A dynamic reaction-restore-type transmission-rate model for COVID-19

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Abstract: COVID-19 became a paradigmatic global pandemic for science, in a real laboratory inserted in reality to understand how some dangerous virus spread can occur in human populations. In this article, a new strategic epidemiological model is proposed, denoted β-SIR. It is because the transmission rate β follows a proper dynamic law, more precisely a reaction-restore type transmission rate model. Some analytical results associated with dynamic consequences are presented for variables of epidemiological interest. It is concluded, observing the geometry of variables plots, such as transmission rate, effective reproductive number, daily new cases, and actives, that pandemic propagation is very sensible to the population behavior, e.g., by adherence to non-pharmaceutical mitigations and loss of compliance levels.

Key-Words: Infection disease, SIR model, variable transmission rate, COVID-19.

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1 Introduction

The use of ordinary differential equations (ODE) in the epidemiological analysis of infectious diseases presents a history of explosive growth, [1], [2], [3], [4], [5], [6], [7], [8], [9], [10], [11], [12], far above other mathematical possibilities, [13], [14], [15], [16], [17], [18], [19], [20], [21]. As a tool, the ODEs stand out for their ability, via interpretation, to describe, explain, and project (i.e., to model), dynamics of contagion and population spread of diseases, which turns them into virtual instruments for testing control actions. Thus, there is the possibility of establishing *ex-ante* evaluation scenarios simulating population interventions (e.g., in crisis contexts), such as mitigation measures or pharmaceutical-type solutions, [22], [23], [24], [25]. There is also the possibility of *ex-post* type analysis, that is, those that seek to determine the necessary conditions of the past to explain the information and data of the present, [26], [27], [28], [29]. Another important aspect is the possibility of determining threshold parameter values with epidemiological significance, such as in herd immunity, knowing the immune fraction to stop an epidemic. They are also an important instrument for optimization (according to certain objectives and restrictions) between alternative health control strategies that have been used in the past or are even in the design stage, [30], [31], [32], [33].

Every expansion has a beginning; let us briefly refer to the initial uses of ODEs as an instrument for epidemiological analysis. Leaving aside the work of the considered founder of demography [34], by introducing the first life table presenting mortality as survival rates [35], a properly mathematical perspective, for the study of the spread and control of infectious (integrating an ODE) appears with the work of [36], in 1786 [37], [38], on smallpox and the effect of variolation. However, it is a chronologically isolated milestone concerning subsequent methodological developments. To our knowledge, it was not until a period at the beginning of the twentieth century that the idea of using mathematical analysis as a methodological possibility for epidemiology began more clearly. A sketch, mainly focused on the contributions of [39], in 1906 [40], the introducer of the law of mass action in a childhood infections paper, including measles, as well as the essential reference to the contribution of [41], with a focus on a mosquito population as vectors, who in the second edition of *The Prevention of Malaria*, published in 1911, builds mathematical models of malaria transmission, [42]. Nevertheless, if we are to seek the characterization of foundational work, that first scientific article that succeeds, by expanding its potential effectively, inaugurating a disci-

pline, Mathematical Epidemiology, is the SIR model (Susceptible - Infected - Recovered) as we know it today, a purification by simplification of the triad of works by [43], [44], [45], in the years 1927, 1932, and 1933, respectively.

To a large extent, the importance and widespread use of the SIR model, as a model of the population dynamics of infectious disease spread, lies in the efficient and effective concurrence of a mechanistic and generic explanatory perspective, on the one hand, and the fair mathematical technicality on the other, [46]. The plausibility of this model has at least two sources: a theoretical argumentation of hypotheses about the process in the abstract and a certain geometric correspondence of the numerical projections with the data. It is this minimalist character, or low cost in complexity, to represent the essentials of the epidemiological process, which allows it to be classified as a strategic model, [47], in contrast to a tactical model that aspires to high fidelity with a specific reference of reality (a certain disease and population, considering many variables and precise relationships, always aspiring to a high predictive capacity).

In the standard SIR model, that is, with constant coefficients, if $\mathcal{R}_0 > 1$, we see that the asset curve has a bell-shaped or unimodal shape. That is, it has a single maximum that occurs when the susceptible fraction (a decreasing function) is equal to the value $1/\mathcal{R}_0$, with $\mathcal{R}_0$ the basic reproductive number or the average number (at the beginning of the disease) of the new infective directly infected by an infective while in said condition. The first estimates (February 2020) for CO-VID-19 of $\mathcal{R}_0$ were in the range $1.40 - 6.49$, see, [48], already around May of the same year, a systematic review reduced it to $2.81 - 3.82$, [49]. Another study for Western European countries, with data until mid-March 2020, places it at $1.90 - 2.60$, [50]. So, for example, with a $\mathcal{R}_0$ equal to 1.5 or 3 for COVID-19, we have that said maximum would imply a susceptible percentage between 33.3% and 66.6%; which respectively implies between 2/3 and 1/3 of the population already infected, which is absolutely far above what was observed for the first wave, in many countries. Thus, the conclusion is that the countries or communities observed in general managed to lower the contagion rate by changing behavior regarding contact with others, obviously in voluntary terms or by mandate of the health authority, through, for example, mitigating measures, non-pharmaceutical type.

A strategic model, that is, a simple and generic one, must have the potential to capture with a good degree of precision the dynamics of the disease. For example, the fact that the intensity of transmission can change over time in response to behavioral changes in the population due to official control interventions. It is the case, for example, in models based on the SIR model, that intervenes in the transmission rate (normally denoted β), which is constant in the standard model, to represent some types of changes, e.g. in the number of close contacts, environmental conditions, or blockades in the passage of pathogens. In this regard, two possibilities are visualized: using an explicit function to represent the dependence of β on time or another variable or indicator, [51], [52], or an implicit one, [53]. In the latter, we represent changes in beta, for example, through a dynamic law for the derivative of beta. The novelty of our paper, inspired by [54], is to introduce a new type of beta-SIR model and to continue to study its dynamical consequences.

There are other possibilities to introduce natural variability. For instance, probability distributions for the intensity of transmission in a given period, have been useful for modeling outbreaks in small-sized populations or for accounting for the volubility of human behavior. In the case of non-uniform transmission, there are also network models, to represent interactions in local groups, e.g., family, friends, or work, which differ in the magnitude of the distance and the interaction times. Another way to incorporate variability is to collect the experience of a type of transmission behavior (adjusting to the data), so that it is an input to project outbreaks in another period in populations with some degree of similarity to that which the experience had. In conclusion, if it is necessary to represent more complex behaviors, one way is to consider the variable beta by abstracting and interpreting the forces and conditioning that intervene in determining the epidemic dynamics.

2 The model

2.1 Some preliminaries

Let us consider an SIR model, i.e., the population is theoretically divided into susceptible, infectious and removed groups, of respective sizes, at a time t, defining functions $S(t)$, $I(t)$ and $R(t)$, such that

$$\begin{cases} S' = -\beta(t)SI/N \\ I' = \beta(t)SI/N - \gamma I \\ R' = \gamma I, \end{cases} \tag{1}$$

in which a temporally variable transmission rate $\beta(\cdot)$ stands out, but with parameters γ (recovery rate) and N (total population $S + I + R$) constant.

The first order condition necessary for a minimum or maximum of the active group, that is, $I' = 0$ at some instant τ, since $I' = \gamma\{\mathcal{R}_e(t) - 1\}I$, with $\mathcal{R}_e = (\beta/\gamma)S/N$, implies $\mathcal{R}_e(\tau) = 1$, or equivalently

$$s(\tau) = \gamma/\beta(\tau), \tag{2}$$

where $s(\cdot) = S(\cdot)/N$, by (1), is a decreasing function.

Then, this equality can occur at several moments, depending on the variability of $\beta(\cdot)$. Thus, the model can break the unimodal character of the typical active SIR curve with β as a constant parameter. Thus, if τ_1 and τ_2, with $\tau_1 < \tau_2$, are two consecutive critical points of $I(\cdot)$, as $s(\tau_1) > s(\tau_2)$, the relation (2) implies $\beta(\tau_1) < \beta(\tau_2)$. Moreover, if there is a peak at τ_1, then there is the possibility of a second peak at a certain τ_3, $\tau_3 > \tau_2$, to the extent that β increases from a minimum at τ_2.

In the literature associated with the mathematical modeling of infectious diseases, several ways are found to incorporate changes in the transmission rate due to environmental variations and human behavior throughout a process of serious epidemic development in a certain population. One way is to assume $\beta(\cdot)$ as a specific and predetermined function of time, as is the case in [55], [56], [57]. However, we also find models that innovate by introducing a dependence on other compromised variables, but still explicitly, as in [58], [59]. However, some works do not predetermine it: In [60], it is assumed that the intrinsic transmission rate changes as a stochastic process with values limited to a limited range of possibilities. In [61], the transmission rates involved are solutions of a differential equation with stochastic noise. In [62], a time-varying transmission rate is considered, assuming loss of adhesion to control actions, as lockdown measures. Here, we will consider an evolving rate $\beta(\cdot)$ according to the theoretical dynamics of the process itself; this is the novelty context of the present work.

In [54], a dynamic law is introduced to express the variability of the transmission rate of the type

$$\beta'(t) \,/\, \beta(t) = \underbrace{\text{restitution}}_{f(\cdot)} - \overbrace{\text{reaction}}^{g(\cdot)} \tag{3}$$

that is, expressing the variability of $\beta(\cdot)$ as a constant tension between the factors that tend to decrease (reaction) and increase (restitution). Observe that, having $\beta(\cdot)$ [time^{-1}], it corresponds to $\beta'(\cdot)$ [time^{-2}], that is, (3) expresses acceleration changes. In this sense, $f(\cdot)$[time^{-1}] and $g(\cdot)$[time^{-1}] correspond to proportions of decrease and increase in transmission rate per time unit.

Both the reaction factor and the restoration factor operate through the way that individuals in the population in question change their behavior regarding aspects that mean a change in the rate of contact with infectious diseases or in the intensity of pathogenic blockade. From the experience with COVID-19, we know that population sectors stopped adhering to mitigation measures for various reasons, many of them sensitive to economic and cultural situations. Although the most widespread would be psychological (pandemic fatigue, [63],[64]), economic (economic insecurity, [65], [66]) and informational (low-risk perception, [67], [68], [69], [70]) types. The existence or expectation of a vaccine and its implementation also alters risk perception.

Regarding the reaction factor, we observe that the recommendations suggest that the health authority, normally advised by a technical team of several experts, use various sources of information and indicators to define the health status of the population and implement mitigation measures, as occurred due to the COVID-19 pandemic. Key information sources include numerical epidemiological data (prevalence, daily cases, reproductive numbers, etc.) that track the strength of the infection and its components, such as causal variables that explain the intensity of the virus spread. It is about diagnosing and projecting to plan an intervention, a deployment of mandates that aim to guarantee the system's medical care capacity for the most serious cases.

A key element in understanding the restitution factor is that, although there may be a significant social effort to reduce the natural (or intrinsic) rate of transmission, the sustainability of this becomes difficult over time. In fact, the greater and longer the exigencies for behavior changes in the population, the more likely it is to be lost in compliance.

2.2 The $\beta(\cdot)$-SIR model

The main novelty of this work is to consider what we denote by β-SIR model, that is, we have the susceptible - infective - removed compartmental model defined by (1), but now, this is coupled with a variable transmission rate $\beta(\cdot)$ following the structure (3), via the differential law:

$$\beta'(t) \,/\, \beta(t) = \underbrace{\nu(\beta_* - \beta(t))}_{f(\beta(t))} - \overbrace{\mu I'(t)}^{g(I'(t))}, \tag{4}$$

with the initial condition $\beta_0 := \beta(0)$, $S_0 := S(0) = N - I_0$, $I_0 := I(0) > 0$ and $R_0 := R(0) = 0$. An important assumption is that $\mathcal{R}_0 = \beta_0/\gamma$ is greater than one, so $I'(0) > 0$. The parameters ν and μ are called the coefficient of restitution and the reaction coefficient, respectively.

Observe that in the formulation of (4) we consider that the relative change in the rate is given by the tension between a reaction factor $g(\cdot)$ which is proportional to a difference in each unit of time, the number of people who become infectious minus those who leave the condition. While, the restitution factor $f(\cdot)$ fulfills the function of putting pressure on the transmission rate to return to a value, called potential or

cultural (the one that would exist if there was no reaction), of the rate equal to β_*. Therefore, if $\beta < \beta^*$ (*resp.* $>$) the restitution factor is positive (*resp.* negative) exerting upward pressure (*resp.* downward).

An important aspect is that the β_* rate is considered the intrinsic rate, which would occur in a virgin population of both infectives and information or actions that have changed the natural (habitual) behavior of people. In this sense, we see that β_* does not necessarily coincide with $\beta_0 := \beta(0)$ and, in general, it can be considered that $\beta_0 \leq \beta_*$.

3 Analytical results

3.1 Case $\nu = 0$

Let us note that equation (4) takes the form $\beta' = -\mu I' \beta$, which deduces the existence of automatism in the control, that is, β decreases if and only if I grows. As the system starts with $I(\cdot)$ increasing, we see that the peak of the assets occurs simultaneously with the minimum value that the transmission rate can reach. Observing that at critical times τ ($I'(\tau)$ and $\beta'(\tau)$ null), we have the equality $\beta''(\tau) = -\mu\, I''(\tau)\beta(\tau)$, we deduce that temporally maxima (*resp.* minima) of $I(\cdot)$ correspond to minimums (*resp.* maximums) of $\beta(\cdot)$.

The literature offers several examples that assume an exponential-type functional expression to establish a decrease in the contagion rate (see, [51], [52]). However, several of them do not offer a return to the original rate, but rather an asymptotic approximation to a new level, although lower than the initial one. In this case, we are assuming, by direct integration of (4), that

$$\beta(t) = \beta_0\, e^{-\mu(I(t)-I_0)}, \quad t \geq t_0. \tag{5}$$

Thus, it is also exponential, but it correlates inversely with the number of assets. In this formulation, which only considers a reaction factor, it also differs in that if the active group is expressed with values below I_0 (e.g., a certain reaction threshold), the value of β increases to β_0. By the way, this is quite an optimistic possibility, in that an arithmetic increase in the active group translates into a geometric reduction in the contagion rate.

In the idea of interpreting the reaction coefficient, let us note that the reaction factor $g(\cdot)$ is equal to μ when $I'(\tau) = 1$ for some instant τ. Linearizing $I(t)$ at this moment, we have $I(t) \sim I'(\tau)(t-\tau)+I(\tau) = (t-\tau)+I(\tau)$, so if $t = \tau + 1$, we have $I(\tau+1) \sim I(\tau)+1$. Then μ is the fraction by which β decreases so that the system does not incorporate one more infectious agent per unit of time. Thus, μ can be considered a uniparametric measure of the intensity of the effort required to lower the transmission rate. In that sense, at the beginning of the propagation (while the susceptible group is approximateable by the entire population), assuming $\mathcal{R}_0 > 1$, if we require the asset differential not exceed a quantity Δ_I before $\mathcal{R}_e = 1$, according to (5), such as $\mathcal{R}_e > \mathcal{R}_0 e^{-\mu\Delta I}$, you must have $\mu \geq \ln(\mathcal{R}_0)/\Delta_I$.

Given a real number x we define its sign, $\sigma(x)$, as -1, 0 or $+1$ depending on whether x is negative, zero, or positive. Now, since $I'/I = \gamma\{\mathcal{R}_e(t) - 1\}$, we have $\sigma(\mathcal{R}_e - 1) = \sigma(I') = -\sigma(\beta')$. So, at the beginning of infectious development, at a certain instant t_0, if $\mathcal{R}_0 = \mathcal{R}_e(t_0) > 1$, then $\beta(\cdot)$ will drop until $I(\cdot)$ reaches its maximum. Furthermore, $\beta(\cdot)$ will recover its initial value β_0, only when $I(\cdot)$ decreases to I_0, that is, at time t when

$$I(t)/I_0 = \exp\left(\int_{t_0}^{t} \{\mathcal{R}_e(a) - 1\} da\right) = 1,$$

$$\text{this is, } \int_{[t_0,t]} \{\mathcal{R}_e(a) - 1\} da = 0.$$

That is, in practical terms, on a graph of the effective reproductive number versus time, β returns to its intrinsic value β_0, when the amount of area that accumulates above and below the level $\mathcal{R}_e = 1$ is the same.

The following result points towards control, as it provides a range for the value of the effective reproductive number, which depends on those eliminated (recovered plus deaths) and on the susceptible fraction of the population also being within a certain range of values.

Theorem 1 *The effective reproductive number, defined by $\mathcal{R}_e(t) = \beta(t)s(t)/\gamma$, with $s(t) = S(t)/N$, for $t \geq t_0$, while $0 \leq p < s(t) < q \leq 1$, satisfies*

$$\Lambda_{q,p}(t) \leq \mathcal{R}_e(t)/\mathcal{R}_0 \leq \Lambda_{p,q}(t), \tag{6}$$

where $\Lambda_{\mathbf{x},\mathbf{y}}(t) = \frac{\mathbf{y}}{\mathbf{x}\mathcal{R}_0+[1-\mathbf{x}\mathcal{R}_0]e^{-\mu R(t)}}$.

Proof: See Appendix A.

Let us observe that $\Lambda_{\mathbf{x},\mathbf{y}}(\cdot)$ is a function such that $\Lambda_{\mathbf{x},\mathbf{y}}(t) \leq 1$, $\Lambda_{\mathbf{x},\mathbf{y}}(t_0) = \mathbf{y}$ and, furthermore $\Lambda'_{\mathbf{x},\mathbf{y}}(t) = -\{\mathbf{x}/\mathbf{y}\}\,\Lambda_{\mathbf{x},\mathbf{y}}(t)\,\mu\,\mathcal{R}_0\,e^{-\mu R(t)}\,R'(t)$, that is, it is decreasing since always $R'(t) \geq 0$. See Figure 1.

3.2 Case $\nu > 0$

Assuming that in (4) the function $I(\cdot)$ is known, its integration as a linear equation leads us to:

Theorem 2 *If we consider in (4) an initial time t_0 and respective conditions $\beta_0 = \beta(t_0)$ and $I_0 = I(t_0)$, then:*

$$\beta(t) = \beta_0 \frac{E(t_0,t)}{1 + \nu\beta_0 \displaystyle\int_{t_0}^{t} E(t_0,\tau)\, d\tau}, \tag{7}$$

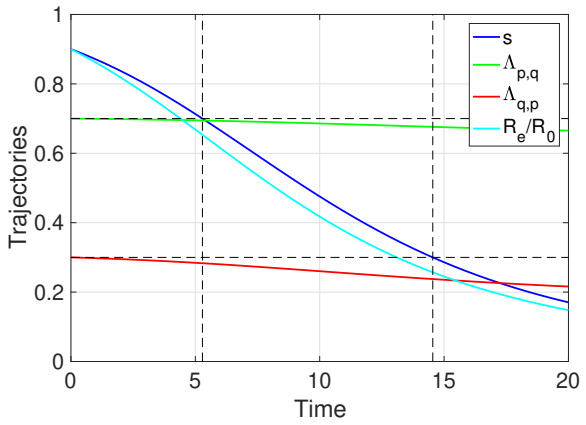

Fig. 1: It can be seen that when $s(t)$ is between p and q, quotient $\mathcal{R}_e/\mathcal{R}_0$ is necessarily between $\Lambda_{q,p}$ and $\Lambda_{p,q}$. It is considered $\beta_0 = 0.3$, $\gamma = 1/14$ $\nu = 0$, $\mu = 0.5$, $p = 0.3$ and $q = 0.7$. The initial condition is $s(0) = 0.9$, $i(0) = 0.1$ and $r(0) = 0$.

where $t \geq t_0$ *and* $E(t_0, t)$ *is equal to* $\exp\{\nu\beta_*(t - t_0) - \mu(I(t) - I_0)\}$.

Proof: See Appendix B.

The importance of Theo. 2 is in the possibility of knowing the transmission rate from the actives's data. Let us note that the function $E(t_0, \cdot)$ in (7), the variable $I(\cdot)$ is equal to $R'(\cdot)/\gamma$, that is, it could be estimated from the daily removed individuals. This rate defines the calculation of the effective reproductive number $\mathcal{R}_e(t) = \beta(t)s(t)/\gamma$, in which $s(t)$ is the susceptible fraction. That is, we have an evaluation of the transmission speed.

Taking into account that $\mathcal{R}_0 > 1$ and that the contagion process starts with a value of β in its intrinsic value β_*, that is, $\beta(t_0) = \beta_*$. Since $\beta'(t_0) = -\mu\gamma[\mathcal{R}_0 - 1]I(t_0)$ is negative, we have that $\beta(\cdot)$ will decrease until it reaches a minimum at a time τ, $\tau > t_0$, in which

$$\begin{aligned} \beta(\tau) = \beta_* - \frac{\mu}{\nu} I'(\tau) \quad \text{and moreover} \\ \beta''(\tau) = -\mu I''(\tau)\beta(\tau). \end{aligned} \tag{8}$$

For example, these equalities tell us how much $\beta(\cdot)$ reduces its value in the period $[t_0, \tau]$, $(\mu/\nu)I'(\tau)$ units. Furthermore, this minimum $\beta(\tau)$ implies an active curve, with, necessarily, a decelerated growth (concave) in a neighborhood of τ, that is, $I''(\tau) < 0$. Since $I' < 0$ implies $\beta' > 0$, this first minimum for $\beta(\cdot)$ is never reached after $I(\cdot)$ presents a first peak or, equivalently, the effective reproductive number reaches or decreases from the desired level.

What can we say about the transmission rate at the time when the effective reproductive number reaches the value one? We know that $\mathcal{R}_e(t) = 1+\gamma^{-1}I'/I$, so $\mathcal{R}_e = 1$ if and only if $I' = 0$, i.e., when $\beta' = \nu(\beta_* - \beta)\beta$. This proves that the peak I_m of $I(\cdot)$ is reached in an instant τ_m after a first minimum of $\beta(\cdot)$, that is, when $\beta(\cdot)$ is retrieving the value. Furthermore, that at that moment $\beta'(\tau_m) \leq \nu\beta_*^2/4$. On the other hand, note that $\mathcal{R}_e'(t) = \gamma^{-1}[I''I - (I')^2]/I^2$ and $\mathcal{R}_e'(t) = (\gamma N)^{-1}(\beta(t)S(t))'$ equals to $(\gamma N)^{-1}(\beta'(t)S(t) + \beta(t)S'(t))$. Using $S' = -\beta SI/N$, by equating and evaluating at τ_m, we obtain $I''/I = [\nu(\beta_* - \beta) - \beta I/N]\beta S/N$. Since $I'' < 0$, it follows that

$$\beta(\tau_m) > \beta_* \frac{\nu N}{\nu N + I_m}.$$

4 Numerical results

The graphical possibilities of transmission rate, effective reproductive number, daily new cases, and infectious are presented matrixly by the rows in Figure 2 and Figure 3, depending on the value of the restoration coefficient: low, medium, and high. This, considering two orders of magnitude for the pair (ν, μ) according to Table 1. Moreover, all these cases regarding the initial condition at instant $t_0 = 0$, with a population of size N that is considered to be decomposed into $(S_0, I_0, R_0) = (N - 1, 1, 0)$.

Table 1: Parameter values for two orders of magnitude (A and B) of the restoration and reaction coefficients, total population, and intrinsic transmission rate.

Case	ν	μ	N	β_*
A	$2/10^1$, $5/10^1$, $8/10^1$	$3/10^1$, $4/10^1$, $5/10^1$, $6/10^1$	10^2	0.65
B	$2/10^2$, $5/10^2$, $8/10^2$	$3/10^4$, $4/10^4$, $5/10^4$, $6/10^4$	10^5	0.45

Now, regarding regularities or patterns in the graphs, let us observe that:

- Without going into detail, the corresponding forms for cases A and B are similar, except that case A represents a comparatively rapid epidemic spread.
- Over time, the transmission rate $\beta(\cdot)$ always decreases towards a flat valley and then begins to recover value as soon as daily cases drop, even exceeding the intrinsic value. Finally, it reaches a peak and ends up asymptotically decaying to β^*. In case A (Figure 2(a-c)) the initial decline is convex, but in case B (Figure 3(a-c)) the decrease begins concavely. We also note that in both cases, A or B, the minimum value to which $\beta(\cdot)$ can drop is seemingly more sensitive to the parameter ν than to μ.

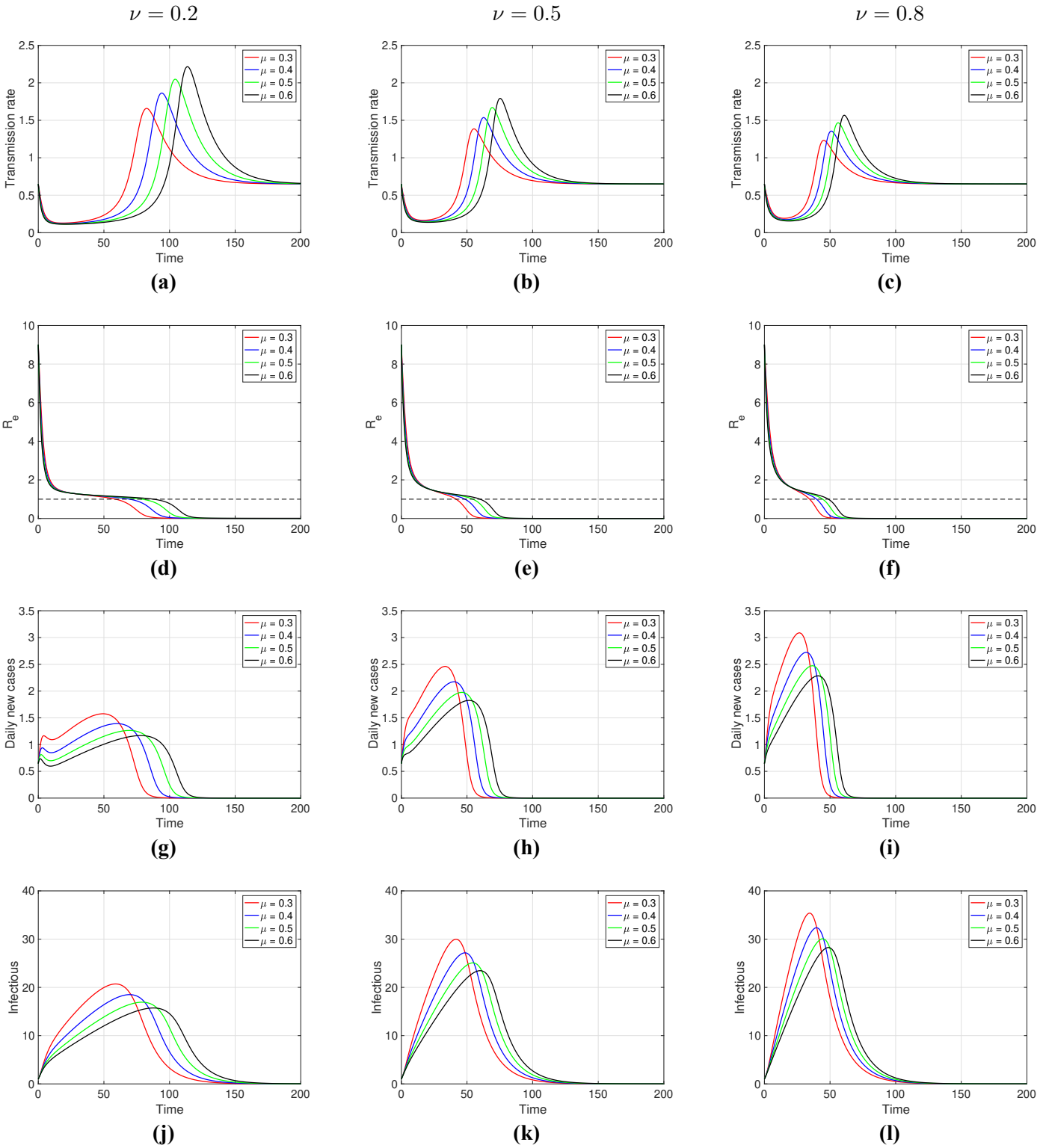

Fig. 2: Simulations of equation (1)+(4) for different values of ν and μ. Conditions initials: $S(0) = 99$, $I(0) = 1$, $R(0) = 0$ and $\beta(0) = 0.65$. $\gamma = 1/14$ and $N = 100$ was considered.

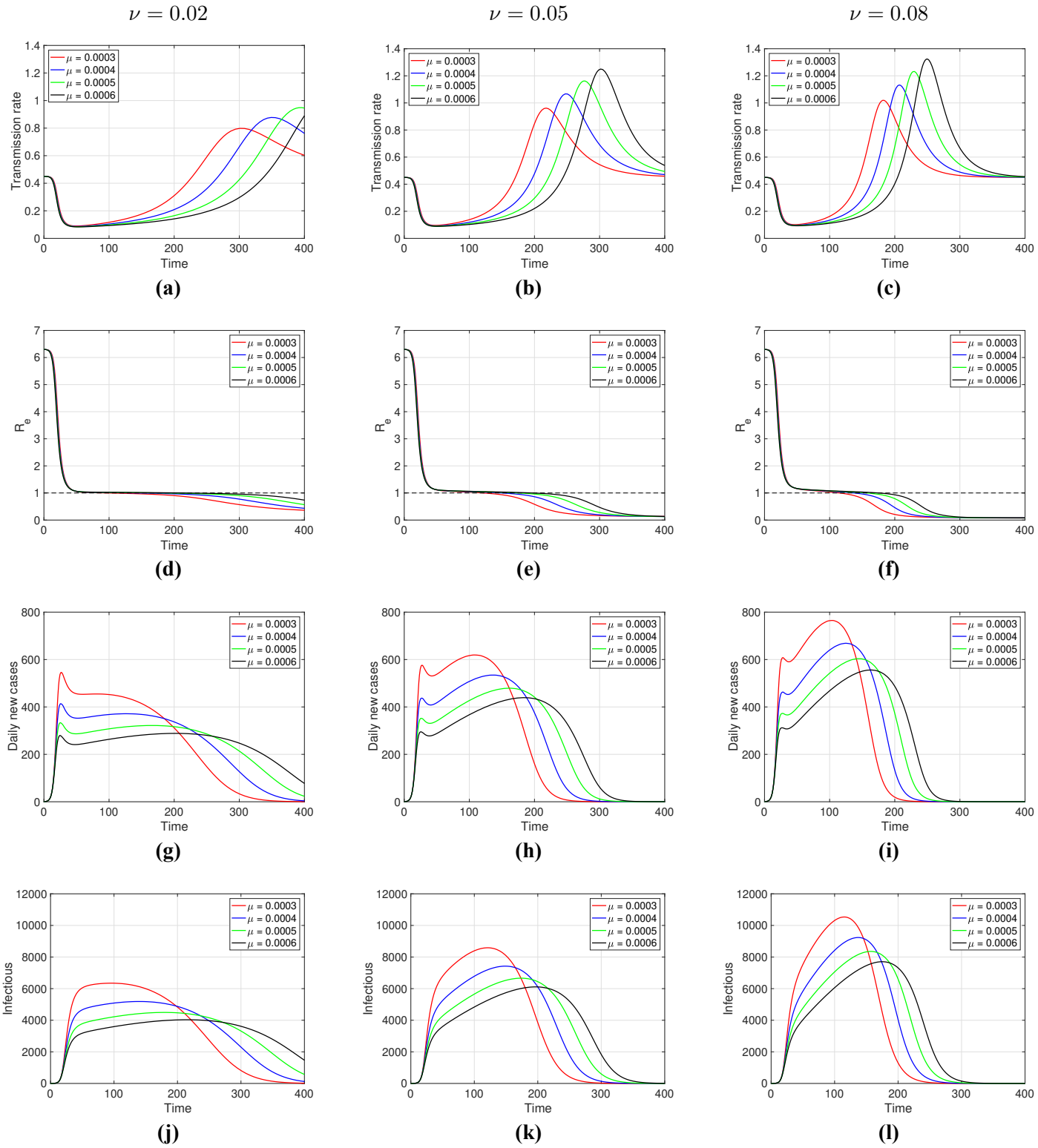

Fig. 3: Simulations of equation (1)+(4) for different values of ν and μ. Conditions initials: $S(0) = 99999$, $I(0) = 1$, $R(0) = 0$ and $\beta(0) = 0.45$. $\gamma = 1/14$ and $N = 100000$ was considered.

- The initial decrease in the rate β is in correspondence with the fall of $\mathcal{R}_e$, but unlike $\beta(\cdot)$ which recovers value, we have that $\mathcal{R}_e$ continues to decrease and tends towards zero, which is only explainable by a significant relative decrease in the susceptible fraction, since $\mathcal{R}_e' < 0$ implies $\beta'/\beta + S'/S < 0$. Another clear pattern of the effective reproductive number is to present a very planar zone when it is close to the value one, a zone that is wider when the coefficient of restitution ν is smaller, compare Figure 2(d-f) or Figure 3(d-f). Furthermore, by fixing ν in each case, the flat zone is wider if the reaction coefficient μ is higher.
- In case B, the daily case curves, Figure 3(g-i), are bimodal with the possibility that the highest peak is the first or the last depending on whether the restoration coefficient ν is respectively higher or lower. Thus, the unimodality inherent to the classic SIR (constant parameters) may not be met. In case A, Figure 2(h,i) the bimodality is not expressed. It is conjectured that there must exist a bound for ν/μ before which these curves are necessarily unimodal.
- For infectious count curves, the effect of a restoration coefficient ν, when fixing the reaction coefficient μ, e.g., the black line for Figure 3(j-i), is a flattened curve for a longer time at lower values of ν. Now, if we set ν, a higher and more advanced asset curve is observed at lower values of μ, that is, at smaller reactions. Observations that are also valid for the curve of new daily cases.

On the other hand, let us note that the total decrease (by reaction) achieved for the beta rate during a certain initial time interval seems to be at the cost of recovering this rate the rest of the time and above the intrinsic value, although approaching it asymptotically, see Figure 4. Indeed, if there exists a unique $t_* > t_0$ in which $\beta(t_*) = \beta_*$, by denoting by $\mathcal{A}_-$ (*resp.* $\mathcal{A}_+$) the area between β_* and $\beta(\cdot)$ over $[t_0, t_*]$ (*resp.* $[t_*, \infty)$) we have:

$$\begin{aligned}\mathcal{A}_- &= \int_{[t_0,t_*]} \{\beta_* - \beta\} \\ &= \nu^{-1} \int_{[t_0,t_*]} \{\beta'/\beta + \mu I'\} \\ &= \mu\nu^{-1}\{I(t_*) - I(t_0)\}\end{aligned}$$

and similarly $A_+ = \mu\nu^{-1} I(t_*)$, so that $\mathcal{A}_+ - \mathcal{A}_- = \mu\nu^{-1} I(t_0)$.

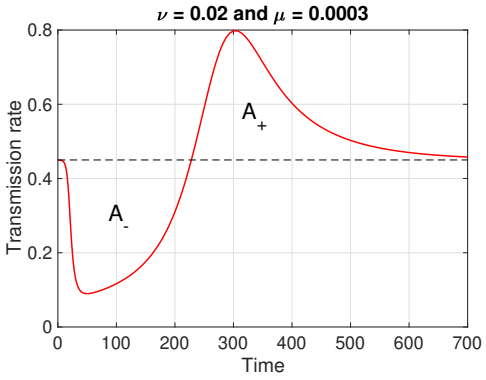

Fig. 4: Initials Conditions: $S(0) = 99999$, $I(0) = 1$, $R(0) = 0$ and $\beta(0) = 0.45$. $\gamma = 1/14$ and $N = 100000$ was considered. In this case $\mathcal{A}_+ - \mathcal{A}_- = \nu/\mu = 0.015$.

5 Discussion

The classical SIR model considers a constant transmission rate. However, in the context of a high- and immediate-risk pandemic (without pharmaceutical alternatives), one of the main sources, if not the main, of the changes in the transmission rate that the population may present during an epidemic is relates to human behavior in terms of adherence and compliance with mitigating measures. In the search for a model that brings together the mechanistic aspects of the SIR but with a phenomenological perspective in terms of simplicity, the assumption of a reaction-restoration tension for the variation of the transmission rate seems reasonable.

How can the transmission rate, as a potential cultural and physical-environmental determinant of a specific population, vary, as it did with COVID-19 before the advent of vaccines? In the first pandemic stage, the general observation (based on pandemic data) is that there was a reaction from authorities and individuals that allowed, based on perception or indicators of the danger of the disease, to reduce its worth. The information that the public received or that was analyzed to decree specific measures is directly or indirectly a reading of the status of individuals concerning the disease, particularly those of population scope, such as the variation of the infectious group size, which is our option in the present work.

In this sense, the main contribution of the work is to model the epidemiological consequences of achieving the health authority and the media to maintain pressure on the system, which manifests itself in a percentage change of decrease in the transmission rate that, as has been said, is proportional to the variation of the infectious group. In other words, having a first dimension or evaluation of the behavior of the system's state variables, both in the case of no resistance to control and in the opposite case.

In particular, in the case of zero resistance to control ($\nu = 0$), for an estimation interval of the remain-

ing susceptible fraction, Theo. 1 provides a bounding band for the effective reproductive number as a fraction of the basic reproductive number and of the removals count (which could be the variable with good measurement possibilities) in principle somewhat crude, as shown in Figure 1, but which could (and it is a mathematical challenge) be improved. Now, by adding a social resistance to mitigation ($\nu \neq 0$), Theo. 2 gives us the rate determination relationship according to active cases through expression (7) which is, accordingly, the generalization of (5). These results, although elementary, have potential usefulness in the development of more tactical models, particularly those that are fed back through data for a specific population. We consider it important to note that when the active group varies downwards, the control acts not only canceling itself but also providing a stimulus for its increase, above the intrinsic value, which makes some sense within the social behavior of reunion again between people. What the comparison between Figure 2 and Figure 3 show is that lower order restoration levels can mean a much slower return to the original or intrinsic transmission rate.

6 Conclusion

The main hypothesis considered is to assume that the reaction factor is proportional to the variation of the active group. Note that this is the group, in terms of size, that directly determines hospital occupancy and the number of deaths. The assumption is that only as this group grows does a downward reaction emerge. So, if it remains constant for some period, such a reaction disappears; finally, if it goes down, the tendency for the rate to rise grows. Another possibility, somewhat close to our case, but which we leave open for analysis in future work, is to assume that the reaction factor $g(\cdot)$ takes the form $g(I, I') = \mu I'/I$, that is, the reaction is sensitive to the percentage variation of the active group.

A general observation is that the β-SIR type models, such as the one analyzed or the β-SEIR versions, maintain a degree of simplicity that helps to understand the mechanics (in terms of the response of the system), not only the contagion of the disease, but also the corresponding "social mechanics", both of the population in general (mainly in the restoration factor), and the work of the health authority (in the reaction factor), the above as long as there are no pharmaceutical possibilities that alter the variables. Let us observe that the type of information or indicators that the authority collects or uses to implement mitigation, directly from the triad (S, I, R) or its derivatives, such as reproductive numbers, is an aspect that could become key, mainly concerning the times and levels that ensure effective management, for example hospital management, in severe cases.

Another key aspect, which could explain a good part of the variability that experience tells us in the data, mainly at the beginning of the epidemic, could be the time delay between the value of the considered indicator(s), the implementation, and the citizen response. In other words, more research is required, associated with the management of health emergencies and human behavior in specific populations, to propose, with greater predictive zeal, functional forms to express variations in the rate of reactive or restoration order.

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Conflicts of Interest
The authors have no conflicts of interest to declare that are relevant to the content of this article.

Appendix A

Note that replacing the second equation of (1) in (4) with $\nu = 0$, we obtain $\beta' = -\mu[\beta s - \gamma]I\,\beta$, with $s(\cdot) = S(\cdot)/N$, the susceptible fraction. By amplifying, by $-1/\beta^2$ and changing the variables $Z = 1/\beta$, we have the equation $Z' + \mu\,\gamma\,I\,Z = \mu\,s\,I$, where together with the expression for $R'(t) = \gamma I$ in (1), we observe that $\mu R' Z = \mu\gamma I Z$, achieving $\left\{Z\,e^{\mu R}\right\}' = [Z' + Z\mu R']e^{\mu R} = \mu s I e^{\mu R}$. Integrating over $[t_0, t]$ and considering $R(t_0) = 0$, we have that,

$$Z(t) = \left\{\frac{1}{\beta_0} + \mu\int_{t_0}^{t} s(a)I(a)e^{\mu R(a)}\,da\right\}\,e^{-\mu R(t)}.$$

Thus, if we consider that $s(a) = \lambda$, we can define

$$Z_\lambda(t) := \left\{\frac{1}{\beta_0} + \mu\lambda\int_{t_0}^{t} I(a)e^{\mu R(a)}\,da\right\}\,e^{-\mu R(t)}.$$

From where, if we consider $p \le s(t) \le q$, we have: $Z_p(t) \le Z(t) \le Z_q(t)$. Then, continuing with the integration for the expression of Z_λ, we see that,

$$\int_{t_0}^{t} I(a)e^{\mu R(a)}\,da =$$

$$\frac{1}{\mu\gamma}\int_{t_0}^{t}[e^{\mu R(a)}]'\,da = \frac{1}{\mu\gamma}[e^{\mu R(t)} - 1],$$

finally

$$Z_\lambda(t) = \frac{\gamma + \lambda\beta_0[e^{\mu R(t)} - 1]}{\gamma\beta_0 e^{\mu R(t)}} =$$

$$\frac{\lambda\mathcal{R}_0 + e^{-\mu R(t)}[1 - \lambda\mathcal{R}_0]}{\beta_0}.$$

Since, $Z_q^{-1} \le \beta(t) \le Z_p^{-1}$, we have

$$\frac{p\mathcal{R}_0}{q\mathcal{R}_0 + [1 - q\mathcal{R}_0]e^{-\mu R(t)}} \le \mathcal{R}_e(t) \le$$

$$\frac{q\mathcal{R}_0}{p\mathcal{R}_0 + [1 - p\mathcal{R}_0]e^{-\mu R(t)}};$$

which ends the proof. $\diamond$

Appendix B

From (7), we define $U(t_0, t) = \nu\beta_*(t - t_0) - \mu(I(t) - I_0)$, so that (4) is equal to $\beta' = \{U'(t_0, t) - \nu\beta\}\beta$. So the change of variables $z(t) = 1/\beta(t)$, implies $z' = \nu - U'(t_0, t)z$. Thus, one has

$$z(t) = z(t_0)\exp\left(-\int_{t_0}^{t} U'(t_0, \tau)\,d\tau\right) +$$

$$\nu\int_{t_0}^{t}\exp\left(-\int_{\tau}^{t} U'(t_0, a)\,da\right)\,d\tau.$$

Now, since $U(t_0, t_0) = 0$ and $U(t_0, t) - U(t_0, \tau) = U(\tau, t)$, by integrating U' into the expression $z(t)$, we obtain

$$\frac{1}{\beta(t)} = \frac{1}{\beta_0}\,\exp\left(-U(t)\right) +$$

$$\nu\int_{t_0}^{t}\exp\left(-U(\tau, t)\right)\,d\tau.$$

Considering $E(t_0, t) = \exp\{U(t_0, t)\}$, isolating $\beta(\cdot)$ completes the proof of Theorem 2. $\diamond$