axioms

MDPI

Article

A Kinematic Approach to the Classical SIR Model

Fernando Córdova-Lepe [1], Juan Pablo Gutiérrez-Jara [2,*] and Katia Vogt-Geisse [3]

[1] Facultad de Ciencias Básicas, Universidad Católica del Maule, Avenida San Miguel 3605, Talca 3480112, Chile; fcordova@ucm.cl
[2] Centro de Investigación de Estudios Avanzados del Maule (CIEAM), Universidad Católica del Maule, Talca 3480112, Chile
[3] Facultad de Ingeniería y Ciencias, Universidad Adolfo Ibáñez, Diagonal Las Torres 2640, Peñalolén, Santiago 7941169, Chile; katia.vogt@uai.cl
* Correspondence: jgutierrez@ucm.cl

Abstract: Given the risk and impact of infectious-contagious X diseases, which are expected to increase in frequency and unpredictability due to climate change and anthropogenic penetration of the wilderness, it is crucial to advance descriptions and explanations that improve the understanding and applicability of current theories. An inferential approach is to find analogies with better-studied contexts from which new questions and hypotheses can be raised through their concepts, propositions, and methods. Kinematics emerges as a promising analog field in physics by interpreting states' changes in a contagion process as a movement. Consequently, this work explores, for a contagion process, the representations and conceptual equivalents for position, displacement, velocity, momentum, and acceleration, introducing some metrics. It also discusses some epistemological aspects and proposes future perspectives.

Keywords: diseases X; SIR model; transmission rate; kinematics

MSC: 34C60

check for updates

Citation: Córdova-Lepe, F.; Gutiérrez-Jara, J.P.; Vogt-Geisse, K. A Kinematic Approach to the Classical SIR Model. *Axioms* **2024**, *13*, 718. https://doi.org/10.3390/axioms13100718

Academic Editor: En-Bing Lin

Received: 6 September 2024
Revised: 10 October 2024
Accepted: 14 October 2024
Published: 16 October 2024

Copyright: © 2024 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (https://creativecommons.org/licenses/by/4.0/).

1. Introduction

Kinematics is defined as the branch of classical mechanics that focuses on the description of the motion of objects, disregarding considerations of their underlying causes [1]. In mathematical models of processes that are viewed as dynamical systems, changes in the value of their variables can be visualized as trajectories of motion within the phase space. In this sense, the language of classical kinematics can be taken to other fields of knowledge, i.e., to describe processes that, in principle, are outside the context of studying the motion of rigid bodies in physics. Therefore, within infectious disease epidemiology, specifically in the study of contagion processes, the description and analysis of these state trajectories over time, using concepts such as position, displacement, velocity, momentum, and acceleration, could become relevant. Even more so if we share the appreciation that: "*Language is not only a communication system but also—or, above all—a tool to articulate thought*" [2]. Thus, new ways of representing and relating concepts can be very useful in leading us along new paths to generate knowledge. Let us note that to know processes in a rigorous sense is to identify them not only by individualizing their specific properties and relations but also by discovering the universality that exists in their particularity, i.e., in the recognition and elaboration of concepts, which is based on "*the adequacy between the object* (process) *and what is thought or known about it*" [3].

A dynamic that can be mathematically modeled is associated with a specific mathematical object, that is, to an ideal concept [4], and as such, does not necessarily admit one single interpretation. Therefore, associated with these models, in addition to narratives, we also have a variety of visualization possibilities. In general, it is essential to note that representations not only serve to understand a phenomenon but also raise new questions

and explanations, which, in the end, add up to the goal of increasing understanding and, therefore, knowledge [5,6]. Thus, there is the possibility that a model of a process may be able to admit, more or less comfortably, a characterization of it by using the concepts and representations of kinematics.

In this sense, the most widely used and well-known base or strategic model [7,8] for the development of infectious processes is the compartmental SIR or (β, γ)-SIR model, which, as a mathematical object, is a two-dimensional and biparametric system of ordinary differential equations (with β being the transmission rate and γ the recovery rate). This model is interpreted not only as an epidemiological model of the contagion of an infectious disease, which is its original meaning but also as a generic model for some types of transmissible phenomena. In addition to its applications in the propagation of epidemics, the SIR model has also been used in various other fields. In this regard, in [9], we find the following list: networks (online social networks [10,11], viral marketing [12], audience applause [13] and diffusion of ideas [14]), informatics (peer-to-peer networks [15] or spread of computer virus [16]), economics and finance (rational expectations [17], financial network contagion [18]), and even science fiction (zombies attack [19]). Today, considering the global experience of the COVID-19 pandemic, the popularity of the SIR model has grown both at the academic level (in Google Scholar, on 3 September 2024, the expression "SIR model" lists 16,100 articles post-2020) and in explanatory and risk communication efforts in the mass media [20]. In academia, there has been (and to some extent continues to be) an explosive development of articles based on the SIR model that explore more tactical and even operational models, i.e., that aspire to a high resolution concerning data that allow them even the possibility of prediction.

As the title indicates, the novelty and contribution of the present work is in proposing a description of the SIR model and its components using the language of physics, in particular kinematics, by the systematic way of constructing analogies [21,22]. This is achieved by identifying the numerical changes in the epidemic compartments with the idea of motion in physical space by using its usual kinetic concepts. In particular, it identifies the vector of compartments with the position vector, visualizes the basic reproductive number with the concept of speed, and examines the variations in the transmission rate with the idea of acceleration. In addition, we visualize the effective reproductive number as the momentum of the susceptible group and introduce the amount of movement for the infectious group equivalently. In addition, the results of the known theory concerning the SIR model are presented from the perspective of these quantities of motion.

A strategic mathematical model is defined as the simplest one that captures the essence of the dynamics defining the differentiating patterns that give identity to a certain type (a whole class) of processes, i.e., in this sense, it is generic. Compared to tactical models, which account for more specific aspects of a process, strategic models are of lower resolution [7]. Still, the architecture of a tactic model is designed from a strategic model, which acts as a template. This is the case of SEIR, SEIRS, SEIQR [23], MSEIR [24], MSEICR [25], mSEIR [26] or other derivative models, which add specific aspects to the SIR model. A model that aims to represent a specific and generally unique reality pursuing a high resolution with the data history and with projection and forecast pretensions is called operational. In these, it is also generally possible, by grouping, to recognize a mother strategic model. The COVID-19 pandemic experience showed us that the SIR model, with constant beta and gamma parameters, does not work as a tactical model [27]. In this sense, the literature offers evidence in two directions: by increasing the epidemiological compartments into which the population studied is divided or by introducing a variable beta transmission rate [28–30]. The long-term objective and, at the same time, the (initial) novelty of this work is the construction of a dynamic theory (in analogy to the Newtonian theory) of contagion processes to provide understanding and analytical support, via a strategic model, to the "forces" that define a variable behavior of the transmission speed, that is, to think about accelerated mobility. Advances in this sense are [31,32]. However, we must first clarify the kinematics of the case of uniform movement or contagion, that is, when the

beta transmission speed is constant in magnitude, a task that is precisely the purpose of this work.

In Section 2 of this paper, we introduce what we have called kinematic epidemiological concepts, namely position, displacement, velocity, momentum, and acceleration. The main results around these concepts, which mainly show a pair of metrics in connection with the idea of velocity, are presented in Section 3. A discussion about these results is discussed in Section 4. The paper closes in Section 5, with some conclusions about the introduced kinematic approach and some comments on its limitations and projections.

2. Epidemiological Kinematic Concepts

Regarding physical motion, in the development of infectious disease, the displacement of pathogens could be performed at different scales of observation [33], for example, microscopically, inside of an individual when viral dynamics are colonizing a particular human organ. Also, there is the macroscopic if the case is the path of movement of such viruses when transported toward a new host who, in turn, as a person, could be moving around in a broader environment. On the other hand, we have the subject of the frequency of the measure registers, e.g., by hours, day, or epidemiological weeks. Appropriate scales have to do with the purpose of the research and the desired accuracy, always seeking consistency [34]. However, we will address the idea of motion as the variation in the state variables tuple in a classical compartmental model—an SIR model. Therefore, we will not refer to the physical movement of pathogens or individuals but to mobility between attributes: the epidemiological conditions inherent to the SIR model. That is the coupled change in the state variables (susceptible–infectious–removed) as functions of time within a geometric space, which is defined by their numerical ranges.

The kinematic framework considers the concurrence of some fundamental elements expressed through the basic concepts associated with the theory. Components that cannot be omitted are space, time, and the existence of at least one moving object. We consider as a moving object; the population to which the SIR model refers; as usual, time will be its independent variable, and space will be a reduction in the phase space associated with the model as a dynamic system. Regarding the main concepts for description, we will consider position, displacement, velocity, and acceleration.

2.1. Position

According to the moving object's size in relation to the magnitude of its displacement, kinematics differentiates between the particle's kinematics and the kinematics of the rigid object. A particle (or material point) is a body of sufficiently small dimensions to be considered in the model as a point in some geometrical space. A rigid body is a system or set of particles whose relative positions do not vary. Note that the particle approach, i.e., when we can disregard the size of the body to consider it as a point, depends on the particular conditions of the problem being studied. What could be the case for a population whose representation is defined by the (β, γ)-SIR model?

For a pair of populations of different sizes, e.g., N_1 and N_2 with $N_2 \gg N_1$, with the same pair of parameters (β, γ), we see that the dynamic trajectories of the absolute values of any of the compartments are, at any given time, very different. However, if we consider the relative values, these trajectories are the same if they share the initial condition and have equal initial percentage values for their three compartments. In other words, they share, as a model, the normalized SIR system of equations

$$s'(t) = -\beta\, s(t)\, i(t), \quad i'(t) = \{\beta\, s(t) - \gamma\} i(t), \quad \text{and} \quad r(t)' = \gamma\, i(t), \tag{1}$$

with some initial condition, at time zero, equal to (s_0, i_0, r_0), with $s_0 + i_0 + r_0 = 1$. In other words, for example, if $S_k(t)$ is the number of those susceptible at time t, $t \geq 0$, for the population N_k, $k \in \{1, 2\}$, then $S_k(t) = N_k\, s(t)$. Thus, it is worth opting for a particle kinematic approach.

It is clear that an epidemiological process produced by an infectious disease and experienced by a closed population of size N is analogous to that of a particle of mass N (*population mass*) that describes a trajectory in space (*epidemiological space*) defined by

$$\Omega := \{(x_s, y_i, z_r) \,:\, x_s, y_i, z_r \geq 0 \quad \text{and} \quad x_s + y_i + z_r = 1\}, \tag{2}$$

being Ω the observer's reference frame. We denote by Ω_* the set where the condition $x_s > 0$ is added to the set in (2).

In this way, the *population position*, $P(t)$, of a population at any time t is given by the coordinate vector $P(t) := (s(t), i(t), r(t))$, where $s(t) + i(t) + r(t) = 1$, which, as mentioned above, represents in its components the percentage of the population in the susceptible, infectious and removed epidemic states.

We have identified the population size with the scalar mass N, but let us observe that this constant mass is the sum of its *susceptible mass* ($m_S = N\,s(t)$), *infective mass* ($m_I(t) = N\,i(t)$) and *removed mass* ($m_R(t) = N\,r(t)$), that is, $N = m_S(t) + m_I(t) + m_R(t)$, at any time $t \geq 0$. Figure 1 shows on its right the *population mass point* highlighted as a 3-tuple of masses (m_s, m_i, m_r) and the *mass epidemiological space*, both concepts as a result of the amplification by N, respectively, of the *population position* (s, i, r) and the *epidemiological space* shown on the left of the figure.

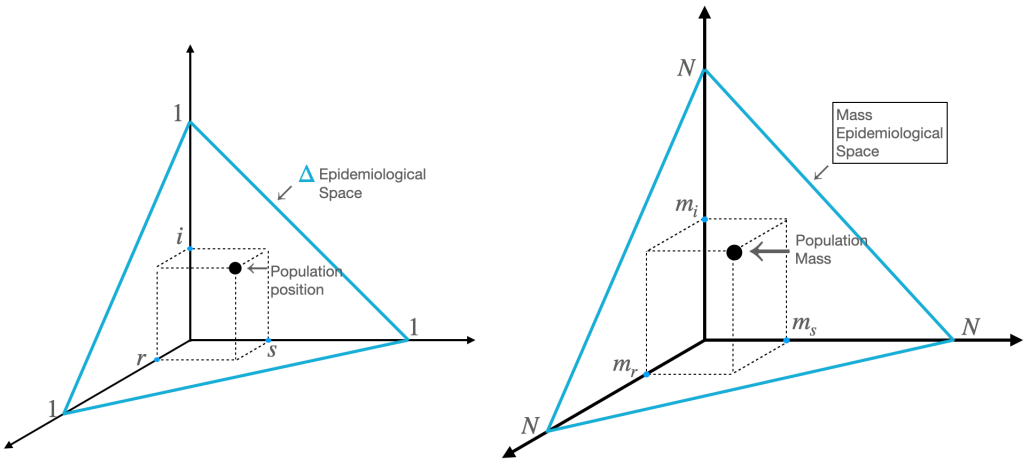


Figure 1. Analogous to a body having a characterization via a position and a mass, the *population position* of a population, at any given instant, is represented on the left-hand side as a point (s, i, r) within the epidemiological space Ω (blue triangular surface). While on the right-hand side (amplifying the population position by the population mass (total population size N), the plot identifies the *population mass point* (m_s, m_i, m_r) within the mass epidemiological space.

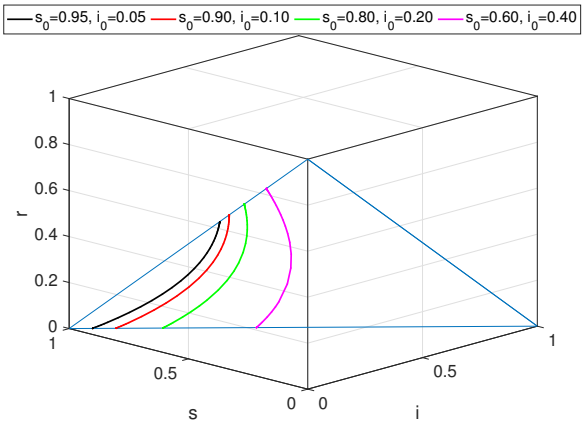
In general, there are two opposite approaches to particle kinematics: From the trajectory, one determines the position, velocity, and acceleration using the mathematical concept of a derivative; or from the acceleration, one deduces (integrating) the velocity and position of the particle. Our work better fits the first approach.

2.2. Displacement

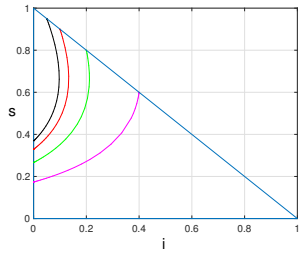
Having Ω as the reference system, we know that the displacement must correspond to the change in position of the population in that system, a "motion of place" (*locus*), hence the word locomotion. Between a pair of time instants t_1 and t_2, with $t_1 < t_2$, the epidemiological position of the population has changed from the population position $P(t_1) = (s(t_1), i(t_1), r(t_1))$ to $P(t_2) = (s(t_2), i(t_2), r(t_2))$, so we can define displacement by the difference $\Delta P(t_1, t_2) := (s(t_2) - s(t_1), i(t_2) - i(t_1), r(t_2) - r(t_1))$, a vector that lays in Ω. On the interval $[t_1, t_2]$, we observe that a population that satisfies (1) indeed defines the parametric curve, $P(t) = (s(t), i(t), r(t))$, that also lays in Ω, which can be proven by

checking that its tangent vector (s', i', r') is perpendicular to the vector $(1, 1, 1)$, normal to the space Ω. In fact, the inner product $\langle (s', i', r'), (1, 1, 1) \rangle$ equals $s' + i' + r' = 0$.

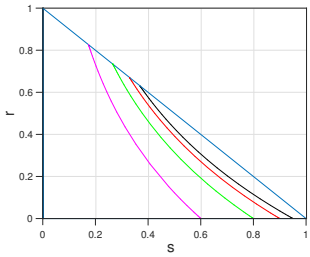
In kinematics, the particle's trajectory is understood as the geometric locus (a subset of the reference space) of the successive locations of the particle. In this sense, the *epidemiological trajectory* of the total population is the graph in the epidemiological space Ω (which always depends on the reference system, that is, a kind of observation point) of the parametric curve $P(t) = (s(t), i(t), r(t))$, with $t \in [0, \infty)$ (see Figure 2).



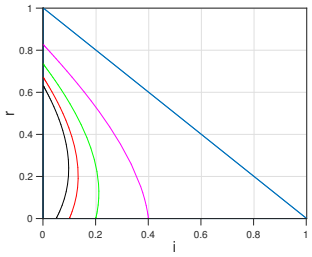
(**a**) Epidemiological trajectories



(**b**) Projection on *is*-plane



(**c**) Projection on *sr*-plane



(**d**) Projection on *ir*-plane

Figure 2. As Ω borders, two diagonal segments D_0 and D_∞ stand out, $D_0 : s + i = 1$ and $D_\infty : s + r = 1$. Therefore, (**a**) represents four *epidemiological trajectories* for $(\beta, \gamma) = (0.3, 0.2)$, i.e., $\beta/\gamma = 1.5$. These are trajectories of system (1) with initial conditions $(s_0, i_0, 0) \in D_0$, and final state values $(s_\infty, 0, r_\infty) \in D_\infty$; for i_0, these represent 5% (black), 10% (red), 20% (green) and 40% (purple) of the total population. (**b**), (**c**) and (**d**) depict, respectively, the projections of the four curves (maintaining the colors) on the coordinate systems (i vs. s), (s vs. r) and (i vs. r). It is observed, as expected, that as the function of time s is decreasing and r is increasing, i may initially grow (black, red and green) but then always decrease to zero.

2.3. Velocity

Velocity, as a concept, involves time; therefore, it is not independent of how time is measured; it is not independent of the "clock" used. However, the two clocks are related through their time units. The differential system (1) involves equations that relate the velocity of the state variables to the states themselves for a specific time clock. What does this system look like for other ways of measuring time, i.e., for other meaningful clocks?

2.3.1. Time Given by the Number of Recovery Periods

An interesting time scale to consider is the time unit given by the (average) duration of the recovery period (recovery time), i.e., γ^{-1}, when it remains relatively constant. This

is, applying the change in variables $\tau = \gamma\, t$, with which system (1) becomes the equivalent dynamical system

$$\begin{cases} s'(\tau) &= -\mathcal{R}_0\, s(\tau)\, i(\tau) \\ i'(\tau) &= +\mathcal{R}_0\, s(\tau)\, i(\tau) - i(\tau) \\ r'(\tau) &= +i(\tau). \end{cases} \tag{3}$$

This system depends on a unique numerical factor (parameter), the basic reproduction number $\mathcal{R}_0 = \beta/\gamma$. In other words, using this standardized clock, the SIR model becomes a uni-parametric model, and the speed of expansion of the process it represents is uniquely determined by $\mathcal{R}_0$.

2.3.2. Time Given by the Number of Removed

It is clear that the variable removed is a strictly increasing function of time. Hence, if the rhythm of time is set by the variable representing removed individuals, i.e., if the change in variables $r = r(t)$ is used, then system (1) becomes

$$\begin{cases} s'(r) &= -\mathcal{R}_0\, s(r) \\ i'(r) &= +\mathcal{R}_0\, s(r) - 1 \\ r'(r) &= +1. \end{cases} \tag{4}$$

In fact, by the chain rule, $s'(r) = s'(t)/r'(t) = -\beta s(t) i(t)/(\gamma i(t)) = -\mathcal{R}_0 s(r)$, $r'(r) = r'(t)/r'(t) = 1$ and, since $s + i + r = 1$, it follows $i'(r) = -s'(r) - r'(r) = \mathcal{R}_0\, s(r) - 1$.

Note that this time measurement linearizes the SIR differential system (1) and allows us to interpret $\mathcal{R}_0$ as the rate of decay of the number of individuals susceptible due to removal. In this representation, it is easier to see the characteristics of a uniform movement (defined by constant parameters).

From system (4) with initial condition $(s_0, i_0, 0)$ (i.e., initially no removed individuals), we obtain through direct solving of the ordinary differential equations the following explicit form:

$$s(r) = s_0\, e^{-\mathcal{R}_0\, r} \quad \text{and} \quad i(r) = i_0 + (1 - e^{-\mathcal{R}_0\, r})\, s_0 - r. \tag{5}$$

Since $i'(r) = 0$ if and only if $s(r) = 1/\mathcal{R}_0$ and $i''(r) = \mathcal{R}_0\, s'(r) = -\mathcal{R}_0^2\, s(r) < 0$, it follows that if $\mathcal{R}_0 > 1$ with $i_0 \sim 0$ (i.e., $s_0 \sim 1$), then the infectious curve $i(r)$ has a unique critical point $r = r_*$, where a global maximum $i_* = i(r_*)$ is attained, and:

$$r_* = \ln(\mathcal{R}_0)/\mathcal{R}_0 \quad \text{and} \quad i_* = 1 - (1 + \ln(\mathcal{R}_0))/\mathcal{R}_0. \tag{6}$$

Furthermore, since

$$\frac{dr_*}{d\mathcal{R}_0} = \frac{1}{\mathcal{R}_0^2}(1 - \ln(\mathcal{R}_0)) \quad \text{and} \quad \frac{di_*}{d\mathcal{R}_0} = \ln(\mathcal{R}_0)/\mathcal{R}_0^2 > 0, \tag{7}$$

we conclude that a higher basic reproduction number implies a higher maximum infectious level i^* (see Figure 3). Figure 3 and Equation (7) also show that the maximum of the removed group is reached when a fraction $1/e$ (36.8%) of the total population N has been infected (removed).

Finally, we observe that for the trajectories of the SIR model in (1), the susceptible variable, $s(\cdot)$, is always decreasing, and hence its complement in the population, that is, the number of cases $1 - s(\cdot)$, is an increasing variable. If this were the time unit, then to define the change in variable $c = 1 - s(t)$, we obtain from system (1) that

$$\begin{cases} s'(c) &= -1 \\ i'(c) &= +1 - 1/\mathcal{R}_e(c) \\ r'(c) &= +1/\mathcal{R}_e(c), \end{cases}$$

where $\mathcal{R}_e := \mathcal{R}_0\, s(c)$ is the well-known *effective reproduction number*. Therefore, if the clock is the number of cases, apart from contrasting the role of $\mathcal{R}_0$ as a dividing factor, compared

to that of a multiplying factor in the other systems, it is not possible for us to conclude anything essentially new using this time unit. Just to confirm, in the SIR model, $\mathcal{R}_e = 1$ is equivalent to $i' = 0$, i.e., $i = i_*$.

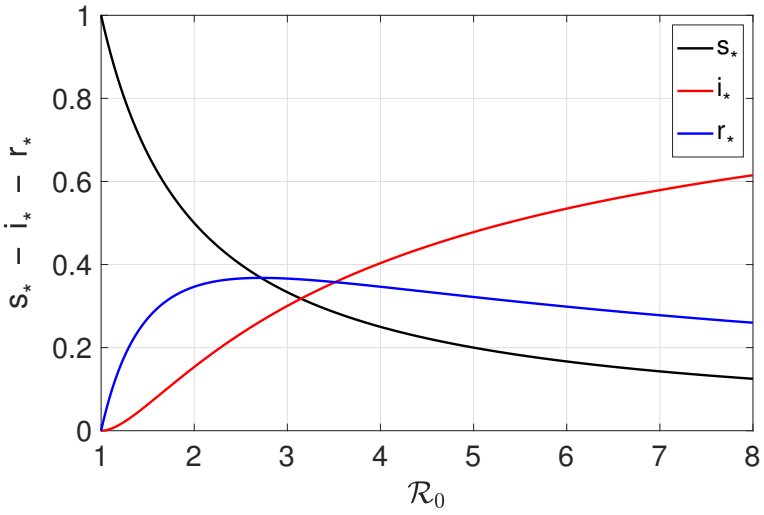


Figure 3. Curves representing the variables s_* (black), i_* (red), and r_* (blue) as functions of $\mathcal{R}_0 > 1$. The figure shows that $r_*(\mathcal{R}_0)$ attains the maximum value $1/e$ at $\mathcal{R}_0 = e$ and that $i_*(\mathcal{R}_0)$ is strictly increasing. For instance, in a population with an $\mathcal{R}_0 = 3$ for a certain contagious disease, it is concluded that the maximum of active individuals can be 30%. About 37% will have contracted the disease at that time, leaving 33% susceptible.

2.3.3. Reproduction and Transmission Speed

The trajectory given by (s, i, r) has an instantaneous velocity $\vec{\mathbf{v}}$ given by (s', i', r') and, as mentioned before, the vector that lies in the Ω space. Furthermore, according to the time scale used, we have $\vec{\mathbf{v}}(t) = \gamma\, i(t)\, \vec{\omega}(t)$, $\vec{\mathbf{v}}(\tau) = i(\tau)\, \vec{\omega}(\tau)$ and $\vec{\mathbf{v}}(r) = \vec{\omega}(r)$, where

$$\vec{\omega}(\cdot) := (-\mathcal{R}_0\, s(\cdot), \mathcal{R}_0\, s(\cdot) - 1, 1). \tag{8}$$

Hence, these velocity vectors, at any given instant, differ only in length. Observe that $\vec{\omega}(r_*) = (-s_0, s_0 - 1, 1)$ and if $s_0 \sim 1$, it reduces to $(-1, 0, 1)$.

Since in the velocity vector $\vec{\omega}$, the variability is given by $s(\cdot)$, in the idea of considering the SIR model as the uniform case, this uniformity (due to a constant velocity) is determined by the basic reproduction number $\mathcal{R}_0$, this is the parameter characterizing the process. We will call $\mathcal{R}_0$ the *reproduction speed*. Although the reproduction number depends on the parameter pair (β, γ), we see in (1) that β defines the flow from the susceptible group to the infectious group, determining a *transmission speed*.

In summary, β determines the speed of movement of the population in terms of transmission but does not completely characterize the change produced in the Ω space unless β equals $\mathcal{R}_0$. For example, this would be the case when using the time scale $\tau = \gamma t$.

2.4. Momentum

Having defined the *transmission speed* as the parameter β in (1), it is possible to continue exploring the analogy with kinematics, for example, by visualizing the movement of the population position as that of a vehicle powered by internal combustion. This vehicle, at speed β, at any time t, has a fuel reserve $m_s(t)$, an activated mass $m_i(t)$, and residual material from the process equal to $m_r(t)$. In this sense, the fact that these masses add up to N (a constant) could be interpreted with the law of mass conservation. On the other hand, given the existence of speed and mass, it is natural to think about momentum.

At least two momentum indicators are observed (the product of a mass and its velocity) for the model (1); these are defined by:

$$f_i(t) := \beta\, i(t), \quad \text{and} \quad f_s(t) := \beta\, s(t). \tag{9}$$

Therefore, understanding the scalar character of β as a speed and the relative masses $i = m_I/N$ and $s = m_S/N$, we will call them *transmission infective momentum* and *transmission susceptible momentum*, at instant t, respectively. The epidemiological literature, since the introduction of the concept by Hugo Muench in [35], calls f_i the force of infection (FOI). Our approach does not share such a name since we eventually seek to work with a Newtonian sense, not the Aristotelian sense of force [36]. However, in our construction, the invitation is to state the analogy with the idea of momentum, which we hope will allow us to advance to postulate a dynamic.

System (1), in the function of those moments, becomes:

$$\left\{ \begin{array}{rcl} f_s' & = & -f_s\, f_i \\ f_i' & = & (f_s - \gamma) f_i \\ r' & = & f_i/\mathcal{R}_0, \end{array} \right. \tag{10}$$

which shows that the *transmission susceptible momentum* always decreases at a relative velocity that is proportional to the *transmission infective momentum*. The latter increases or decreases depending on if f_s is greater or less than γ. This condition is equivalent to the effective reproduction number $\mathcal{R}_e := (\beta/\gamma)s(t) = f_s/\gamma$ being greater or less than one.

System (10) in differential form implies that $\mathrm{d}f_i = \gamma\{1/\mathcal{R}_e - 1\}\,\mathrm{d}\mathcal{R}_e$. Then, through direct integration, we conclude that the process follows a trajectory that lays on the integral curve

$$f_i(t) - f_i(0) = (f_s(0) - f_s(t)) - \gamma \ln(f_s(0)/f_s(t)). \tag{11}$$

Observe that when dividing (11) by γ, we can rewrite it as

$$g_i(t) - g_i(0) = (\mathcal{R}_e(0) - \mathcal{R}_e(t)) - \ln(\mathcal{R}_e(0)/\mathcal{R}_e(t)), \tag{12}$$

where $g_i(t) := \mathcal{R}_0\, i(t)$ and $g_s(t) := \mathcal{R}_0\, s(t) = \mathcal{R}_e(t)$, which we denominate, respectively, *reproduction infective momentum* and *reproduction susceptible momentum*, at time t. From (12), if $s_0 \sim 1$, then $\mathcal{R}_e(0) \sim \mathcal{R}_0$ and $g_i(0) \sim 0$, hence $g_i = (\mathcal{R}_0 - \mathcal{R}_e) - \ln(\mathcal{R}_0/\mathcal{R}_e)$.

2.5. Acceleration

The trajectory of a uniform process (defined by constant parameters β and γ), when assuming γ is fixed, is determined by modifying β, i.e., by modifying the transmission velocity. In the SIR model, β characterizes the population according to the transmission velocity of the disease that affects it. It determines the pattern of close contacts and transmissibility, dependent on cultural and environmental factors. Therefore, if in the presence of the disease, the population decides or is mandated to change behavior, or if the environment responsible for the transmission changes significantly, the value of β, intrinsic to the population, will change. Mathematically, this means that $\beta' = 0$ will no longer hold. Therefore, having understood β as velocity, β' can be naturally named *transmission acceleration*, consequently giving rise to a non-uniform movement process. Undoubtedly, the presence of high-risk diseases with high intrinsic transmission velocity that produces $\mathcal{R}_0 > 1$ forces health authorities to implement measures to reduce β. Due to the recent COVID-19 pandemic, one can find several alternative ways in the literature to model a variable behavior of the velocity β; see [28,37–49].

In this sense, and inspired by the β-SEIR model introduced in [45], one can observe that introducing a new differential equation describing the dynamics of β implicitly introduces variations in the transmission velocity to break the uniform process, and efforts are being made to achieve a non-zero acceleration (e.g., to reduce the reproduction number), through which one is implicitly exploring the terminology of reaction and restoration force.

We must declare that the concept of acceleration is key to the authors' future purpose, which is first to establish a kinematic theory of contagion processes, with what is presented in this article as the first advances in the context of a SIR representation. Although, subsequently, in the mean-time, the main objective is to support a dynamic theory of contagion processes that allows us to install the language of forces, for example, of a resultant force (or net force) that argues for the introduction of variable transmission rates (beta) (i.e., non-zero acceleration). For example, obtaining the first equation of the beta–SEIR system (5) in [45] or of the beta–SIR system (1) of [49] as the formal result after the concurrence of three forces: the infection force, the mitigation force and, in addition, a restoration force.

A natural extension is to denote $\mathcal{R}_0'$ the *reproduction acceleration*. This acceleration is related to the *transmission acceleration* through the equation $\mathcal{R}_0'/\mathcal{R}_0 = \beta'/\beta - \gamma'/\gamma$, which relates the relative changes in $\mathcal{R}_0$, β and γ when differentiating $\mathcal{R}_0 = \beta/\gamma$.

Table 1 describes the relation of epidemiological concepts with their analogies in kinematics.

Table 1. Relation of epidemiological concepts resulting from analogies with kinematics.

Name	Definition	Notation
Population mass	Total population size	N
Epidemiological space	$\{(x_s, x_i, x_r) \in \mathbb{R}_0^+ : x_s + x_i + x_r = 1\}$	Ω
Population position	$(s(t), i(t), r(t))$	$P(t)$
Epidemiological trajectory	$(s(\cdot), i(\cdot), r(\cdot))$	$P(\cdot)$
Susceptible mass	$N\,s(t)$	$m_s(t)$
Infective mass	$N\,i(t)$	$m_i(t)$
Removed mass	$N\,r(t)$	$m_r(t)$
Population mass point	$(m_s(t), m_i(t), m_r(t))$	$P_m(t)$
Mass epidemiological space	$\{(y_s, y_i, y_r) \in \mathbb{R}_0^+ : y_s + y_i + y_r = N\}$	Ω_N
Epidemiological trajectory	$\{(s(t), i(t), r(t)) : t \geq 0\}$	$P(\cdot)$
Displacement	$\varphi(s_2) - \varphi(s_1)$	$\Phi(s_1, s_2)$
Reproduction speed or Basic reproduction number	$\mathcal{R}_0(\cdot)$	$\mathcal{R}_0(\cdot)$
Transmission speed	$\beta(\cdot)$	$\beta(\cdot)$
Transmission infective momentum	$\beta\,i$	f_i
Transmission susceptible momentum	$\beta\,s$	f_s
Reproduction infective momentum	$\mathcal{R}_0\,i$	$\tilde{f}_i$
Reproduction susceptible momentum or Effective reproduction number	$\mathcal{R}_0\,s$	$\tilde{f}_s$
Transmission acceleration	$\beta'(\cdot)$	$\beta'(\cdot)$
Reproduction acceleration	$\mathcal{R}_0'(\cdot)$	$\mathcal{R}_0'(\cdot)$

3. Results

To more appropriately quantify the magnitude of a displacement, and inspired by the right-hand side of Equation (12), we define the function $\varphi : (0, 1] \to (0, \infty]$ by

$$\varphi(x) := (x - 1) - \ln(x). \tag{13}$$

Some properties of $\varphi(\cdot)$ (see Figure 4) that will be useful later are:

(a) $\varphi(\cdot) \geq 0$.
In fact, the graph of the logarithm function in $(0, 1]$ lays below its tangent line $y(x) := x - 1$ taken at the point $(1, 0)$.

(b) $\varphi(x) = 0$ if and only if $x = 1$.
If we assume $\varphi(x) = 0$, this implies that $y(\cdot)$ defined as in (a) satisfies $y(x) = \ln(x)$.

Due to the concavity of the logarithm function, this only occurs at the tangent point, i.e., when $x = 1$.

(c) $\varphi(x_1) = \varphi(x_2)$ if and only if $x_1 = x_2$.
We can define the following function to describe the difference between the two images as follows:

$$\Phi(x_1, x_2) := \varphi(x_2) - \varphi(x_1) = \left(1 - \frac{\ln(x_2) - \ln(x_1)}{x_2 - x_1}\right)(x_2 - x_1). \tag{14}$$

If we assume $\varphi(x_1) = \varphi(x_2)$, i.e., $\Phi(x_1, x_2) = 0$ and $x_1 \neq x_2$, we obtain a contradiction since, according to the mean value theorem, there exists x between x_1 and x_2 such that $\Phi(x_1, x_2) = \varphi'(x)(x_2 - x_1) = (1 - \ln'(x))(x_2 - x_1) = (1 - x)(x_1 - x_2)/x \neq 0$.

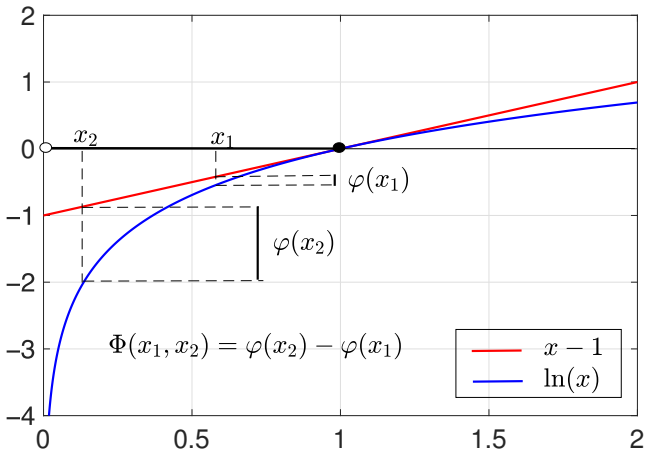


Figure 4. Function $\varphi(x) := (x - 1) - \ln(x)$, for $x \in (0, 1]$, represents the distance between the tangent line (red line) at $(1, 0)$ of the logarithm function and its image (blue curve).

Observe that the function $\Phi : (0, 1] \times (0, 1] \to \mathbb{R}$ defined in (c) by Equation (14) satisfies the following properties:

(A) $\Phi(x_1, x_2) > 0$ if $x_1 > x_2$.
(B) $\Phi(x_1, x_2) = 0$ if and only if $x_1 = x_2$.
(C) $\Phi(x_1, x_2) = -\Phi(x_2, x_1)$.
(D) $\Phi(x_1, x_2) = \Phi(x_1, x) + \Phi(x, x_2)$ for all $x \in (0, 1]$.

With these properties ((A)–(D)), it is easy to show that $((0, 1], |\Phi(\cdot, \cdot)|)$ defines a metric space (e.g., (D) shows the triangle inequality). Even more, given $X_k = (s_k, i_k, r_k) \in \Omega^\circ$, where $\Omega^\circ := \Omega \setminus \{(0, x_i, x_r) \in \Omega\}$, $k \in \{1, 2\}$, and the function

$$\mathbf{d}(X_1, X_2) = |i_2 - i_1| + |\Phi(s_1, s_2)|, \tag{15}$$

then we have the following statement:

Theorem 1. *The pair* $(\Omega^\circ, \mathbf{d})$, *with* $\mathbf{d}(\cdot, \cdot)$ *defined by (15) forms a metric space.*

Proof. Let us consider some generic $X_i = (s_k, i_k, r_k) \in \Omega^\circ$, $k \in \{1, 2, 3\}$. It is clear that (i) $\mathbf{d}(X_1, X_2) \geq 0$. (ii) The proposition $\mathbf{d}(X_1, X_2) = 0$, i.e., $|i_2 - i_1| + |\Phi(s_1, s_2)| = 0$ is equivalent to $i_1 = i$ and $\Phi(s_1, s_2) =$. However, by the above property (B) for Φ, this is equivalent to $(i_1, s_1) = (i_2, s - 2)$, and obviously $r_1 = 1 - (s_1 + i_i) = 1 - (s_2 + i_2) = r_2$. (iii) By property (C) for Φ, it follows $\mathbf{d}(X_1, X_2) = \mathbf{d}(X_2, X_1)$. (iv) The triangular inequality $\mathbf{d}(X_1, X_3) \leq \mathbf{d}(X_1, X_2) + \mathbf{d}(X_2, X_3)$ follows from property (D) of Φ and the use of the absolute value properties. □

Figure 5 shows an example of a neighborhood defined by the metric of the previous theorem; it has its center at the point $(1/e, 1/e, 1-2/e)$, and its radius is $1/8$.

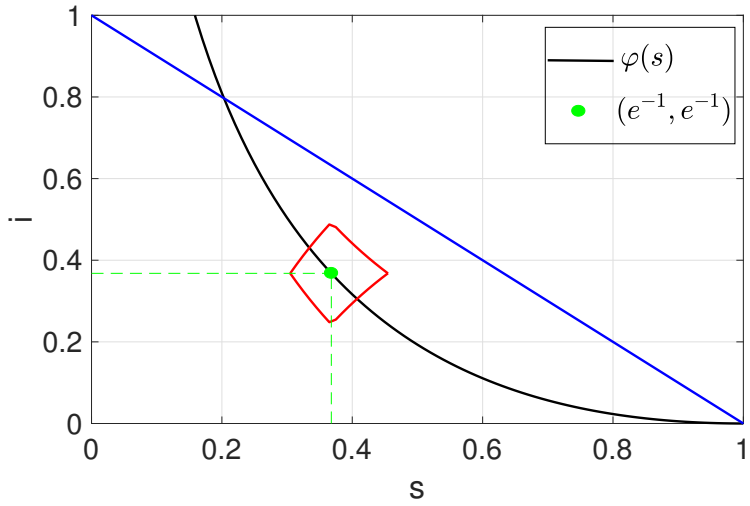


Figure 5. The graph of the $\varphi(\cdot)$ function is shown in black. The straight line $s + i = 1$ is shown in blue. The interior of the region bounded by the red graph shows the neighborhood formed by the points (s, i, r), $r = 1 - s - i$, whose distance $\mathbf{d}$ from the center (in green) $(e^{-1}, e^{-1}, 1 - 2e^{-1})$ is less than $1/8$. The "sides" of this neighborhood are obtained as parallel transports of the graphs of $\varphi(s)$ or $-\varphi(s)$.

Theorem 2. *If $P(t) = (s(t), i(t), r(t))$, $t \geq 0$, represents a trajectory of the population determined by system (1) and if the time instants t_1 and t_2, are such that $0 \leq t_1 < t_2$, then $s_1 > s_2$ and*

$$\Phi(s_1, s_2) := \varphi(s_2) - \varphi(s_1) = (\mathcal{R}_0 - 1)(s_1 - s_2) + \mathcal{R}_0(i_1 - i_2) > 0. \tag{16}$$

where $x_k := x(t_k)$, $k \in \{1, 2\}$ and $x \in \{s, i, r\}$.

Proof. The proof is a direct application of $\Phi(s_1, s_2) = (s_2 - s_1) - \ln(s_2/s_1)$ and the equality (12), but simplifying by β/γ and considering $s_1 = s(0)$, and $s_2 = s(t)$, from where $\ln(s_2/s_1) = \mathcal{R}_0\{(s_2 - s_1) + (i_2 - i_1)\}$. Therefore, it follows (16). □

Thinking about the representation given by (4), when the number of removed gives the clock, let us consider the notation $\Delta x(r) := x(r+1) - x(r)$, i.e., the marginal variation, where $x(\cdot)$ is some function of the variable r, such as $s(\cdot)$ or $i(\cdot)$.

Theorem 3. *If $P(t) = (s(t), i(t), r(t))$, $t \geq 0$, represents a trajectory of the population determined by system (1) and if the time instants t_1 and t_2 are such that $0 \leq t_1 < t_2$, $r(t_1) = r$ and $r(t_2) = r + 1$, for some $r > 0$, then $\Delta\mathbf{d}(r) = (\mathcal{R}_0 - 1) + \{|\Delta i(r)| - \Delta i(r)\}$, where $\mathbf{d}(r) = \mathbf{d}(P(t_1), P(t_2))$. In particular, if $r < r_*$, then*

$$\Delta\mathbf{d}(r) = \mathcal{R}_0 - 1. \tag{17}$$

Note that if t_1 is large enough, then $\Delta i(r) \sim 0$, so $\Delta\mathbf{d}(r) \sim \mathcal{R}_0 - 1$.

Proof. Since $s(\cdot) = 1 - i(\cdot) - r(\cdot)$, for any pair of instants t_1 and t_2, we have $s(t_1) - s(t_2) = -\{i(t_1) - i(t_2)\} - \{r(t_1) - r(t_2)\}$. Thus, by (16), $\Phi(s(t_1), s(t_2))$ is equal to $\{i(t_1) - i(t_2)\} + (\mathcal{R}_0 - 1)\{r(t_2) - r(t_1)\}$. Therefore, by (15), $\mathbf{d}(P(t_1), P(t_2))$ is equal to $|i(t_2) - i(t_1)| + |\{i(t_1) - i(t_2)\} + (\mathcal{R}_0 - 1)|$, given that $\{r(t_2) - r(t_1)\} = (r+1) - r = 1$. Therefore, changing the clock,

$$\Delta\mathbf{d}(r) = (\mathcal{R}_0 - 1) + \{|\Delta i(r)| - \Delta i(r)\}.$$

Now, if $r < r_*$, then $i(\cdot)$ increases on $[t_1, t_2]$. Therefore, $\Delta i(r) = i(t_2) - i(t_1) > 0$, so (17) is immediate. □

Now, for any r_1 and r_2, $0 < r_1 < r_2$, from the equality satisfying $\mathbf{d}(P(r_1), P(r_2))$ in the above proof and the mean value theorem, we can assert that $\mathbf{d}(P(r_1), P(r_2))/(r_2 - r_1)$ is equal to $i'(\tilde{r})$, with $r_1 < \tilde{r} < r_2$. Noting that $i'(\tilde{r}) < \mathcal{R}_0\, s(\tilde{r}) - 1 < \mathcal{R}_0 - 1$, it follows that:

$$\mathbf{d}(P(r_1), P(r_2))/(r_2 - r_1) = (\mathcal{R}_0 - 1) \; + \; |i'(\tilde{r})|\{1 - sgn(i'(\tilde{r}))\}.$$

In particular, if $r_2 < r_*$, then $\mathbf{d}(P(r_1), P(r_2))/(r_2 - r_1) = \mathcal{R}_0 - 1$.

Let us assume that our time interval is $[0, t_*]$, with $i(t_*) = i_*$. Taking into account an initial condition $P(0) = (1, i_0, 0)$, if we define $\mathbf{D}(t) = \mathbf{d}(P(0), P(t))$, we have $\mathbf{D}(t) = \{i(t) - i_0\} + \varphi(s(t))$, since $\varphi(1) = 0$. Then, if $0 \le t_1 < t_2 \le t_*$, we have $\mathbf{D}(t_2) - \mathbf{D}(t_2) = \mathbf{d}(P(t_1), P(t_2))$, given that $i(t_2) > i(t_1)$ and $\varphi(s(t_2)) > \varphi(s(t_1))$. Therefore, $\{\mathbf{D}(t_1) - \mathbf{D}(t_2)\}/\{r_2 - r_1\} = \mathcal{R}_0 - 1$. This is, $\partial \mathbf{D}/\partial r = \mathcal{R}_0 - 1$ or in its integral form

$$\mathbf{D}(r) = (\mathcal{R}_0 - 1)\, r, \;\; 0 \le r \le r_*. \tag{18}$$

That is an explicit linear form for the total displacement as a function of the removal flow.

Theorem 4. *If $P(t) = (s(t), i(t), r(t))$, $t \ge 0$, represents a trajectory of the population determined by system (1) and if the time instants t_1 and t_2, are such that $0 \le t_1 < t_2$, then*

$$\Phi(s(t_1), s(t_2)) = \beta \int_{t_1}^{t_2} \{1 - s\} i \, dt. \tag{19}$$

Proof. Equation (19) is obtained first by integrating $[t_1, t_2]$ on the equality $\beta\{1 - s\}i = \mathcal{R}_0 r' + s'$, which is obtained directly from the first and third equations of system (1)). Therefore, it follows $\int_{[t_1, t_2]} (1 - s) i \, dt = \mathcal{R}_0(r(t_2) - r(t_1)) + (s(t_2) - s(t_1))$. Then, the proof finishes using the expression in (16). □

Returning to the concept of velocity, using the definition of displacement that we have introduced, we see that given a trajectory, velocity is a measure of the rhythm of the displacement produced in a certain time period, typically through the quotient of the displacement per length of the period of time passed. Hence, assuming differentiability, we have that if $v(t)$ is the limit when $h \to 0$, of $\Phi(s(t), s(t + h))/h$, then, due to (19), we obtain $v(t) = -\{1 - s(t)\}\, s'(t)/s(t)$. Considering an SIR trajectory, this would be

$$v(t) = -\beta\{1 - s(t)\}\, i(t). \tag{20}$$

This velocity is proportional to the product between individuals that have lost susceptibility and the size of the infectious (active) group. Furthermore, this reaffirms that the transmission rate β, as far as being a rate, is an essential velocity parameter.

4. Discussion

4.1. From the Epistemological Viewpoint

This work aims to form a proposal to describe contagion processes through the construction of analogies (recognized as the first inferential mechanism) with processes inherent to physical phenomena, such as those related to the movement of a particle. In doing so, we propose exploring the language that kinematics provides as mechanics' first branch. Undoubtedly, this route involves oversimplified assumptions—a cost always inherent to modeling. Thus, trying to put our approach in an epistemological theoretical context, we frame it in the vision inaugurated by Charles Pierce. In his vision of the hypothetical-deductive method, he adds to the context of validation (induction–deduction) the context of discovery with the corresponding analogy–abduction interactions. That is, following the

route that tries to answer questions such as: What does it look like (in this case, the contagion process)? To what other processes in well-known areas does it resemble? Therefore, the idea is to advance in "abduction" (see below) and to propose and create an explanatory hypothesis [50,51].

This formalist proposal will be incomplete as long as the conceptual elements presented, all abstract, do not end in their operational equivalents, that is, that they admit measurements to be sustained in the mentioned validation context. This work expects to generate a change in perspective in favor of the opportunity for new questions and hypotheses. "When a scientist encounters an unknown phenomenon, he or she must resort to analogical thinking and ask him or herself: "What does it look like?..." This inferential mechanism, of undoubted value in discoveries, is called *abduction*. When this hypothesis is sufficiently validated, it becomes part of the heritage of a discipline; it is used as a doctrinal referential framework, from which it is inferred (*deduction*), and thus science is done with the hypothetical-deductive method" [52].

4.2. From the Kinematic Viewpoint

The intention of all science is always to obtain the best approximation to the phenomenon under study through a model, which remains true when the language expressing such model is mathematical or mathematized, such as that of physics, i.e., something more precise, opening or expanding the space of significance. Therefore, important is the perspective that results from considering the (β, γ)-SIR model as the visualization of changes between compartments as a uniform movement (defined by constant parameters). It contains a single determinant, a single constant parameter, namely, the basic reproductive number, which determines a constant base speed of reproduction of the phenomenon. At this point, understanding the changes in this parameter, in particular, in its transmission velocity component, i.e., β, leads to the upcoming challenge of development efforts—-especially in the conceptualization of ideas—which allow us to describe the forces that activate and drive population behavior and that explain the aforementioned changes. In other words, the perspective introduced cannot be considered closed, even in its abductive stage, as long as we do not expand it to a more robust one, for example, by passing from a kinematic to a dynamic perspective, to complete the mechanics of the transmission processes associated with infectious-contagious diseases.

5. Conclusions

The World Health Organization (WHO) uses the term "disease X" to refer to an emerging pathology associated with an unknown pathogen, the importance of which is such that it could be the precursor to a future pandemic. Increasing human incursion into wild ecosystems increases the likelihood of transmission of zoonotic pathogens among the human population. In this context, humankind must have a broader and more diverse set of tools for understanding and managing such risks through its professionals and specialized agencies.

This work presents the first part of a Newtonian mechanical theory of contagion, which seeks to diversify the understanding of the processes of spreading infectious diseases. More specifically, a minimal decalogue of kinematic concepts is established, focused on the description of motion, to advance later (future) works toward dynamics that address the causal forces and moments involved. The methodology employed consisted of elaborating a correspondence through an analogy between the kinematics of uniform rectilinear motion and the epidemic processes that use the SIR-type compartment model in their modeling. In other words, analogous relationships are established between their most important concepts.

In this sense, a natural space for the movement was defined, understanding by movement the change in the relative values of the state vector (the position), which is composed of the fraction of susceptible (s), infectious (i), and removed (r), in the set Ω of the trios (s, i, r) of non-negative components, subject to the condition $s + i + r = 1$. As a main result,

this space was endowed with a metric (see Equation (15)) that allows us to conclude that the displacement in Ω for each new individual removed is a constant equivalent to the excess with respect to one of the basic reproductive numbers (see Equation (17)), so we also call $\mathcal{R}_0$ the reproduction speed.

It should be noted that by considering the velocity at an instant as the tangent vector to the trajectory of states at that time, we have shown that the velocity is equal to the expression of the equation in Equation (8) when time is measured as a function of the flow of individuals removed. This velocity vector can be expressed as $(-\mathcal{R}_e, \mathcal{R}_e - 1, 1)$, where $\mathcal{R}_e$ represents the effective reproductive number. On the other hand, two concepts that will gain relevance in future mechanics of contagion are what we have termed "transmission infective momentum" (f_i) and "transmission susceptible momentum" (f_s), which, under this analogy, represent the amount of movement of susceptible and infective groups, respectively. Although the current literature refers to f_i as an infective force, this should be interpreted in an Aristotelian sense (mass times velocity) and not as a force in the Newtonian context (mass times acceleration).

Assuming $\mathcal{R}_0(t) := \beta(t)/\gamma$, we define $\mathcal{R}_0'$ as the "reproduction acceleration", determined by the "transmission acceleration" $\beta'(\cdot)$. This concept will become relevant in future mechanics proposals since it will facilitate the transition to the concept of force.

In summary, this proposal introduces a novel theoretical way to address contagion processes in kinematic terms and offers potential for elevation and justification through constructing a dynamic contagion theory. This approach corresponds to our immediate future project, which, as the authors' state, is already in advanced stages and aims to elucidate the causalities underlying epidemic mobility. It is also hoped that this dynamic theory will prove helpful in the foundation of more tactical or operational mathematical models.

Author Contributions: Conceptualization, F.C.-L.; methodology, F.C.-L., J.P.G.-J. and K.V.-G.; software, J.P.G.-J.; validation, F.C.-L., J.P.G.-J. and K.V.-G.; formal analysis, F.C.-L.; investigation, F.C.-L., J.P.G.-J. and K.V.-G.; resources, F.C.-L. and J.P.G.-J.; data curation, F.C.-L. and J.P.G.-J.; writing—original draft preparation, F.C.-L., J.P.G.-J. and K.V.-G.; writing—review and editing, F.C.-L., J.P.G.-J. and K.V.-G.; visualization, F.C.-L., J.P.G.-J. and K.V.-G.; supervision, F.C.-L.; project administration, F.C.-L. and J.P.G.-J.; funding acquisition, F.C.-L., J.P.G.-J. and K.V.-G. All authors have read and agreed to the published version of the manuscript.

Funding: This research was funded by Agencia Nacional de Investigación y Desarrollo (ANID) of Chile, FONDECYT Regular, grant number 1231256.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: The data presented in this study are available on request from the corresponding author.

Acknowledgments: The following study was supported by Universidad Católica del Maule.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. The Editors of Encyclopaedia Britannica. *Kinematics*; Encyclopedia Britannica: Edinburgh, UK, 2024.
2. Mariño, X. *La Conquista del Lenguaje: Una Mirada a la Evolución de la Mente Simbólica*; Shackleton Books: Oxford, UK, 2020.
3. Ángel Rodríguez, M. *El Proceso de la Ciencia*; Fondo Editorial de la UIGV: Lima, Perú, 2010.
4. Córdova-Lepe, F. Some ontological and epistemic aspects for modeling in interdisciplinary research. *Rev. Model. Mat. Sist. Biol.* **2023**, *3*, 1.
5. Treagust, D.F. The importance of multiple representations for teaching and learning science. In *Education Research Highlights in Mathematics, Science and Technology*; International Society for Research in Education and Science: Konya, Türkiye, 2018; pp. 215–222.
6. Opfermann, M.; Schmeck, A.; Fischer, H.E. Multiple representations in physics and science education—Why should we use them? In *Multiple Representations in Physics Education*; Springer: Cham, Switzerland, 2017; pp. 1–22.
7. Ramos-Jiliberto, R. *Deja a la Estructura Hablar: Modelización y Análisis de Sistemas Naturales, Sociales y Socioecológicos*; Ediciones UM: Santiago, Chile, 2020.

8. Conway, G.R. Strategic models. In *Pest and Pathogen Control: Strategic, Tactical and Policy Models*; John Willey & Sons: New York, NY, USA, 1984; pp. 15–28.
9. Rodrigues, H.S. Application of SIR epidemiological model: New trends. *arXiv* **2016**, arXiv:1611.02565.
10. Zhao, L.; Cui, H.; Qiu, X.; Wang, X.; Wang, J. SIR rumor spreading model in the new media age. *Phys. A Stat. Mech. Its Appl.* **2013**, *392*, 995–1003. [CrossRef]
11. Qiu, L.; Jia, W.; Niu, W.; Zhang, M.; Liu, S. SIR-IM: SIR rumor spreading model with influence mechanism in social networks. *Soft Comput.* **2021**, *25*, 13949–13958. [CrossRef]
12. Kandhway, K.; Kuri, J. How to run a campaign: Optimal control of SIS and SIR information epidemics. *Appl. Math. Comput.* **2014**, *231*, 79–92. [CrossRef]
13. Mann, R.P.; Faria, J.; Sumpter, D.J.; Krause, J. The dynamics of audience applause. *J. R. Soc. Interface* **2013**, *10*, 20130466. [CrossRef]
14. Bettencourt, L.M.; Cintrón-Arias, A.; Kaiser, D.I.; Castillo-Chávez, C. The power of a good idea: Quantitative modeling of the spread of ideas from epidemiological models. *Phys. A Stat. Mech. Its Appl.* **2006**, *364*, 513–536. [CrossRef]
15. Leibnitz, K.; Hoßfeld, T.; Wakamiya, N.; Murata, M. Modeling of epidemic diffusion in peer-to-peer file-sharing networks. In Proceedings of the Biologically Inspired Approaches to Advanced Information Technology: Second International Workshop, BioADIT 2006, Osaka, Japan, 26–27 January 2006; pp. 322–329.
16. Zhu, Q.; Yang, X.; Ren, J. Modeling and analysis of the spread of computer virus. *Commun. Nonlinear Sci. Numer. Simul.* **2012**, *17*, 5117–5124. [CrossRef]
17. Aadland, D.; Finnoff, D.; Huang, K.X. The Dynamics of Economic Epidemiology Equilibria. 2011. Available online: https://ideas.repec.org/p/pra/mprapa/29299.html (accessed on 21 July 2024).
18. Elliott, M.; Golub, B.; Jackson, M.O. Financial networks and contagion. *Am. Econ. Rev.* **2014**, *104*, 3115–3153. [CrossRef]
19. Munz, P.; Hudea, I.; Imad, J.; Smith, R.J. When zombies attack!: Mathematical modelling of an outbreak of zombie infection. *Infect. Dis. Model. Res. Prog.* **2009**, *4*, 133–150.
20. Cuesta-Herrera, L.; Pastenes, L.; Cordova-Lepe, F.; Arencibia, A.; Torres-Mantilla, H.; Gutierrez-Jara, J. Analysis of SEIR-type models used at the beginning of COVID-19 pandemic reported in high-impact journals. *Medwave* **2022**, *22*, 2552. [CrossRef]
21. Dreistadt, R. An analysis of the use of analogies and metaphors in science. *J. Psychol.* **1968**, *68*, 97–116. [CrossRef] [PubMed]
22. Gentner, D.; Jeziorski, M. 11. Historical shifts in the use of analogy in Science. In *Psychology of Science: Contributions to Metascience*; Cambridge University Press: Cambridge, UK, 1989; pp. 296–325.
23. Zhu, C.C.; Zhu, J. Spread trend of COVID-19 epidemic outbreak in China: Using exponential attractor method in a spatial heterogeneous SEIQR model. *Math. Biosci. Eng.* **2020**, *17*, 3062–3087. [CrossRef] [PubMed]
24. Inaba, H. Age-structured homogeneous epidemic systems with application to the MSEIR epidemic model. *J. Math. Biol.* **2007**, *54*, 101–146. [CrossRef]
25. Suriani, S.; Toaha, S.; Kasbawati, K. MSEICR Fractional Order Mathematical Model of The Spread Hepatitis B. *J. Mat. Stat. Dan Komputasi* **2021**, *17*, 314–324. [CrossRef]
26. Chen, D.; Yang, Y.; Zhang, Y.; Yu, W. Prediction of COVID-19 spread by sliding mSEIR observer. *Sci. China Inf. Sci.* **2020**, *63*, 1–13. [CrossRef]
27. Moein, S.; Nickaeen, N.; Roointan, A.; Borhani, N.; Heidary, Z.; Javanmard, S.H.; Ghaisari, J.; Gheisari, Y. Inefficiency of SIR models in forecasting COVID-19 epidemic: A case study of Isfahan. *Sci. Rep.* **2021**, *11*, 4725. [CrossRef]
28. Kolokolnikov, T.; Iron, D. Law of mass action and saturation in SIR model with application to Coronavirus modelling. *Infect. Dis. Model.* **2021**, *6*, 91–97. [CrossRef]
29. Law, K.B.; Peariasamy, K.M.; Gill, B.S.; Singh, S.; Sundram, B.M.; Rajendran, K.; Abdullah, N.H. Tracking the early depleting transmission dynamics of COVID-19 with a time-varying SIR model. *Sci. Rep.* **2020**, *10*, 21721. [CrossRef]
30. Taghvaei, A.; Georgiou, T.T.; Norton, L.; Tannenbaum, A. Fractional SIR epidemiological models. *Sci. Rep.* **2020**, *10*, 20882. [CrossRef]
31. Córdova-Lepe, F. On the ways to consider a variable transmission rate in a pandemic strategic SEIR model. In Proceedings of the Winter School on Mathematical Modelling in Epidemiology and Medicine, Valparaíso, Chile, 19–24 June 2023.
32. Córdova-Lepe, F. A Newtonian approach to the dynamics of the spread of high-risk infectious diseases. In Proceedings of the International Workshop "Mathematical Modelling of Epidemiological Dynamics", Le Havre, France, 17–21 June 2024.
33. Koopman, J.S.; Lynch, J.W. Individual causal models and population system models in epidemiology. *Am. J. Public Health* **1999**, *89*, 1170–1174. [CrossRef] [PubMed]
34. Onstad, D.W. Temporal and spatial scales in epidemiological concepts. *J. Theor. Biol.* **1992**, *158*, 495–515. [CrossRef] [PubMed]
35. Muench, H. *Catalytic Models in Epidemiology*; Harvard University Press: Cambridge, MA, USA, 1959.
36. Jammer, M. *Concepts of Force: A Study in the Foundations of Dynamics*; Courier Corporation: Chelmsford, MA, USA, 1999.
37. Towers, S.; Patterson-Lomba, O.; Castillo-Chavez, C. Temporal variations in the effective reproduction number of the 2014 West Africa Ebola outbreak. *PLoS Curr.* **2014**, *6*. [CrossRef] [PubMed]
38. Cowling, B.J.; Lau, M.S.; Ho, L.M.; Chuang, S.K.; Tsang, T.; Liu, S.H.; Leung, P.Y.; Lo, S.V.; Lau, E.H. The effective reproduction number of pandemic influenza: Prospective estimation. *Epidemiology* **2010**, *21*, 842–846. [CrossRef]
39. Tariq, A.; Undurraga, E.A.; Laborde, C.C.; Vogt-Geisse, K.; Luo, R.; Rothenberg, R.; Chowell, G. Transmission dynamics and control of COVID-19 in Chile, March–October, 2020. *PLoS Neglected Trop. Dis.* **2021**, *15*, e0009070. [CrossRef]

40. Santamaría, L.; Hortal, J. COVID-19 effective reproduction number dropped during Spain's nationwide dropdown, then spiked at lower-incidence regions. *Sci. Total Environ.* **2021**, *751*, 142257. [CrossRef]
41. Tang, B.; Bragazzi, N.L.; Li, Q.; Tang, S.; Xiao, Y.; Wu, J. An updated estimation of the risk of transmission of the novel coronavirus (2019-nCov). *Infect. Dis. Model.* **2020**, *5*, 248–255. [CrossRef]
42. Wang, X.; Gao, D.; Wang, J. Influence of human behavior on cholera dynamics. *Math. Biosci.* **2015**, *267*, 41–52. [CrossRef]
43. Córdova-Lepe, F.; Gutiérrez-Aguilar, R.; Gutiérrez-Jara, J.P. Number of COVID-19 cases in Chile at 120 days with data at 21/03/2020 and threshold of daily effort to flatten the epi-curve. *Medwave* **2020**, *20*, e7861. [CrossRef]
44. Cabrera, M.; Córdova-Lepe, F.; Gutiérrez-Jara, J.P.; Vogt-Geisse, K. An SIR-type epidemiological model that integrates social distancing as a dynamic law based on point prevalence and socio-behavioral factors. *Sci. Rep.* **2021**, *11*, 10170. [CrossRef]
45. Córdova-Lepe, F.; Vogt-Geisse, K. Adding a reaction-restoration type transmission rate dynamic-law to the basic SEIR COVID-19 model. *PLoS ONE* **2022**, *17*, e0269843. [CrossRef] [PubMed]
46. Gutiérrez-Jara, J.P.; Saracini, C. Risk perception influence on vaccination program on COVID-19 in Chile: A mathematical model. *Int. J. Environ. Res. Public Health* **2022**, *19*, 2022. [CrossRef] [PubMed]
47. Gutiérrez-Jara, J.P.; Vogt-Geisse, K.; Cabrera, M.; Córdova-Lepe, F.; Muñoz-Quezada, M.T. Effects of human mobility and behavior on disease transmission in a COVID-19 mathematical model. *Sci. Rep.* **2022**, *12*, 10840. [CrossRef] [PubMed]
48. Córdova-Lepe, F.; Gutiérrez-Jara, J.P. A dynamic reaction-restore-type transmission-rate model for COVID-19. *WSEAS Trans. Biol. Biomed.* **2024**, *22*, 118–130. [CrossRef]
49. Córdova-Lepe, F.; Gutiérrez-Jara, J.; Chowell, G. Influence of the Effective Reproduction Number on the SIR Model with a Dynamic Transmission Rate. *Mathematics* **2024**, *12*, 1793. [CrossRef]
50. Nubiola, J.C.S. Peirce y la Abducción. Analogía Filosófica XII/1. Available online: https://www.unav.es/gep/AN/ANIndice.html (accessed on 15 August 2024).
51. Martín, P.; del Carmen, M. Abducción, método científico e Historia. Un acercamiento al pensamiento de Charles Pierce. *Páginas* **2015**, *7*, 125–141. [CrossRef]
52. Carli, A. *La Ciencia Como Herramienta Guía para la Investigación y la Realización de Informes, Monografías y Tesis Científicas*; Biblios: Buenos Aires, Argentina, 2008.

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.