CME LIVE OPEN ACCESS

Journal of
Gynecology and Reproductive Health

Review Article ISSN: 2574-2728

The Prized Son Theory: A Neo-Darwinian Explanation for Sex and Aging

João Carlos Holland de Barcellos*

University of São Paulo, Brazil.

Corresponding Author: João Carlos Holland de Barcellos. University of São Paulo, Brazil.

Received: 2025 Aug 01 **Accepted:** 2025 Aug 21 **Published:** 2025 Aug 30

Abstract

After highlighting where some of the most well-known theories about aging and death fall short, we 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.

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

1. Introduction

1.1. The Enduring Mystery of Aging and Sex

This section contextualizes the profound biological mystery surrounding the evolutionary causes of aging and sexual reproduction. It highlights the historical challenges in formulating a universally accepted Darwinian explanation for these phenomena, which, at first glance, seem to contradict the principle of maximizing individual fitness.

1.2. The Paradox of Senescence and Sexual Reproduction

The evolutionary cause of aging, or senescence, remains one of the great mysteries in science, particularly within the field of biology. Despite over 300 theories cataloged by gerontologists like Zhores Medvedev, most of them focus on the physical mechanisms of aging, rather than their Darwinian causes. The absence of a satisfactory explanation for the evolutionary origins of aging is a notable gap in biological understanding. In parallel, sexual reproduction presents its own set of paradoxes. Although it is a widespread mode of reproduction among multicellular organisms, it entails significant costs for the individual. Such costs include the need to find a partner, greater energy expenditure, and the transmission of only half of a parent's genes to the offspring, in contrast to the 100% efficiency of asexual reproduction. The prevalence of sexual reproduction, despite these apparent disadvantages, suggests the existence of substantial evolutionary advantages that outweigh these individual costs.

1.3. Definition of Key Terms

For a clear understanding of the proposed theory, it is fundamental to establish the definitions of the central terms

- **Aging (Senescence):** In the context of this study, the term aging is synonymous with senescence. It is defined as the 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.
- **Immortal Organisms:** Refers to organisms that do not die from aging. This does not imply they are invulnerable to external causes of death, such as lack of food, predator attacks, accidents, diseases, or hostile environments. It simply means they do not undergo senescence, i.e., 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.
- **Mortal Organisms:** Describes organisms that undergo the process of 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.

1.4. The Primordial State Life Began as Immortal

The investigation into the origin of life is crucial for understanding the evolution of aging. Modern theories indicate that life emerged approximately four billion years ago from a replicating molecule, possibly a proto-RNA, formed randomly in the primitive environment known as the primordial soup. These initial replicators created copies of themselves – clones – using the available molecules. The imperfection in the copies resulted in mutations, leading to variations in survival and reproduction capacity. The essential conditions for Darwinian evolution were present: Reproduction, Variability, and Natural Selection. The struggle for replication continued, and at some point, a mutant replicator developed a protective layer against attacks from other replicators, giving rise to the first cell. This cellular replicator, like bacteria, dominated primitive life, exhausting the primordial soup of non-cellular replicators. The crucial point is that life, in its simplest form, began as immortal. There was no internal mechanism for aging. Therefore, immortality is presented as the most fundamental and simple characteristic for existence. From this premise, aging is not an inevitable consequence of biological wear and tear, but rather a characteristic that must have actively evolved. This establishes a fundamental challenge for any theory of aging: to explain why immortality, the default state, gave way to mortality in many complex organisms.

1.5. Introducing the Prized Son Theory, a Neo-Darwinian Perspective

The Prized Son Theory (also referred to as Awarded Offspring Theory) is proposed as a new approach to explain the cause of senescence at a neo-Darwinian level, i.e., through genetic adaptation via natural selection. The theory argues that aging is a result of programmed death, which would be evolutionarily advantageous for genes in sexually reproducing organisms, as it would allow the elimination of the bodies that carry them. A central aspect of this theory is the proposal of a mechanism that circumvents the problematic concept of group selection. Traditionally, group selection, where a trait benefits the group at the expense of the individual, is viewed with skepticism in evolutionary biology for contradicting the maximization of individual fitness. The Prized Son Theory seeks to overcome this barrier by focusing on benefits at the genetic level. This integrated approach implies a more holistic understanding of evolution, where aging and sexual reproduction are not isolated phenomena, but rather interdependent adaptations that arise from shared selective pressures.

2. Foundations of the Prized Son Theory Gene-Centered Evolution

This section delves into the theoretical foundation of the Prized Son Theory, emphasizing its distinction from traditional Darwinism by adopting a neo-Darwinian, gene-centered view of evolution. It illustrates how natural selection operates at the gene level, even when this conflicts with the survival or reproduction of the individual organism.

2.1. Orthodox Darwinism vs Neo-Darwinism

Orthodox, or classical, Darwinism focuses on natural selection acting on the fitness of the individual organism, defined as its ability to survive and reproduce. However, the currently most accepted view in biology is Neo-Darwinism, which postulates that natural selection operates primarily on genes, considering the gene as the central unit of evolutionary dynamics. While in most cases what is advantageous for the individual organism is also advantageous for the genes that compose it, and vice versa, there are situations where the survival of the organism conflicts with the survival of its genes.

2.2. The Concept of Gene Fitness

To address these conflicts, the theory introduces the concept of gene fitness (or fitness of a subgroup of genes). Gene fitness is defined as the capacity that a gene confers to the phenotype to increase its own frequency in the population's gene pool. This distinction is crucial, as it allows for the analysis of the evolution of traits that may appear disadvantageous to the individual but are beneficial for the propagation of its genes.

2.3. Conflicts Between the Individual Organism and Gene Survival

To illustrate the dynamics of gene fitness, the theory presents hypothetical and real examples of conflicts between the individual organism and its genes

2.3.1. Conflicts Favoring the Individual at the Expense of Genes (Rare in Nature)

- **Infanticide:** A mutant organism that feeds on its own offspring might survive and reproduce more than other organisms without this mutation. However, its genes would not benefit, unless in situations of extreme food scarcity, where the survival of the organism, even practicing infanticide, would be advantageous for the genes.
- **Cannibalism (Within the Species):** An organism that habitually hunts and consumes indiscriminately members of its own species tends to harm its genes because it would be destroying its own genetic lineage unnecessarily.
- **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, would see the frequency of her genes decrease in the population's gene pool compared to mothers who protect their offspring.
- **Observation:** The rarity of these behaviors in nature provides empirical validation for the gene-centered perspective. Behaviors that harm gene fitness are strongly selected against.

2.4. Conflicts Favoring Genes at the Expense of the Organism (Common in Nature)

- **Altruistic Mother:** A mother who risks her life to defend her offspring. This behavior can favor her genes, present in her descendants, even if it entails high risk for the individual organism, as long as the probability of saving the offspring is equally high.
- **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.
- **Observation:** The frequency of these examples in nature demonstrates that actions that increase gene fitness can be selected, even if they harm the individual organism. This observation is not just a theoretical preference, but a framework that better explains observed biological phenomena.

2.5. Parental Altruism as a Gene-Driven Strategy

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 demonstrated. 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 descendants, even if it may harm her as an individual organism. The evolutionary benefit conferred to genes over the individual organism justifies parental altruism.

2.6. Reinterpreting Group Selection Through Gene Fitness

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 shares its prey with others in the pride without all lions adopting the same behavior would be disadvantaged, unless the pride was composed of its own family, with a high degree of genetic sharing. The Prized Son Theory directly addresses this controversy, stating that it will break through the barrier of group selection. The solution lies in demonstrating how traits that benefit the group can, ultimately, provide an advantage at the genetic level, such as through kin selection or the Awarded Offspring mechanism. This positions the theory as a sophisticated attempt to reconcile seemingly contradictory levels of selection, offering a more nuanced understanding of how complex social and life history traits can evolve. Group selection, therefore, can only be considered a valid neo-Darwinian explanation if there is a mechanism that proves the benefit to the organism's gene that promotes benefits for the group at the expense of the individual organism.

3. Critiques of Pre-existing Theories of Aging

This section systematically evaluates the main theories of aging that have gained some acceptance in the scientific community, highlighting their specific deficiencies in providing a comprehensive Darwinian explanation, particularly in light of the gene-centered and adaptive perspective of the Prized Son Theory.

3.1. General Deficiencies of Existing Theories

Many of the existing theories on aging are, at best, incomplete, as they rely solely on internal mechanisms (physical causes) and neglect evolutionary influences. These theories frequently fail to explain the enormous differences in aging rates among distinct species. Furthermore, they do not offer convincing reasons for the inability of somatic cells in multicellular organisms to regenerate indefinitely, unlike immortal bacteria or germ cells. A robust theory of aging should, ideally, identify the evolutionary causes or selective pressures that favored its emergence. It should also answer fundamental questions such as: why do some species age rapidly while others do not, or do so very slowly? Why does aging predominantly occur in sexually reproducing organisms, while asexual organisms hardly age (like multicellular anemones and jellyfish)? And why do somatic cells not produce more telomerase, as germ cells do, to avoid aging? The recurrence of these questions without satisfactory answers points to a fundamental gap in existing approaches.

3.2. Specific Critiques

3.2.1. Theory of Free Radicals (Harman, 1954)

This theory proposes that aging occurs due to an excess of free radicals – ionized molecules, usually oxygen compounds – produced as byproducts of cellular metabolism.

• **Critique:** The theory fails to explain why cells degenerated by free radicals could not be replaced by non-degenerated ones, as normally occurs with dead somatic cells. Furthermore, it would imply that animals with higher metabolic rates would age more rapidly, which is contradicted by many examples in nature. It also does not explain why athletes would age faster than sedentary individuals, which is not always true either.

3.2.2. Theory of the Good of the Species (Weismann, 1882):

August Weismann proposed that aging results from programmed death for the benefit of the species, freeing up resources for younger, more adapted individuals.

• **Critique:** This theory is fundamentally flawed due to its reliance on group selection without a mechanism that overcomes the loss of individual fitness. If an immortal mutant emerged, it would outcompete mortals in reproduction, leading to an immortal population, even if this were detrimental to the species as a whole. The fitness of the individual organism, in this case, would outweigh the benefit of the species.

3.2.3. Theory of Accumulated Damage (Medawar & Haldane, 1952)

Sir Peter Medawar and J. Haldane proposed that aging occurs due to the accumulation of genetic damage that manifests later in life, under low selective pressure.

• **Critique:** The theory does not explain why such genes would spread in initially immortal species, suffering from the same problem of group selection as Weismann's hypothesis. Any gene that reduces the organism's fitness, even if late-acting, should be negatively selected. Additionally, it does not establish a correlation with sexual reproduction.

3.2.4. Theory of Antagonistic Pleiotropy (Williams, 1957):

George Williams formulated that senescence can be explained by alleles that benefit the organism in youth (e.g., enhanced vision) but harm it later (e.g., cataracts), being maintained due to the initial reproductive advantage.

• **Critique:** It does not explain the absence of this effect in asexual species, the large differences in longevity in very similar species, or why the organism could not maintain the level of gene activity that benefited it in youth. Most importantly, the theory does not demonstrate why immortal organisms, which did not inherit these genes and thus would not suffer these symptoms in adulthood, could not compensate for weaker youth performance with greater vitality in their infinite adult phase.

3.2.5. Theory of the Disposable Soma (Kirkwood, 1977)

Thomas Kirkwood suggested that, given the high mortality from external factors, energy should be directed towards reproductive capacity instead of maintaining the organism indefinitely.

• **Critique:** The theory does not quantify the energy needed for cellular repair versus reproduction to conclude that the expenditure would be unfeasible. It seems contradictory to discard an experienced adult in its reproductive years to replace it with younger, inexperienced individuals, who will require time and energy to reach reproductive maturity. Furthermore, young organisms are more prone to dying than experienced adults.

3.2.6. Theories of Evolvability (Goldsmith, Mitteldorf, Travis, Bowles)

These theories, based on Weismann's Good of the Species idea, propose that aging increases the rate of species evolution, allowing for a faster replacement by more adapted generations.

• **Critique:** They suffer from the same paradox of group selection as Weismann's theory, as they lack a neo-Darwinian mechanism that explains how a trait disadvantageous to the individual, which reduces its fitness, could spread through the species, even if it benefited the group as a whole.

3.2.7. Theory of the Sexual Cause (Clark)

William R. Clark refined the Disposable Soma theory, suggesting that aging began with protozoa that segregated germinal (micronucleus) and somatic (macronucleus) DNA, with somatic DNA being destroyed after reproduction due to damage.

• **Critique:** It contains logical errors: there is no evolutionary need to program cell death if it occurs naturally due to the accumulation of mutations or wear and tear. Furthermore, it does not present reasons that would render somatic DNA repair impossible, given that bacteria and germ cells are immortal.

A critical and recurring flaw identified in many of these theories is the inability to provide a neo-Darwinian mechanism that explains how a trait detrimental to individual fitness (such as programmed death or the costs of sexual reproduction) could spread and become fixed in a population if it only benefited the group. This convergence of critiques on established theories highlights a fundamental theoretical gap that the Prized Son Theory proposes to fill, offering a direct gene-level mechanism to overcome the paradox of group selection. The persistence of the concept of programmed death in various theories, despite the difficulties in explaining it, suggests that the idea that aging is an active and genetically controlled process maintains a strong intuitive appeal. The challenge, which the Prized Son Theory seeks to address, is to provide a robust mechanism for the evolution and persistence of such a program, given its apparent individual disadvantage.

4. Sexual Reproduction: The Awarded Offspring Mechanism

This section elaborates on the Prized Son Theory's central explanation for the evolutionary advantage of sexual reproduction: the Awarded Offspring mechanism. It compares sexual and asexual reproduction, critiques existing theories on sex, and details how sexual recombination facilitates the rapid emergence and spread of highly fit super-organisms.

4.1. Advantages and Disadvantages of Sexual vs. Asexual Reproduction

Sexual reproduction, despite its individual costs, offers significant advantages compared to asexual reproduction, as summarized in Table I.

Characteristic	Sexual Reproduction	Asexual Reproduction
Genetic Variability	(+) Greater (mixing of genes from parents)	(-) Lesser (offspring are clones)
Elimination of Harmful Mutations	(+) Easier (organisms with double harmful mutations are eliminated more rapidly)	(-) Does not eliminate harmful mutations easily
Dissemination of Beneficial Mutations	(+) Faster through males (a male with high fitness can inseminate multiple females)	(-) Does not spread beneficial mutations to all (each cell generates its own lineage)
Selection	(+) Sexual selection favors adaptive characteristics and promotes the extinction of less adaptive ones	(-) There is no sexual selection
Difficulty of Reproduction	(-) Greater (need to find a partner)	(+) Easier (no need to seek sexual partners)
Energy Expenditure	(-) Greater (production of males, gamete transport)	(+) Lower
Efficiency of Genetic Transmission	(-) Each offspring carries only half of the chromosomes from a parent	(+) Each offspring carries all the chromosomes from the parent (100% efficiency)
Generation of Super-Organisms	(+) A child can inherit two or more beneficial mutations from each of its parents, becoming a super-organism	(-) A double beneficial mutation depends directly on the quantity of offspring and the time it takes for this to happen (beneficial mutation rare, double even rarer)

Table I: Advantages and Disadvantages of Sexual/Asexual Reproduction

Citation: de Barcellos, J. C. H. (2025). The Prized Son Theory: A Neo-Darwinian Explanation for Sex and Aging. *J Gynecol Reprod Health, 3*(2), 1-7.

Although sexual reproduction implies disadvantages such as greater difficulty and energy expenditure to find a partner, in addition to transferring only half of the chromosomes to the offspring, its advantages, especially the ability to generate super-organisms, are fundamental for the long-term adaptability of the species.

4.2. Critique of the Red Queen Theory

The Red Queen Theory, proposed by William Donald Hamilton, attempts to explain the necessity of sexual reproduction as a way for multicellular organisms to defend themselves against bacterial infections. It argues that, due to the faster growth and mutation rate of bacteria, multicellular animals would need to acquire genetic variability more rapidly to protect themselves. However, the Prized Son Theory presents a critique of this view. Most mutations are harmless or harmful, with beneficial mutations being much rarer. Thus, the mixing of genes through sexual reproduction should, in theory, produce more less-adapted organisms than more-adapted ones, and with lower resistance to bacteria than higher resistance. Furthermore, similar to Weismann's Good of the Species theory, Hamilton's theory does not provide a mechanism that explains how this group selection could occur in terms of genetic benefit, i.e., how a sexuality gene could fare better than an asexuality gene. The main deficiency lies in the lack of a direct mechanism that demonstrates how the individual costs of sexual reproduction are compensated by a benefit at the genetic level that ensures its propagation.

4.3. The Awarded Offspring Mechanism

The key to understanding sexual reproduction and aging, according to the theory, lies in what is called the Awarded Offspring Theory. This mechanism proposes that sexual reproduction allows the fusion of two or more beneficial mutations from distinct organisms into a single organism, resulting in a super-organism with extremely high fitness. This occurs without the need for an extensive waiting period, as would be the case in asexual reproduction. An elucidating example is the difficulty bacteria have in surviving two types of antibiotics administered simultaneously, due to the absence of sexual reproduction. For a bacterium to develop resistance to two drugs, an impossible population density would be required for the double mutations to occur by chance. If bacteria possessed sexual reproduction, a bacterium resistant to the first antibiotic could mate with another resistant to the second, producing a super bacterium resistant to both, which would then proliferate.

This perspective diverges from Geoffrey Miller's view in The Mating Mind, which suggests that sexuality primarily serves to discard mutations, especially harmful ones. While Miller argues that sexual reproduction ensures that some offspring will have very good genes by concentrating harmful mutations in others that die, the Prized Son Theory refines this idea. Harmful mutations are naturally eliminated by selection; sexuality is not strictly necessary for this. The true 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. The mechanism for the dissemination of the sexual allele would be as follows: an allele that induces sexual reproduction would generate gametes that would meet other mutant gametes carrying different beneficial mutations. This would result in a super-organism with a double beneficial mutation and 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. This ability to produce super-organisms provides the direct benefit at the gene level that resolves the paradox of group selection for sexual reproduction. The individual, and its genes, that participates in sexual reproduction, despite the individual costs, obtains a greater probability of producing highly fit offspring that will successfully perpetuate its genes. This is the central mechanism by which sexual reproduction, an apparently costly trait for the individual, becomes evolutionarily stable.

5. Aging as an Evolutionary Adaptation: Creating Vacancies for Super-Organisms

This section explores how the Prized Son Theory explains aging as an evolutionary adaptation. It argues that programmed death is a crucial mechanism for generational renewal, which, in stable environments, is essential for the evolution and adaptability of species, especially through the creation of vacancies for super-organisms.

5.1. Equilibrium Condition and the Problem of Immortality

In a reasonably stable environment, the birth rate of a species should, on average, match its death rate to maintain population equilibrium. If the birth rate were consistently higher, the population would proliferate until exhausting available resources; if it were lower, the species would go extinct. In this equilibrium condition, if a species were immortal (without programmed death in its DNA), the only births that could survive and reach maturity would be those replacing individuals who die from accidental causes (fights, accidents, predators, diseases, etc.). In an extreme scenario where, adult organisms of an ageless species also did not die from other causes (besides starvation), all potential births would starve to death before reaching maturity, as there would be no food resources for the unborn offspring. Consequently, evolution would not occur, as there would be no opportunity for the birth of mutant organisms, which are the raw material for evolution. A species that does not evolve is doomed to extinction because it cannot adapt to environmental changes or to competition with other species, especially against rapidly adapting bacteria (Red Queen Theory).

5.2. Aging as a Death Gene

If a mutant gene (or group of genes) that caused the death of the organism after it had passed its reproductive phase appeared, giving it enough time to transmit this gene to the next generation, this death gene could benefit from the increase in the death rate it caused. When the aging gene kills the organism (which has already had its offspring), it creates a vacancy (releases the occupied space and used food resources) within the local group, allowing some infant or

Citation: de Barcellos, J. C. H. (2025). The Prized Son Theory: A Neo-Darwinian Explanation for Sex and Aging. *J Gynecol Reprod Health, 3*(2), 1-7.

unborn organism to reach maturity. Crucially, this vacancy has the chance to be filled by a super-organism, i.e., an individual who inherits two or more beneficial mutations from its parents, assuming that this species reproduces sexually. Since death occurs within the local group, there is a higher probability that this vacancy will be filled by a relative of the deceased organism, rather than a stranger. This means that the gene that causes death by aging has a higher chance of filling the vacancy, as it is geographically closer to the released resources than a distant allele that does not 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 an awarded offspring once to quickly spread through the population, benefiting both the group's evolutionary rate and the aging gene(s) itself. This explanation implies a component of kin or spatial selection for the dissemination of the aging gene, refining the mechanism beyond simple population renewal.

5.3. Instability of Immortal Populations and Optimal Aging Rate

A population composed of immortal individuals, even if super-organisms, would tend to have a low evolutionary rate. This region of immortals would have low adaptability and could be quickly overtaken by more adaptable organisms, specifically the group of mortal organisms. It is therefore concluded 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. Table II summarizes the advantages and disadvantages of immortality

Characteristic	Mortal Organisms (Age)	Immortal Organisms (Do not age)
Beneficial Mutation Rate	(+) Higher (higher death rate allows more births, each new birth can carry a new mutation)	(-) Lower (lower mortality rate prevents new births from surviving)
Harmful Mutation Rate	(-) Higher	(+) Lower
Species Evolutionary Rate	(+) Highest (the population set is replaced more quickly by new generations)	(-) Lowest (old organisms tend to stay alive and consume resources that could serve new ones)

Table II - Advantages and Disadvantages of Immortality

Exportar para as Planilhas

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 allows the organism to live too long, it prevents others from being born and surviving. There must, therefore, be an optimal level of aging that allows organisms to have an optimal number of offspring while also permitting the species to evolve. In short, the Prized Son Theory proposes that

• 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 genes only needs to hitch a ride once on an awarded 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 would not be spread as vigorously as in sexual reproduction (as the rate of awarded offspring is higher in sexual reproduction) [1-11].

6. Conclusion

The Prized Son Theory offers a comprehensive neo-Darwinian explanation for the paradoxes of aging and sexual reproduction, positioning both as crucial adaptations for long-term genetic perpetuation. By centering on gene fitness, the theory proposes an elegant mechanism, the Awarded Offspring, that resolves the challenge of group selection. Sexual reproduction, despite its individual costs, is revealed as a driver of evolution by facilitating the emergence and rapid dissemination of super-organisms with beneficial mutations. Aging, in turn, emerges as a programmed strategy to create the necessary vacancies, allowing these super-organism to thrive and drive the species' adaptability.

References

1. Biological Aging.
2. Rose, M. R. (2004). Biological immortality. The scientific conquest of death. *Essays on infinite lifespans, Buenos Aires: Libros en Red*, 17-28.
3. Franken, J. (2021). The Hayflick Limit. *River Teeth: A Journal of Nonfiction Narrative, 22*(2), 107-111.
4. Hornsby, P. J. (2007). Telomerase and the aging process. *Experimental gerontology, 42*(7), 575-581.

5. Harman, D. (1992). Free radical theory of aging. *Mutation Research/DNAging, 275*(3-6), 257-266.
6. Li, S., Vazquez, J. M., & Sudmant, P. H. (2023). The evolution of aging and lifespan. *Trends in Genetics, 39*(11), 830-843.
7. Teixeira, I. N. D. A. O., & Guariento, M. E. (2010). Biology of aging: theories, mechanisms, and perspectives. *Ciencia & saude coletiva, 15*(6), 2845.
8. Clark, W. R. (1998). *Sex and the Origins of Death.* Oxford University Press.
9. Blute, M. (2003). The Mating Mind: How Sexual Choice Shaped the Evolution of Human Nature.
10. Dolnick, E. (1991). Why do women outlive men. *The Washing-ton Post Health*, 10-13.
11. Fletcher, J. A., & Doebeli, M. (2009). A simple and general explanation for the evolution of altruism. *Proceedings of the Royal Society B: Biological Sciences, 276*(1654), 13-19.