Suplemento de la Revista Mexicana de Física **1** (4) 59–66 SEPTEMBER-OCTOBER 2020

General thermodynamic efficiency loss, aging and Gompertz mortality law

Jorge A. Montemayor-Aldrete and Rafael F. Márquez-Caballé
Departamento de Estado Sólido, Instituto de Física, Universidad Nacional Autónoma de México,
Circuito de la Investigación Científica s/n, Ciudad Universitaria, 04510, Ciudad de México, México.

Received 5 May 2020; accepted 7 June 2020

A simple and general thermodynamic theory is applied to describe the irreversible aspects of the continuous process of functional efficiency loss, which occurs in dissipative biological structures after they reach maturity [1]. This theory, among other things, follows Prigogine [2] by considering that these dissipative structures perform their functions and carry out cyclical processes per se since they are self-organizing away from equilibrium. By using the irreversible thermodynamic theory of aging by Montemayor-Aldrete et al. [1], we have obtained results such as the following: The accumulated damage that occurs in dissipative biological structures after they reach maturity, which is the product of linear loss of functional efficiency with time, leads to the law of exponential mortality rate by Gompertz. Also, an extension of the irreversible aging theory of self-organized dissipative systems for the case of living organisms of variable body temperatures has been developed.

Keywords: Aging processes; Gompertz mortality rate; non-equilibrium thermodynamics; biophysics.

PACS: 05.70.Ln; 89.75.Fb; 87.10.-e; 87.18.-h

1. Introduction

Since prehistoric times, death and aging have been of great interest to humanity [3-6]. This concern about death is not unique to humans, it also exists in some of the higher mammal species [7-10]. With Tito Lucrecio Caro, the first prescientific document written without mention myths or fantasies about human or animal aging appears [11], and with Lavoisier, the path of scientific research on the aging of both, humans and animals, begins [12]. Nowadays many theories address the issue of aging, see [1,13-26]. The possible applications of the existing theories are multiple, the references [27-31] give an idea of the breath of the fields where they can be used to solve specific problems. Many theoretical works of irreversible thermodynamics about the aging of living beings have been developed [1,13,15,18,22-26].

Recently, one of them [1] has been used to explain why, on average, women live longer than men. The percentage discrepancy between the experimental value for the population of Europe and the numerical calculation was 2.5%. In addition, this theory allowed to describe experimental data about the linear fall of the functionality of several human body organs overtime after the first 30 years of life. This theory was recently used to determine the parameter of functional efficiency loss by functional cycle, α, for 71 living being's species as a function of mass covering 18 orders of magnitude [32]. Among other results, the mathematical adjustment allowed us to conclude that there is a minimum in the value of the α parameter for a 23.3 kilograms mass which is close enough to the Homo sapiens one.

In the theoretical framework published in the Montemayor-Aldrete *et al* [1] theory we will address two problems: First, the existing relationship between the aging rate of living organisms and the death rate in a population as a function of age will be established. Second, an extension of our irreversible aging theory of self-organized dissipative systems will be developed for the case of living organisms of variable body temperatures.

2. Theory

Before introducing our subject, we are going to summarize the main elements of the theory due to Montemayor-Aldrete *et al* [1]; this material will be presented with some additions and precisions that will make it clearer from a physical point of view.

All "macroscopic things", that more than things are macroscopic processes, together with the living beings that surround us, are subject to cyclic dissipative processes of diverse nature [2]. Everyday life provides us with a wealth of empirical evidence that many macroscopic objects and the living systems that surround us, at some point in their existence begin to age or deteriorate. And eventually, they suffer the fatal failure, that is, the cease of energy and matter exchange between the system and its surroundings it means that the dissipative structure disintegrates rapidly until it reaches a state of thermodynamic equilibrium.

From Prigogine [2], we know that the appearance of dissipative structures is due to the stationary state instability, which as a result of a bifurcation, the systems become self-organized far away from equilibrium. According to Nieto-Villar *et al.* [18]., essentially, such dissipative structures also emerge as a consequence of chaotic regimes that operate on the threshold of stationary states instabilities and are maintained by dissipating energy and mass to the environment. After biological beings reach sexual maturity, the chaotic regime tends to disappear, and the aging processes begin. Furthermore, experimental evidence indicates the functionality of the organs many living systems decays linearly as time goes by after reaching sexual maturity [33]; and eventually, the corresponding self-organizing dissipative systems stop

working, and the dissipative structures degrade on their way to reach thermodynamic equilibrium.

According to Th. De Donder, the second thermodynamic law allows to define the entropy of a system in its differential form, dS, as

$$dS \equiv dS_i + dS_e, \tag{1}$$

where dS_e is the entropy flux between the system and the environment and dS_i is the production of entropy during its evolution due to the irreversible processes [34], and by taking the temporal expression for the entropy production rate $\dot{S} \equiv (d_i S/dt)$ is obtained

$$\dot{S} \equiv \frac{d_i S}{dt} \equiv \sum_i J_i X_i. \tag{2}$$

Where J_i and X_i are respectively the flows and the generalized forces. On the following, the rate of entropy production, $d_i S/dt$ will be denoted by $\dot{S}$.

In such expression, it is possible to define the well-known Rayleigh dissipation function [35] as,

$$\Phi_R \equiv T\dot{S}, \tag{3}$$

which is not only applicable to dissipative biological systems but also to frictional heat production in Lagrangian systems; and in general, represents the heat production inside a system and it can be measured experimentally. For biological dissipative structures [1], far from the thermodynamic equilibrium, it is convenient to write the Eq. (3) per unit volume or mass, ϕ_R,

$$\phi_R = T\dot{s}. \tag{4}$$

For humans, there are two different outlines about the number of chronological stages that the entropy production rate goes through over their life span. On one hand, the first proposal due to Aoki [36] is based on his appreciation that

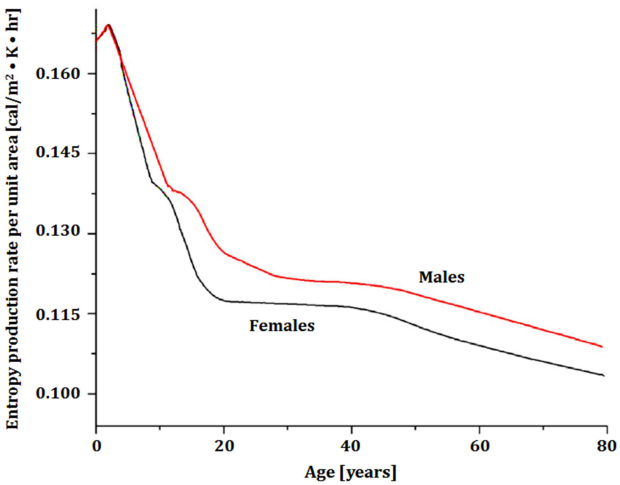

FIGURE 1. The rate of entropy production per surface area, for the healthy humans under basal conditions for both sex (upper curve for males and lower curve for females) and different age. This graph was elaborated with data taken from [1].

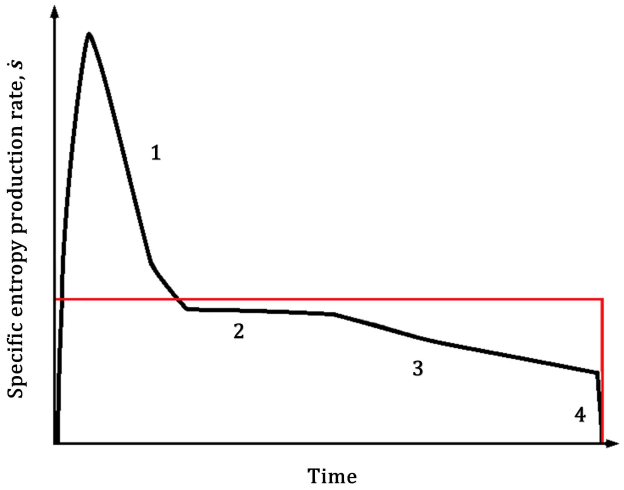

FIGURE 2. General schematic diagram corresponding to the specific entropy production rate, $\dot{s}$, versus time for a dissipative biological structures. 1) Initial transitory stage, with $\dot{s} > 0$, which corresponds to the growth of the structure, 2) Steady state that corresponds to a physical situation in which the production rate of dissipative structure is equal to the rate of its destruction. 3) The aging stage, where the destruction rate dominates the production processes. 4) The failure stage. Note: The rectangle has the same area as the schematized real function that describes the specific entropy production rate as a function of time.

the entropy production rate does not reach a steady-state value, and based on such proposition, he determines that there are three chronological stages; while on the other hand, Montemayor-Aldrete *et al*. [1] consider that for human beings the stationary states for the entropy production rate per unit body surface, exist, for the healthy humans under basal conditions for both sex and different age see Fig. 1. Based on such foundation, they consider that in a general qualitative way there are four chronological stages, not only for human beings but for all kinds of living beings. In Fig. 2, the general schematic behavior of the different chronological stages that occur in the living self-organized dissipative structures, can be observed: 1) An initial transitory stage, where the entropy production rate per unit volume, $\dot{s}$, grows more or less exponentially; after some time, the entropy production rate reaches a maximum value and begins to decrease. In this stage the organism develops and increases in size as time goes on, becoming larger and larger. 2) Then we arrive at a stationary stage or stable dissipative state, where $\dot{s}$ is approximately constant; this stage corresponds to a dissipative state where the dissipative structure rates of construction and destruction are equals to each other. At this stage, the specimen reaches its adult condition, and starting from there, aging occurs first at a small, almost imperceptible rate. 3) Aging in the adult stage corresponds to a stage which is characterized by an approximately linear decrease in $\dot{s}$ over time, corresponding to average linear decreases in the physiological functions of all organs and subsystems of the living being. 4) Finally, a short duration dissipative state corresponding to a catastrophic or destructive failure of the dissipative biological structure occurs.

All living dissipative systems develop functional cycles. Of these, the most important functional cycles are those related to the nutrients and oxygen assimilation rates (if applicable) for all and every one of the cells that constitute each living being. The heartbeat rate promotes the distribution of specific forms of free energy per unit of time throughout the body, which allows them to satisfy the needs of their systems. However, cycle after cycle, all living beings gradually decrease their capacity to perform work due to their functional disorder increase, which makes it increasingly difficult for the dissipative structure to obtain free energy and nutrients absorption from the outside, and finally, the catastrophic failure occurs [37, 38]. For non-living dissipative systems, these processes could be defined as a process of intrinsic progressive loss of functionality, which conducts to the ceasing of working properly and eventually to failure [39]. In other words: *Each dissipative structure that operates through the performance of functional cycles, necessarily generates a net amount of functional entropy or functional disorder in each cycle; and therefore, the structure cannot last beyond a maximum time of continuous work, which is characteristic of each structure. Or, the whole energy amount dissipated as heat by any dissipative structure during its total continuous operating time has a maximum value characteristic of each structure* [1]. This finite time of continuous operation is due to the increase in functional entropy or functional disorder that eventually causes the fatal breakdown or interruption of the dissipative machine operation.

If we consider that the whole process, corresponding to basal metabolic conditions, occurs at a constant absolute temperature T, (the treatment corresponding to basal metabolic conditions for variable body temperatures will be found in Appendix 1), for each dissipative system, in particular, the area under the curve entropy production rate per unit volume over the total time of continuous operation is the maximum entropy per unit mass generated during the total time of continuous operation. In mathematical language:

$$\int_{t_i}^{t_f} \frac{ds}{dt} * dt = \frac{K_s}{T}, \tag{5}$$

with K_s as the specific energy (per unit mass) dissipated during the total process of work for each type of dissipative machine or living system, where t_i and t_f are respectively the initial and final times. Using the mean value theorem of calculus, we have:

$$\overline{\frac{ds}{dt}}(t_f - t_i) = \frac{K_s}{T}, \tag{6}$$

where $\overline{ds}/dt$ is the average value of the entropy production rate specific to our system between the times t_i and t_f. Substituting the Rayleigh dissipation function per unit mass; $\phi_r = T\dot{s}$, (which represents the heat production rate within a system per unit mass), we get $(\dot{\phi_R}/T)(t_f - t_i) = K_s/T$, therefore, the total operating time of the dissipative system is given by:

$$(t_f - t_i) = \frac{K_s}{\overline{\phi_R}}. \tag{7}$$

This expression shows directly that the dissipative system has a maximum of continuous operation, which is inversely proportional to the average of the (specific) Rayleigh dissipation function of the system, and directly proportional to its constant K_s, which is characteristic of every type of dissipative system.

It is clear that all dissipative structures perform functional cycles, which are repeated over and over again during their total continuous operation time, before each structure fails. One can define an average value τ for the duration of the dissipative cycles between t_i and t_f, through the following analysis: $t_f - (t_f - \delta t) + (t_f - \delta t) - (t_f - 2\delta t) + ... + (t_f - (n-1)\delta t) - (t_f - n\delta t) = n\delta t = (t_f - t_i)$. If we consider the maximum number of cycles performed by the dissipative system as $N_{\max}$, then $n\delta t = (t_f - t_i) = N_{\max}\tau$. Where is it clear that $n \ll N_{\max}$, and $t \gg \tau$. If we define τ_i as the cycle duration for each of the time intervals given by the n groups of cycles, then it is easy to obtain the following expression:

$$\frac{\tau_1 * [t_f - (t_f - \delta t)]}{\tau_1} + ... + \frac{\tau_n * \delta t}{\tau_n} = \sum_{i=1}^{n} n_i * \tau_i = (t_f - t_i) = N_{\max}\tau,$$

and therefore we have the average value τ of how long a dissipative cycle lasts, between t_i and t_f given by $\tau = (\sum_{i=1}^{n} n_i * \tau_i / N_{\max})$. Then, from Eq. (6) we have:

$$\frac{\overline{\phi_R}}{T}\tau\left(\frac{t_f - t_i}{\tau}\right) = \frac{K_s}{T}, \tag{8}$$

Eq. (8) indicates that each time τ, the living machine performs one of the $N_{\max} = [(t_f - t_i)/\tau]$ cycles, an amount of specific entropy equal to $(\overline{\phi_R}/T)\tau$ is produced.

On one hand, there is wide experimental evidence in biology, which indicates that the functionality of the organs in many living systems declines linearly over time after reaching sexual maturity [33]. On the other hand, the operation of the different organs of a self-organized living system requires the arrival of a certain free energy rate which allows the operation of the different organs and whose dissipation is the source of the corresponding entropy production rate. Thus, the union of the experimental evidence [33] and the theory of Prigogine [34] allow us to conclude that for many living systems the entropy production rate linearly declines as time goes by once they reach sexual maturity. Based on the above physical and phenomenological considerations, we can ponder that for times greater than that in which macroscopic signs of aging begin to be observed, t_{sd}, it is possible to perform an approximation on $\dot{s}(t)$, which can be expressed as a function relative to the value of steady-state entropy production rate, $\overline{\dot{s}_{ss}}$, like a bilinear function of α and t, $\dot{s} = \dot{s}(\alpha, t)$, where α it is defined as a thermodynamic aging parameter

that measures the functional decay of the dissipative systems in each cycle.

Therefore, $\dot{s}(t)$ for times greater than the time at which the stationary state begins to decay, t_{sd}, we must be able to express it as a development in Taylor series at first order according to the temporal difference between time t and the time t_{sd}. This development must be done around the value of the entropy production rate at steady state, $\overline{\dot{s}}_{ss}$, and the sign of the linear term proportional to $(t - t_{sd})$ must be negative. Such negative contribution must grow as the number of cycles of continuous operation grows, therefore such term must be divided by τ, so that we have the quotient $(t - t_{sd})/\tau$ to count the number of functional cycles performed between t_{sd} and t, where $t > t_{sd}$; and finally we need to define a symbol, α, to describe the thermodynamic aging parameter that measures the functional decay of the dissipative system in each cycle. Therefore, the equation that allows to express in a simple way $\dot{s}(t)$ during the decay stage of the stationary states due to aging is the following,

$$\dot{s(t)} = \overline{\dot{s}}_{ss} \left[1 - \alpha * \left(\frac{t - t_{sd}}{\tau} \right) \right], \tag{9}$$

where t_{sd} expresses the time in which the stationary state begins to decay. In addition, a new macroscopic physical variable related to the stationary state of entropy production called the *remaining fractional functionality* can be defined as,

$$\Pi_{Re}(t) \equiv \frac{\dot{s}(t)}{\overline{\dot{s}}_{ss}}. \tag{10}$$

The residual functionality, $\Pi_{Re}(t)$, in a physical sense, is not only the remaining functionality of any organ; but also, and fundamentally it is the quotient of the entropy production rate at time t and the steady-state entropy production rate of the whole dissipative system, or could also be evaluated for some particular organ. So,

$$\Pi_{Re}(t) = \left[1 - \alpha * \left(\frac{t - t_{sd}}{\tau} \right) \right]. \tag{11}$$

This equation allows to evaluate α, with the experimental knowledge of $\Pi_{Re}(t)$.

From the above equation it is also clear that when the dissipative system collapses, $\Pi_{Re}(t) = 0$. This result implies that α can also be evaluated from the total failure condition, which is expressed as follows,

$$\alpha = \frac{\tau}{t_f - t_{sd}}. \tag{12}$$

Hence, we have two equations from which we can determine the thermodynamic aging parameter α, Eqs. (11), and (12).

Once the development of the theory has allowed to define a thermodynamic aging parameter α, that measures the functional decay of the dissipative system in each cycle, it is immediate to ask what relationship exists between such thermodynamic parameter and the mortality rate of members of a population with a specific age within a population of a biological system. This problem will be covered in the next section.

2.1. Gompertz mortality law and Survival curves, obtained as a consequence of the previous irreversible thermodynamic theory of aging

In 1820, the actuary Benjamin Gompertz found that the mortality rate behavior of adult human populations as a function of time obeys a geometric progression law. Since then, it has been a valuable tool in demography and other scientific disciplines [40].

In modern notation [41], Gompertz mortality rate $\dot{R}(t)$ can be written as,

$$\dot{R(t)} = \dot{R}_o e^{\alpha_G t}, \tag{13}$$

where $\dot{R(t)}$ is the mortality rate in adulthood at time t, $\dot{R}_o$ is the mortality rate at initial time t_o, and α_G is the constant that describes the monotonic acceleration of aging. The experimental advantages of presenting Eq. (13) in logarithmic form are evident. It is easy to see that the expression for the surviving population of a group at time t, $S_{\text{sur}G}(t)$, associated with Gompertz's law is,

$$S_{\text{sur}G}(t) \equiv \exp \left[\frac{\dot{R}_o}{\alpha_G} * \left(1 - e^{\alpha_G t} \right) \right]. \tag{14}$$

According to Ricklefs and Scheuerlein [42], aging is a continuous decline in physiological functions with age, after maturity. Our theory through Eq. (11), agrees with the previous statement. Such equation, which refers to the residual functionality of any organ from any of all species, $\Pi_{Re}(t)$, is the result of the tendency that the second law of thermodynamics imposes on any self-organized dissipative system, to gradually age after upon reaching sexual maturity, such processes eventually lead to the dissipative system ceasing to exist as such; and reach the state of thermodynamic equilibrium. Similar considerations to those expressed by Ricklefs and Scheuerlein, although more precisely, they are due to Ames *et al.* [43]: "The degenerative diseases associated with aging include: cancer, cardiovascular disease, immune-system decline, brain dysfunction, and cataracts". Accordingly, with De and Ghosh [44], "aging is characterized by a progressive loss of physiological integrity, leading to impaired function and increased vulnerability to death. This deterioration is the primary risk factor for major human pathologies including cancer, diabetes, cardiovascular disorders, and neurodegenerative diseases". The decrease in physiological functions of any organism with age manifests itself in the populations of each species as an increase in the mortality rate at advanced ages. Many functions have been used to adjust experimental data related to death events in populations, among which the functions of Gompertz and Weibull stand out [42]. In this section, we will demonstrate that the Gompertz mortality law is a physical consequence of the irreversible theory of aging of Montemayor-Aldrete *et al.* [1].

From Eqs. (10)-(12) is straightforward to show that,

$$\dot{s}(t) = \bar{\dot{s}}_{ss}\left[1 - \left(\frac{t - t_{sd}}{t_f - t_{sd}}\right)\right]. \tag{15}$$

Defining the functional damage function $D(t)$ as:

$$D(t) \equiv \left(\frac{t - t_{sd}}{t_f - t_{sd}}\right), \tag{16}$$

it is possible to write the expression that relates the fractional damage with the residual fractional functionality as:

$$\Pi_{Re}(t) + D(t) = 1. \tag{17}$$

From Eqs. (12) and (16), the functional damage at time t, $D(t)$ can be written as,

$$D(t) = \alpha * \left(\frac{t - t_{sd}}{\tau}\right). \tag{18}$$

At this point we can ask ourselves: What is the death average probability for an individual member of a population whose age is given by a time t? This must be proportional to the accumulated damage which starts at the time t_{sd}, when the stationary state begins to decay. It should be noted that in large populations, subsets formed by people suffering from chronic diseases can be presented and can be globally characterized by their corresponding values of thermodynamic aging parameter α, which measures the functional decay of the dissipative system in each cycle. In what follows for these subsets, the value of α would simply be seen experimentally, which describes the accelerated aging of each of these subsets. In mathematical language we have,

$$\frac{P_{rob}(t)}{\delta t} = \beta\left[C + D(t)\right], \tag{19}$$

with $\beta > 0$ as a constant, and $C > 0$ also s a constant. In the first-time increment, $\delta t = t_1 - t_{sd}$, then $D(t_1 = t_{sd} + \delta t) = \alpha(\delta t/\tau)$ and Eq. (16) gives: $(P_{rob}(t)/\delta t) = \beta C(1 + (\alpha/C) * (\delta t/\tau))$. After n increments of magnitude δt each, we have,

$$\frac{P_{rob}(t)}{n\delta t} = \beta C\left(1 + \frac{\alpha}{C} * \frac{\delta t}{\tau}\right)^n, \tag{20}$$

with $n\delta t = dt$, $n \gg 1$ and $(\alpha/C\tau) \ll 1$, finally:

$$\dot{R}(t)_{Th} \equiv \frac{P_{rob}(t)}{dt} = \beta C * e^{\left(\frac{\alpha}{C\tau}\right)t}$$
$$\equiv \dot{R}(0)_{Th} * e^{\left(\frac{\alpha}{C\tau}\right)t}. \tag{21}$$

From the comparison between Eq. (21) with Eq. (13), the following equivalences are presented between theoretical and experimental parameters that appear in the expression of the Gompertz mortality rate:

$$\dot{R}(t)_{Th} \equiv \frac{P_{rob}(t)}{dt} = \dot{R}(t), \tag{22}$$

$$(\beta C)_{Th} = (\dot{R}_0)_{\exp}, \tag{23}$$

$$\left(\frac{\alpha}{C\tau}\right)_{Th} = \alpha_{G\exp}. \tag{24}$$

In addition, the corresponding Gompertz model for survival at time, t, is given according to the following analysis: For a population N the change in time for the fraction of the surviving population at age, $S_{\text{sur}}(t)$, is $(dS_{\text{sur}}(t)/dt)$; which can be written as the negative of the product of the mortality rate, multiplied by the fraction of the surviving population (both at time t), according to: $(dS_{\text{sur}}(t)/dt) = -\dot{R}(t) * S_{\text{sur}}(t)$, which is equivalent to:

$$\frac{1}{S_{\text{sur}}(t)} * \frac{dS_{\text{sur}}(t)}{dt} = -\dot{R}(0)_{Th} * e^{\left(\frac{\alpha}{C\tau}\right)t}. \tag{25}$$

After integrating between the limits for the following values the fraction of the survival rate to time $S_{\text{sur}}(0) = 1$ and $S_{\text{sur}}(t)$, which correspond respectively to the times $t = 0$ and t, we obtain the expression for survival at the time t, $S_{\text{sur}}(t)$, which is:

$$S_{\text{sur}}(t) = \exp\left[\left(\frac{\beta C^2\tau}{\alpha}\right) * \left(1 - e^{\left(\frac{\alpha}{C\tau}\right)t}\right)\right]. \tag{26}$$

If for a population N, we consider the fraction of the surviving population corresponding to an individual of the N, such as $S_{\text{sur}}(t_{\text{sol}})$, then the solution of the following expression contributes approximately to the maximum life-time for the members of such species t_{sol}, the subscript indicates the life span,

$$S_{\text{sur}}(t_{\text{sol}}) = \frac{1}{N} = \exp\left[\left(\frac{\beta C^2\tau}{\alpha}\right) * \left(1 - e^{\left(\frac{\alpha}{C\tau}\right)t}\right)\right], \tag{27}$$

the solution of the above equation for t_{sol} is:

$$t_{\text{sol}} = \left(\frac{C\tau}{\alpha}\right) * \ln\left[1 - \left(\frac{\alpha}{\beta C^2\tau}\right)\ln\left(\frac{1}{N}\right)\right], \tag{28}$$

and the comparison between Eq. (27) and (14) gives us the following equivalence between the survival functions coefficients given by one and another equation:

$$\left(\frac{\beta C^2\tau}{\alpha}\right)_{Th} = \frac{\dot{R}_0}{\alpha_G}. \tag{29}$$

At this stage, it is immediate to observe that Eq. (21), which corresponds to the law of the exponential growth rate of Gompertz mortality, has been obtained by using an irreversible thermodynamic aging theory [1]. In addition, such Equation provides a solid theoretical foundation to the historical consideration that the parameter, α_G , which appears in Eq. (13), is called the Gompertz aging parameter [45-47]. We consider that this demonstration gives a great physical foundation to Gompertz mortality law, as compared to other theories of mortality [33,48-56].

In addition, the previous analysis resolves from the root a paradox that consists on the linear decay of living systems organs functionality over time after they reach sexual maturity; and simultaneously, these populations experimentally obey the exponential law of mortality growth rate with age [33]. It should be noted that the rate of linear decay of the metabolic rate for human populations in adulthood is higher in men than for women [1, 57].

3. Discussion

According to Nieto-Villar *et al.*, in general, the aging theories can be grouped into three major groups: Those related to the action of reactive oxygen species, the theories that establish a link between metabolic rate and the longevity of organisms, and those that focus the aging processes in an irreversible thermodynamic scheme [31]. Despite their differences, essentially the three types of theory groups consider that aging leads to progressive loss of physiological integrity, leading to impaired function and increased vulnerability to death. Within the framework of our theory, the different degenerative diseases associated with the aging of a population are specific manifestations of the general loss of thermodynamic efficiency over time. Of course, the theory allows both the analysis of the macroscopic thermodynamic manifestations of a specific disease in a subset of the total population that suffers from it; as well as the comparison of their loss rate of functional efficiency with another subset of the population of the same age that is healthy. However, it should be remembered that our theory, as any other phenomenological irreversible theory of aging, only provides general tools for the experimental and phenomenological macroscopic study of different irreversible aspects of aging. In other words, this theory does not replace in any way the concrete study of the different chronic-degenerative diseases by means of theoretical approaches about microscopic-statistical mechanisms of physiochemical and cellular biophysics applied to complete organisms or organs thereof. It is clear that the second law asserts that a natural process runs only in one-time direction and is not reversible [34]. The theory of aging presented and applied here can be classified within the group of irreversible thermodynamic theory. Our proof of the Gompertz equation proof, which is based in the physical consideration that a growing cumulative damage occurs due to the continuous process of loss of the functional efficiency, is a further systematic confirmation of the irreversible nature of aging, which for the portion of the population that has reached maturity or adulthood gives rise to the law of exponential growth in the death rate over time. This result gives to Gompertz the expression of a great theoretical advantage in relation to other biological phenomenological expressions used to describe the mortality rate as a function of time.

We have extended our irreversible theory for the case of aging occurring under conditions of variable body temperatures, which will allow the study of living systems such as not homoeothermic animals, and plants including their ecosystems such as , trees, and forests. The approach here developed, see for instance Eqs. (11), (13) and (14) can be applied to contribute to the study of different problems, such as the long-term (and short -term) effect caused by air pollution in big cities or by lung virus infections on the aging rate of human lungs.

4. Conclusions

First, we have demonstrated that cumulative damage due to the continuous process of functional efficiency loss, which occurs in mature biological dissipative systems, leads to the Gompertz mortality law. This demonstration solves a paradox: The fact that the functionality of the organs of multicellular living beings that have reached maturity decays linearly over time and that simultaneously their populations obey the exponential Gompertz's mortality law over the elapsed time.

Second, an extension of the irreversible aging theory of self-organized dissipative systems for the case of living organisms of variable body temperatures has been obtained, which in principle allows to study the living systems such as not homoeothermic animals, plants, trees, and forests.

Appendix A

In the case of the operation of biological dissipative systems working under variable body temperatures, the physical treatment of the problem mentioned in Section 1 has two possible approaches.

A). In the case where the body temperature changes are very small and rather correspond to small oscillations around an average value, it is immediate that the average temperature, $\overline{T}_{\text{lin}}$, that we must take into account in the analysis of the problem is as follows:

$$\overline{T}_{\text{lin}} = \frac{\sum_i^f T_i l_i}{\sum_i^f l_i} \tag{A.1}$$

Where T_i is for the temperature corresponding to the quantity of cycles denoted by l_i, during which the operation temperature is different; $l_i \ll N_{\max}$, and $N_{\max} = \sum_i^f l_i$ is the total number of cycles performed by the dissipative system during its continuous operation, and f s the number of time segments with different temperatures.

B). If the temperature variations are large, then Eq. (5) appears as follows,

$$\int_{t_i}^{t_f} \frac{ds}{dt} * dt = \frac{K_s}{\overline{\overline{T}}} \tag{A.2}$$

Where $\overline{T}$ is the average temperature, which will be defined by the following analysis. Considering Eqs. (1), (2) and the definition of the Rayleigh dissipation function [34] as, $\Phi_R = T\dot{S}$, Eq. (3) which in general, represents the production of heat inside a system and it can be measured experimentally; we have that under those conditions, we can define $\dot{S}$ as before by Eqs. (2), (3), and then also the specific entropy production rate is given by Eq. (4), therefore Eq. (5) becomes,

$$\int_{t_i}^{t_f} \frac{ds}{dt} * dt \cong \sum_{t_i}^{t_f} \frac{1}{T_j} \frac{\Delta s_j}{\Delta t_j} * \Delta t_j \tag{A.3}$$

On the following the rate of entropy production, (d_iS/dt) will be denoted by $\dot{S}$.

In other words,

$$\Delta S_{fi} = \frac{K_s}{\overline{\overline{T}}} \tag{A.4}$$

From Eqs. (A.3) and (A.4) it is possible to write,

$$\frac{1}{\overline{\overline{T}}} = \frac{\sum_{t_i}^{t_f} \frac{1}{T_i} * \Delta s_j}{\sum_{t_i}^{t_f} \Delta s_j} \tag{A.5}$$

If we want to write Eq. (A.5) similarly to Eq. (6), then we obtain the following result,

$$\dot{S}_{\text{Aver,fi}} * (t_f - t_i) = \frac{K_s}{\overline{\overline{T}}} \tag{A.6}$$

With $\dot{S}_{\text{Aver,fi}} \equiv (\Delta S_{fi}/(t_f - t_i))$, which means that $\dot{S}_{\text{Aver,fi}} = (\sum_{t_i}^{t_f} \Delta S_j/(t_f - t_i)$, and as $(t_f - t_i) = N_{\max} * \tau$; then $\Delta S_j = S_j * n_j * \tau$. And therefore, $\dot{S}_{\text{Aver,fi}}$ is given by

$$\dot{S}_{\text{Aver,fi}} = \frac{\sum_{t_i}^{t_f} \dot{S}_j * n_j}{\left(N_{\max} = \sum_i^f n_j\right)} \tag{A.7}$$

Acknowledgments

We dedicate this work to the memory of our fellow colleague Germinal Cocho, who was not only a very intrepid pioneer in the analysis of complex systems, but also a model to follow for his great professionalism and social commitment to the basic problems of the great majority of humanity.

We are grateful with Edgar Muñoz-Sánchez and M. del Socorro Ponce-Orozco for their kind support in preparing and correcting the writing, respectively. We thank the referee for their helpful comments and interesting suggestions.

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