**Caution coupling explains linear phase of Covid-19 curves**

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Understanding of epidemics through modelling is gained both through highly simplified population models such as the early SIR models [1, 2] (distinguishing Susceptible, Infective, and Recovered individuals) and its extensions both to more differentiated models like SEIR [3-6] and SIDARTHE [7, 8], and to include other population structure such as age with a matrix of social mixing [9, 10], stochastic effects [11, 12] and spatial [13-15] or other network structure [16-18]. In addition, in connection with detailed population data (big data) [19, 20], cause and effect can be analysed in detailed agent models [21, 22]. However, certain key features of the Covid-19 pandemic data affecting us all have not been explained simply. In particular, the remarkably common extended linear phase in countries’ cumulative confirmed cases is poorly understood. While small world network effects may account for non-exponential early growth [16], they do not explain the linear saturation effect well below levels required for “herd immunity”. In this letter, we show that central features of this societal response complexity can indeed be understood using the S(E)IR(D) family of models by means of a single additional deterministic coupling, one reflecting societal introspection: *caution*. As society reacts to and endeavours to contain the strong effects of the Covid-19 virus on health, social well-being and the economy, it may appear that explanation requires every aspect of society to be modelled in detail: from the use of public transport, religious practises and child play patterns to political decision making, the media and legislature[23]. However, the prolonged near linear response both in the cumulative number of Corona cases as well as those in individual populations can be understood as a generic human response to the epidemic in the age of information. This insight may in turn help us to focus societal measures and responses more effectively.

In March 2020, a study in Oxford [24] suggested, using an SIR model, that a much larger portion of society may be infected than normally inferred from the testing data, which was biased to symptom carriers. This was in-line with standard epidemiological modelling that relates the epidemic proliferation peak and subsequent decline to the advent of a significant fraction of the population becoming immune to the disease. Contemplating the linear global data, see Fig. 1, the author was struck by the similarities with bacterial viral proliferation kinetics inside a single cell and in evolution experiments *in vitro*, which systematically show exponential growth giving way to linear growth in the absence of immunity [Ref]. The fundamental reason for this is specific resource limitation, for example of the Qß viral replicase enzymes responsible for copying the RNA viral genome [Ref]. This is, like the nesting site limitation in bird populations, a straightforward thing: when most replicase enzymes are occupied in the copying process with a particular RNA genome, they are not available for proliferation of other RNA, and the throughput of copied RNA becomes a constant, limited by the throughput of such copying enzymes. The author wondered if a related mechanism could account for the observed linear growth phase for Covid-19 in societies shown in Fig. 1.

We add an endogenous informed human cautionary response to simple compartmentalized models in the following way. We split the population of susceptible individuals S into two classes S and Sc, the former acting unaffectedly and hence experiencing exposure to the virus at the initial rate, the latter exercising informed caution and hence reducing their exposure to the virus by a factor c0<1. We then identify the subpopulation C in the deterministic population model most indicative of the negative impact of the disease for the population, as the trigger for this response. For example, in the case of the simple SIR model, it is the class C=I of infected individuals, and more realistically in the SEI3R model, which adds an exposed class E of not yet infectious individuals and two additional classes I2 and I3 of infectious individuals (in hospital and in intensive care respectively), it is the class C=I3. Although one may expect that the number of deaths also dramatically influences the execution of caution, it is a cumulative category which does not reflect the current situation as does I or I3. In fact, the daily number of deaths would be an appropriate indicator but this is simply proportional to I in the SIR model and I3 in the SEI3R model.

To capture the response to this cautionary trigger, we first employ a simple reaction mechanism: a reversible caution binding mechanism. As we will see below, we could also use a threshold trigger response familiar in nerve cells. The simple reversible caution model involves both a transition of unaffected susceptible individuals S to cautious individuals Sc, with a rate proportional to the product of their densities in the population (as in the law of mass action for chemical interactions, this captures the frequency of interactions), as well as a reverse transition from cautious individuals to individuals acting unaffectedly, proportional to the density of cautious individuals (resulting in a natural single exponential decay of caution in time). The mechanism and equations of two representative endogenous caution models are shown in Fig. 2. Note that in this first approach, we do not distinguish between cautious and non-cautious exposed or infected individuals I. We have tested the impact of making this distinction in a second set of models, also shown in Fig. 2, which share the prediction of a linear phase, with a stronger impact of caution. Once in hospital we assume naturally that all individuals are exercising (or having exercised for them by hospital staff) a significant degree of caution.

An alternative threshold trigger response replaces the product law by a threshold activation rate of the form where is the difference between the endogeneous cautionary trigger signal introduced above and a threshold value (e.g. 25% of hospital ICUs being occupied) and describes the sharpness of the transition. While this may be required to fit accurately the sudden press amplified responses to reports of rising death tolls, it is not essential to explain the observed long linear response phases.

We employed a straightforward python-based modelling strategy making use of either direct ordinary differential equation encoding and the lmfit package or the modelling package GomPy with its interface to the SciPy minimize fitting procedure via least squares. The results of our modelling are shown, firstly for representative variation of the caution parameters in Fig. 3, showing the range of response forms that the model can account for and then fitted to the country response data shown in Fig. 4.

This phenomenom is not restricted to US or Russian responses. Whereas some countries, with geographic advantages in isolation, by executing radical containment policies, have managed to reduce the Covid-19 virus to a very low level, the far more generic growth response is a transition from an exponential (or possibly power law [16]) phase to a relatively constant rate of growth, often with 100s or 1000s of cases per day, in some cases after an initial overshoot. The generic linear phases of growth are surveyed in data taken from Johns Hopkins University database, using a rolling average over 7 days to remove prominent weekly variations in reporting and some fluctuations, in Fig 2 for the 10 most affected countries (US, Brazil, Russia, India, UK, Spain, Italy, Peru, France, and Iran) and six others selected because they represent a strong variety of government responses to the pandemic (Sweden, Germany, Netherlands, Poland South Korea, Australia).

The simplest generic structure of epidemics, captured also by the SIR model, is initial exponential growth tempered by the rise of so-called herd-immunity in the population.

We briefly address a potential critique of our result arising from limitations in the testing procedure. If the number of available tests is limited in a country, then this could result in a saturation in the number of confirmed cases per day, resulting in an apparent linear growth. Although such limitations have occurred and are visible for example in the data from …, we argue that our overall conclusions are independent of this effect. Firstly, overall testing levels in a significant number of countries have exceeded by a large factor the number of positive cases. Secondly, the linear trend also occurs in the number of deaths. Thirdly, the numbers of tests have been increasing in most countries and the linear trend persists.

The implications of these results are instructive. Firstly, it is no longer herd immunity but cautionary measures that is the primary limiter of spread of the disease: the latter have effect at much lower disease frequencies than herd immunity which would only be achieved much later after an enormous societal cost. Secondly, the strongly asymmetric peak responses to the daily case statistics and the linear population responses in cumulative case indicators (and in deaths) result from an active regulation of the degree of caution exercised in the population. As evidenced by the case of Sweden this is not entirely dependent on government legislation, which itself is in response to fears of repercussions based on severity indicators like the number of deaths, but also occurs naturally in the population, for example through the ubiquitous distribution of statistics concerning ICU cases. Depending on the timescale of relaxation of caution, the response can also involve second waves and longer-term oscillation. Thirdly, the natural human response, and as we have seen also government mediation of this apart from in a few nations, is not to maintain strict caution measures up until disease eradication. The successive relaxation of measures, even close to disease eradication results in very long periods of nearly constant case frequencies, consistent with the cautionary regulation process that we describe in this paper. Examples of this include Germany, Australia, South Korea, Switzerland and many other states.

**Figures**

Fig.1 Common linear phase of growth in individual countries

Fig.2 The mechanism and equations of two representative endogenous caution models.

Fig. 3 Effect of caution feedback for representative variation of the caution parameters

Fig. 4 Fit to country data for caution arising from critical societal burden in SCEI3R model

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