 2017), one concludes that the probability of failure for the glass element of area A under generic tensile stress can be written in the general form:

$$P_{Aeff}^F\left(\sigma_{\max}\right) = 1 - \exp\left[-A_{eff}\left(\frac{\sigma_{\max}}{\eta_0}\right)^m\right], m = 2(\alpha-1), \eta_0 = \frac{K_{Icr}}{\eta^{1/2}Y} \tag{26}$$

where $\sigma_{\max}$ is the maximum tensile component of stress throughout the body and A_{eff} is the "effective area", i.e., an appropriate rescaling of the area A to take into account the variability of stress from point to point and the fact that it is not equibiaxial.

It is not clear, at the time of the present writing, how this re-scaling towards the effective area could be translated into a statistical theory of epidemics. In any case, the analysis suggests that when the spreading of the epidemics in the territory is not uniform and the effects on the population depend on factors that are not considered by the simple criterion (19), an expression of the type (18) is still valid, but the nominal age A_n should be rescaled, while σ_{max} should represent a measure of the maximum level of the epidemic within the territory.

The Case of the COVID-19 Epidemics in Italy

Between February 1st and November 30th, 2020, 1.651.229 COVID-19 cases have been diagnosed in Italy (https://www.epicentro.iss.it/coronavirus). The epidemic spread scenario can be schematized in three phases. The first phase (first wave), from February to the end of May 2020, was characterized by a very rapid spread of cases and deaths and by a strong territorial concentration in the North of the country. A transition phase occurred in the summer season, from June to mid-September, when the spread was very limited. By starting from the end of September, the cases have quickly increased again (second wave).

Most of the models that consider the evolution of epidemics are based on the number of infected people and/or deaths from contagion (Becker and Dietz, 1995; Fraser, 2007; House and Keeling, 2008; Goldstein *et al*., 2009; Ross *et al*., 2010). However, the identification of all cases and deaths attributable to a new virus is very difficult. In the observation period, the official number of deaths induced by COVID-19 in Italy was 57.647, while the difference between the total number of deaths in 2020 and the average number in the same months in the years 2015-2019 was about 84.000. This underlines the importance of using objective parameters, such as raw mortality, to estimate the effects of epidemics and monitor their evolution.

The Relative Index of Epidemics

The expression for the probability of death (21), by recalling that $\eta_0 = K_{cr}/(Y\,\eta^{1/\beta})$ and that η is given by (12), can be re-written in the form:

$$P_{A_r}^{D}(\sigma) = 1 - \exp\left[\frac{F(A_r)}{\Delta A_n}\left(\frac{Y\sigma}{K_{cr}}\right)^{m}\right] \tag{27}$$

that, after some simple analytical steps, becomes:

$$\ln\ln\left[\frac{1}{1-P^{D}(A_r,\sigma)}\right] = \ln\left[\frac{F(A_r)}{\Delta A_n}\right] + (\alpha-1)\beta\ln\left[\frac{Y\sigma}{K_{Ic}}\right] \tag{28}$$

which represents a straight line $Z = X + Q$ in the plane $Z = \ln\ln\left\{\left[1-P_{Ar}^{D}(\sigma)\right]^{-1}\right\} - X = \ln\,[F(A_r)/\Delta A_n]$, referred to as the epidemic Weibull plane. This is the counterpart of the Weibull plane (Weibull, 1939), commonly used for the graphical estimation of the Weibull parameters (Pisano and Royer Carfagni, 2015). Hence, according to the proposed theory, measured mortality rates, ordered by nominal age, should be aligned in the epidemic Weibull plane, which represents the criterion for the estimation of the parameters γ_1 and γ_2 of the renormalization law (2). The procedure consists in finding the renormalization law that provides the best fit to the experimental points in one particular state of the epidemic. Then, it must be verified that the same normalization provides a reasonable alignment in the other states under study.

The intercept of the interpolation line (28) with the ordinate axis in the epidemic Weibull plane is given by:

$$Q = (\alpha-1)\beta\ln\left[\frac{Y\sigma}{K_{cr}}\right] \tag{29}$$

In general, $Q < 0$, since $\alpha > 1$, $\beta > 0$, and $Y\,\sigma/K_{cr} < 1$. Remarkably, the higher the intercept Q is, the higher the death rate among the elderly compared to the young turns out to be and vice-versa. Hence, the proposed model accounts for the distribution of the deaths and, through the intercept Q, for the shifting in mortality rates between age classes. Recall that the values for α and K_{cr}, which is a measure of human ´´toughness", are not affected by the level of epidemics. The limit conditions where the whole population dies from the epidemic is reached when the value of the product $\sigma \cdot Y$ is such that the ratio $Y\,\sigma/K_{cr}$ approaches the unit value and Q tends to become null. In another word, in this case, the minimum level of damage $\delta = \delta_{min}$ is sufficient to cause death.

The inverse of the absolute value of Q, i.e., $-1/Q$, might be considered an effective measure of the level of epidemics, but a quantitative estimate can only be obtained in relative terms. Let the reference configuration, labeled as "0", be characterized by the parameters β_0, Y_0, σ_0, while the configuration under study, labeled as "1", by β_1, Y_1, σ_1. Once the best-fit line in the epidemic Weibull plane for the configurations “0” and “1” and the respective intercepts on the ordinate axis Q_0 and Q_1 are determined from the analysis of the mortality data, assuming that α and K_{cr} are the same for both configurations, the “epidemic ratio” r:

$$r = \frac{-1/Q_1}{-1/Q_0} = \frac{Q_0}{Q_1} = \frac{\beta_0}{\beta_1}\frac{\ln\left[\frac{Y_0\,\sigma_0}{K_{cr}}\right]}{\ln\left[\frac{Y_1\,\sigma_1}{K_{cr}}\right]} = \frac{\beta_0}{\beta_1}\frac{\ln\left[\frac{K_{cr}}{Y_0\,\sigma_0}\right]}{\ln\left[\frac{K_{cr}}{Y_1\,\sigma_1}\right]} \tag{30}$$

can represent a relative measure of the strength of the epidemics. Then, as a measure of the distance of r from the unit, the "relative index of the epidemic” I_e is defined as:

$$I_e = 100(r-1) = 100\frac{Q_0 - Q_1}{Q_1} \tag{31}$$

where factor 100 is introduced to obtain numbers that are easier to read.

Hence, $I_e > 0$ indicates that the "strength" of the epidemic is higher in situation "1" than in situation "0". Of course, I_e will reach negative numbers when the epidemic is milder in the configuration under analysis than in the reference configuration. If there are no changes in the ability at curing the disease, i.e., if $\beta_0 = \beta_1$, an increase of I_e could be due to a major spread of the epidemic (increasing Y) or to an increased lethality of the virus (increasing σ) and vice-versa. Changes in the capacity of curing diseases can greatly alter I_e, i.e., the effects of the epidemic, when $Y_1\sigma_1 = Y_0\sigma_0$; e.g., an effective vaccine would lead to an increase of β accompanied by a decrease of I_e, whereas exceeding the capacity of intensive care units would lead to the opposite effect.

Observe that it is customary to consider excess mortality as an effective indicator of the impact of an epidemic. However, this cannot account for the demographic structure of the population and the distribution of deaths by age. These represent the main

conceptual differences between excess mortality and the relative index of epidemics. In particular, the theory explicitly considers the greater aptitude of the elderly to develop life-threatening diseases during epidemics. Hence, equal variations in the number of deaths in the young and in the elderly represent different signs of the impact of an epidemic, which cannot be captured by excess mortality.

First Wave

Contagion in Italy officially started at the end of February: ten municipalities in the province of *Lodi* (Lombardia region) and one in Padova (Veneto region) were quarantined since February 24th. National quarantine was imposed on March 9th, while the release of the lockdown gradually started on May 4th. The evolution of the relative index of epidemics is here analyzed in the most populated (more than 2 million inhabitants) Italian regions, which are Campania, Emilia Romagna, Lazio, Lombardia, Piemonte, Puglia, Sicilia, Toscana, and Veneto, geographically located as in Fig. 2.

From January to June, setting the single month as a unit of time Δt, the renormalization law $A_n = F(A_r)$ in *ante* COVID-19 conditions, representing the configuration of comparison (configuration "0"), was derived from the averages of the number of dead, sorted by age, in the same periods of the years from 2015 to 2019. For all the Italian municipalities, these data are available online at https://www.istat.it/it/archivio, while the total population at the beginning of each month at http://demo.istat.it, together with the demographic structure referring to a certain solar year. The solar years of age were grouped in nine sets ($A_r = \{0-10, 10-20, ..., 80-90, >90\}$) and the ages at the center of the intervals and 100 for ages higher than 90 were assumed to be the ages representatives of the groups. The ratios between the number of deaths and peer population give the probabilities of death for each i-*th* set, which were averaged in the period 2015-2019 for each considered month.

Recall that the theoretical scaling between death probabilities at any two real ages is given by Eq. (27). Then, parameters γ_1 and γ_2 of (9) for each considered month were calibrated by equating measured and expected death probabilities given by Eq. (27) at the point $A_{r,2} = 45$. The assumed form of renormalization turned out to be accurate in all the regions under analysis. For the sake of example, the results of calibration for Campania are shown in Fig. 3.

Fig. 2: Geographic location of the various Italian regions under investigation

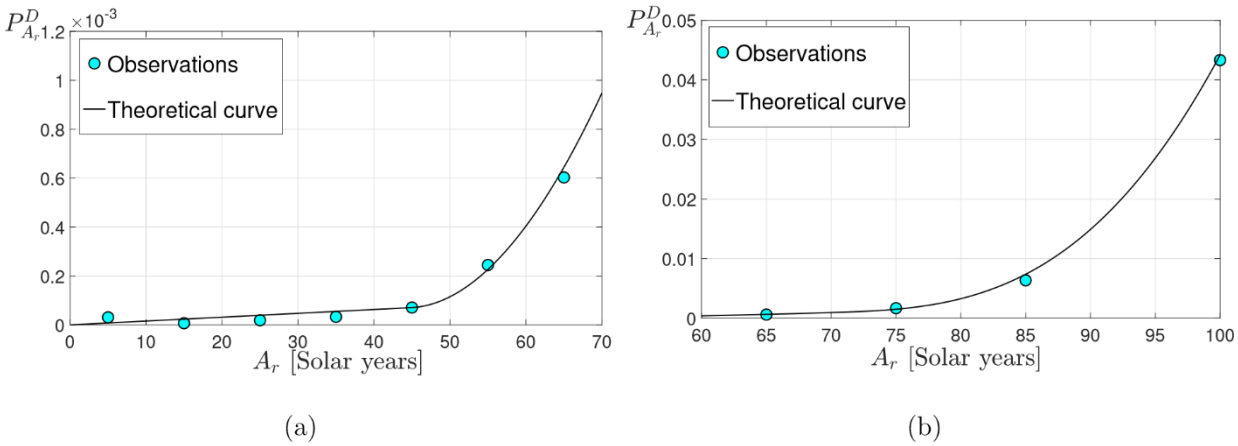

Fig. 3: Observed probability of death $P^D_{A_r}$ in the month as a function of real age A_r. Comparison with the theoretical predictions. Region *Campania* in the configuration "0" (average of 2015-2019) for March. (a) Left-hand-side branch; (b) right-hand-side branch

The agreement between measured probabilities of death and theoretical expectations is very good, for all the considered regions, apart from the death probabilities associated with ages much lower than 40 years. This might be because these are strongly sensitive to small variations in the number of deaths. Furthermore, the probability of deaths in the range of 0-10 is always higher than the theoretical predictions because deaths at birth are not associated with natural degradation of the human body and hence not covered by the proposed theory. The same is true for deaths from non-epidemic causes, such as road accidents, drugs, or homicides.

Then, the value of the intercept Q in the configuration "0" (Q_0) is graphically obtained from the line of the type $Z = X + Q$ (28) that best fits the measured points in the epidemic Weibull plane $Z = \ln\ln\{[1 - P^D(A_r,\sigma)]^{-1}\} - X = \ln[F(A_r)/\Delta A_n]$.

Passing to the configuration "1", i.e., the periods of observation in 2020, the effects of the epidemic are gathered in the product $Y\,\sigma$, since parameters γ_1 and γ_2 do not vary according to the theoretical model. Again, the number of deaths sorted by age and the total population are available at https://www.istat.it/it/archivio and http://demo.istat.it, respectively. Then, the graphical procedure is repeated to obtain the value of the intercept Q_1 in the configuration "1".

For the sake of illustration, the graphical regressions associated with the Campania region in march for the configurations "0" and "1" are shown in Fig. 4(a) and 4(b), respectively. Notice that the fitting is very good, apart from the lower age classes, for which the mortality rates are not well captured by the theory for the reasons explained above. The good fitting confirms the goodness of the theory.

Finally, the relative index of epidemic I_e has been calculated from (31). The monthly variation of this index, within the six months corresponding to the first wave of COVID-19, is shown in Fig. 5.

March was the month of the epidemic peak in most of the considered regions, while the peak was reached in April in Piemonte, Puglia, and Toscana. Lombardia was by far the most infected region, while the epidemic was mild in the central regions (Lazio, Toscana) and very limited in the south (Campania, Puglia, Sicilia). This was mainly the consequence of the lockdown countermeasures, which were simultaneously imposed on the whole national territory. In the North, where the epidemic had already started, the lockdown was only partially effective; in the South, where the epidemic had not yet spread, the timely activation of the lockdown prevented the diffusion of the infection.

Observe that there are negative values for I_e in all the considered regions in January, February, and June; in May, I_e is greater than one in Lombardia only. A negative value indicates that the severity of the epidemic in configuration "1" (2020) is lower than in configuration "0" (previous years). Recall that the theory does not consider COVID-19 as the only type of epidemic: there may be mortality from other sources, such as influenza. A negative index may indicate that, when the COVID-19 virus had not yet spread, the level of mortality in 2020 was less than in previous years or, similarly, that the lockdown did limit the spread of other viruses, other than COVID-19, concerning previous years.

Second Wave

Summer 2020 represented a transition phase for the epidemic in Italy, in which the spread of the virus was very limited. The index of the epidemic for such a period is not significant and, hence, it is not recorded.

A significant increase in the cases of infection was observed starting from the end of September. Differently from the wave that occurred during Spring, the spread of the virus was quite uniform on the whole national territory, probably because the lockdown was released everywhere. Starting from November 6th, the restrictive countermeasures to prevent the spread of the virus were differentiated in the Italian regions on basis of 21 epidemic indicators. In particular, the growth of the epidemic reproduction number R_t was considered the main indicator from which the countermeasures were defined. Regions and autonomous provinces were classified into three areas (red, orange, and yellow) corresponding to three different risk scenarios. The classification was based on ordinances weekly released by the Italian Ministry of Health.

For the nine most populated regions, we have calculated the relative index of epidemics I_e from September 28th to December 27th, by setting the single week as the period of observation, coherently with the timescale of the classification of the risk. The index is calculated from raw mortality data, released by ISTAT and available at http://www.istat.it/it/archivio. The results are illustrated in Fig. 6.

By comparing Fig. 5 and 6, it is evident that the regional differences are much more limited. The highest impact of the second wave is recorded in Piemonte, where the relative index of epidemics reached a value higher than 4, which is however much lower than the peak reached in Lombardia during the first wave. The maximum value reached by I_e in Campania, Lombardia, and Veneto is about 3, a little lower than 3 in *Puglia* and about 2 in the other considered regions. The peak has been reached in the second half of November; only Veneto presents a trend very different from the other regions, characterized by a peak about in the middle of December.

Recall that the relative index of epidemics depends upon the level of spread Y, as per Eq. (30). Regarding the Susceptible-Infectious-Recovered (SIR) model (Kermack and McKendrick, 1927), which details how a virus is transmitted from an infected person to a healthy one

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(Grassly and Fraser, 2006; 2008), Y could be associated with the ratio between the number of infected I and the number of susceptible S. Hence, we can conjecture that Y is strictly associated with the epidemic reproduction number R_t, defined in the SIR theory as the average number of secondary cases per infectious case in a population made up of both susceptible and non-susceptible hosts. Figure 7 shows the weekly variation of R_t, estimated by the researchers of the Bruno Kessler Foundation on behalf of the Italian authorities (ISS). By comparing Fig. 6 and 7, it should be observed that the peaks in I_e are delayed by about 4 weeks concerning R_t and that I_e starts to decrease when R_t becomes lower than unity.

The maximum values of R_t estimated for Lombardia and Piemonte is very close to one another, but the difference in terms of maximum I_e is noteworthy. Observe that the ascending branch of the R_t function for Piemonte is less steep than that for Lombardia. Hence, the value reached by R_t in Piemonte has been higher than unity for more weeks than in Lombardia: this might justify the difference in terms of I_e. Compare now the situation between Lombardia and Campania. The trend for I_e is quite similar, but this is not the case in terms of R_t. Concerning the theory presented here, this might be due to a different capacity of the regional health systems at curing the disease (parameter β) or to a different lethality of the virus (parameter σ). However, such finding might be as well a consequence of a non-correct estimation of the index R_t. Furthermore, it is of interest to notice that the R_t trend for Veneto is very similar to that of the other regions, apart from the fact that it remains higher than one, although only slightly, even in December; on the other hand, the trend for I_e is very different and the time delay between the peaks of I_e and R_t is of the order of 7 weeks. This difference may again be attributed to the particular way in which the local authorities from Veneto organized their actions.

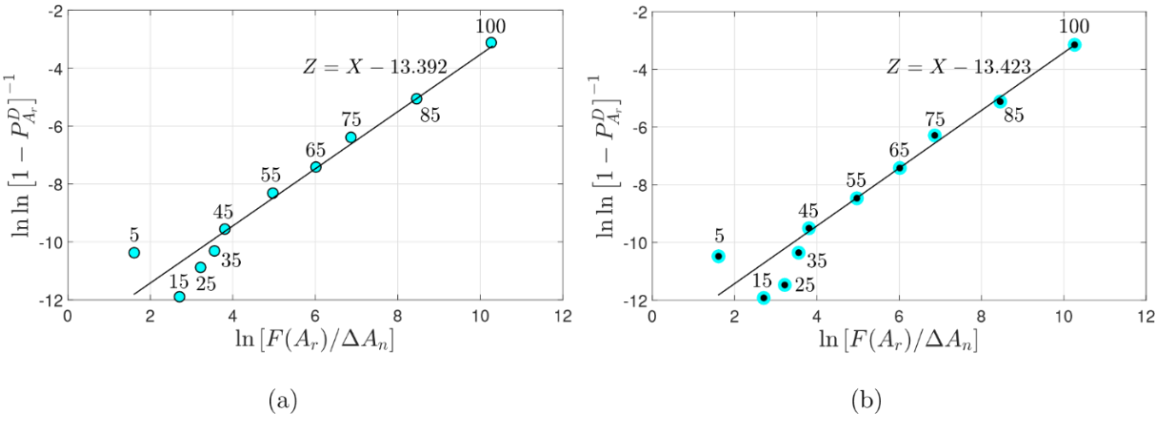

Fig. 4: Graphical regressions in the epidemic Weibull plane of the probability of death as a function of the renormalized age for the Campania region in March. (a) Configuration "0"; (b) configuration "1"

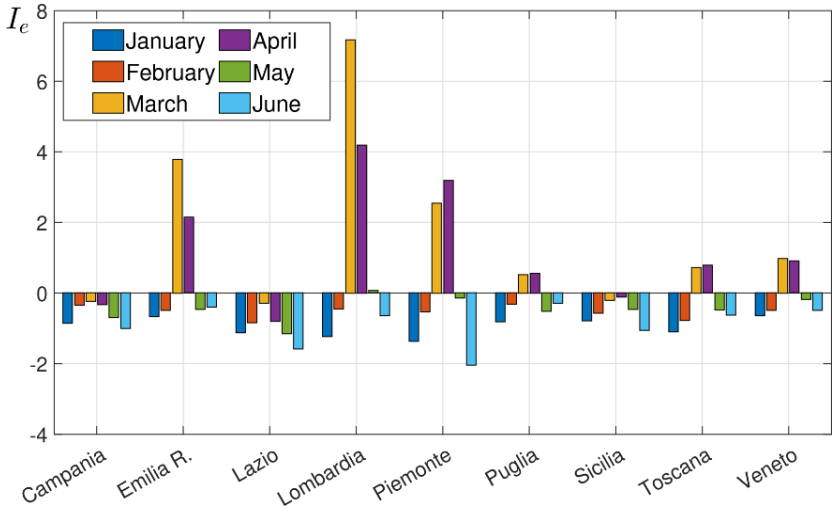

Fig. 5: Values of the relative index of epidemic I_e from January 2020 to June 2020 in the most populated Italian regions concerning *ante* COVID-19 conditions, which refer to average mortality in the same months of previous years (2015-2019)

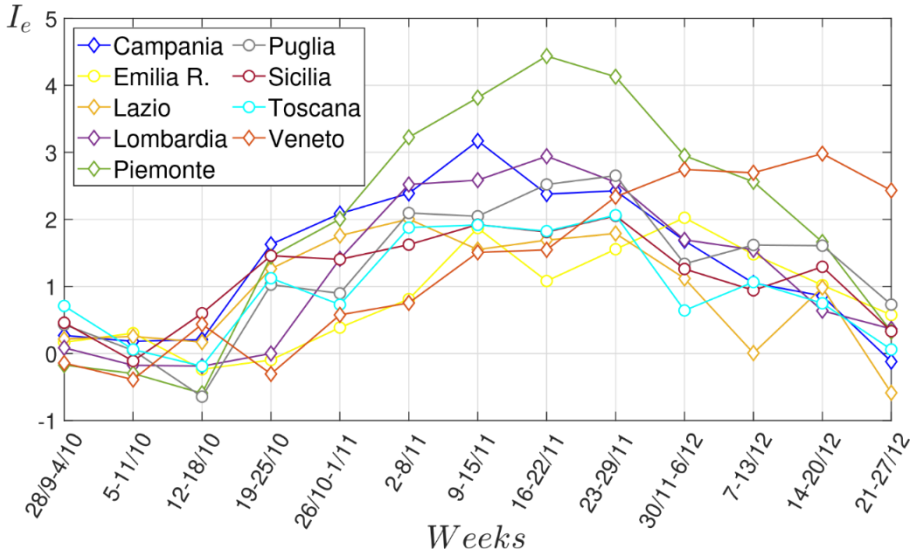

Fig. 6: Weekly variation of the relative index of epidemic I_e from September 28^{th,} 2020 to December 27^{th,} 2020 in the most populated Italian regions. *Ante* COVID-19 conditions of comparison refer to average mortality in the same weeks of previous years (2015-2019)

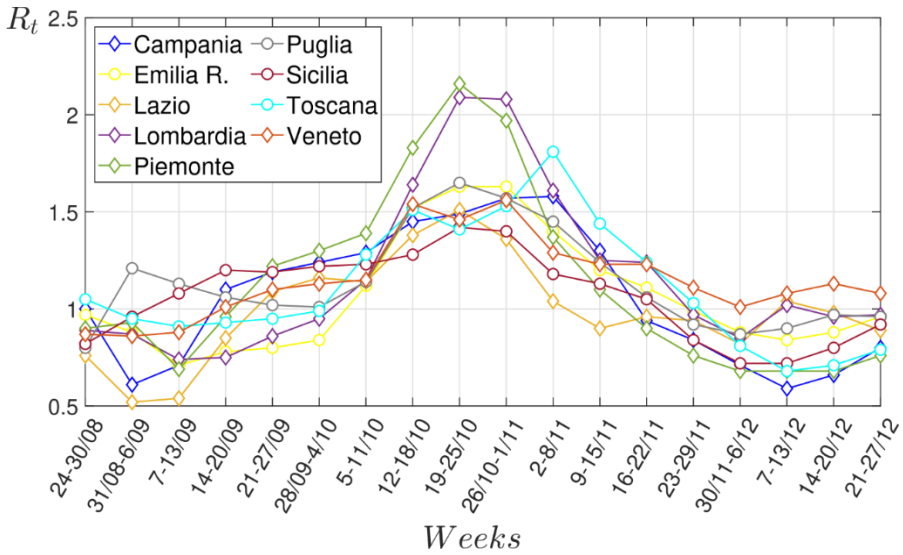

Fig. 7: Values of the epidemic reproduction index estimated by the researchers of the Bruno Kessler Foundation on behalf of the Italian ISS from August 24^{th,} 2020 to December 27^{th,} 2020. Calculations made on a weekly base

The algorithm used by the researchers of the Bruno Kessler Foundation refers to the methodology described in the article available at https://arxiv.org/ftp/arxiv/papers/2003/2003.09320.pdf.

A few examples of calculation can be found online at https://www.epicentro.iss.it/coronavirus/opendata. The week is set as the period of observation. The value of the new daily infections is not used for the calculation of R_t, whereas this is calculated on basis of symptomatic infected only. Since positive swabs of one day refer to dates of onset of symptoms, distributed in the two weeks before, calculation of R_t requires time to collect and select data. Furthermore, the values are strongly influenced by the quality of data (Azmon *et al.*, 2014; Gamado *et al.*, 2014). Since I_e, on the other hand, is obtained from raw mortality data, its estimation is more objective.

Therefore, it would be desirable to derive an analytical correlation between Y and R_t and, as a consequence of this, between R_t and I_e. In this case, the relative index of the epidemic could also be used as an instrument for assessing the goodness of the estimate of R_t.

Conclusion

The presented statistical model allows the definition of an index of the epidemic, directly calculated from the records of the number of deaths, classified by age, during the period of the epidemic, and the comparison with the corresponding data in previous conditions. The model relies upon the definition of the nominal age, which represents the rescaling of the real age, statistically associated with the probability of developing a pathology that is critical for a certain level of the epidemic. One of the major advantages of this procedure is that it is not necessary to recognize which are deaths directly associated with one specific form of the epidemic, since only the rough mortality data, sorted by age, enter into the calculations.

The model borrows concepts from consolidated models in the probabilistic mechanics of brittle materials, where the stress takes the place of the level of epidemic and the nominal age represents the size of the body. The probability of finding a crack of a given size in the material plays the role in the probability of developing a critical pathology in nominal life. The size effect experimentally verified in the mechanics of materials, derives from the fact that the larger the body, the higher the probability of finding a defect of critical size; this is conceptually similar to the increased probability of finding a critical pathology in an older than in a younger person. The criterion from linear elastic fracture mechanics, which correlates the applied stress with the critical size of the crack, has similarities with the criterion used to define the critical pathology for the level of the epidemic. The theoretical calculations provide a statistical law a` la Weibull, of the same type encountered in the mechanics of brittle materials, to describe the probability of death as a function of the level of the epidemic for any value of the nominal age.

To verify the reliability of the model and to show its potentialities, the statistical data regarding deaths that occurred in the most populated regions of Italy during the two waves of the COVID-19 epidemic, have been processed and commented on. The model provides results in perfect agreement with the observations. Moreover, since it furnishes a quantitative estimate of the effects of the epidemic according to our capability of offering reliable cures, the theory could be developed for predictive purposes. To this aim, much research is still needed to provide, in particular, specific mathematical expressions, as a function of other more specialized state variables, for the various factors that influence the values reached by the relative index of epidemics.

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Author's Contributions

Gabriele Pisano: Contributed to the development of the theoretical model, the analysis of data and writing the manuscript.

Antonio Bonati: Contributed to the analysis of data and writing the manuscript.

Gianni Royer Carfagni: Organized the study and contributed to the development of the theoretical model.

Ethics

This article is original and contains unpublished material. The corresponding author confirms that all of the other authors have read and approved the manuscript and no ethical issues involved.

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