Sex and Aging: The Theory of the 'Prized Son'

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"*Everything becomes clear after the mystery is unraveled.*"(unknow authr)

Summary: After highlighting where some of the most well-known theories about aging and death fall short, we will propose a new theory that explains sexual reproduction and aging. In this theory, both sexual reproduction and senescence emerge as Darwinian adaptations. A mechanism that circumvents group selection is also suggested. We will then develop the "Equation of Death," which establishes species longevity as a function of parameters related to their prey and predators.

Keywords: Aging, Sex, Death, Evolution, Senescence, Adaptation, Selective Pressure, Reproduction, Sexual, Asexual, Theory of Aging, Longevity, Biological Clock, Programmed Death, Equation of Death.

1- Definitions

In this text, we will use the term "aging" as a synonym for "senescence." Senescence is defined as a gradual accumulation of degenerative changes in the organism that inexorably lead to death. Alternatively, it can be described as "the progressive deterioration of almost all functions of the organism over time." [1]

We will also use the term "immortal" to refer to organisms that do not die from aging. This does not mean they cannot die due to lack of food, predator attacks, accidents, diseases, hostile environments, or other external causes. It simply means they do not undergo senescence, meaning they do not have a programmed death in their DNA, nor do their vital functions significantly decline over time, leading the organism to death. Bacteria, for example, are considered immortal in this sense, as they do not age.

Similarly, we will use the term "mortal" to describe organisms that undergo aging, meaning they have instructions in their DNA to die after a certain period of time or experience a significant decline in vital functions over time, inevitably leading to death. Mammals, for instance, always age and die.

2- Introduction

The evolutionary cause of aging is still regarded as one of the great mysteries in science, particularly within the field of biology. Numerous theories have attempted to explain it: "*The Russian gerontologist Zhores Medvedev cataloged over 300 of them. However, a large number of these theories are not truly concerned with the causes but rather with the mechanics of aging*." [1]

Despite the substantial number of theories, only a handful of them have gained any acceptance within the scientific community. Unfortunately, none of them have provided a satisfactory explanation for the Darwinian causes of aging. The theory we will present, which I've named the "***Prized Son Theory***," aims to address this issue by explaining the cause of senescence at the neo-Darwinian level, i.e., through genetic adaptation via natural selection. In this new theory, we will argue that aging is a result

of "programmed death," as it would be evolutionarily advantageous for genes in sexually reproducing organisms to eliminate the bodies that carry them.

To comprehend the evolutionary process underlying aging, we must start at the beginning: the origin of life.

3- The Beginning

Modern theories about the origin of life [2] indicate that it began around four billion years ago, originating from a replicating molecule. According to these modern theories, this replicating entity should have been something akin to a proto-RNA, formed by chance in the primitive environment of that time, known as the "primordial soup."

The early replicators created copies of themselves—clones—using the molecules present in this "primordial soup." Since the copies were not always perfect, mutations occurred, causing some copies to have better or worse copying abilities than their parent molecules. Those that were more successful in surviving and reproducing left behind more copies of themselves. The necessary conditions for Darwinian evolution were present: Reproduction, Variability, and Natural Selection.

The struggle for replication continued relentlessly. At some point, a mutant replicator must have emerged that developed a protective layer against attacks from other replicators—the first cell. This cellular replicator succeeded so well with its protective layer that it virtually dominated early life. The primordial soup likely ended up with only cellular replicators—such as bacteria [3]. Later on, some mutant bacteria "realized" that if they grouped together in colonies, they would have a better chance of survival. These colonies would evolve into the first multicellular organisms.

4- Bacterias

Bacteria are immortal. They reproduce through fission: a bacterium divides into two (two identical clones), and each of these clones divides into two, and so on, growing exponentially over time if there are no environmental constraints.

The crucial point to grasp is that ***life began as immortal***. There was no internal mechanism for aging. Thus, the simplest characteristic for existence is immortality.

5- Causes and Mechanisms

It's important to differentiate between evolutionary causes and the physical causes that lead to aging (internal mechanisms of senescence). Evolutionary causes always lead to some internal mechanism (physical causes) that trigger the aging process. For instance, the feeling of fear might cause trembling, sweating, and shivering. We can attribute this to hormones like adrenaline and cortisol that prepare the body for fight or flight. However, this would be more of a physical cause of the fear process rather than its evolutionary cause. The evolutionary cause would be an adaptive genetic perception of danger: Organisms with genes enabling them to perceive danger were more likely to survive than those lacking such genes. Thus, genes inducing organisms to perceive and react to danger had greater evolutionary success than those without them. In short, the

evolutionary (Darwinian) cause of fear would be the detection of danger, and the physical causes would involve the release of specific hormones to prepare the body for action.

6- Hayflick Limit

Presently, the "Hayflick Limit" [4] is considered the most significant physical cause of aging—the so-called "biological clock." In 1961, Dr. Leonard Hayflick discovered that there is a maximum number of cell divisions—around 50—that each somatic cell can undergo in the human species. Beyond this limit, the cell stops dividing and dies.

The internal mechanism responsible for this limitation is based on the telomeres of chromosomes. In species with linear chromosomes, like humans, each chromosome has a termination at both ends known as a telomere. With each cell division, these telomeres become shorter. This means that the chromosomes in daughter cells have shorter telomeres than those in the parent cells, and consequently, they have a shorter lifespan as well. Chromosomes without telomeres lose their function, leading to cell death [5].

7- Germ Cells and Telomerase

Not all cells in the body are subject to the Hayflick limit. Germ cells, the gametes (sperm and egg cells), do not experience telomere shortening. This is because these cells produce an enzyme—telomerase—that prevents telomere reduction [6]. Somatic cells also produce this enzyme, but at insufficient levels to completely repair the telomeres. In gametes, the production of telomerase is higher, and as a result, they do not age. Germ cells are, therefore, considered immortal. Individuals with a deficiency in telomerase production may experience premature aging, as observed in the condition known as *progeria* [28]. This disease provides strong evidence of the role of telomeres in the aging process:

"*Telomere maintenance is implicated in chromosome stabilization and cellular immortalization. Telomerase, which catalyzes de novo telomere synthesis, is activated in germ cells and many cancers.*" [7]

8- A Good Theory of Aging

A good theory of aging should provide, if they exist, the evolutionary causes or selective pressures that favored the emergence of aging. It should also address the following questions:

a) Why do some species age rapidly while others do not, or age very slowly?

b) Why does aging predominantly occur in sexually reproducing organisms, while asexual organisms hardly age? (Multicellular asexual organisms like anemones and jellyfish, for instance, do not seem to age) [11].

c) Why do somatic cells not produce more telomerase, like germ cells do, in order to avoid aging as well?

9- Main Theories of Aging

Theories of aging solely based on internal mechanisms, disregarding evolutionary influences, are at best incomplete. These theories, aside from failing to explain the enormous differences in the aging timelines among different species, do not offer reasons for why the organism itself doesn't regenerate: If bacteria are single cells and can live indefinitely without aging, why can't the somatic cells within a multicellular organism do the same? [9]

Before delving into the core of the new theory, it's advisable to present some of the main theories about aging and demonstrate why they fall short in addressing the issue of explaining the evolutionary causes of aging. It's worth noting that theories exclusively based on internal mechanisms are far from providing a Darwinian-level explanation, as the evidence suggests a genetic influence in the process, indicating that such genes were subject to natural selection.

9.1- Theory of "Free Radicals"

This theory, proposed in 1954 [8] [6], suggests that aging occurs due to an excess of free radicals—ionized molecules, usually oxygen compounds—produced and released in the body as byproducts of cellular metabolism (mitochondria).

According to this theory, free radicals are responsible for aging, as they lead to cell degeneration and ultimately death. While it's true that cell degeneration can accelerate aging, this theory fails to explain why cells killed by free radicals couldn't be replaced by non-degenerated ones, as normally occurs with dead somatic cells. This theory should imply that animals with higher metabolic rates age more rapidly, as they would produce more free radicals. However, many animals defy this rule [1]. It would also suggest that athletes age much faster than sedentary individuals, which isn't always true either. Therefore, although free radicals might harm cells and contribute to aging, they fall short as an all-encompassing theory of the aging process.

9.2- "Good of the Species" Theory (Weismann)

August Weismann (1834-1914) [10] proposed in 1882 that aging results from "programmed death"—a mechanism encoded in DNA that leads cells to die—and that it evolved through natural selection to benefit the good of the species, even if this had a negative effect on the organism's fitness (survival and reproduction capacity). Weismann believed that by removing older members from the population, more resources would be available for the younger individuals, who presumably would be better adapted to the environment than their parents, thus favoring the species' evolution as a whole[10].

This theory, also known as the "Weismann theory" [1], has an unresolved flaw: It relies on "group selection," which, as we'll see, should not be used unless well-founded.

To understand why group selection, in this case "death for the good of the species," is problematic, let's consider a population of the same species composed of mortal and immortal organisms, initially in equal numbers and equilibrium. In this scenario, if one organism dies, it can be replaced by either a mortal or an immortal offspring. The

probability of mortals dying is higher since they age and die. The probability of replacement by an offspring of an immortal is also higher because there could be several older mortals struggling to reproduce. Therefore, seemingly, the population would gradually become immortal, even if this was harmful to the species as a whole.

Now, suppose there's a population entirely composed of mortal organisms. Let's say an immortal mutant organism is born—one that doesn't age. With higher fitness, this organism could continue reproducing and having offspring when other organisms of its age group are already dead due to aging. In other words, this immortal organism would seemingly have a better chance of having its offspring replace deceased organisms than the mortal ones. Consequently, without a mechanism to counteract this logic, over time, the population would tend to become entirely immortal, even if it's harmful to the entire population as a whole. The organism's fitness, in this case, would outweigh the species' benefit.

Thus, without any mechanism explaining how group selection would favor mortals over immortals, "group selection" as used in this theory appears to contradict Darwinian mechanisms of fitness. For this reason, this theory also did not gain traction.

9.3- "Accumulated Damage" Theory (P. Medawar and J. Haldane)

Sir Peter Medawar (1915-1987), a Nobel laureate in medicine, was a professor of zoology and anatomy at the University of London [10]. In 1952, Medawar and J. Haldane wrote an article proposing a theory to explain aging through the accumulation of damage in the genome. This accumulation of damage would be possible if such damage only appeared later in the organism's life [11], allowing these genes to experience low selective pressure. For instance, if a severe genetic disease caused by a mutation in a particular gene appeared during puberty, before sexual maturity, this gene would be strongly selected against because the organism wouldn't have time to reach sexual maturity and reproduce. Thus, if harmful mutations express early, they are less likely to pass to the next generation, making them rarer. The opposite is also true: The later a harmful gene expresses, the greater the chance it remains in the population since the organism could have many offspring before the gene eventually expresses itself and kills the organism. Thus, harmful mutations expressing late could slowly accumulate in the population's genome, and according to Medawar, this accumulation would be responsible for aging [10] [11].

This theory has several positives: It explains aging from a genetic standpoint, utilizes Darwinian theory to explain the model, and empirical data partially supports it.

Despite these positives, the theory has significant drawbacks: Organisms started as immortal, not mortal. Therefore, any gene that reduces the organism's fitness should be negatively selected, even if it appears later. For example, consider an initially immortal species and imagine a mutant organism with a gene that kills it at age 50. This organism cannot have more offspring because it's dead. This wouldn't be the case for other organisms of the species, and therefore, their competitors would leave more descendants. There's no reason for this gene to spread and bring about mortality and aging. This is the same argument refuting the Weissman hypothesis (9.2). Additionally, this theory doesn't explain why some species don't age while others do. It also doesn't correlate sexual reproduction with aging, as all evidence seems to indicate.

9.4- "Antagonistic Pleiotropy" Theory (G. Williams)

In 1957, George Williams, a professor at the University of Michigan, formulated a theory where senescence could be explained by a phenomenon called "antagonistic pleiotropy." Pleiotropy refers to a gene influencing multiple distinct traits in an organism. The essence of this theory is that some alleles can benefit the organism with respect to a certain trait in its youth—such as enhanced vision—while simultaneously harming it in another trait later in life, like causing cataracts [10]. Consequently, the gene would be beneficial (more so than the normal allele) at the start of the organism's sexual life, allowing it to have high fitness during youth and produce more offspring than organisms without this mutation. However, after a certain time, this gene would act negatively on another trait, harming the organism. Nevertheless, the gene would have already been passed on to new generations, as it was advantageous to the organism during the early reproductive phase.

While this theory is logical and seemingly consistent, it still has some deficiencies: It doesn't explain why this effect doesn't occur in asexual species. It doesn't answer why very similar species (like some bird and fish species) with similar genes have vastly different lifespans [10]. It doesn't clarify why the organism couldn't maintain the same level of gene activity that benefited it during its youth and high-fitness phase, suddenly altering it and decreasing its adaptability. Most importantly, the theory doesn't demonstrate why immortal organisms, which didn't inherit these genes and thus wouldn't suffer these symptoms in adulthood, couldn't compensate for their weaker youth performance with greater vitality in their infinite adult phase.

9.5- "Disposable Soma" Theory (T. Kirkwood)

In 1977, Thomas Kirkwood, then a statistician, published an article titled "Disposable Soma." "Soma" refers to the part of the body composed of somatic cells, meaning non-germ cells. According to Kirkwood, as organisms experience high mortality due to external factors (predators, diseases, accidents, starvation, etc.), it wouldn't be productive to keep the organism alive beyond its expected lifespan [13]. Thus, energy should be used to enhance reproductive capacity rather than maintaining an organism indefinitely. In other words, the organism could possess an internal DNA repair mechanism to keep it alive, but this would consume energy that could be better used for reproduction. Since organisms tend to die due to external causes, it wouldn't be worth the cost of keeping them alive beyond necessity [10].

One problem with this theory is that it doesn't quantify how much energy is needed to repair cellular damage compared to the energy expended on reproduction to conclude that the expenditure would be unfeasible. Additionally, organisms at the beginning of life are much more likely to die than experienced adults. This is without accounting for the time and energy needed to reach puberty for the commencement of reproductive life. Thus, it seems contradictory to discard an experienced adult in its reproductive years to replace it with younger, inexperienced individuals who will need time and energy before starting their reproductive lives. Even if an adult is more energy-costly, if it possesses high fitness due to its experience and immortality, it could spread its genes much more effectively, even if the DNA repair mechanism requires more energy.

9.6- Theories of "Evolvability"

In his article "The Evolution of Aging" [10], Theodore C. Goldsmith, a NASA engineer, provides a comprehensive explanation of the main aging theories and discusses several scientists and scholars (e.g., J. Mitteldorf, J. Travis, J. Bowles) who support the so-called "Evolvability Theories."

These theories are built upon Weismann's "*Good of the Species*" theory, where "good" is defined as an increased rate of the species' evolution. Therefore, it would be beneficial for the species if its members aged and died, as aging would allow new generations, theoretically more adapted and evolved, to replace the older ones at a faster rate than an immortal population—faster than a non-aging species. Consequently, aging organisms would increase the "*evolution rate*" of the aging species as a whole, benefiting the group.

This is indeed advantageous for the species. However, the problem with group-benefiting theories at the expense of the individual (group selection) is that, as seen in section 9.2, they often lack a "neo-Darwinian" mechanism (gene-based fitness) or a Darwinian mechanism (organism-based fitness) that can resolve the paradox of "group selection." According to Darwin's theory, better-adapted organisms with higher fitness are more likely to survive and leave more descendants than less-adapted ones. Therefore, a characteristic that would be disadvantageous to an individual—reducing its fitness—even if beneficial to the group as a whole, shouldn't spread across the species. In essence, when group selection comes at the cost of individual organisms, a mechanism explaining the paradox of fitness loss is necessary for it to be valid. Unfortunately, this is not the case for the "Evolvability Theories" outlined by Goldsmith.

9.7- "Sexual Cause" Theory (W. Clark)

In his book "Sex and the Origins of Death," William R. Clark, a professor in the Department of Molecular, Cell, and Developmental Biology at the University of California, further refines the "Disposable Soma" theory (9.5) from a neo-Darwinian perspective—based on genes [14].

In this reinterpretation of the "Disposable Soma" theory, Clark explains that aging began early in Earth's history with our earliest ancestors, called *protozoa*—single-celled organisms with a nucleus containing linear DNA with telomeres protecting the ends. Clark doesn't explain why it was advantageous for protozoa to change their circular chromosome to linear ones. Nonetheless, gene incorporation from parasitic bacteria into protozoa occurred, leading to a symbiotic relationship, as seen with mitochondria. This enabled protozoa to grow and develop specialized structures like protective shells (cytoskeletons) and feeding structures (microtubules) [17], or even the ability to form colonies, some of which later evolved into multicellular organisms.

With the advent of sexual reproduction, which benefits genes and the species in numerous ways (as we'll see in the next topic), protozoa experience the segregation of DNA into distinct nuclei for the first time. The micronucleus contains germinal DNA

used solely during reproduction, while the macronucleus contains somatic DNA used for daily cell maintenance.

According to Clark, somatic DNA experiences more degradation than germinal DNA, and since the latter is passed to the next generation, there's no need to repair somatic DNA, which can accumulate harmful mutations and should therefore be destroyed. During sexual reproduction, the following process occurs:

"*...and then the old macronucleus, isolated at one end of the cell, begins to degenerate and dies... What do ciliate protozoa have to do with humans?... A lot, because it's only by looking at sexual reproduction in protozoa like the paramecium that we can see the generation of DNA that is not passed on to the next generation. This segregation of DNA into two compartments does not occur in bacteria or other organisms that reproduce asexually. And what happens to the excess DNA that is not used in reproduction? It's destroyed. In fact, it can be argued that it's in the programmed death of the macronuclei of primitive eukaryotes like paramecia that our own bodily death is foreshadowed.*" [18]

Clark explains that the programmed death of macronuclei (corresponding to somatic cells in the body) is necessary because they are probably heavily damaged and no longer required after reproduction.

However, this conclusion contains two logical errors: First, there's no need to program cell death when it will happen naturally due to the accumulation of mutations or cell wear and tear. It would be like an engineer designing an elaborate and expensive bomb on a Martian rover to explode when the rover's battery depletes and it becomes inoperative. If the rover will become inoperative on its own, it's illogical to spend time and resources on a device that would cause it to explode after it's no longer useful. Similarly, Clark doesn't demonstrate the evolutionary necessity or any natural cause for programming the death of somatic cells when they are destined to lead the organism to death anyway. Second, he doesn't present reasons that would render somatic DNA repair impossible, as it could indeed be achieved since, like bacteria, somatic cells also divide by fission. If bacteria and germ cells are immortal, then, in theory, protozoa could be as well. If bacteria can reproduce indefinitely, protozoa, in principle, could do the same.

10 - The "Awarded Offspring" Theory (Jocax)

In order to understand the "Awarded Offspring" theory, let's first comprehend the advantages (+) and disadvantages (-) of Sexual and Asexual Reproduction. Table (I) below summarizes the main differences:

Reprodução Sexuada	Reprodução Assexuada
(+) Greater Genetic Variability. (there's mixing of genes from parents)	(-) Lesser Genetic Variability. (the offspring are clones)
(+) Eliminates harmful mutations from the species more easily. (Organisms inheriting double harmful mutations tend to be eliminated more rapidly).	(-) Doesn't eliminate harmful mutations easily.
(+) Spreads beneficial mutations more rapidly through males. (A single male with high fitness and good adaptation can inseminate multiple females.)	(-) Não espalha mutações benéficas a todos. (Cada célula gera sua própria linhagem).
(+) Seleção sexual favorece encontro de características adaptativas e promove a extinção das menos adaptativas. (As fêmeas escolhem os 'melhores' machos)	(-) There is no sexual selection.
(-) Greater difficulty in reproduction, as there is a need to find a partner for it. (A sexual partner is not always available)	(+) Easier reproduction, as there is no need to seek sexual partners.
(-) Greater energy expenditure for reproduction. (The energy required to produce a male is substantial, and their role is solely to transport gametes for females to generate new organisms).	(+) Lower energy expenditure for reproduction.
(-) Each offspring carries only half of the chromosomes from a parent. (Meiosis segregates parental chromosomes and combines them in offspring with half from each parent).	(+) Each offspring carries all the chromosomes from the parent. A 100% transmission efficiency.
(*+*) A child can inherit two or more beneficial mutations from each of its parents. pais e tornar-se um 'super-organismo'. (O filho pode ser premiado com duas ou mais mutações benéficas de cada um de seus pais).	(-) A double beneficial mutation depends directly on the quantity of offspring and the time it takes for this to happen. (If a beneficial mutation is rare, a double beneficial mutation is even rarer).

Table I - Advantages and Disadvantages of Sexual/ Asexual Reproduction

Let's also summarize the advantages and disadvantages between mortal organisms (which age and die) and Immortals (which die for reasons other than aging). Comparisons need to be made within a population in relative equilibrium, that is, of relatively constant size, stable over time. In this table II, mortality is not related to the type of reproduction.

Mortal Organisms (Age)	Immortal Organisms (Do not age)
(+) Higher beneficial mutation rate. (A higher death rate allows more births to occur. Each new birth can carry a new mutation)	(-) Lower beneficial mutation rate. (A lower mortality rate prevents new births from surviving.)
(-) Higher rate of harmful mutations. (idem)	(+) Lower rate of harmful mutations.
(*+*) Highest evolutionary rate of the species. (The population set is replaced more quickly by new generations.)	(-) Lowest evolutionary rate of the species. (Old organisms tend to stay alive and consume resources that could serve new ones.)

Table II - Advantages and disadvantages of immortality

10.1- Introduction

The theory of the "Favored Offspring" is based on two aging theories: the theory of the "Good of the Species" (in Item 9.2) and the theory of "Evolvability" (in Item 9.6). However, unlike these theories, instead of using orthodox Darwinism, where selection acts on the individual organism, we will employ neo-Darwinism, where natural selection operates on genes, and from there, we will break through the barrier of group selection.

In most cases, there are no conflicts between orthodox Darwinism, centered on the "fitness" of the individual organism, and neo-Darwinism, based on genes. Generally, what is advantageous for the individual organism is also advantageous for the genes that compose it, and vice versa. Thus, the selective pressures that act on the individual organism also apply to its genes. For example, if an organism exhibits high adaptability ("fitness" = the ability to survive and reproduce) to its environment, it is expected that its genes will increase in frequency in the species' genetic pool in the next generation. However, the organism and its genes are not always in complete agreement. There are cases, as we will see below, where the survival of the organism conflicts with the survival of its genes.

10.2- Examples of conflicts

To illustrate some conflicts between the individual organism and its genes, let's consider some hypothetical cases:

*10.2.1- Conflicts favoring the individual at the expense of genes:***

1-*The Infanticide*: A mutant organism that tends to feed on its own offspring. It might survive and reproduce more than other organisms without this mutation, but its genes wouldn't benefit. This behavior is very rare because by feeding on its own offspring, it would likely decrease the frequency of its genes in the gene pool. Of course, this practice could be beneficial to the genes and the organism if it is in a situation of extreme lack of food, which would otherwise lead to the offspring's demise. In this particular case, the survival of the organism even if it practices infanticide would be advantageous to the genes.

2-*The Cannibal*: An organism that habitually hunts and consumes both members of its own species and those of other species indiscriminately. It might survive and reproduce more efficiently than organisms that feed exclusively on different species. However, this behavior tends to harm its genes because it would be destroying its own genes unnecessarily.

3-*The Coward Mother*: A mother that lacks "maternal instinct" and doesn't risk her physical integrity for her offspring, even if the probability of harm is very low. In this case, her genes would also decrease in the population's gene pool compared to those mothers who protect their offspring, even though this behavior would be favorable for her own survival and reproduction.

10.2.2- *Conflicts favoring genes at the expense of the organism*

1-*The Altruistic Mother*: A mother who risks her life to defend her offspring. This occurs in nature when the mother instinctively believes that risking her life could ensure the survival of her offspring. This instinct can favor her genes even if the individual risk to her life is high, as long as the probability of saving the offspring is equally high.

2-*Sexual Suicide*: In some species like the praying mantis and certain spiders, males allow themselves to be devoured by females in exchange for successful copulation. Fertilization can result in hundreds of offspring, thus being advantageous for the genes, even at the cost of the male's life.

These examples serve to understand the contrast between orthodox Darwinism – centered on the individual organism – and neo-Darwinism, centered on genes. The examples favoring genes at the expense of the individual organism are real and occur frequently in nature, while the examples harming genes are not common. Neo-Darwinism is currently the most accepted view in biology, and the examples above are easily explained by considering the gene, rather than the individual organism (or even the species), as the central unit of evolutionary dynamics.

10.3- “Gene fitness”

We could then introduce the concept of "gene fitness" (or "fitness" of a subgroup of genes), which refers to the adaptability or degree of suitability of the gene(s), as opposed to the "fitness" of the organism.

"Gene fitness" can be defined as the capacity that a gene confers to the phenotype to increase its own frequency in the population's gene pool.

In the first three cases from the previous example (10.2.1), we can see that there would be a reduction in the "gene fitness" or the fitness of the subgroup of genes that induce the organism to attack or harm organisms that share a significant amount of genes with it. On the other hand, there would be an increase in gene fitness in cases where genes benefit, even at the risk of the individual organism's survival (Examples 10.2.2). Therefore, we can conclude that actions or predispositions that harm "gene fitness," if they exist, must be very rare, and for this reason, the examples above (10.2.1) do not occur in nature. Conversely, the opposite should be true: actions or predispositions that increase "gene fitness," even if they harm the individual organism, should be more readily found in nature.

10.4- "Parental Altruism"

The term "Parental Altruism" refers to the set of genetic predispositions that lead an individual organism to assist others of its species, even if it may harm itself. The degree of relatedness tends to influence the degree of altruism displayed towards others. The most familiar example is that of a mother risking her physical integrity or even her life to protect her offspring from a dangerous predator.

Such behavior can benefit her genes, which are present in her offspring, even if it may harm her as an individual organism. The evolutionary benefit conferred to genes over the individual organism justifies parental altruism.

10.5- "Group Selection"

"Group selection" can be defined as actions, practices, instinctive predispositions, or behavioral phenotypic traits that benefit the group as a whole (population or species) at the expense of the individual organism. In terms of classical Darwinism, centered on the individual organism, this concept is almost impossible to accept because it contradicts the principles of classical natural selection, which grants greater adaptability to organisms with higher "fitness."

For example, a lion that, instead of consuming its prey, shares the meat with others in the pride would be benefiting the group, but it might suffer greatly and have limited chances of survival and reproduction, unless all lions adopted the same behavior! Otherwise, such a practice would be detrimental to it.

However, this altruistic behavior could thrive if the pride he shared the food with consisted of his **own family**, where there is a high degree of genetic sharing. Otherwise, this practice wouldn't be explainable either.

Thus, group selection cannot be considered a valid neo-Darwinian explanation unless there is a mechanism that proves the benefit to the organism's gene that promotes group benefits at the expense of the individual organism.

10.6- A Reprodução Sexuada

Sexual reproduction can be considered a form of group selection, as it tends to harm the individual organism while conferring benefits to the group as a whole (Table-I).

But why would sexual selection "harm" the individual?

For several reasons: The organism needs to search for, and might not even find, partners to reproduce with. In asexual reproduction, this doesn't happen. The organism needs to expend more energy in the search and might not find a mate for reproduction; in asexual reproduction, this isn't necessary.

Genes create males, who don't become pregnant! Their sole biological function would be to carry germinal material to females: a waste of energy. Furthermore, the organism only transfers half of its chromosomes to each offspring; in asexual reproduction, it transfers 100% of the genes, twice as much.

So, why does sexual reproduction exist? What mechanism would offset the individual "harm"?

William Donald Hamilton, a biologist and member of the Royal Society of London, proposed a theory known as the "*Red Queen Theory*." In this theory, Hamilton aims to explain the necessity of sexual reproduction as a way for multicellular organisms to defend themselves against bacterial infections [19], [22]. Since the growth rate of bacteria, and therefore also mutations, is faster than that of multicellular animals, the latter would need to acquire variability more rapidly to protect themselves from these bacterial mutations. One way to achieve this would be through sexual reproduction, where genetic variability could counterbalance the rapid bacterial mutations.

However, we know that the vast majority of mutations are either harmless or harmful. Beneficial mutations are much rarer. Thus, the mixing of genes through sexual reproduction should, therefore, produce more organisms that are less adapted rather than more adapted, more organisms with lower resistance to bacteria than with higher resistance. Furthermore, similar to Weissman's theory of the "Good of the Species," Hamilton's theory does not provide a mechanism that explains how this group selection could occur in terms of gene benefit: how, for example, a "sexuality gene" could fare better than an "asexuality gene."

To address these questions and those related to aging, as we will see, let's move on to our next topic.

10.7 - The Theory of the Gifted Offspring

One answer, which could be the key to these questions, is what I refer to as the "*Theory of the Gifted Offspring*": Sexual reproduction allows the merging of two or more beneficial mutations from distinct organisms into a single organism, producing a high "*fitness*" "*super-organism*" without the need to wait for an extensive period, as is the case in asexual reproduction.

10.7.1 - In Sexual Reproduction

Asexual reproduction does not allow for the occurrence of a double beneficial mutation in the same organism without considerable luck! Let's consider, for example, how difficult it is for bacteria to survive two types of antibiotics administered simultaneously due to lacking sexual reproduction:

"*...Compared to other bacteria, H. pylori is a highly mutable microorganism. Its mutation frequency depends on the marker considered and varies greatly among different bacterial lineages. For instance, concerning rifampicin, extremely high mutation rates were found in some strains, 3x10-5, while in others, much lower rates, 4x10-8, were observed. Regarding erythromycin, the mutation frequency is lower, ranging from 1x10-7 to 5x10-9. The higher the bacterial population at the site of infection, the greater the chance of resistance mutations occurring, eventually being selectable during antibiotic therapy. Considering the average mutation rates from the above example, to find bacteria resistant to two drugs, a population density around 1x1014 would be required, which is impossible. This highlights the importance of antibiotic combinations...*" [20]

If these bacteria possessed sexual reproduction, a bacterium resistant to the first antibiotic could mate with another one resistant to the second antibiotic, producing a super-bacterium resistant to both antibiotics, which would then proliferate.

The idea behind the "Theory of the Gifted Offspring" is that it matters less to genes the number of survivors in the next generation than their long-term survival capability. It should be worthwhile for genes to sacrifice the ease of asexual reproduction if it results in a higher ability to perpetuate genes. G. Miller encapsulates this perspective well in his book "The Mating Mind," referring to sexuality as a means to discard mutations, particularly harmful ones:

"*...To prevent mutations from accumulating over time, sexual reproduction takes some risks. ... Most offspring will inherit nearly the same number of mutations from their parents. However, some may be lucky: They might inherit below-average numbers of mutations from both parents and have far better genes than average. They should survive and reproduce very well. Their mutation-free genes will spread through future generations. Other offspring may be unlucky: They might inherit an above-average load of mutations from both parents and might not develop at all or might die in childhood. When they die, they take a large number of mutations with them into evolutionary oblivion. This effect is highly significant. By endowing the next generation with uneven numbers of mutations, sexual reproduction ensures that at least some of the offspring will have very good genes... **As long-term evolution is a competition in which the***

winner takes all, it's more important to produce a few offspring that have a good chance of doing well than a larger number of mediocre offspring..." [21]

My critique about sexuality existing to eliminate harmful mutations also applies here: Harmful mutations are naturally eliminated. Sexuality is unnecessary for this purpose. Just as there are bacteria without harmful mutations, there can also be offspring from sexual reproduction organisms without them. Just as harmful mutations affect the bacteria that carry them, they can also affect sexually reproducing organisms, reducing their fitness and impeding the survival of the mutant gene in the long term.

The role of males in sexual reproduction would be to enable the dissemination of beneficial mutations throughout the population at a much higher rate than in asexual reproduction.

What mechanism would enable sexual reproduction?

To answer this, let's assume that within the same species, there's an allele, a gene, that induces sexual reproduction, for example, by releasing gametes into the aquatic environment, which would join to form new organisms. There's also the asexual allele that induces asexual reproduction. We then have two alleles (sexual and asexual) competing within the same species for survival. Mutations occur in both subgroups. A mutant asexual offspring has the same chances of receiving a mutation as a sexual offspring. Whether the mutation is good or bad will benefit or harm both in the same way. But suppose this mutant offspring generates gametes that will meet another mutant gamete with a different beneficial mutation.

We now have a super-organism, a mutant with a double beneficial mutation, an entity with extremely high fitness, causing the sexual allele to have a much higher chance of survival and reproduction than its asexual competitor. This could, in the long term, lead to its fixation within the species.

10.7.2- On Aging

Also, we can use the theory of the "Gifted Offspring" to explain aging. To understand the process, let's consider a hypothetical and quite bizarre scenario: Suppose a government offers a million-dollar prize to the family of someone who commits suicide. However, to discourage the act, the prize wouldn't be given immediately after the suicide, and the family wouldn't know the reason for receiving the prize. Thus, if suicide had a genetic origin, these genes could be present in some other member of the same family (siblings share, on average, 50% of chromosomes), and as a result, the awarded value would ultimately favor all the genes of that family, including, and especially, the genes responsible for the suicide.

What can we deduce from the bizarre story above?

That a phenotypic trait that impairs the organism's fitness, even if that trait might lead to death, could thrive in the population if it confers a sufficiently significant benefit to the group in which this organism shares its genes. We saw this when studying conflicts between the individual organism and its genes in section 10.2.2 above.

In a reasonably stable environment, a species' birth rate should, on average, match its death rate. If this weren't the case, if the birth rate were consistently higher than the death rate, the species would proliferate until there were no natural resources left to sustain it. Conversely, if the death rate were consistently higher than the birth rate, the species would go extinct [10]. Thus, let's analyze aging assuming that species are in a relative state of balance; the species can grow up to the limit of the food resources in its habitat, which is the normal situation:

Birth Rate = Death Rate

Therefore, within this equilibrium condition, if a species were immortal, that is, if there were no programmed death in its DNA (no aging), *the only births that could survive and reach maturity would be those replacing individuals who die from accidental* causes such as fights, accidents, predators, diseases, etc.

However, let's now consider a hypothetical extreme scenario where adult organisms of an ageless species also do not die from other causes (aside from starvation). In this case, all potential births in this species would starve to death before reaching maturity since there would be no food resources for the unborn offspring! Thus, *evolution would not occur*, as there would be no opportunity for mutant organisms, which are the raw material for evolution, to be born.

In this **extreme scenario, the species would be stuck in time**, **unable to evolve** and a species that does not evolve is doomed to extinction because it cannot adapt to environmental changes or competition with other species, especially when competing against disease-causing bacteria that mutate rapidly (Red Queen Theory).

Consequently, *a species where adults do not die will eventually become extinct*. However, as we've seen, accidental deaths can still occur, allowing births and, therefore, possible beneficial mutations to reach maturity and be transmitted to the species' gene pool. Nonetheless, mutations are rare, and beneficial mutations are even rarer. For this reason, the accidental death rate might not be sufficiently high for the required number of beneficial mutations needed for species adaptation to be achieved.

If a mutant death gene (or group of genes) appeared that would kill the organism after it had passed its reproductive phase, giving the organism enough time to pass this gene to the next generation, this "death gene" could benefit from the increase it caused in the death rate.

This presents a case of group selection: The "**aging gene**" would be beneficial to the group by allowing the increase of 'good' mutants and thus the evolutionary rate of the species, benefiting it. However, it would harm the individual organism by killing it, reducing its fitness. For this to be possible, there must be a mechanism that outweighs the loss of fitness in the individual organism and, in return, increases the fitness of the gene. This is what will occur, as we will see:

When the "aging gene" kills the organism (which has already had its offspring), it creates a "vacancy" (releases the space occupied and the food resources used) within the local group, allowing some infant or unborn organism to reach maturity. It's important

to note that the resources this organism occupied are generally geographically closer to organisms sharing its genes, like its children and relatives.

Furthermore, it creates the possibility that the organism now able to mature is a "super-organism," meaning that with the vacancy created by this death, there's a chance that the new organism inherits two or more beneficial mutations from its parents, **assuming this species reproduces sexually!**

In other words, we have a case analogous to the suicide scenario in our previous bizarre example, where the family received a million dollars if any child committed suicide. In our case, the parent dies, and the grand prize goes to whoever is bestowed with a "gifted offspring" possessing two or more beneficial mutations.

Since death occurs within the local group, there's a higher probability that this vacancy will be filled by a relative of the deceased organism rather than a complete stranger. This means that the gene causing death by aging has a higher chance of filling the vacancy, as it's geographically closer to the left resources, than a distant allele that doesn't share many genes.

If the "super-organism" that fills the vacancy and reaches sexual maturity has sufficiently high fitness, it will propagate its genes much more vigorously than a normal organism. **Thus, the "aging gene" only needs to hitch a ride on a "gifted offspring" once to quickly spread through the population, benefiting the group's evolutionary rate and now also its own "aging gene".**

Now let's suppose the opposite occurs: a population consisting of mortal individuals, and a mutant immortal organism (which does not age) is born, also being a "super-organism." In this case, as in the previous scenario, the immortality gene should spread rapidly within the local environment due to the fitness conferred by the double beneficial mutation. In this region where this immortal organism resides, there's a tendency for other immortals to emerge, leading to a low evolutionary rate. This implies that the region of immortals has low adaptability and could quickly be overtaken by more adaptable organisms, specifically the group of mortal organisms. Thus, we can conclude that the group of immortal organisms, due to their lower evolutionary rate, is unstable and will likely be replaced by mortal organisms with higher evolutionary adaptability.

It's interesting to observe that the aging rate (biological clock time), correlated with the species' evolutionary rate, should also experience selective pressure for adjustment. Aging should occur at a pace that, if it kills the organism too early, harms the genes by preventing them from having an adequate number of offspring. If it lets the organism live too long, it prevents others from being born and surviving. There must be, therefore, an optimal level of aging that allows organisms to have an optimal number of offspring while also permitting the species to evolve.

In short:

- In a balanced and relatively stable environment, aging is beneficial to the species because it increases the death rate, thereby raising the species' evolutionary rate: its adaptability grows.

- Aging, as an evolutionary adaptation, should primarily occur in species with sexual reproduction, as it enables the occurrence of "super-organisms" (organisms carrying two or more beneficial mutations) that can spread beneficial mutations much more rapidly and effectively than in asexual beings.

- The "aging gene" only needs to hitch a ride once on a "gifted offspring" to spread throughout the group and benefit both the species and the aging gene(s) itself.

- A subgroup of immortal organisms would have an unstable existence because, by not adapting at the same rate as mortal organisms, it would tend to be replaced by the latter.

- Multicellular species with asexual reproduction would have much fewer benefits from senescence because although aging and death slightly increase the possibility of new beneficial mutations, they wouldn't be spread as vigorously as in sexual reproduction (as the rate of "gifted offspring" is higher in sexual reproduction).

11- The “Equation of Death”

- With this theory, we can now outline the "Equation of Death." This equation would assess the lifespan of sexually reproducing species – their longevity – based on some of their characteristics, as well as those of their prey and predators within a stable environment.

- Let's suppose there is an environment, such as an island, where prey and predators coexist, not necessarily peacefully, as predators feed on prey to survive. A principle applies to all natural environments:

"*If there is an abundant resource that can be utilized by life, and if a 'predator' that benefits from this resource emerges, its population will grow.*"

Sunlight is an abundant resource that covers our environment and can be used by life. Therefore, following our principle, if a 'predator' for this light emerges, such as a plant that uses this sunlight, its population will grow as long as this resource is available.

Now, in this example, we have another abundant resource: plants. In the same way, herbivores will emerge and thrive by using these plants as a food source.

After that, various types of carnivores will appear, creating a pyramid of prey and predators with vegetation at the base, followed by herbivores, and various species of carnivores.

The highest level of this food pyramid would be a predator that feeds on other carnivores but isn't preyed upon by any other organism. This could be the case for eagles or lions, for instance.

It's important to note that the current human environment isn't stable due to rapid population growth, which disrupts the balance. However, terrestrial organisms' genes have adapted, for the most part of the four billion years that life has existed on Earth, during periods of relative equilibrium. Significant transformations generally occur in short time frames.

11.1-Initial Conditions

Let's assume that our environment is relatively stable, meaning it's in dynamic equilibrium: organisms die and are born, but their relative frequency remains constant. In other words, the total number of organisms of any species inhabiting our environment remains constant over time. Of course, the hypothesis of a stable ecosystem doesn't hold on evolutionary timescales. Over millions of years, new species tend to disappear, and new ones emerge. Our timescale is therefore shorter. So, by our initial hypothesis of a balanced environment, we have for all species:

Birth Rate = Death Rate (I)

If the birth rate were higher than the death rate, the population would continuously grow until exhausting the available resources. If the birth rate were lower than the death rate, the population would decrease until extinction. As we've studied previously, we can also assume that all sexually reproducing species have programmed death, meaning they age and die. Let's define some variables relative to any given moment in this environment:

L = Longevity of a particular species X, meaning its average lifespan before dying of old age.
Nt = The total number of organisms of species X.
Nu = Number of organisms of X that have or will reproduce.
Nn = Number of organisms of X that will die without reproducing.

Then:

Nt = Nu + Nn (II)

If we define:

G = Average number of pregnancies per reproducing organism (over a period L). That is, G is the total number of pregnancies during period L, divided by the quantity of reproducing organisms (G = Total Pregnancies / Nu). Thus, over period L, reproducing organisms will generate (G * F) offspring each.

F = Average number of offspring per pregnancy per reproducing organism.

We should also note that within the period L (Longevity = lifespan of species X organism), all organisms of species X from a given moment will be dead (either of old age or by predators) and replaced by their descendants. For example, a newly born organism of species X will be dead after time L, whether due to predators or aging. Thus, in a period L, all organisms from a particular moment will be dead, and the entire population will be replaced by the offspring of reproducing organisms:

$$\mathbf{Nt = Nu * (G * F)} \qquad \text{(III)}$$

11.2- The Evolutionary Rate

Due to the constant turnover of organisms as a result of death, beneficial mutations occur, and species evolve. If a species doesn't evolve, meaning it no longer presents beneficial mutations, it becomes stagnant and can be extincted by predators that continue to evolve or replaced by another species that consumes the same resources it used to consume. Therefore, for equilibrium to be maintained, the rate of evolution of a prey species must be at least equivalent to the rate of evolution of its predators. The reverse is also true: The rate of evolution of a predator must at least keep up with the rate of evolution of its prey (at least the least evolving prey). For example, if the prey evolves by adopting defense mechanisms against the predator, and the predator doesn't evolve to match, it will starve.

However, the rate of evolution of a species is proportional to the rate of beneficial mutations the species undergoes, and the rate of beneficial mutations is proportional to the mutation rate, which in turn is proportional to the birth rate of those who reproduce within that species. Thus, the rate of evolution of the species is proportional to the rate of useful births, meaning the organisms that will reproduce, as organisms that don't reproduce cannot pass on their mutations and won't contribute to the "evolvability" of the species.

So, for any species, it holds:

$$\mathbf{\text{Evolution Rate} = k * \text{Rate of Useful Births}} \qquad \mathbf{(IV)}$$

Where k is the constant that converts the birth rate into the rate of beneficial mutations.

If we define:

Tx = Evolution rate of species X, we can rewrite (IV) in terms of our variables:

$$Tx = k * Nu / L \quad (Va)$$

If we further define:
T(i) = Evolution rate of the i-th predator
N(i) = Rate of useful births (that will reproduce) of the i-th predator
L(i) = Longevity (maximum lifespan) of the i-th predator

Using these variables, formula (IV) can also be rewritten for the predators of species X:

$$\mathbf{\text{Evolution Rate of the i-th Predator} = k * N(i) / L(i)} \qquad \mathbf{(Vb)}$$

11.3- The Base of the Pyramid

To remain in balance, in relation to its predators, the evolutionary rate of a species must be proportional to the sum of the evolutionary rates of its predators. If K(i) (a number that varies from 0% to 100%) denotes the degree of importance of species X in the menu of the ith predator (=predator (i)), we will have:

Evolutionary Rate of X = Sum{ K(i) * Evolutionary Rate of Predator(i) } (VI)

K(i)=Constant that defines the degree of importance of species X for the i-th predator's diet. This is necessary because species X may not be the main menu of the predator(a), but it may be the main diet of the predator(b) so that its evolutionary rate contributes more to the evolutionary rate of X.

Putting (VI) in terms of our variables, and using equations (Va) and (Vb), we have:

Nu/L = Sum{ K(i) * N(i) / L(i) } (VII)

If we now use equation (III), we can isolate the longevity L, and we will have the lifespan per aging as a function of the predator parameters, the size of the population of X and the average number of offspring per organism:

L = (Nt / (G*F)) / Sum{ k(i) * N(i) / L(i) } (VIII)

This formula only works for organisms at the bottom of the food pyramid, as it only considers predators of species X, and not the general case. We can simplify it a bit to do a qualitative analysis.

Let's simplify equation (VIII), assuming that the predators have the same birth rate and longevity, that is, that k(i)*N(i)/L(i) = k1*N1/L1. If we have "M" species of predators of X, where X is its only diet (k(i)=1), and knowing that N1 = Nt1/(G1F1), formula (VIII) will be simplified to:
L = L1*(Nt /N1) / (G*F) / M (IX)

We can infer, in this case, that the longevity L of prey at the base of the food pyramid is:

- inversely proportional to the number of offspring per organism (G*F).
- inversely proportional to the number of predators (M).
- proportional to the longevity of their predators (L1).
- proportional to the size of the population relative to the predators (Nt/N1).

11.4- The Top of the Pyramid

Now we will calculate the formula for the longevity of species X, when X is at the top of the food chain, like, for example, an eagle, or a lion.

There is an asymmetry between being at the base of the pyramid or being at its top, because the menu of the predator at the top, being varied, will not be at the mercy of a single species of prey, whereas from the point of view of the prey, any of its predators could, in principle, exterminate it. That is, for a prey, any of its predators could, if it evolved faster than X, drive it to extinction. Thus, in the case that X is a predator at the top of the pyramid, with several types of prey available, the rapid evolution of one of these prey may not starve the predator, since it could feed on the other prey on its menu.

Predator's Tx X = Sum of the evolutionary rate of its Prey (X)

If Z(i) denotes the weight factor, a number between 0 and 1, which indicates how important the i-th prey is to the menu of predator X. Then we can rewrite equation (X) in terms of these variables:

Tx = Sum{ Z(i) * Tx_da_Presa(i) } (XI)

Using (Va and Vb) and (III) and (XI), we will have the Longevity formula for the top predator of the food chain as a function of the parameters of its prey:

L = (Nt / (G*F)) / Sum{ Z(i)* N(i) / L(i) } (XII)

For the purposes of analysis, let's simplify the formula of the predator, above, and consider that all its prey have the same importance in its food menu and that their populations and life span are the same, that is: N(i) = N1 and L(i)=L1. We will then have for the longevity of the top predator of the pyramid:
L = (Nt/N1) * L1 / (G*F) (XIII)

That is, the longevity of the top predator in the food chain is:

- Inversely proportional to the average number of children (G*F).
- Proportional to the average longevity of its prey (L1).
- Proportional to their relative population (Nt/N1).

11.5- The Equation of Death

that our species X can have multiple prey and multiple predators.

In this case, we are going to calculate the longevity in the middle of the food chain, that is, we are going to consider the general case, in which case the species is "squeezed" between the evolution of its prey and also that of its predators. The evolutionary rate of the species should be the combination of equations (VI) and (XI), where we will have:

Evolutionary Rate of X = Sum{ Evolutionary Rate of Predators and Prey} (XIV)

Using (XI), (VII) and (V), (XIV), and denoting Predators{} as the sum in relation to X's predators and Prey{} as the sum in relation to their preys, we have:

$$Nu/L = Predators\{ K(i)*N(i) / L(i)\}+Preys\{ Z(i)* N(i) / L(i) \} \quad (XV)$$

Isolating the longevity variable L, and we finally have the

"Equation of Death":

$$L=(Nt/(G*F)) / [Predators\{K(i)*N(i)/L(i)\}+Preys\{Z(i)*N(i)/L(i)\}] \quad (XVI)$$

Where:
L = Longevity of species X.
Nt = Population size of species X.
G = Average number of pregnancies per organism of species X.
F = Average number of children per pregnancy.
Predators{} = Sum over predators of species X.
K(i) = Number between 0 and 1 indicating the degree of importance of X in the menu of the ith predator. The sum of K(i) need not be unity.
N(i) = Size of the breeding population of the i-th species. If it is inside Predator{} it will be a predator species of X, if it is inside Prey{} it will be a prey species of X. That is: N(i)=Nt(i)/(F(i)*G (i))
L(i) = Longevity of the i-th species.
Z(i) = Weight factor in weighting. Indicates how important the i-th prey is in feeding X.

11.6- Empirical Verification

We can now compare our equation with experimental data on animal longevity [24]. For a predator, equation (XVI) can be simplified in the form of equation (VIII). In it we verify that the longevity is proportional to the average age of its prey, and proportional to the size of its population in relation to that of its prey:

A Tiger lives, on average, 17 years. And some of their prey are: Pigs 12 years old; Goats 17; Boar 17, Monkey 13. Which seems to go along with the equation.

A Rat lives, on average, 4 years but there are a huge number of predators that feed on it. According to equation (IX) its lifespan is inversely proportional to the number of predators, which are many. Therefore, this would explain its low longevity.

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