



Human Aging as a Creational Good: Interactions between Theology and Molecular Biology

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A Christian theology of human aging faces significant challenges. First, aging receives far less theological attention than the heavily related and well-established themes of creation, sin, death, and imago Dei. Second, Christian theology discordantly supports two fundamental yet polarized claims: that human aging is a good of creation, and that it is an effect of humanity's fall. Third, it lags in its engagement with the science of aging. This article counteracts these obstacles by integrating Christian theology with molecular biology. Aging generating lifespan ("human biological aging," or HBA) is distinguished from aging leading to life expectancy. Intrinsic molecular pathways driving HBA are orchestrated, functional systems spanning multiple biological strata. Surprisingly, aging processes are indivisible from life, health, and growth processes. These considerations build a case for understanding HBA as a good of divine creation, clarify interpretations of humanity's fall, challenge transhumanist aspirations of age reversal or cybernetic immortality, inform theology–evolutionary biology dialogue, and promote a holistic view of human life through time.



Introduction

The United Nations predicts that by 2050, those over sixty-five will more than double children under five (United Nations 2022), while those eighty years or over are projected to be the fastest growing of any population fraction (United Nations 2017), delivering enormous economic and ethical challenges to societies.

For several reasons, it seems the world is not ready for this “tsunami” of elderly. Of all the life stages, old age is arguably the sole stage that can evoke aversion, expressed in the three-fold response of fear of decline and morbidity, disgust by the non-aged over the unpleasantness of aged bodily decline, and hatred in the form of elder abuse (Kolnai 1998). For those experiencing the genesis of aging’s decline, “aging anxiety” is known to set in (Lasher and Faulkender 1993). For those still young, ageism keeps aging at arm’s length (de Lange 2015, 77). Moreover, some in the scientific community aim to stop or even reverse the aging process, with the inference being that aging is a defect or disease that needs to be conquered (Gems 2011; Sinclair 2019, xxi, 67, 81, 83). Overall, then, humanity continues to struggle to accept and assimilate the aged and the aging process.

Western Christian theology faces three challenges in its attempts to respond to the realities of an elderly tsunami, society’s aversion toward the aged, and the aging process. The first is a dearth of engagement with the theme of aging. Compared to the well-established themes of creation, sin, death, and imago Dei, much less is said about the closely related theme of aging. Representing this observation, from over twenty-five surveyed systematic and biblical theologians—including theo-anthropological specialists and such influential thinkers as Jürgen Moltmann, Dietrich Bonhoeffer, Carl Henry, Stanley Grenz, Alexander Schmemmann, Richard McBrien, N. T. Wright, Rudolf Bultmann, Reinhold Niebuhr, Marc Allen Cortez, John McCarthy, Hans Schwarz, Charles Sherlock, David Hugh Kelsey, John MacQuarrie, J. Wentzel van Huyssteen, and Ted Peters—there is no analysis of human aging.

The second challenge is with the particular core question of theological causation: Is human aging a good of creation or an effect of sin in the world? The answer is critical and with far-reaching consequences. The concepts of whether aging is of God or not, whether aging is a creational good or an effect of sin, are disparate, even antithetical. An answer favoring aging’s link with sin can feed palpable negativity toward aging, while an answer supporting human aging as a creational good can serve as a strong foundation for theology’s engagement to counter that negativity.

Augustine of Hippo’s (2002, 62–67) allegorical interpretation of old age in *On Genesis: A Refutation of the Manichees*, John Calvin (1960b, 403), Karl Barth (1958b, 324–618), and Paul Tillich (1964, 77–83, 144–58) all arrive at a perspective on aging—from radically different approaches—that places it

within good creational processes. Yet, Augustine, later in *The City of God* (2003, 518, 519, 520, 524, 537, 539), Thomas Aquinas (1920, 490–92), Karl Rahner (1965, 14, 15, 33, 34), Wolfhart Pannenberg (1991, 96ff, 172), and John Zizioulas (2008, 98–101) alike claim that human aging is a consequence of humanity's sin. Such strongly polarized views reveal a problem in need of resolution.

The third challenge is the need for further engagement with the science of aging. With Pannenberg (1993, 33), "If theologians want to conceive of God as the Creator of the real world, they cannot possibly bypass the scientific description of that world." Pannenberg (1985) does bring aging into dialogue with science in his *Anthropology in Theological Perspective*, yet since that 1985 work, considerable growth has occurred in our scientific understanding of aging. More recently, the influential ethicist Gilbert Meilaender (2013) asks the question "Should we live forever?" in his book on aging and lifespan. Yet, his choice to effectively overlook the scientific question "How do we age?" in favor of the question of purpose "Why do we age?" appears to bypass the possibility that the science of aging can inform the question of whether aging is one of the goods of creation or inform theology's engagement with the ethics of aging and longevity enhancement.

In light of Christian theology's need for internal cohesion and a consistent response to the process of human aging, this article engages Christian theology with molecular biology to support the claim that human aging is a creational good. This article thus majors on one dimension to the field of aging: that of the interaction of human aging as a matter of theological causation with human aging as a matter of physical causation. Other perspectives on, and dimensions to, human aging, for example, the pastoral, phenomenological, or sociological, warrant much attention but are not the focus of this article.

The significance and implications of the claim that human aging is a creational good are explored for such themes as humanity's fall, evolutionary biology, transhumanism, and perspectives on the aged human life.

Human Aging: Critical Distinctions Guide a Nuanced Perspective

The interplay of factors at genetic, cellular, somatic, and environmental levels makes the aging process far more complex than Christian theology's delineation of aging as simply "aging." Therefore, the following builds critical distinctions to guide a more nuanced perspective.

Distinguishing Lifespan from Life Expectancy

Human lifespan is the maximum length of time the human organism can live and changes little with chronological period or environment or across geographical boundaries. Gerontologically, the human lifespan has long been considered to be about 120 years (Bromley 1974, 25; Hayflick 1998), with recent additional scientific support that this limit is "fixed and subject to natural constraints" (Dong, Milholland, and Vijg 2016). Interestingly, this span is consistent with

the traditional Hebrew blessing “Ad me’ah ve-essrim shana” (may you live for 120 years) and has biblical support from Genesis 6:1–4, where the 120-year limit is to be interpreted not as God’s wrathful castigation against sin but as the divine parent-like maintenance of boundaries and protection within those boundaries (Westermann 1984, 367–76). Thus, a degree of consistency can be found between biology’s outlook on lifespan as a natural limit and theology’s outlook as a divinely appointed limit.

Life expectancy, on the other hand, is the length of time a human can *expect* to live and is highly variable across time, environment, and geography. Extrinsic factors negatively modulate the aging process and can cause an end to life before 120 years (Baltes and Smith 2003). Observations of improved health-span and longer life expectancy in some settings are indications of life expectancy’s plasticity, and of how those extrinsic factors can themselves be modulated to influence life expectancy. Figure 1 distinguishes life expectancy from lifespan, highlighting some extrinsic factors.

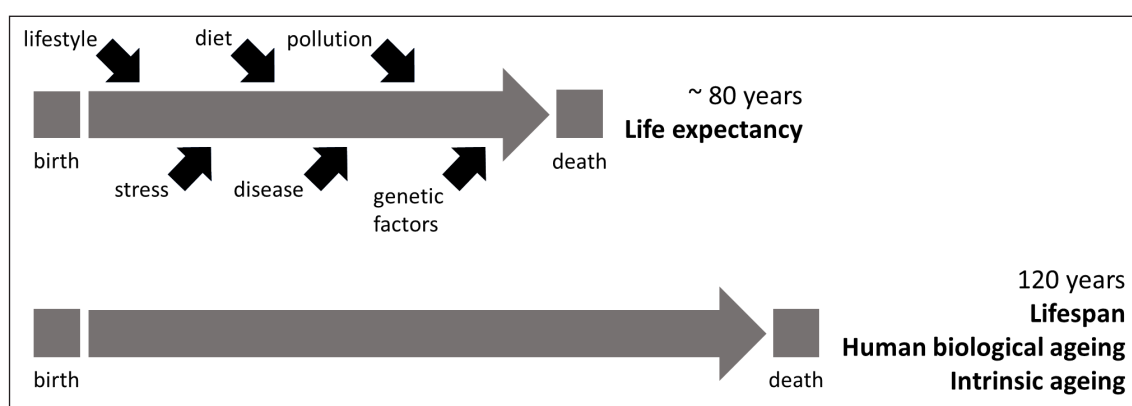


Figure 1: Idealized model comparing life expectancy with lifespan. Medical science attributes an approximate 120-year limit to human lifespan, determined by intrinsic processes that drive human biological aging. Life expectancy has a modulated aging process determined in part by many more extrinsic factors. Extrinsic factors shown here are examples, not intended to be exhaustive, and positioned arbitrarily.

With that important distinction comes questions of aging. The causal link between aging and natural physical death is well accepted: aging precipitates natural physical death (World Health Organization 2022). But the nuance between aging that leads to lifespan and aging that leads to life expectancy is critical. It is here that molecular biology offers explanations that also have theological relevance. This article focuses on molecular biological processes that are intrinsic to the human body, proposing that those processes are causally connected to aging that determines lifespan. It is this aging that this article terms “human biological ageing,” or HBA. Aging that determines life expectancy is the topic of subsequent investigation.

Organismal Aging and Population Aging

The study of aging in populations considers a myriad of extrinsic factors that require epidemiological tools for analysis. Changes in the aging of a population may not necessarily apply to every individual in that population, so there will be limits to how these inform the nature of the aging process. This article focuses on the determinants of aging in the human organism rather than the aging of populations.

Biological Aging and Age-Related Disease

Is age-associated disease an integral part of the aging process (such that they cannot be separated even by definition), or can aging be a process independent of age-related diseases? This question has plied the minds of some of the finest researchers in this field, Robin Holliday and Leonard Hayflick, who offer opposing answers in the *Journals of Gerontology: A, Biological Science* (Holliday 2004; Hayflick 2004) and elsewhere (Hayflick 2000).

Hayflick, who argues for a clear demarcation between biological aging and age-related diseases, has the more convincing argument for his following reasons: (a) the pervasive occurrence of aging across virtually all species; (b) aging's occurrence even within species removed from the wild and protected from disease; (c) that not all features of aging apply to disease; and (d) medicine's improvement of the treatment of age-related diseases—to the point of sometimes overcoming them—yet aging is not thereby prevented (though health-span and life expectancy may improve). To those substantial arguments I add the following: (e) the claim that all human aging necessarily and by definition includes age-associated disease is refuted by one or more examples to the contrary, such as the documentation of natural death without obvious pathology in both nonhuman and human organisms. A notable study is that of Isaio Shimokawa et al., where rats (an organism valued for its human-like physiology) were fed certain diets. At necropsy, 25 percent of rats that died at old age did not show signs of pathology that could cause death (Shimokawa et al. 1993). For humans, one such example is that of Jeanne Calment, who died aged 122 years with no illness recorded on her death record (Robine and Allard 2003). Moreover, old-age death without disease is accepted in gerontological literature (Cavanaugh and Blanchard-Fields 2011, 16). And (f), a demarcation between aging and disease is evidenced in the current and disturbing trend worldwide of ten different cancers that traditionally have been associated with later aging now markedly increasing in incidence in the thirty- to fifty-year-old age bracket (Mauri et al. 2024). Overall, this article accounts for these considerations as a reasonable argument for the position that aging and age-related diseases are distinct.

Collectively, then, these distinctions add nuance to a perspective on aging—distinctions that are necessary as further on the article provides molecular biological evidence to show that HBA leading to lifespan can be theologically considered a good of creation.

At the Molecular Level, Human Biological Aging is a Functional, Orchestrated System

Telomere Erosion Is Genetically Programmed

Since the seminal papers of Leonard Hayflick and P. S. Moorhead in the 1960s (Hayflick and Moorhead 1961; Hayflick 1965), the gradual erosion or shortening of proliferative cells' chromosomal telomeric repeats as a function not of time but of the number of cell cycles has been confirmed in humans and across species, *in vivo* and *in vitro*, and from thousands of publications. It is well understood that this is significant for cell and tissue aging: cells have limited lifespans determined by the length of telomeres at the ends of chromosomes. Once a critical telomere erosion point is reached, cells undergo death or a nonfunctional state termed senescence. After a certain number of cell divisions, tissues comprised of proliferative cell populations eventually decline into reduced function or dysfunction, a process that partly but definitely contributes to organismal aging, diagrammed in Figure 2.

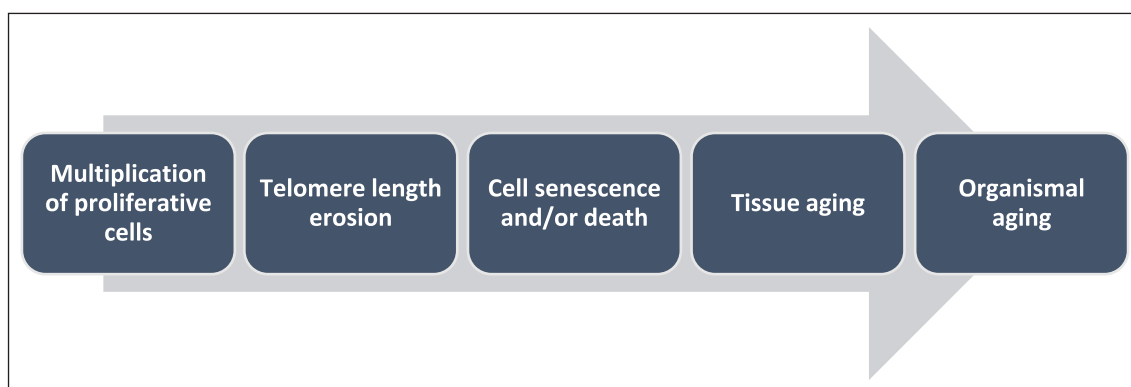


Figure 2: The basic process of telomere-based aging.

Intuitively, one may evaluate telomere “erosion” as a random process, but it is not. The family of enzymes responsible for copying chromosomes, DNA polymerases, have extraordinary copying fidelity—by the enzyme’s proofreading ability, an error rate of one mismatch per three thousand million nucleotides (Korona, LeCompte, and Pursell 2010; Thomas et al. 1991). That copying accuracy is a function of the gene sequences encoding the polymerases and determining the enzymes’ tertiary structures and catalytic sites. Thus, DNA polymerase accuracy is genetically programmed.

However, due to “inherent properties” (Chan and Blackburn 2004) in the way this copying machinery works, as proposed famously by James Watson and Francis Crick in 1953 (Watson and Crick 1953) and confirmed, this DNA polymerase family cannot copy the chromosomal ends completely. Known as the end replication problem, the critical consequence is that with every cell division, progressive shortening of the telomere’s building blocks occurs. The more division, the more the loss of telomeric repeats. This would be inevitable

in all normal somatic dividing cells. The point to emphasize here is that, like polymerase accuracy, telomere erosion is also genetically programmed.

Telomere-Induced Cell Senescence and Apoptosis Are Coordinated, Functional Systems

Through normal cell division, telomere erosion to the critical threshold length leads to either replicative cell senescence or cell death (apoptosis). When either of these events dominate over cell population renewal, then tissue renewal and homeostasis are disrupted, and a tissue's regenerative capacity deteriorates. Both telomere-induced cell senescence and apoptosis are nonrandom and highly coordinated, with the key intermediate step being the initiation of the DNA damage response pathway (DDR). As an overview, a scheme showing the flow of action from telomere erosion to cell senescence or death and organismal aging is shown in Figure 3 (Itahana, Dimri, and Campisi 2001; Multani and Chang 2007).

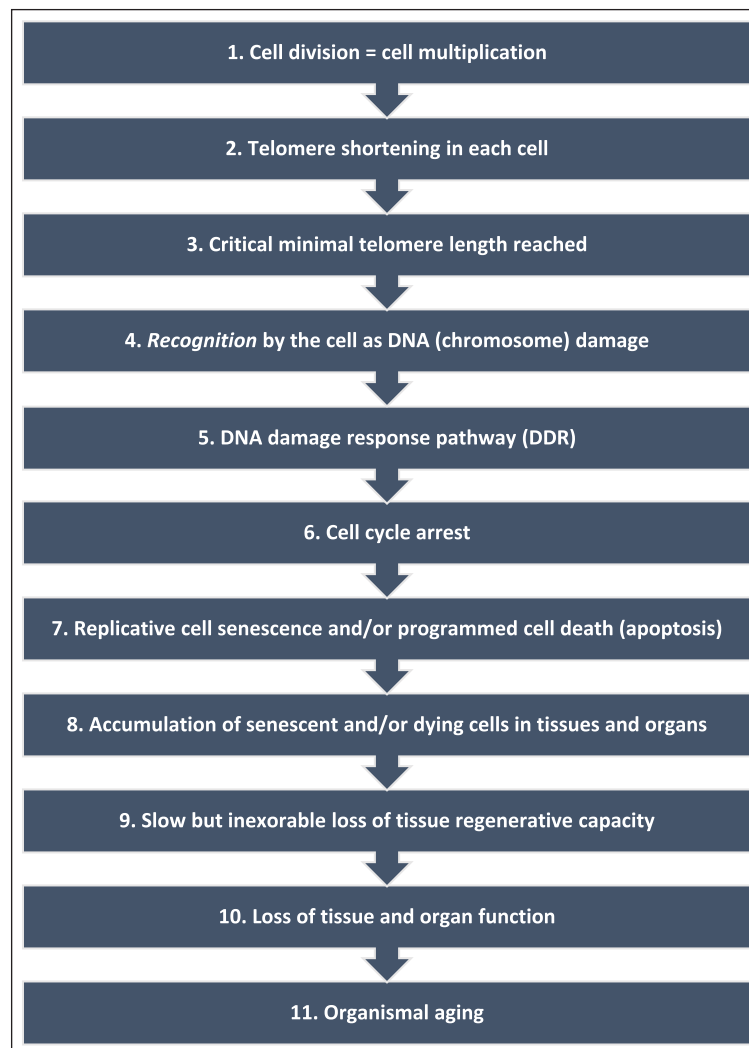


Figure 3: Downstream consequences of telomere erosion contributing to organismal aging.

Within Figure 3's overview, the DDR is central. Fabrizio d'Adda di Fagagna, Soo-Hwang Teo, and Stephen P. Jackson (2004) consider the DDR "an integrated and highly coordinated set of events" akin to a classical signal transduction pathway, described in categories of stimulus, sensors, transduction and amplification, and output. This pathway's purposeful coordination is outlined in Figure 4, simplified because the coordination of the DDR involves over twenty proteins (see Figure 2 in d'Adda di Fagagna [2008]).

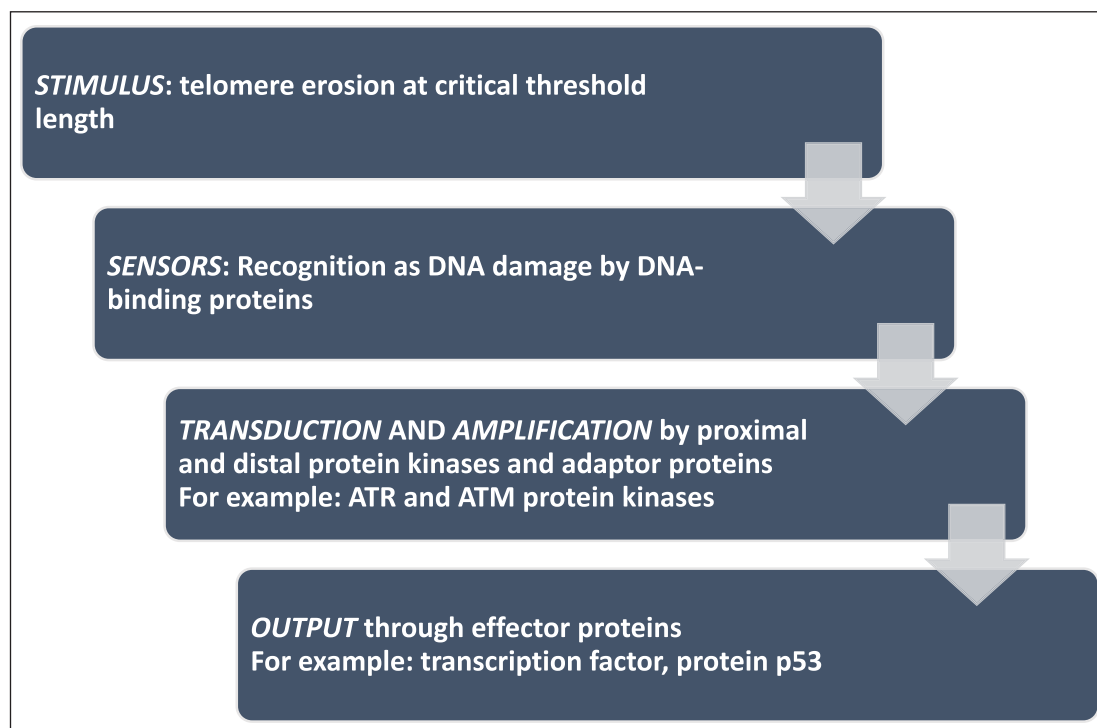


Figure 4: Coordinated events in the DDR. ATR and ATM are protein kinases, signal transduction enzymes that transmit information along specific response pathways such as the DDR.

Telomere erosion to the critical threshold, recognized as DNA damage, can also induce apoptosis, the major form of programmed cell death (Rich, Allen, and Wyllie 2000). Figure 5 summarizes this.

Apoptosis amounts to a series of coordinated molecular events and is far from random, purposeless, or accidental. It is, even in itself, a functional system.

The Absence of Telomerase in Somatic Cells Is Genetically Programmed

Elizabeth Blackburn's laboratory found that telomeric sequences are added to the ends of *Tetrahymena* chromosomes (Blackburn et al. 1983). This led to the discovery of a unique enzyme complex named telomerase, which, coupled with an RNA component, synthesizes telomeric building blocks and preserves telomere length (Greider and Blackburn 1985). Humans, as with many other organisms, possess a gene that encodes a similar telomerase (Morin 1989).

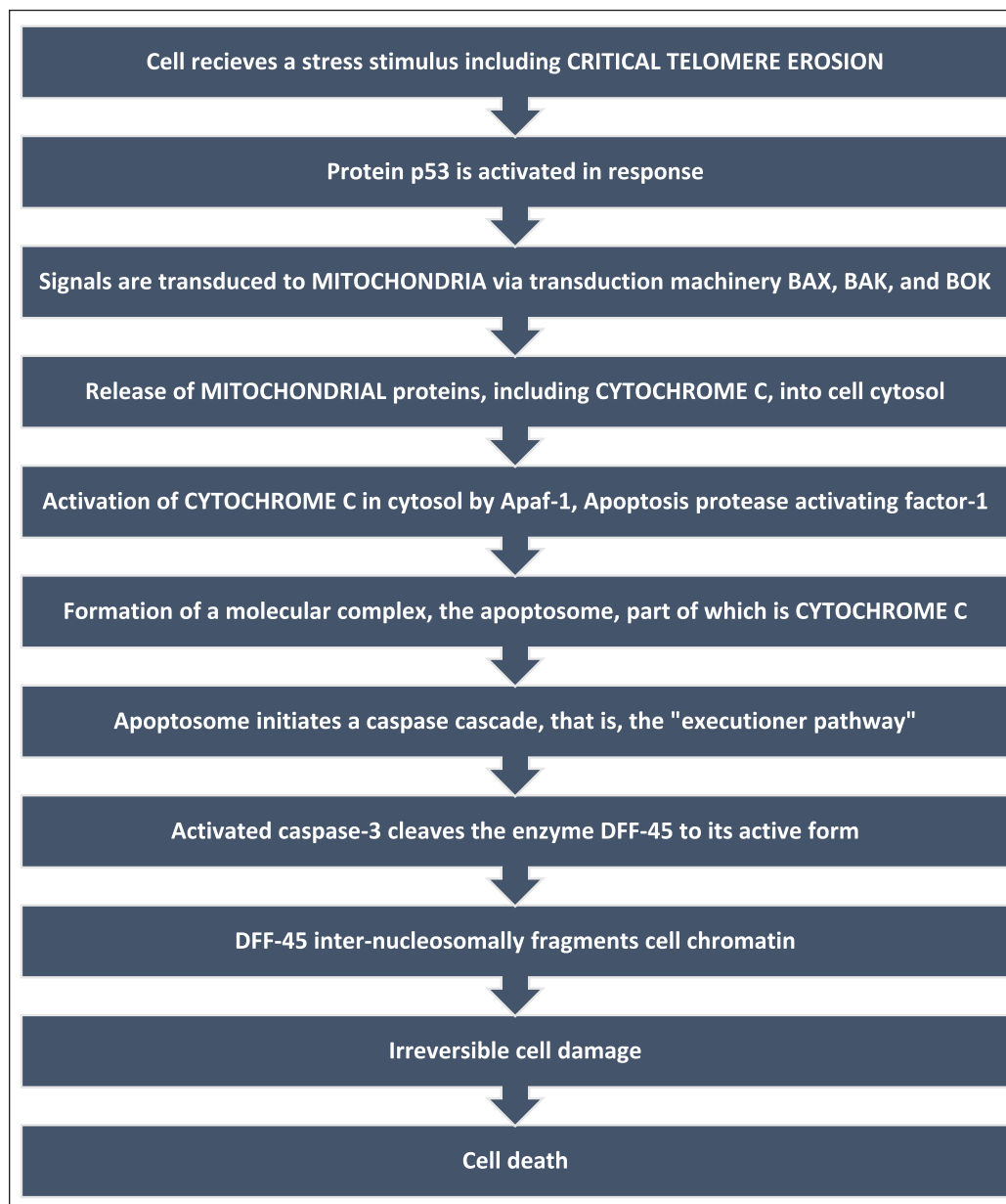


Figure 5: Condensed summary of the coordinated and defined molecular process from telomere erosion to apoptosis (Liu et al. 1998; Widlak et al. 2000; Lindsten et al. 2000; Goldstein et al. 2000; X. Wang 2001; Wei et al. 2001; Scorrano et al. 2002; Penninger and Kroemer 2003; Hill, Adrain, and Martin 2003; Arnoult et al. 2003; Saelens et al. 2004; Hooker et al. 2012).

In humans, however, telomerase is active in germline cells but inactive in most somatic cell types (Kim et al. 1994; Wright et al. 1996). Either the genes, though present, are switched off and the telomerase complex is not expressed, or telomerase expression is at low levels. It is this absence or reduction of telomerase in most somatic human cells that comprises the other key component, along with DNA polymerase's inherent end replication problem, that prevents the preservation of telomere length as cell populations divide.

Absence or reduction of telomerase is not an accidental, uncontrolled, or random process. Rather, it is genetically programmed, predominantly during embryogenesis. Significant changes occur in the expression of telomerase in the somatic cells of the developing embryo, fetus, and newborn. The existence of a program would point to a purposeful role for telomerase *non*-expression and therefore telomere erosion in postnatal human beings.

Regarding early-stage embryonic development, from work with murine, bovine, and human cells, telomerase expression is elevated and telomere elongation occurs at the morula to blastocyst transition (reviewed in Ozturk, Sozen, and Demir 2013). Yet, a remarkable reversal of telomerase expression eventuates during longer-term human gestation. Woodring Wright et al. (1996) found that at sixteen weeks of gestation, activity is found in a broad range of tissues yet becomes undetectable in the same tissues only a few months after birth. Further work (Ulaner and Giudice 1997; Ulaner et al. 1998) delineated the consistent dependency of telomerase levels on gestational and post-gestational age in different tissues.

Thus, over the longer term of gestation, a cessation of telomerase activity in somatic tissues is the normal phenomenon. In light of the sheer precision of embryogenesis and the replicability of telomerase expression across the embryogenesis of species and different organisms, there is little if no support for a random, uncontrolled cessation of telomerase expression. Rather, the reasonable conclusion is that telomerase non-expression in postnatal organisms is a genetically controlled developmental program, contributing to telomere erosion in postnatal somatic tissues.

The Central Nervous System Is a Functional Control Center for Aging

Evidence presented so far has focused on intracellular systems of organismal aging. However, the recent work of Guo Zhang et al. (2013) focused on the systemic control pathways in an organism, in particular the hypothalamus of the central nervous system, showing this control center to have a programmatic role in the aging process and in lifespan determination.

In initial experiments, male and female mice (like rats, a classic humanlike physiological model) of a set middle age were engineered to have either artificially lower or higher levels of hypothalamic NF- κ B, a key transcriptional regulator. Compared with control mice, mice with artificially inhibited NF- κ B lived longer and showed better cognitive function and muscle endurance, thicker muscle fibers, thicker skin, and more bone mass. Elevated NF- κ B levels produced the opposite effects. From these experiments, Zhang et al. were able to conclude that both the protein that switches on NF- κ B activation (IKK- β) and NF- κ B itself form a “driving force” in aging by the hypothalamus.

Following this, middle–old aged mice were genetically engineered to have a nonfunctional IKK- β gene in hypothalamic microglial cells. This prevented the

age-associated increase in microglial cell numbers in the hypothalamus; moreover, TNF- α production in both microglia and in neurons was not induced as age progressed. From these results, the researchers formed a hypothesis: that removing IKK- β from hypothalamic microglia will slow the systemic aging process. When tested, indeed this was the case. Thus, modulating the levels of IKK- β and of NF- κ B in the hypothalamus causally affected aging in the whole mouse.

But what might brain IKK- β and NF- κ B be acting on at the molecular level to effect systemic aging? The release of hypothalamic Gonadotropin-releasing hormone (GnRH) for the stimulation of sex hormone production gives GnRH its fundamental role in sexual development and reproduction. But in this work, Zhang et al. (2013, 215) “revealed a direct link between IKK- β and NF- κ B activation and GnRH decline” that drives sex-independent systemic aging. Importantly, GnRH-related age changes occurred in the brain and beyond in the periphery.

Stages in the mechanistic pathway are outlined in Figure 6.

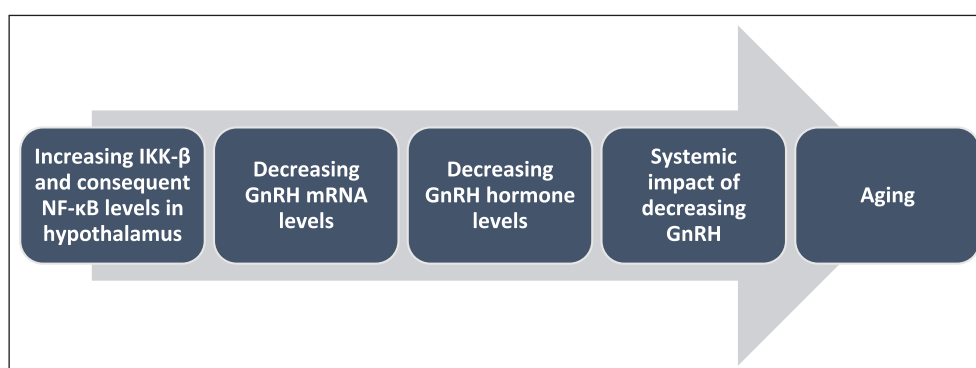


Figure 6: Hypothalamic control of systemic aging via GnRH.

Zhang et al. (2013, 215) concluded this important work by stating: “Our findings provide a proof of principle to the hypothesis that ageing is a life event that is programmed by the hypothalamus.”

Further explorations of a mechanism for the systemic control of aging have come from subsequent work on neural stem cells of the mediobasal hypothalamic region, an area “crucial for the neuroendocrine regulation of the physiological homeostasis of the whole body” (Zhang et al. 2017, 52). Here, Yalin Zhang et al. (2017, 2018) showed that, within normal physiology, hypothalamic adult neural stem cells secrete exosomal miRNAs into cerebrospinal fluid to control aging speed. Subsequent work has drawn attention to what molecular events occur upstream to control the release of exosomal miRNAs by htNSCs. Work by Yu-Zhong Xiao et al. (2020) showed that depletion of a long noncoding RNA, Hnscr, is sufficient to drive the senescence of htNSCs and aging-like phenotypes in mice. “Mechanistically, Hnscr binds to Y-box protein 1 (YB-1) to prevent its degradation and thus the attenuation of transcription of the senescence marker gene *p16^{INK4A}*” (Xiao et al. 2020). In exploring the functional processes

affected by Hnscr deficiency, the most significantly altered biological processes included signal pathways involved in cell senescence and apoptosis, inflammatory responses, glucose metabolism, and protein degradation.

Overall, this work forms a highly significant contribution to understanding aging as a programmed life event.

Molecular Biology Reveals an Indivisibility between Life–Health–Growth Processes and Aging Processes

Considering these molecular processes in isolation from one another hides a remarkable fact worth developing: on multiple biological strata—on the levels of molecule, process and pathway, organelle, organ, and organism of the whole human—the aging process is indivisible from life, health, and growth processes.

At the molecule level, DDR proteins, for example ATM and ATR, are not only essential for the DNA damage response triggered by telomere erosion but are also critical factors in telomere maintenance and homeostasis; in other words, they play roles in genome integrity (part of cell health) *and* cell demise, and which role is channeled depends on how critically short the telomeres are (Shiloh 2003, 2014). Transcription factor p53 is another example. Known as the “the guardian of the genome,” it acts on many levels to regulate genome integrity and cell safety, survival, and health (Vousden and Lane 2007; Park et al. 2016), with studies also showing its involvement in the more organismal-level functions of development, metabolism, and reproduction (Rufini et al. 2013). But p53 also acts to remove damaged cells by participating in the DNA damage response and activating the processes of senescence and of apoptosis (Lane 1992; Sahin and Depinho 2010; Royds and Iacopetta 2006; Multani and Chang 2007). This strong link (Yu and Zhang 2003; Villunger et al. 2003; Chen, Hales, and Ozanne 2007) shows p53 to be both a center of action (Yee and Vousden 2005) and a center of control (Laptenko and Prives 2006; Braithwaite, Del Sal, and Lu 2006). Connected with p53’s role in activating senescence and apoptosis, evidence supports the claim that p53 regulates organismal aging (Tyner et al. 2002; Rufini et al. 2013). Importantly, as with ATM and ATR, p53 fundamentally contributes to both cell life/health and cell demise.

At molecular pathway and process levels, apoptosis, the major form of programmed cell death, directly contributes to mammalian embryogenesis and normal and healthy postnatal growth and occurs even in mature organismal tissues in both vertebrates and invertebrates (Staley, Blaschke, and Chun 1997). John Kerr, A. H. Wyllie, and A. R. Currie (1972) consider apoptosis to have a “complementary but opposite role to mitosis [cell division] in the regulation of animal cell populations”; apoptosis occurs in order for normal growth to occur (Judah, Ahmed, and McLean 1965; Kerr 1971).

At the organelle level within cells, mitochondria use respiratory oxygen to generate the universal energy molecule of living cells, ATP, in a process termed oxidative phosphorylation, or cellular respiration (Newmeyer and

Ferguson-Miller 2003). ATP synthesis requires the activity of a series of protein complexes embedded in the mitochondrion's inner membrane that form the electron transport chain. The protein cytochrome c is a critical component of these complexes and therefore intimately involved in the life process of cellular respiration.

Yet, mitochondria and cytochrome c are also essential to the intrinsic apoptosis pathway. Mitochondrial cytochrome c forms part of the apoptosome, which initiates the “executioner” pathway of apoptosis. Thus, mitochondria and their molecules, cytochrome c, drive cell life; they also drive cell death (Ozoren and El-Deiry 2002; Khosravi-Far and Esposti 2004).

At the organ level, the mammalian brain's hypothalamus is essential for life, health, and growth processes, being a master regulator for reproduction and sexual development in both males and females, energy metabolism, body temperature, and circadian rhythms. But as we have seen, Zhang et al.'s (2013) work broke new ground by showing this control center to have a programmatic role in the aging process and in lifespan control.

At the organism level, the creation of a living human body during embryogenesis involves the activation and coordination of aging–death processes: the genetic program of cessation of telomerase expression in somatic cells, and apoptosis.

The indivisibility between life–health–growth processes and aging processes across multiple biological strata is shown in Figure 7.

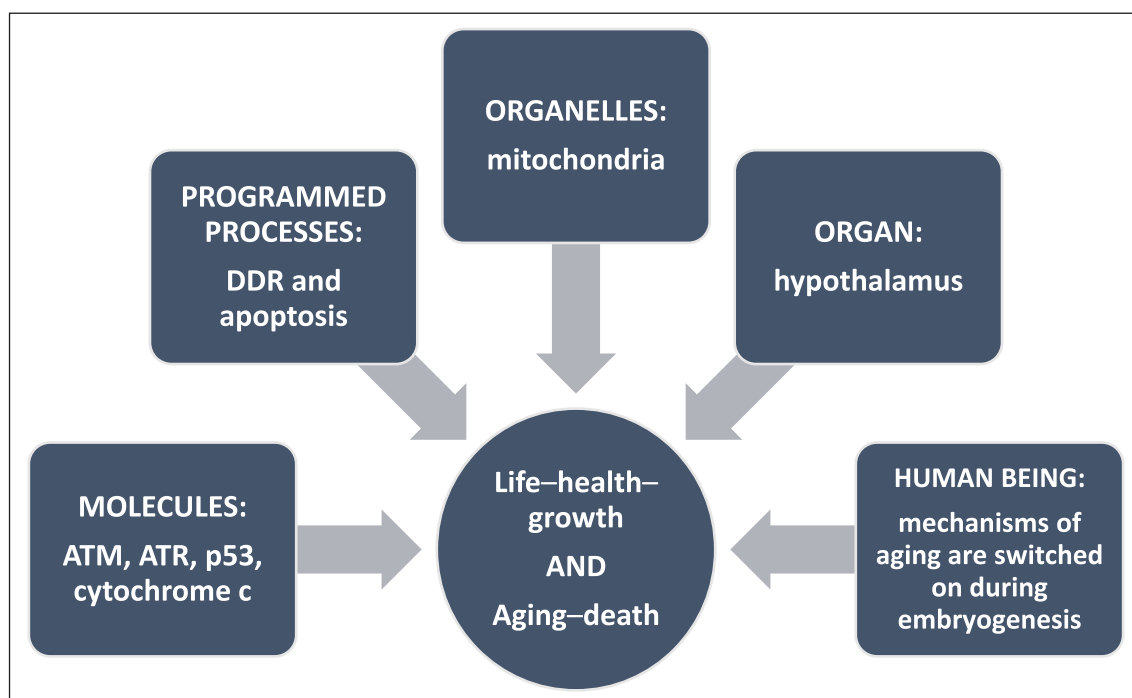


Figure 7: The indivisibility of life and aging–death processes across different biological strata.

Christian Theology's Interaction with the Molecular Biology of Human Biological Aging

This article now interacts Christian theology with molecular biology around the central proposal that HBA—aging leading to lifespan—is one of the goods of divine creation.

The growth in the understanding of human aging at the molecular level brings us closer to understanding the biological nature of the human being, of the human through time, of humanity's finitude, and of natural physical death. Thus, we are penetrating with some success into mysteries that preoccupy much of theology. This should get theology's attention. Moreover, an interaction of this sort seeks to contribute resolution in one of the two broad themes in the ongoing science–religion debate identified by Nicholas Spencer (2023, 7), that being “the nature and status of the human.”

Functional, Programmed Systems of Human Biological Aging Support the Claim That Aging Is Evidence of Divine Creative Activity

As noted, Christian theology has polarized responses as to whether aging is of God or an effect of sin. Is human aging “good” in the biblical creation sense? I am concerned here with the seminal creation data of Genesis 1:1–2:3 and its explicit, prominent, and repetitive “approval” formula that proclaims creation's categories as “good” and the whole as “very good.” With attention to this and other scriptures, and to the exegetical parameters of literary and historical context, authorial intent, textual boundaries, style, and structure, “creational good” is defined as being (a) willed and accomplished by God the master craftsman, (b) reflecting the goodness and character of God, (c) having harmony and complementarity, (d) forming part of the relational good of creation, and (e) being fit for purpose (Hooker 2021, 50–60).

Several of these descriptors reflect the ancient Hebrew understanding of a functional ontology for creation rather than the modern Western material ontology. John Walton (2009, 26–28, 36–45) brings this out well: to the ancient Hebrews, a “good” created entity existed “by virtue of it having a function in an ordered system.” Here, Walton echoes the Hebrew conception of an ordered system as “society and culture,” yet the concept of an ordered, functional system cannot exclude other discoverable ordered systems such as those in biological systems discussed here.

Considering the molecular biology of aging in light of the aforementioned, the answer is yes, there is support for a biblical “goodness” in aging's molecular processes. We observe genetic control, purpose, direction, orchestration and complex organization, intracellular and intercellular communication, sensors, and the transduction of information, all of which argue for functional systematization.

Yet, some may challenge this. In witnessing inbuilt molecular mechanisms of aging, could we be witnessing the changes in the human body that are the

very marks of the effect of humanity's fall? Has science discovered theology's concept of a cosmic fall? If this is so, then the "post-fall" changes to every relevant gene in every cell of every human—forming part of the normal genetic code—make every human being, including post-fall Adam, fundamentally and physically unlike pre-fall Adam and would have major implications (some may say devastating implications) for our understanding of imago Dei.

The more appropriate response has the stronger argument: the inseparability of coordinated molecular processes of life and those of aging–death leads us down an altogether different path. Aging's coordinated molecular phenomena exist on five strata of physical existence: at the levels of molecule, process and pathway, organelle, organ, and organism. One may be inclined to say "remove apoptosis, remove cytochrome c and p53 and mitochondria and the hypothalamus—all those entities known to cause death and aging—and we will have a more 'vigorous' human being." But this cannot be ventured because these very entities are essential to life, health, and growth; without these entities, there would be no living human organism. Thus, science confronts theology with the probability that aging is exquisitely and intricately built into life.

Altogether, the evidence and implications of that evidence build a reasonable argument for evaluating HBA (a) as an intrinsic biological life process and physiological program that determines lifespan, (b) not as a random process nor one determined exclusively by external factors, and (c) as an assertion, with Hayflick, that "aging is not a disease" but rather that it is good in the biblical creation sense.

Astonishment, Reverence, and Humility toward the Unexpected Are Appropriate Theologically Driven Responses to Human Biological Aging

Beyond an alignment of the molecular data with the concept of human aging as good in the biblical creation sense, it is appropriate to gauge how theology may interact in a deeper way. William Desmond's three modalities of wonder—curiosity, perplexity, and astonishment—provide a framework for interaction (Tyson 2023). With Desmond, science is often scientific in its self-belief. Here, finding answers is driven by curiosity. This seems harmless enough, but it is a drive that believes all is determinate, that science can find "a determinate solution to a determinate problem," driving as on a hamster wheel a "ceaseless accumulation of determinate cognitions" (Desmond 2023, 20, 24).

Scientific self-belief leaves out the other modalities of perplexity and astonishment, and to Desmond (2023, 25), this is a mistake. Penetratingly, Desmond proposes that in its contraction down to mere curiosity, science expands its concept of knowing to an "idolatrous knowing" where the "too-muchness" in the "given being" is dismissed. Without science retaining astonishment and perplexity in its enterprise, the determinate vanquishes both

the indeterminate and “overdeterminate,” idolatrously becoming a project of “self-determination.” There is much to appreciate here in Desmond’s assessment.

With Paul Tyson (2023, 11), knowledge in science is to be subordinated to worship. Theology has a role here: to speak reverence, to preserve astonishment. Notably, Desmond (2023, 25) selects the word “astonishment” because it stresses “*the emphatic beyond expectation.*” Astonishment involves the unexpected. The unexpected calls us to stop, step off the ceaseless hamster wheel fueled by curiosity, take a seat to reflect, and adopt an attitude of humility.

Within this framework, then, HBA as a divine creative good warrants reflection. Inbuilt aging processes, and their intricacy, evoke astonishment. The aging process is inseparably intertwined with human biological life. This indivisibility is, quite reasonably, astounding, perhaps even perplexing. There is room for reverence here.

A considered humility is also appropriate. First, perhaps surprisingly, the various orchestrated intrinsic molecular processes move humans toward biological instability. Humans seek life, health, and growth, yet this article argues that humans cannot achieve these fine goals without also aging. Second, the observed is further witness to how humans share biological solidarity with other creatures. Just as many animals age and die through intrinsic coordinated processes, so do humans, as exemplified by the programmed cell death process of apoptosis that occurs in a vast range of organisms from insects to mammals (Staley, Blaschke, and Chun 1997) and in modified form even in plants (Wang et al. 1996; Dickman et al. 2017). Moreover, unlike humans, some species of metazoa negligibly age. Aside from the obvious problems this creates for Christian theology’s cosmic fall theory, we must ask: How can humanity, the “pinnacle of creation,” age and die faster than a lobster? The fact is that within the broad range of aging rates in the animal kingdom, humans fall somewhere—without fanfare—in the middle, which rather dilutes the perspective of an innate biological superiority of humans over other lifeforms and steers us away from potential idolatrous anthropocentrism.

Christian Theology Challenges Transhumanist Goals to Overcome Aging

Within Desmond’s framework is a valuable assessment of the effect science’s contraction of wonder to mere curiosity has on the nature of gaining new knowledge. He notes: “[I]here are ways of questioning that lack reverence for the thing questioned” (Desmond 2023, 37). The “idolatrous knowing” of the scientific enterprise is observed in how science perceives the knowledge of human aging and what questions it asks of aging. To ask such questions as “How can we understand aging to master it, to stop or reverse it, perceiving it as defect or disease?” may well lack that reverence, belonging to that “impious” category of question.

These perceptions are found among prominent scientists in the aging field. David Gems summarizes the 2010 meeting of the Royal Society of London for Improving Natural Knowledge in his 2011 paper, writing for the nineteen scientists of that meeting. Here he promotes aging as a disease (Gems 2011): “The evolutionary theory provides the bleak insight that aging serves no purpose in terms of fitness, but instead is a lethal genetic disease that afflicts all human beings.” Likewise, David Sinclair, in *Lifespan: Why We Age and Why We Don't Have To*, promotes the perspective that aging is a disease and that therefore aging and death are to be overcome (Sinclair 2019, xvii, xiv, xxi, 67, 81, 83). More generally, scientific efforts have been underway for some decades to reverse aging (Fossel 2015, 187–202).

Contrary to these perspectives, on the basis of the scientific data presented here and its alignment with aging as one of the goods of creation, it is appropriate to seriously question both transhumanist goals of age reversal and cybernetic immortality, and the evaluation of human biological aging as a disease.

The Molecular Biology of Human Biological Aging Challenges Theology's Cosmic Fall Theory

The inseparability of aging processes and life, health, and growth processes adds clarity to interpretations of the consequences of humanity's fall. Relevant here is Christian theology's cosmic fall theory, which claims that the disobedience of the first humans not only effected a break in humanity's relationship with God but also directly brought about an intrinsic physical change in all creation, leading to (among other effects) dissolution and degeneration, including aging.

As John Bimson (2006) has noted, this doctrine has a history almost as long as Christianity itself. The theory continues and develops through the centuries with such influential and prolific thinkers as the post-Nicene John Chrysostom (349–407AD; Chrysostom 1979, 444), John Calvin (1965, 119, 174; 1960a, 173–74), and several influential contemporary biblical theologians (Cassuto 1961, 163–69; Barth 1968, 29, 308–9; Mathews 2002, 249ff.; Murray 1968, 302–4; Godet 1977, 314; Beker 1980, 149, 232; Bruce 1985, 170; Ziesler 1989, 219–21; Fitzmyer 1993, 505, 507, 509; Moo 1991, 554; 1996, 517; Kruse 2012, 347–48; Merrill 2016). Calvin, for example, speaks of the world's “degeneration,” “corruption,” and “wretched defilement,” Barth of the “present misery” of every “particle of the world,” Frederic Louis Godet of the “sombre hue” of nature, and Joseph A. Fitzmyer of creation's present “lack of beauty, vitality, and strength.” Thus, this theory is highly influential and still a force to be reckoned with in traditional Christian theology.

Biblical commentators mentioned here as well as Augustine, Aquinas, Pannenberg, Rahner, and Zizioulas noted earlier—all effectively championing aging as an effect of the fall—may well find the molecular data astonishingly, perhaps perplexedly, unexpected because that data, from a theological standpoint, disputes the concept of HBA as an effect of sin.

The Molecular Biology of Human Biological Aging Challenges the Doctrine of Physical Death as the Consequence of Humanity's Fall

As noted earlier, the accepted relationship is that aging precipitates natural physical death. Yet, the traditional position within much of Western theology forges a strong causal link between humanity's fall and human physical death: sin leads to natural physical death. Louis Berkhof (1996, 258–61, 669–70) goes so far as to say, “The position of the Church has always been that death in the full sense of the word, including physical death, is not only the consequence but the penalty of sin.” Is human natural physical death the consequence of humanity's fall or, in stark contrast, a part of God's good creation? This is a fundamental yet problematic and troubling question; Barth (1958a, 593) calls its resolution “extraordinarily difficult.”

A few contemporary examples within theology illustrate the challenge. In his interpretations of Paul the apocalypticist, J. Christiaan Beker supports physical death and a cosmic fall as consequences of humanity's fall but at the same time highlights an important problem: in Pauline theology, in Christ, sin has been overcome, but death remains. “Paul's apocalyptic thinking connects death so closely with sin that cosmic death is the inevitable result of sin.” Yet, Beker goes on: “Death executes its reign over the created order like an infectious disease even after its ally, sin, has been defeated by Christ. The relation of suffering and finitude to sin and death remains unclear. There seems to be a residue of death in the created order that is not directly related to sin” (Zizioulas 2008, 101). This article argues that natural physical death—as the precipitant of aging—is more reasonably explained thus: (a) while agreeing with Beker that natural death is “not directly related to sin”; yet, (b) contra Beker, there is no need to conceptualize physical death as a problematic “residue;” rather, natural physical death, as the precipitant of a creationally good aging, actually resides within God's good creation (Beker 1980, 215, 222).

In another example, Zizioulas draws a strong line between, on the one hand, unlimited life and God, and on the other hand, finitude, dissolution, and physical death. It could be construed from statements as such as “Biology sees death as a process that begins at birth, and links aging to reproduction. The mystery of life and phenomenon of death are bound together for life bears death within itself” (Zizioulas 2008, 98) that aging is claimed as a normal and good part of creation. This would be an incorrect assessment, however, for Zizioulas (2008, 98–101) goes on to claim that aging, dissolution, and physical death are direct consequences of the fall: “This counterfeit life . . . which carries death within it, is the outcome of the fall. It is a poor, evil and intolerable form of life. The Christian view is that death is never good, it is always an outrage.” The work of this article is contra Zizioulas: from the biology of the natural human being, one cannot separate molecular processes of aging from those of life. Against Zizioulas (2008, 101), who says that “related to God, creation would have life without limit,” limit is inseparably intertwined with this good creation.

These examples briefly highlight the struggle to get the link right between natural physical death and the fall. But on a more constructive note, one should probe whether support for the central claim of this article can be found in alternative avenues of investigation. In fact, support comes from exegesis of the Eden story (Genesis 2:4b–3:24), to which we now briefly turn.

Two foci are significant in the construction of further support¹: the meaning of YHWH's warning of the death sentence in Genesis 2:16–17 and the Yahwist source's transformation of ancient Near Eastern (ANE) motifs for life, death, and the tree of life. Adhering to the principle that literary structure communicates meaning, Jerome Walsh's synchronic exegesis reveals the Eden story to be a play of seven scenes. The strong chiasmic structure of this text is built from both form (Walsh 1977) and antiparallel themes (Hooker 2021, 324–50). The play presents aetiology symbolically (symbolism here is an artful means of expressing realities; that is, the fall is historical). The chiasm places the death threat of 2:17 in scene one, with the eating of the forbidden fruit forming the central scene four. There is wide agreement that, in Nils Lund's words, "identical ideas are distributed across the passage *at the extremes and centre*" (Lund 1942, emphasis added; Blomberg 1989; Thomson 1995); thus, scenes one and seven with the central scene four. Significant for our discussions, the chiasm informs the meaning of death in scene one's death threat as follows: death is tied explicitly to the eating in scene one and implicitly (via scene one's threat) to the eating in the central scene four. Yet, it must be asked: Is there an antiparallel thematic counterpart in scene seven for the theme of death in scene one? Affirmatively, "the sentence of death threatened" in scene one has its counterpart as "the sentence of death realized" in scene seven. Scene seven's fulfillment of the death threatened in scene one is the expulsion from Eden, complemented by its out-workings predicted in scene six, a concept that excludes physical death. The exclusion of physical death from the death threat, then, argues for a demarcation between natural physical death and sin's consequence, and for a creationally "good" pre-fall human mortality.

Semantic, literary, and historical considerations augment such a conclusion (Hooker 2021, 340–47). Of particular note is the impact from narrative tension. As a classic case of prolepsis, scene four's 2:16–17 is extremely important for the movement of the narrative. The reader is guided to ask, and awaits answers to, two questions: "Will humans eat the fruit?" and "What will happen when they eat the fruit?" The answer to the first question comes at scene four; however, where is the answer to the second question? On the basis of proleptic fulfillment of narrative tension, it is problematic to contend that the death in 2:16–17 is physical death. Then there is no fulfillment in the Eden story, and one must wait several hundred years for Adam's unremarkable, anticlimactic death—not in the Yahwist but in the Priestly account. It makes much more literary sense to interpret death's threatened sentence as fulfilled at scene one's mirrored scene one, yielding a resolution internal to the story.

The perspective that the Eden story is polemic against ANE concepts of life and death adds to these considerations. The Eden story takes several themes from ANE myths and refutes or transforms them. In particular, negating what is virtually a maxim among ANE tales that eternal life is the prerogative only of the gods, in the Eden story, it is YHWH's intention for humanity to be gifted life from the tree of life. Humanity dwells in the heavenly abode with God, and with uninhibited access to this tree, of which, in the strong case made by Herman Obbink (1928), Adam and Eve freely eat before the fall. However, within the sanctuary symbolism of the narrative, elaborated by Gordon Wenham (1994), "life" is transformed from the everlasting physical immortality of ANE tales to definitively one of life with God in the sanctuary, a life of intimate friendship. Bearing this transformation of "life" in mind, it is reasonable to venture that the Eden story has also transformed the typical ANE concept of "death" as physical death into a spiritual death. Death's meaning is the very component of the story that is deliberately held over in tension, awaiting explanation in scene seven and supplying an atypical answer to the question, "What does death, the penalty for sin, look like?"

Can These Molecular Biological Investigations Offer Improved Consonance between Christian Theology and Evolutionary Biology?

Creaturely Aging and the Problems of Cosmic Suffering and Natural Selection

Away from exegetical precision, we are drawn to the sweeping panorama of an evolving cosmos and its joining of humans with all creatures. As Peters (2010, 929) assesses, even more challenging for the theistic evolutionist than Genesis interpretation is what he calls the "entangled knot" of the problem of suffering, including the raw, unfeeling process of natural selection. Here, he concurs with Philip Hefner when Hefner (1993, 42–43) states, "nowhere do the problems of theodicy bear down with greater weight and urgency than in the consideration of the processes of natural selection."

Could the phenomenon of aging be a component of natural selection? Quite possibly. Aging may be included because the overarching reality here is one of vulnerability—vulnerability plays out in all of predation and extinction, and fundamentally so in aging. Predation may be fast and aging slow, yet predation is random and aging inescapable. Aging lessens fitness, cuts off potential, diminishes strength, elevates degradation, enhances vulnerability to physical failure and disease, and even creates death.

Aging, then, would appear to serve natural selection. Even if it did not, it can comfortably be placed within the broader phenomenon of cosmic suffering and disintegration. But in doing so, we have here a meeting of two apparently disparate realities: the randomness, apparent meaninglessness, and cruelty of suffering and natural selection with the control, direction, ordered programming, and functional systematization of the mechanisms of aging. This is worth exploring further.

“Free Process” or “Only Way” Arguments for Life’s Ambiguity?

Theodicy’s grappling with creaturely suffering opens up the concept of life’s ambiguity. Life is “shot through with ambiguity” (Southgate 2008a, 71), an expression that describes the presence of the natural world’s good *and* evil: fulfillment with lack of fulfillment, integration with disintegration, life with destruction. Resolution for this theological problem comes in two forms. Originally expounded by John Polkinghorne (1989, 66–68) and cultivated by others including Hefner (1993, 117), the first is the “free process” defense: creaturely freedom requires kenosis, God’s self-removal, whereby “creation is free to create itself in a continuing process of self-alteration” (Peters 2018, 696). The second, the “only way” argument, posits that God is not removed but constrained to a certain way that melds the negatives of life with the positives. Bringing the claim of this article into the equation, then, argues against the first defense: one could well say that, by the creationally good mechanisms of aging, the steady, inexorable tapping of the hammer of aging is God’s inescapable tool to enact this divine work. God is not removed but very much present. This standpoint also backs up Robert John Russell’s (2008, 214) concept of the providence of God: “Essentially what science describes without reference to God is precisely what God, working invisibly in, with, and through the processes of nature, is accomplishing.”

Nevertheless, the claim of this article could nuance some arguments for the second defense. In Nancey Murphy’s (2007, 140) version of the only way defense, she writes that creaturely limitations are, as constraints within natural evil, “unavoidable by-products of choices God had to make.” Yet, the mechanisms of aging investigated here speak more clearly of deliberate divine intent than divine restriction.

Aging, Deep Incarnation, and the Theology of the Cross

To further integrate divine presence and action with the problem of evolutionary suffering, Peters is one among several interpreters in the theology–science space who advocate an interpretation of the natural world through the cross (for multi-author dialogue with Denis Edwards, a major exponent of deep incarnation, see Peters and Marie Turner [2020]). Peters (2010, 930): “We look at the cross whereon a dying man suffers, and we see the God of life and healing.” The cross’s second manifestation conveys to us that the healing, life-giving God is also the suffering God, meeting and suffering with all creation. Yet, from the investigations of this article, there is a complementary way in which theistic evolution can meld the concepts of a good creation—concepts reflective of the character of God—with the phenomena of suffering and disintegration. The aging process becomes a mirror. Just as the healing and power of the divine life merge with struggle and suffering in the nature of God, so too is this seen in aging: life processes merge with processes that are the very source of weakness,

even death. Though not explicitly noting aging, Ernst Conradie's (2020, 48–49) remark is particularly apt here: "Godself becomes incarnate in the very tissue of biological existence."

Undoubtedly, the absorbing theology–evolutionary biology discussion warrants much more investigation than this article can provide. The answer to this section's question, a question of consonance, appears to be that the investigations here do move towards improved consonance between theology and evolutionary biology. But, indeed, it exposes further challenges. One of them is the concept of "natural evil" in light of this article's claims. To define the sufferings among nonhuman and human creatures alike as natural evil is common theological parlance (see, for example, Southgate 2008b; Peters 2018). Yet, if aging is a creational good, I would venture that this warrants a reappraisal of what theology designates as evil. Another challenge is, of course, one of eschatology: how can we integrate creationally good aging processes with theology's vision of a new creation devoid of loss, pain, and suffering? Aptly, Peters (2018, 708) remarks, "Theodicy must find an eschatological, not a logical, resolution."

A Theology–Molecular Biology Integration for Aging Supports a Holistic View of Human Life through the Passage of Time

Moving very briefly to a psychosocial perspective, contemporary Western society individualizes, atomizes, and fragments human life. Life is defined as stages: young, middle, and old are the obvious. But our fragmentation goes much further; even prenatal life has fetal stages that serve to validate when or when not to abort. This defined fragmentation shapes our behavior toward others. Aged care medicine is not immune to this, as physician Karen Hitchcock (2015, 132–33) observes: "Medicine takes the ill and chops them up as if a person is a walking textbook of distinct chapters: lung, heart, liver, brain . . . cigarettes smoked, alcohol consumed, blood sugar level." She goes on to rightly criticize the prejudiced fragmentation of hospital care into care for the young above care for the old; ageism asserts itself in the lesser priority given to the sick elderly because "they have had their turn."

However, if aging is intertwined with life processes, then the stage of life we call elderly cannot be—must not be—separated from other stages of life we may popularly think of as "good." Thus, we would speak of the whole person through the whole of his or her time, a perspective that has the potential to transform aged care medicine. Simone de Beauvoir (1996) echoes this thought of continuity when she says, "What should a society be, so that in his last years a man might still be a man? The answer is simply: he would always have to have been treated as a man" (quoted in Hitchcock 2015, 131). The creational goodness of human aging provides the foundation upon which de Beauvoir's answer of continuity can rest.

The work of this article informs pastoral theology's response to the models of human limitation, such as medical models of disability, D. Creamer's "limits" model of disability, and societal models of normalcy. The inseparability of aging processes from life processes, and the positioning of aging as a good of creation, helps correct what Michael Mawson (2023), I think rightly, sees as society's and even medicine's "misguided" perception of aging as a "problem" and shifts it back into the category of "normal" human life.

Conclusion

In its theology of aging, Christian theology needs internal cohesion and a consistent response to contemporary negative societal mindsets on the aged and aging. Unfortunately, these needs are hindered by the three problems of a distinct lack of development in a foundational theology of aging, an adherence to two antithetical claims regarding the origin of aging, and a lack of engagement with the rapid progress in the science of aging. This article has sought to contribute to engagement and resolution in these areas by integrating Christian theology and molecular biology. Critical distinctions have been highlighted in order to clarify the complexities of aging. Aging generating lifespan (HBA) has been distinguished from aging leading to life expectancy. Intrinsic molecular pathways have been explained that drive HBA; these processes are orchestrated, functional systems and occur across multiple biological strata. Significantly, aging processes are seen as indivisible from life, health, and growth processes. These considerations build a case for understanding HBA as a good of divine creation, add clarity to interpretations of the consequences of humanity's fall, steer efforts away from transhumanist goals of age reversal and cybernetic immortality, inform theodicy's study of cosmic suffering, and drive a holistic view of human life through the passage of time.

The core idea of human aging as a good of creation is not comprehensively served by this work; there are omissions persuasive of the need for a more considerable science–theology synthesis. A biblical theology of aging is warranted and more needs to be said to bring the limitations, losses, and sufferings of aging into a pastoral goodness of aging. Perhaps critically, this article has not engaged with aging that leads to life expectancy. This is an omission that may seem surprising considering life expectancy and not lifespan is the almost universally existential fact for human beings. It has been a deliberate step to focus on the intrinsic factors driving human biological aging in this article. Yet, there are extrinsic factors modulating aging rates, and these factors must be considered when examining life expectancy. The question must be faced as to whether, and how, these factors can integrate into a theology of aging and theology's doctrine of sin. The complexity of this field, including the significance of epigenetics, means this must be the task of further investigation.

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Note

- ¹ From Herman Obbink's (1928) robust argument, the phrase "lest he . . . take also of the tree of life and eat and live forever—" (Genesis 3:22) is best not used to indicate a pre-fall mortality.

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