A Simple Planning Problem for COVID-19 Lockdown, Testing and Tracing: Comment

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1 Introduction

Alvarez, Argente and Lippi (2021) – henceforth AAL – offer an analysis of lockdown policies to manage COVID19. The planner problem formulated by AAL is pioneering work, which has appeared early on in the pandemic and proved extremely valuable, serving as the seminal paper for a strand of studies analyzing dynamic interventions in the COVID19 context. Two main features characterize the planner problem. First, lockdowns mitigate disease growth and reduce deaths but at the same time disrupt economic activity. Second, the planner is subject to a set of assumptions about epidemiological dynamics.

Since the planner framework involves solving a dynamic optimal control problem, there is a natural trade-off in the degree of richness feasible for the two model components, the economic and the epidemiological. In the case of AAL, this trade-off is resolved by choosing a very flexible policy instrument that allows the degree of lockdowns to vary in each period while keeping the epidemiological model extremely simple.

In this comment we highlight the fact that a number of the simplifying assumptions underlying the SIR model used by AAL are critical when it comes to optimal policy design. While this specification is attractive for its simplicity and allows for a rich formulation of the economic aspects of the planner problem, one should use it with caution, apply correct parameterization, and be aware of the policy implications of using a mis-specified model.

2 The SIR18 Specification

AAL describe epidemiological dynamics using a set of equations known as the SIR model. The first main equation represents the decrease in the pool of susceptibles ($S(t)$) as a result of new infections that occur when infected ($I(t)$) and susceptibles meet and interact such that a new infection is generated. The rate at which such meetings happen is captured by the transmission rate $\beta(t)$.

$$\dot{S}(t) = -\beta(t) \cdot I(t) \cdot S(t) \tag{1}$$

The second main equation describes the evolution of the stock of infected people, which in this model is identical to the stock of people who are infectious. New infections are exactly the outflow from the pool of susceptibles as in (1) and people stop being infected and infectious at rate γ, encompassing both recovery and death:

$$\dot{I}(t) = -\beta(t) \cdot I(t) \cdot S(t) - \gamma I(t) \tag{2}$$

Individuals flowing out of the infected pool, $-\gamma I(t)$, due to either death or recovery, enter the absorbing state R, standing for Resolved.

AAL parameterize this model using two numbers. First, they set $\gamma = 1/18$, based on data of a duration of 18 days till death. Similar numbers have been used by many papers.[1] Second, they set the value of $\beta(t)$ to 0.13 implying a reproduction number of 2.34. This follows from the definition of the reproduction parameter:[2]

$$\mathcal{R}(t) = \frac{\beta(t)}{\gamma}$$

An important point to note about the above SIR specification is that it has one parameter, γ, capture two distinct disease properties – the duration of the infectiousness period and the duration of illness till death/recovery. In reality, these durations are vastly different, as we explain below, and cannot be captured with one number. To highlight the fact that the duration of infectiousness period is set to 18 in AAL, we call this specification $SIR18$.

3 Misspecification and the Speed of Disease

In Bar-On et al. (2021) we provide a detailed description of an epidemiologically-grounded model of disease dynamics and use it in an optimal policy framework. Due to the modelling trade-off described above, we examine lockdown policy instruments which are less elaborate than the one used by AAL.

While we do not claim that AAL should have used an elaborate epidemiological model, we argue that a simple correction to the $SIR18$ specification is warranted in order to bring it closer to the epidemiological evidence, with important implications for the optimal policy path and welfare outcomes. This would not constrain the flexibility of the policy instrument, a prominent feature of AAL.

To derive this correction, and keeping our critique of epidemiological dynamics modelling to a minimum, we focus on two duration numbers that appeared early on in the pandemic in two papers in Science, co-written by researchers from China (Tsinguha, Hong Kong, and others), from the U.S. (Harvard, Princeton, Columbia, Penn State, UC Davis, and NIH), and the U.K. (Imperial, Oxford, and Southampton); see Tian et al (2020) and Li et al (2020). These numbers warrant a different SIR specification than the one used by AAL.

First, several days pass before people who get infected start spreading the disease themselves. This time span is called the latency period. It has been known to epidemiologists since very early in the pandemic and lasts around 3 days.

Second, the duration of the infectiousness period is relatively short, with peak infectiousness lasting around 4 days, after which a person virtually stops to participate in the generation of new cases but has not yet recovered or died.

[1] See CDC estimates at https://www.cdc.gov/coronavirus/2019-ncov/hcp/planning-scenarios.html#definitions Table 2. The median number of days for adults ranges between 16 to 19.

[2] See the discussion in Bar-On et al. (2021) for details about this important parameter and its values.

How should one parameterize the SIR model given these two features of the disease? It seems reasonable to use a parameterization that replicates correct disease dynamics. Wallinga and Lipsitch (2007) derive the relationships between the speed of disease growth, the basic reproduction number, and durations of various states for a number of epidemiological specifications. In particular, they show that for any given value of the basic reproduction number $\mathcal{R}_0$, the speed of the disease growth in a SIR model will approximate the model with the latent and infectious period as described above, if the parameter γ in SIR is set to be the sum of the above two periods,[3] that is, 7. We call this specification $SIR7$.

While $SIR7$ is accurate in terms of the disease speed, it deviates by definition from the documented overall duration of the disease till death, of approximately 18 days. However, this deviation is of second-order importance in the AAL context, as it does not alter the cumulative amount of deaths in the objective function of the planner. It just shifts these deaths backwards in time by less than two weeks, affecting the way the value of deaths is discounted in the objective function, a negligible effect given the two-year planning horizon in AAL.

The reproduction parameter $\mathcal{R}(t)$ reflects the number of new infections generated by an infected person over the course of her illness. Assuming that people are infectious for 18 days, as in AAL, while preserving reasonable values of the reproduction number (like the AAL value of 2.34) implies that the transmission rate $\beta(t)$ is relatively low, ($\beta(t) = 0.13$ in AAL), and as a result, the disease spreads relatively slowly through the population. By contrast, in the $SIR7$ specification, in order to match the same reproduction number of 2.34 with an infectiousness period of 7 days only, one has to conclude that the disease spreads much faster ($\beta(t) = 2.34 \cdot \frac{1}{7} = 0.33$). In fact, for a given value of the reproduction number $\mathcal{R}(t)$, the transmission rate $\beta(t)$ is proportional to the inverse of the infectiousness period. By assuming an infectiousness period almost 3-fold longer, one ends up assuming a disease that is 3 times slower than the real one.

To illustrate the point, Figure 1 presents the dynamics of an unmitigated disease under the AAL specification ($SIR18$) and under the corrected ($SIR7$) specification.

Figure 1

One can immediately see that the epidemic is much more aggressive under the correct specification with the peak arriving two months earlier than in the mis-specified $SIR18$ model.

[3] As shown in Wallinga and Lipsitch (2007, p. 601), in the model with distinct latent and infectious periods, the basic reproduction number $\mathcal{R}_0$, the disease growth rate λ and the latent and the infectiousness periods durations (d_{lat} and d_{inf}) are linked: $\mathcal{R}_0 \approx 1 + (d_{lat} + d_{\text{inf}}) \cdot \lambda$. For the SIR model, Wallinga and Lipsitch (2007) show that: $\mathcal{R}_0 \approx 1 + \frac{1}{\gamma} \cdot \lambda$. So that the dynamics of SIR will be identical to the dynamics of the model with latent and infectious periods iff: $\frac{1}{\gamma} = d_{lat} + d_{\text{inf}}$

4 Policy Implications

In their paper, AAL analyze the optimal path of lockdown policy under the $SIR18$ model. For example, for the baseline case (see their Figure 1 Panel A and upper panel of Table 1), the authors present an optimal lockdown path and find that it will lead to a cumulative welfare loss (loss of lives and loss of output) equivalent to 28% of annual GDP. The cumulative death rate at the end of two years will be around 0.7% of the population, which is $2,310,000$ people in US terms.

However, as we have shown above, the dynamics of COVID19 in reality are much faster than the $SIR18$ dynamics assumed by the planner in AAL. Basing policy on a counter-factually slow disease while dealing with a fast disease has grave consequences.

To illustrate this point, we do the following simple exercise. We take a planner as in AAL[4], who derives an optimal lockdown policy under $SIR18$. We then apply this policy with a disease that in fact evolves according to $SIR7$. We denote this scenario 'AAL' and compare it to the planner who faces exactly the same objective function but derives the lockdown policy from the epidemiological model of $SIR7$, and applies it to a disease that actually evolves according to $SIR7$. We call this scenario 'corrected' to highlight the fact that the policy is derived on assumptions that better reflect actual disease dynamics.

Figure 2 below presents the results of the exercise.

Figure 2

Two main conclusions arise from Figure 2.

First, the top panel shows that optimal lockdown policy based on the properties of the disease looks very different from the AAL policy: the degree of lockdown rises faster, to a level that is higher by almost 10 percentage points. Subsequently, lockdown in the AAL policy is removed quite fast so that by day 50 the economy is fully released, whereas under the correct policy over one-third of the population remains locked at day 50, and the restrictions remain in place till day 80, an entire month longer than under the AAL policy.

Second, the AAL policy leads to worse outcomes in terms of the death toll. The bottom panel of Figure 2 shows that the cumulative death toll is 0.57% under the correct policy, and 0.72% under the AAL policy. In US terms, the correct policy saves almost half a million lives relative to the other one.

5 Conclusions

While simplifications of full-fledged epidemiological models are extremely useful when deriving optimal policy, especially given the proven importance of

[4]We only consider the baseline parameter values used by AAL and the case of no tracing and testing and no quarantine, for simplicity and because these extensions do not affect our results. We use the AAL Matlab code made available by the authors online.

endogenous feedbacks between individual behavior and policy, one should use such simplifications with caution. Dropping some epidemiological parameters and assigning values to others has direct consequences for implied disease dynamics, and eventually, for optimal interventions and the resulting outcomes. One should be aware of such consequences for modelling choices.

In Economics, we are used to debating parameter values and to performing sensitivity analysis when the true value of the parameter in question is unknown (e.g., the coefficient of risk aversion in the utility function or bargaining power in labor market settings). The case of modelling epidemics in Economics is different in two respects. First, basic disease properties like the ones discussed above become the focus of epidemiological research as soon as the epidemic starts to unfold. Therefore, the magnitudes of the key parameters become known in the professional literature quite quickly and with reasonable precision, as is evident from the aforementioned epidemiological studies for the case of COVID19. Second, one of the main outcomes we are interested in when analyzing policy during the epidemic is the death toll. The latter is extremely sensitive to seemingly innocuous assumptions about disease dynamics, as we have demonstrated. Due to the extremely important nature of this outcome, inaccurate specifications that are eventually biasing policy and leading to high death tolls cannot be taken as legitimate variations on correctly specified models.

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