Differential Games in Spread of Covid-19

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Abstract—Given the ongoing Covid-19 pandemic, it is of interest to understand how the disease spread is affected by planner (government) intervention and population behaviors. In this work, the spread of Covid-19 is modeled as a differential game between the planner and population. Susceptible-Infected-Recovered (SIR) disease dynamics are modified to incorporate these choices. In this framework we characterize the joint equilibrium exposure profile between the planner and population. Additionally, as in case of Covid-19, the role of asymptomatic carriers, inadequacies in testing, contact tracing and quarantining can lead to a significant underestimate of the true infected numbers as compared to just the detected numbers. Therefore, it is vital to model the true infected numbers within the context of choices made by individuals within the population. To incorporate this, we extend our framework by modifying the dynamics to include additional sub-compartments of ‘undetected infected’ and ‘detected infected’ in the disease dynamics.

I. INTRODUCTION

Infectious diseases spread because of interactions between the infected and the susceptible. At an individual level, a simple strategy to reduce the possibility of transmission is to voluntarily reduce ones interaction with others, i.e., to do social distancing. A central planner aims to impose constraints to individual behavior so as to maximize the total societal welfare. To model the disease spread effectively it is important to combine the choices of both the planner and the individuals in a unified way. The main goal of this work is to formulate a game theoretic framework in which one can analyze and characterize the resulting equilibrium between the individuals and the planner. A secondary goal is to modify the disease spread dynamics so as to account for the spread of disease by infected individuals who are not detected and isolated from the susceptibles. This also leads to incorporating the individual’s estimate of infection in the disease spread model. We present some simulation results based on these results to demonstrate how population behavior, planner control, detection rates and trust in the reported numbers play a key role in the disease spread.

A. Related Work

In mathematical epidemiology the spread of diseases is often modeled through various compartmental models. The simplest of them is the SIR model [2]. A considerable literature has been built up to include many extensions and variations to this basic model (see for example [3], [4], [5] and references therein). An issue with these models is that they don’t capture interventions of government nor individual choices. These decisions can have a significant impact on the disease spread trajectory.

Prior to the outbreak of Covid-19 some applications of optimal control in field of epidemiology include masking rates to prevent swine flu [6], treatment rates in dengue transmission [7], etc. Since the Covid-19 pandemic began there have been a considerable number of works which formulate optimal Non-Pharmaceutical Intervention (NPI) as a control problem. [8] proposed a a lockdown that tapers down gradually. [9] has a multi-group SIR model in which the authors look at the optimal control problem for the planner can do age-specific targeted lockdowns. They show that the optimal solution is to enforce stringent lockdowns for the older section of the population. [10] shows that intermittent lockdowns may be optimal for a class of utility functions in low sero-prevalence scenarios. In a line of work closely related to the current work, [11] and [12] combine game theoretic equilibrium analysis based on utility considerations of the individuals with the SIR model. It is clear from disease trajectories of many countries that it is not sufficient to study a control problem for a planner or the equilibrium strategies of the population separately. In all these models, they don’t study combined interaction between individual choices and governmental policies. The above models also don’t incorporate how perceptions of the extent of disease spread affects the further spread of the disease. Please refer to [1] for further references and details.

II. PRELIMINARIES

A. Game theoretic setup

We assume that the game is played till a finite horizon time $T$. This $T$ can be interpreted as the idealized vaccine arrival time wherein the vaccine affects the entire population instantly and puts an end to the disease.

The planner tries to control the spread of disease by imposing constraints on individual exposure choices. The individual tries to modify their behavior by either reducing or increasing their exposure fraction at any time $t$ while complying with the constraints imposed by the planner.

A strategy $A$ $\in$ $C[0,T]$ $\nonumber^1$ of the planner is such that $A_2$ $\in$ $[0,1]$, $\forall t \in [0,T]$. $A_i$ sets the maximum permissible exposure of an individual at time $t$. $A_i$ represents the restrictions imposed by the planner on the individual’s

\nonumber^1C[0,T]$ refers to the space of continuous functions taking values from $[0,T]$ to $\mathbb{R}$.

\nonumber^2A_i = A(t)$, i.e, the value of the function $A$ at time $t$. Similarly for $g_i$.  
exposure profiles in the form of lockdowns, closing down schools, restricting public transport and other NPIs. At times, also consider the strategy \( g^\alpha \) of a canonical individual \( \alpha \) differing from the symmetric strategy \( g \) employed by the rest of the population.

B. Dynamics of Disease Spread

The evolution of susceptible fraction \( S_t \in [0,1] \) and the infected fraction \( I_t \in [0,1] \) is given by:

\[
\begin{align*}
\frac{dS_t}{dt} &= -\beta g_t^2 S_t I_t \\
\frac{dI_t}{dt} &= \beta g_t^2 S_t I_t - \gamma I_t
\end{align*}
\]

with the initial conditions \( S_0 = 1 - \epsilon, I_0 = \epsilon \). \( \epsilon \) is the initial fraction of infection in the population, \( \beta \) is the probability of getting infected per interaction with an infected individual and \( \gamma \) is the recovery rate from infection. If the entire population plays a uniform exposure fraction \( g_t \), the effective susceptible and infected fractions are \( g_t S_t \) and \( g_t I_t \) respectively, and the total number of interactions between the susceptibles and infected is \( g_t^2 S_t I_t \). This explains the quadratic dependence on \( g_t \). We note that if all individuals played \( g_t = 1, \forall t \in [0,T] \) then we get the standard SIR model.

A canonical individual \( \alpha \), will get infected at an unknown random time \( \tau_\alpha \). We assume that each individual has an estimate for their own survival probability which they estimate through a hazard rate model

\[
\frac{dP(\tau_\alpha > t)}{dt} = -\beta g_t^\alpha g_t P(\tau_\alpha > t)
\]

Here \( g_t^\alpha \) is the exposure fraction strategy played by the individual \( \alpha \) and \( P(\tau_\alpha > t) \) is the survival probability at time \( t \). This estimate of the survival probability and the trade off between benefits and risks of exposure will drive the individual’s exposure strategy.

C. Cost Functionals of Players

For simplicity, we assume \( \alpha \) gets a linear rate of benefit \( B \) per unit time from interacting with other individuals in the society. We also assume that upon contracting the disease, the individual suffers a one time cost \( C \). Thus, \( \alpha \) minimizes the following cost functional \( J_\alpha \) where ( in what follows \( \mathbb{1}_E \) is the characteristic function of a set \( E \))

\[
J_\alpha(g, g^\alpha, A) = \mathbb{E}\left[ -\int_0^{T\wedge \tau_\alpha} B g_t^\alpha ds + C \mathbb{1}_{\{\tau_\alpha \leq T\}} \right].
\]

The expectation above is with respect to the \( \alpha \)'s survival probability defined in (2) above. An equivalent formulation of the functional\(^5\) more suited for optimal control methods we seek to apply is

\[
J_\alpha(g, g^\alpha, A) = \int_0^T P(\tau_\alpha > t) g_t^\alpha \left\{ -B + C \beta g_t I_t \right\} dt.
\]

In the objective functionals above, we impose the restriction \( g_t^\alpha, g_t \leq A_t \).

An individual gets a benefit of \(-B g_t \Delta t\) and has an expected cost of infection \( C \beta g_t I_t \) from an exposure strategy of \( g_t \) in the interval \([t, t+\Delta t]\). Thus, a measure of cost borne by the entire society is then \((-B + C \beta g_t I_t) g_t S_t \Delta t\) during this interval. The above discussion motivates the planner’s functional to be

\[
J_P(g, A) = \int_0^T S_t g_t \left\{ -B + C \beta g_t I_t \right\} dt
\]

III. MAIN RESULTS

A. Pure Population Equilibrium Without Detection

We shall assume that there is no control from the planner, i.e., \( A_t = 1 \) for all \( t \in [0, T] \). It is only a game between the individuals of the population. An equilibrium result similar to that mentioned in this subsection can be found in [11]. To derive the equilibrium exposure strategy we use the Pontryagin Minimum Principle (PMP) (see section 3.3 in [13]). Let \( \alpha \) play the strategy profile \( g_t^\alpha \) and let the rest of the population play \( g_{eq,t} \). Then in equilibrium we have

\[
J_\alpha(g_{eq}, g_{eq}^\alpha, 1) \leq J_\alpha(g_{eq}, g^\alpha, 1)
\]

\[
g_{eq}^\alpha = g_{eq,t}
\]

The first condition is the definition of an equilibrium (Nash) while the second equation follows from the symmetric assumption on the individual’s exposure profile.

Theorem 1: For the dynamical game without a central planner the equilibrium exposure profile must be of the form:

\[
g_{eq,t}^\alpha = g_{eq,t} = \min \left( \frac{B}{\beta I_t(C - \lambda_t)}, 1 \right) \mathbb{1}_{\{C > \lambda_t\}} + \mathbb{1}_{\{C \leq \lambda_t\}}
\]

The corresponding dynamics are given in (1-2). The differential equation for the adjoint variable \( \lambda_t \) is given by

\[
\frac{d\lambda_t}{dt} = g_{eq,t} (B - \beta I_t g_{eq,t} (C - \lambda_t))
\]

with boundary conditions: \( S_0 = 1 - \epsilon, I_0 = \epsilon, \lambda_T = 0 \).

\(^3\)\( g_t, \beta, \epsilon, B \) and \( C \) are positive real constants.

\(^4\)\( P(\tau_\alpha > t) \) is the probability of getting infected before \( t \).

\(^5\)This form shows the explicit dependence of \( J^\alpha \) on \( g, g^\alpha \) and \( A \).
**Proof Sketch.** For the complete proof please refer to [1, Theorem 1]. We provide only a proof sketch here.

1. Consider $\alpha$’s control problem assuming the rest of the population plays $g_{eq,t}$. The control $g^\alpha\epsilon$ shows up in the Hamiltonian from the cost (3) and equation (2).
2. Minimize the Hamiltonian w.r.t $g^\alpha$ while imposing $g^\alpha_{eq,t} = g_{eq,t}$.
3. The adjoint boundary conditions follow from transversality conditions of PMP.

One can show that (see [1, Appendix A] for proof)

$$\lambda_t = E \left[ \int_{t}^{T} -BG_g^{\alpha} ds + C \mathbb{1}_{\tau_a \leq t} \right] \text{ where } \tau_a > t \tag{6}$$

Thus, $\lambda_t$ can be interpreted as the future expected cost given the individual has survived till time $t$. With this interpretation the equilibrium profile in Theorem 1 implies that whenever either the total number of infections or the future expected costs are high then the population starts social distancing until the number of infections decrease.

**Lemma 2:** $\lambda_t$ is a non-decreasing function of $t$. More precisely whenever $g_{eq,t} < 1$ then $\lambda_t$ is a constant and is strictly increasing whenever $g_{eq,t} = 1$.

**Proof:** This is easily verified by looking at the expressions for $g_{eq,t}$ and $\frac{d\lambda_t}{dt}$ from Theorem 1.

Intuitively, this is because for any arbitrarily small interval the equilibrium strategy must accrue more benefit than a strategy of complete social distancing, i.e., $g_t = 0$ within the same time interval.

**Lemma 2** indicates that in equilibrium the dynamics is a hybrid one with the value of $g_{eq,t}$ triggering the switch between the states of social distancing and normal behavior. When $g_{eq,t} = 1$ we have normal behavior with no social distancing and the dynamics of $S_t, I_t$ is just the same as the SIR model.

**B. Equilibrium with Population and Planner**

In this case the planner is trying to optimally set a threshold to minimize its own cost function. The constraint on the planner is that if it sets a high enough threshold then individuals behavior may follow the pure population equilibrium from the previous subsection (and hence the threshold $A_t$ becomes non-binding). We have the constraints that $0 \leq g_t \leq A_t$ and $0 \leq A_t \leq g_{eq,t}^{pop}$\footnote{$g_{eq,t}^{pop}$ refers to the pure population equilibrium described in the (5).}. In this case at equilibrium we must have

$$J_\alpha(g_{eq}, g_{eq,t}^{pop}, A_{eq,t}) \leq J_\alpha(g_{eq}, g^\alpha, A_{eq,t}),$$

$$J_P(g_{eq}, A_{eq}) \leq J_P(g_{eq}, A),$$

$$g_{eq,t}^{pop} = g_{eq,t}.$$  \tag{7}

As the set of admissible controls for the players vary with both time and state we use a generalized version of PMP (Theorem 3.1 in [14]). We have the following result characterizing the equilibrium between population and planner:

**Theorem 3:** The population-central planner game has the following equilibrium profile:

$$g_{eq,t} = \min \left( \frac{B}{\beta \epsilon_t(C - \lambda_t)}, A_{eq,t} \right) \mathbb{1}_{\{C > \lambda_t\}} + A_{eq,t} \mathbb{1}_{\{C \leq \lambda_t\}}$$

$$A_{eq,t} = \min \left( \frac{B}{2\beta \epsilon_t(C - \lambda_{1,t} + \lambda_{2,t})}, g_{eq,t}^{pop} \right) \mathbb{1}_{\{C + \lambda_{2,t} > \lambda_{1,t}\}}$$

$$+ g_{eq,t}^{pop} \mathbb{1}_{\{C + \lambda_{2,t} \leq \lambda_{1,t}\}} \tag{8}$$

with $g_{eq,t}^{pop}$ as defined in (5) of Theorem 1. The corresponding dynamics are given in (1-2). The differential equation for the adjoint variables is given by

$$\frac{d\lambda_t}{dt} = g_{eq,t}(B - \beta g_{eq,t} \epsilon_t(C - \lambda_t)),$$

$$\frac{d\lambda_{1,t}}{dt} = g_{eq,t}(B - \beta g_{eq,t} \epsilon_t(C + \lambda_{2,t} - \lambda_{1,t})),$$

$$\frac{d\lambda_{2,t}}{dt} = -\beta g_{eq,t} \epsilon_t S_t(C - \lambda_{1,t} + \lambda_{2,t}) + \gamma \lambda_{2,t},$$

with boundary conditions: $S_0 = 1 - \epsilon, I_0 = \epsilon, \lambda_T = 0, \lambda_{1,T} = 0, \lambda_{2,T} = 0$.

**Proof Sketch.** For the complete proof please refer to [1, Theorem 4]. We provide only a proof sketch here.

1. Consider $\alpha$’s control problem assuming the rest of the population plays $g_{eq,t}$ and the planner control $A_t$. Minimize the Hamiltonian w.r.t $g^\alpha$.
2. Now consider the planner’s control problem with restriction $g_{eq,t}^{pop}$. The planner’s Hamiltonian will include the adjoint variable in (5).

It can be easily seen that $A_{eq,t} = g_{eq,t}$ and hence the planner’s threshold is always binding. The exposure profile of the population is also seen to be the net result of the strategic choices of the planner and the population.

**C. Detection of Infection**

In this section we partition the infected group $I_t$ into two subgroups - $I_{d,t}$, the undetected group of infected and $I_{d,t}$, the detected group of infected. We assume that once the infected are detected they are effectively quarantined and no longer infect the susceptibles. Hence, an infected individual
either recovers without being detected or gets quarantined after detection. An infected individual is modeled to remain infectious for a period of $\frac{1}{\gamma}$ and has a probability of being detected in this period. For an individual $\alpha$, conditioned on the event $\tau_\alpha = t$, we assume a probability density of detection over the period $(t, t + \frac{1}{\gamma})$. $\tau_d$ denotes the random time of detection once $\alpha$ is infected. Thus $\tau_d \in [0, \frac{1}{\gamma}]$. For simplicity, we shall assume that the probability of detection is uniform over $[0, \frac{1}{\gamma}]$. Thus, the individual’s objective functional becomes:

$$
E \left[ \int_0^{(\tau_\alpha + \tau_d)\wedge T} \left(-Bg_\alpha^t dt + C I(\tau_\alpha \leq T)\right) \right]
$$

Here $\frac{1}{\eta}$ (with $\eta > 1$) captures the probability of detection and $\gamma$ is a parameter in the model. Setting $M_t := \gamma \int_{t-\frac{1}{\eta}}^t P(\tau_\alpha > r)dr$, we can rewrite (see [1, section III-C]) the individual’s objective functional as (superscript $d$ stands for detected):

$$
J'_\alpha(g, g^a, A) = -B \int_0^T g_\alpha^t \left\{ P(\tau_\alpha > t - \frac{1}{\gamma}) \left(1 - \frac{1}{\eta} \right) + \frac{M_t}{\eta} \right\} dt + CP(\tau_\alpha \leq T).
$$

(9)

The individual $\alpha$ has knowledge only of $I_{d,t}$ and makes an estimate of $I_{u,t}$ from $I_{d,t}$. For simplicity, we assume that the estimate has the form:

$$
\widehat{I}_{u,t} = \kappa I_{d,t}.
$$

$\kappa$ encapsulates the trust the population has on the reported detected numbers. Now as $P(\tau_\alpha > t)$ is linked to the individuals perception of infection, we must modify (2) to:

$$
\frac{dP(\tau_\alpha > t)}{dt} = -\beta g_\alpha^t g_t \widehat{I}_{u,t} P(\tau_\alpha > t),
$$

$$
= -\beta g_\alpha^t g_t \kappa I_{d,t} P(\tau_\alpha > t).
$$

The state equations for the individual (set $P_t := P(\tau_\alpha > t)$) are:

$$
\frac{dS_t}{dt} = -\beta g_\alpha^t g_t I_{u,t},
$$

$$
\frac{dI_{u,t}}{dt} = \beta g_\alpha^t g_t I_{u,t} - \gamma(1 + \frac{1}{\eta}) I_{u,t},
$$

$$
\frac{dI_{d,t}}{dt} = \frac{\gamma}{\eta} I_{u,t} - \gamma I_{d,t},
$$

$$
\frac{dP_t}{dt} = -\beta g_\alpha^t g_t \kappa I_{d,t} P_t,
$$

$$
\frac{dM_t}{dt} = \gamma(P_t - P_{t-1/\gamma}).
$$

(10)

New infections are caused by the interaction between the susceptibles and undetected infected. These new infections are initially always assumed to be undetected. Then, some of the undetected infected move to $I_{d,t}$ due to the detection density $\gamma$.

The control formulation now has constant delays in state variable $P_t$ for both the objective functional and state equations. These types of control problems are called Retarded Optimal Control Problems (ROCP). We shall use a version of the minimum principle for this ROCP (see theorem 4.2 in [14])

$$
T
$$

(11)

Theorem 4: The population game with detection has the following equilibrium profile:

$$
R_{eq,t} = \min \left( \frac{B((1 - 1/\eta)P_{t-1/\gamma} + M_t/\eta)}{\beta P_t \kappa I_{d,t} (C - \lambda_t)}, 1 \right) I_{\{C > \lambda_t\}} + \mathbb{1}_{\{C \leq \lambda_t\}},
$$

with boundary conditions: $S_0 = 1 - \epsilon, I_{u,0} = \epsilon, I_{d,0} = 0, \lambda_T = 0$.

Proof Sketch. For the complete proof please refer to [1, Theorem 5]. We provide only a proof sketch here.

We apply theorem 4.2 from [14] to the ROCP of the individual $\alpha$. Compared to Theorem 1, the Hamiltonian also incorporates the delayed state variables. Consequently, the adjoint equations and the optimal control depend on these delayed variables.

IV. SIMULATION RESULTS

The theorems derived in section III provide a basis for simulating a dynamical system with initial infection. The equilibrium solution to the game leads to solving a system a differential equations with a two point boundary condition. This is fairly typical in optimal control and is due to the PMP (see [1, Section IV] for more details of the simulation).

Table I: Parameter values used in simulations.

| $T$  | $\epsilon$ | $B$ | $C$ | $\beta$ | $\gamma$ |
|------|------------|-----|-----|--------|--------|
| 400  | 1.65e-08   | 0.01| 1   | 0.2    | 0.05   |

The values for the various parameters in the simulation are given in Table I above. The parameters in the detection case - $\eta$, the probability of detection, and $\kappa$, trust in detected
infected. As the infection numbers inevitably rise the population voluntarily reduce exposure below even the planner’s threshold. This results in the peak infection becoming plateaued in a manner similar to population equilibrium. The social distancing and thresholding is gradually reduced as we approach T. Compared to the population equilibrium case the peak infection is delayed and the economic impact (as measured by exposure time) is reduced.

High detection ($\eta = 1$) and high trust ($\kappa = 1$) leads to a lower peak than SIR but unlike the population or population planner cases the peak is not prolonged. The peak infection is delayed compared to SIR. There is no social distancing due to high levels of detection and trust. This reduces the adverse economic impact due NPIs.

In figures 2a and 2b, we plot the effects of different detection rates ($\eta$) and trust levels($\kappa$) on the spread of disease. When detection rates are high ($\eta = 1$) but trust ($\kappa = 32$) is low (dashed dark green), then as soon as infected numbers peak the population drastically reduces exposure. This is due to the low trust in the detected numbers. The population believes the planner is doing a poor job of the detection even though, in reality, the detection rates are high. This leads to unnecessary economic loss. In the low detection ($\eta = 5$) but high trust ($\kappa = 1$) (light green) case, the disease spread curve is very close to the SIR situation. The population is misinformed about the disease spread due to high trust and low detection. The undetected infected comprise the majority of infected numbers and continue to spread the disease unhindered. This is similar to the SIR situation where there is neither planner control nor any social distancing. In the low detection ($\eta = 5$) and low trust ($\kappa = 32$) (dashed light green) case, the population reduces exposure completely as soon as the infection numbers start to peak. However, compared to the high detection case, infected numbers are higher since most of them go undetected.

The various comparisons seem to suggest ideally for controlling disease spread one needs to have high detection rates with transparency in the reported numbers so that the population has confidence in the reported numbers. If the detection numbers are low and the planner tries to underplay the poor detection it could result in the worst possible scenario - an unmitigated disease spread which could stress the medical resources at peak infection. In practice, it is often difficult to achieve very high detection rates due to asymptomatic carriers, testing errors etc. and hence must be combined with some moderate amount of planner control in form of lockdowns and imposing social restrictions. The population also has an important role to play by voluntarily reducing exposure and other NPIs (wearing numbers, are varied to understand their impact on the disease spread.

In figures 1a and 1b, we plot the infection and exposure fraction, respectively, versus time. SIR (blue) shows a sharp peak infection (indicative of exponential decline in susceptibles). This is the worst case scenario - no social distancing, no detection, no quarantining and no planner control. It has the highest peak infection whose onset is advanced compared to other scenarios. Population equilibrium (black) shows a prolonged infection peak. The exposure profile indicates that the population starts to socially distance approaching peak infection and only stops it close to $T$. In planner-population (red), the planner initially sets a moderate threshold to control the spread of the disease. This results in a delayed peak. As the infection numbers inevitably rise the population voluntarily reduce exposure below even the planner’s threshold. This results in the peak infection becoming plateaued in a manner similar to population equilibrium. The social distancing and thresholding is gradually reduced as we approach T. Compared to the population equilibrium case the peak infection is delayed and the economic impact (as measured by exposure time) is reduced.

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Detection rates and the trust in the detected numbers play a crucial role in the spread of disease - too little detection with unfounded trust in detected numbers can lead to an unmitigated spread of the disease while too little trust with high detection leads to unnecessary loss of economic activity. Simulation results supporting these conclusions are presented.

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