Science Prism

Illuminating Science, Empowering Minds

Issue 18
7 May 2026.

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Welcome to *Science Prism* — your gateway to exploring the brilliance of science and discovery. Like a prism dispersing light into a spectrum of colours, we distil complex scientific ideas into clear, engaging, and thought-provoking insights. Delivered monthly, *Science Prism* is designed for students, researchers, and curious minds, bringing you the latest breakthroughs, timeless questions, and actionable ideas to fuel your curiosity and drive innovation. Guided by our tagline, "*Illuminating Science, Empowering Minds,*" we aim to inspire fresh perspectives and connect the transformative power of science with the world around us. Join us each month as we explore knowledge, spark curiosity, and celebrate discovery, one insight at a time.

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Audio Summary Of The Entire Issue

In this Issue

Welcome to the May 2026 Issue of *Science Prism*

If April explored how science advances through dialogue and shared experience, this issue turns inward towards one of biology's most fundamental questions: how living systems maintain balance, repair damage, and gradually change over time.

This issue opens with Ageing: When Cells Lose Precision, an exploration of ageing as a gradual loss of biological accuracy that begins silently within cells long before visible changes appear. Moving through DNA repair, cellular senescence, inflammation, stress biology, mitochondrial decline, and epigenetics, the article presents ageing as an interconnected process shaped by both cellular mechanisms and environmental influences.

In *Science Spectrum*, Dr Malini Srivastava continues the Changing the Narrative on Suicide series with an important reflection on lived experience in suicide prevention. The article highlights why listening to survivors is essential for building compassionate, human-centred approaches to mental health care.

This month's *Science Shapers* section features *Dr Yoshinori Ohsumi*, recipient of the *2016 Nobel Prize in Physiology or Medicine*, whose pioneering discoveries on autophagy transformed our understanding of cellular maintenance, stress adaptation, and ageing biology.

We are also delighted to announce that *Science Prism* now has a dedicated website, which will serve as the new home of the newsletter. Readers can now access both current and previous issues through the platform, creating a growing archive of articles and scientific discussions.

Together, this issue reflects a shared theme: resilience, whether in cells or in people, depends on the ability to repair, adapt, and remain connected through change.

With curiosity and continuity,
The *Science Prism* Team.

***Science Prism* Current Issue:**
https://sciencearc.org/science-prism/

AGEING: WHEN CELLS LOSE PRECISION
A Journey from Molecular Signals to the Fate of the Human Body

Ageing is not merely a visible change but a gradual loss of cellular precision. It involves subtle molecular changes that often go unnoticed for years but collectively influence the health and function of the entire body. By linking processes such as damage, repair, signalling, and cellular behaviour, it portrays ageing as an interconnected biological journey rather than a single cause. Most importantly, it encourages us to rethink ageing not as an inevitable decline alone, but as a process that we can better understand and potentially influence.

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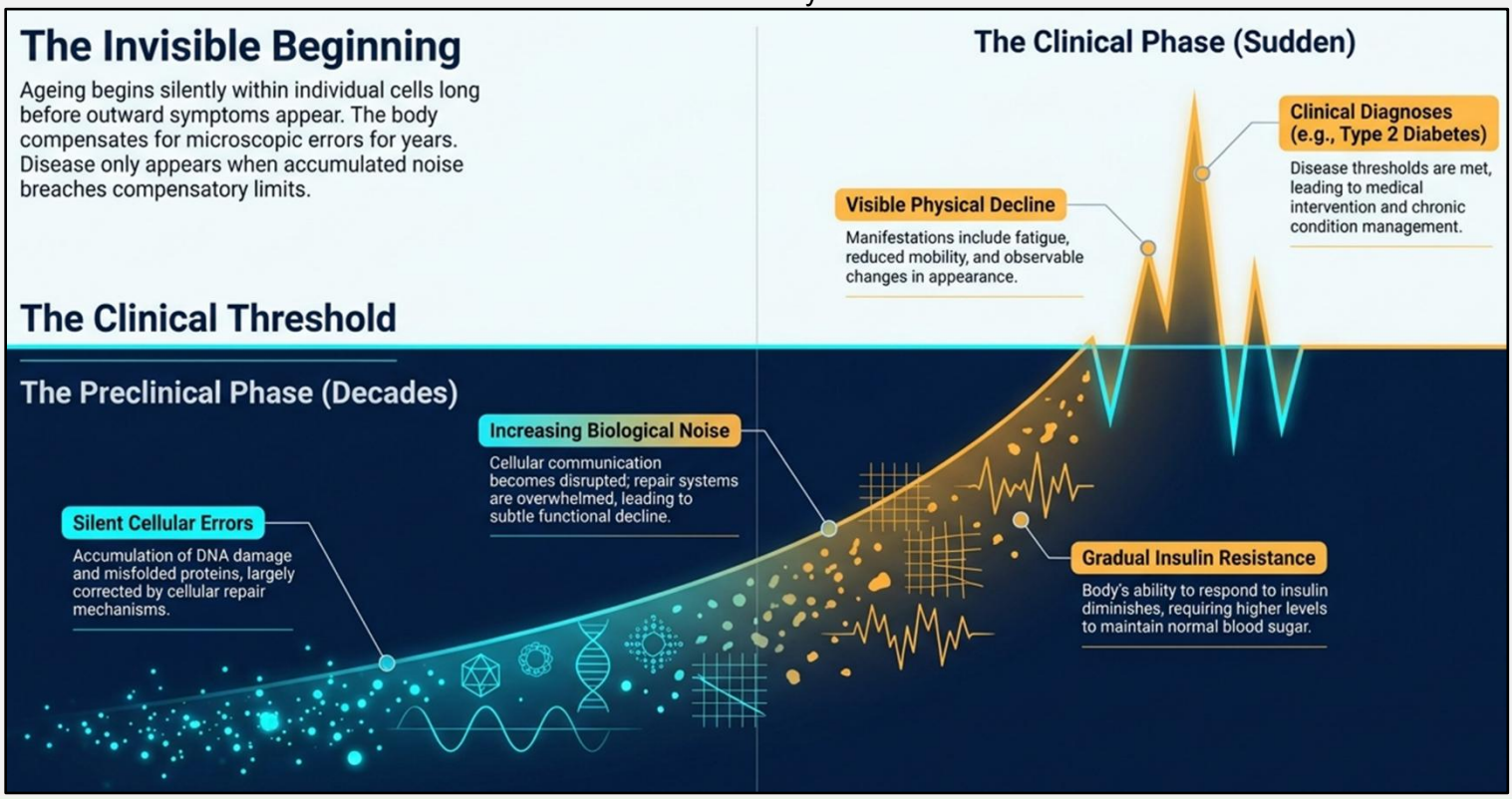
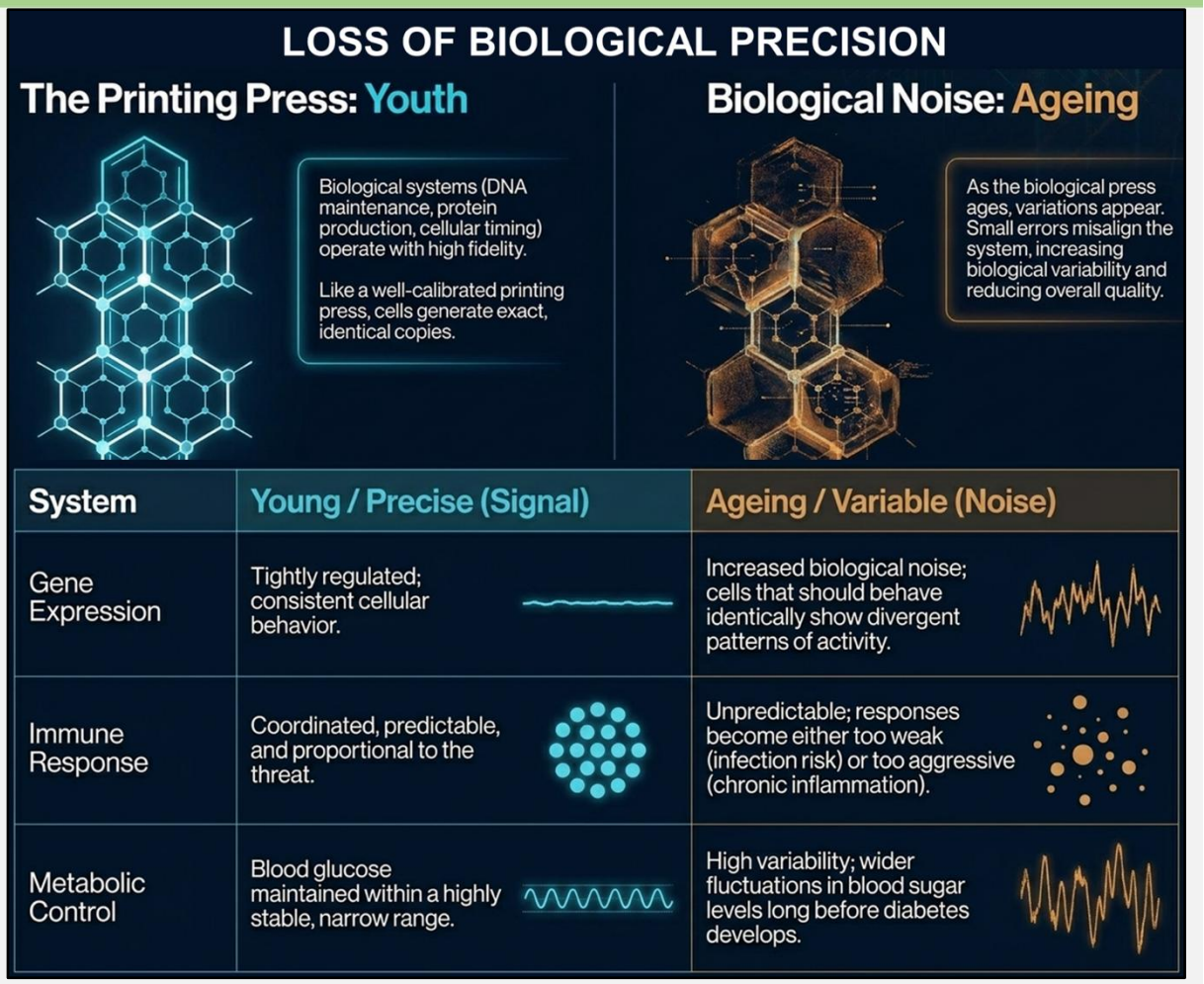
The Invisible Beginning of Ageing. Most people recognise ageing only when it becomes visible. A person may notice that they tire more easily, recover more slowly from illness, or observe changes in skin or hair. These visible signs are often treated as the beginning of the ageing process. However, scientific evidence shows that ageing begins much earlier, long before any outward changes appear. Ageing begins at a microscopic level, within individual cells. It is not a sudden transition but a gradual process that unfolds over time. Researchers studying ageing in humans and model organisms consistently observe that biological changes begin silently and accumulate long before clinical symptoms develop. Cells in our body are constantly active. They divide, repair damage, respond to signals, and maintain internal balance. Each of these processes requires high accuracy, which is maintained effectively in early life.

The body can often compensate for minor changes, with some processes adjusting when others become less accurate, helping to preserve overall function, which is why ageing remains unnoticed for many years. But this compensation has its limits; as more changes build up, the system struggles to maintain harmony. Eventually, these accumulated changes lead to noticeable differences. Biological alterations start well before clinical symptoms emerge. Therefore, the silent beginning of ageing is a crucial phase, during which small changes build up and influence future outcomes. Recognising this phase shifts the focus from visible ageing to understanding the underlying processes, emphasising the importance of studying cellular function over time rather than waiting for symptoms to appear.

Biological Precision: The Foundation of Health. To understand ageing, it is essential to understand what is being lost. The key concept and foundation of health is biological precision, the ability of cells and systems to perform their functions accurately, consistently, and in coordination. Imagine a coordinated team managing a complex task in which every member must act correctly and on time. Even minor errors or loss of coordination can affect overall performance. Similarly, in the body, cells function as part of a coordinated system rather than as isolated units. Each cell



communicates with others via chemical signals, responds to environmental changes, and contributes to the functioning of tissues and organs. For this system to work, several processes must occur with precision. One important process is DNA maintenance. DNA contains the instructions for cellular function, and cells must copy and maintain this information accurately. Another critical process is protein production. Proteins carry out most cellular functions, and they must be produced with the correct structure and in the correct amounts, since errors in protein structure or production can lead to cellular dysfunction. Cells also rely on highly regulated signalling pathways to communicate via a network of hormones, neurotransmitters, and other molecules, guiding cellular behaviour, and in young and healthy systems, responses to signals are predictable and proportional.



Precision also involves timing. Many biological processes follow rhythmic patterns. For example, hormone levels fluctuate throughout the day, producing rhythms that ensure that processes occur at the appropriate time. Health can therefore be understood as a state in which these processes are maintained with high precision. Evidence from clinical studies supports this view. In healthy individuals, physiological parameters such as blood glucose levels, body temperature, and hormone levels are maintained within a narrow range. This stability reflects precise regulation. When precision is lost, variability increases, as often observed in ageing and disease. This increased variability is associated with a higher risk of cardiovascular disease.

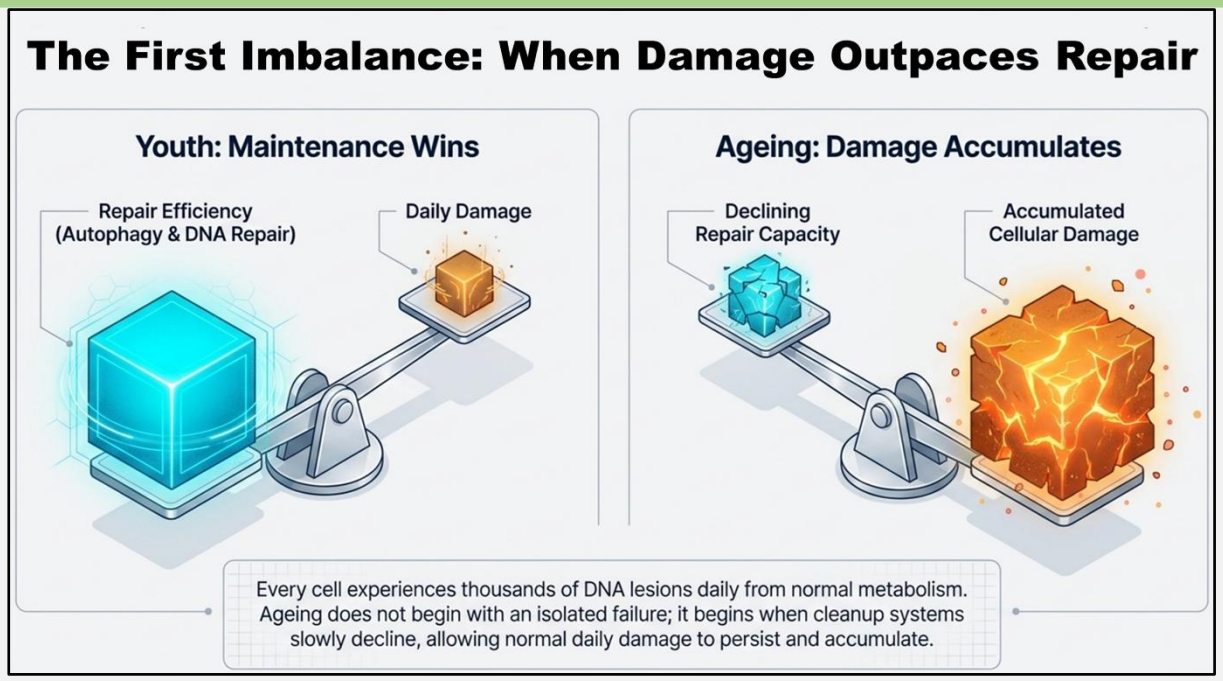
Another example comes from the immune system. In younger individuals, immune responses are well coordinated. With age, responses may become less predictable. Some responses may be weaker, increasing susceptibility to infection. Others may be excessive, contributing to inflammation. These observations suggest that loss of precision is a key feature of ageing.

At the cellular level, this loss of precision can be understood as increased variability and reduced reliability. Processes that were once tightly controlled become more variable. A helpful analogy is that of a printing press. In a well-maintained press, each copy is identical. If the machine becomes less precise, variations begin to appear. Some copies may have errors, others may be slightly misaligned. The overall quality declines.

In the body, cells function like such a system. When precision is maintained, function is stable. When precision declines, variability increases. It is important to note that biological systems are designed to tolerate some level of variability. Small variations do not necessarily lead to dysfunction. However, when variability increases beyond a certain point, it begins to affect overall function. Experimental evidence supports this concept. Studies in ageing biology have shown that increased variability in gene expression, protein levels, and cellular responses is a consistent feature of ageing across different organisms. This suggests that maintaining precision is essential for long-term health.

Damage and Repair: The First Imbalance. One of the most fundamental processes underlying ageing is the balance between damage and repair. Damage is a constant feature of life. Every cell in the body

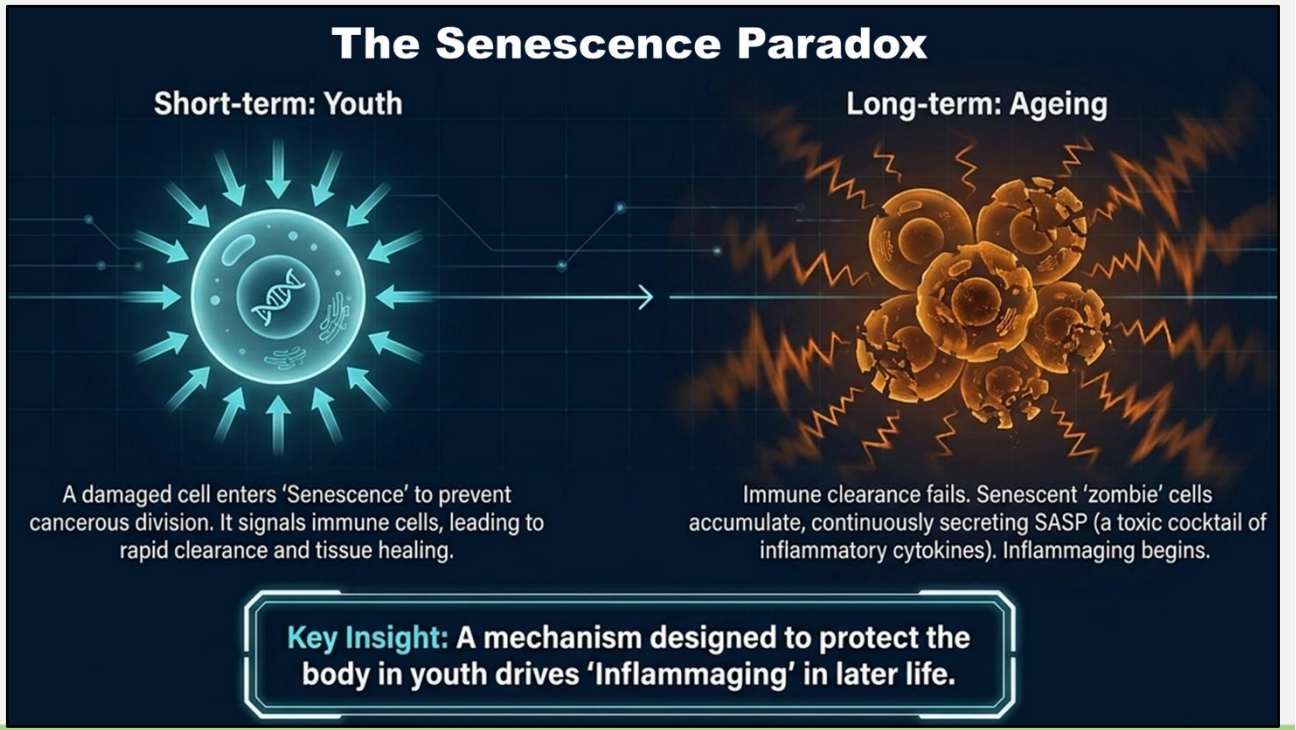
experiences damage continuously, which arises from both normal metabolic processes and external factors. For example, during energy production, cells generate reactive oxygen species that can damage DNA, proteins, and lipids. Environmental factors such as ultraviolet radiation and pollutants also contribute to cellular damage.



Experimental studies have shown that DNA damage occurs frequently in cells, with estimates suggesting that each cell experiences thousands of DNA lesions per day. Despite this, cells continue to function because they possess efficient repair mechanisms that detect and correct damage. Research has demonstrated that defects in DNA repair lead to accelerated ageing and increased disease risk. For example, individuals with genetic defects in DNA repair pathways often develop conditions characterised by early-onset features of ageing. This provides strong evidence that repair mechanisms are essential for maintaining cellular function. In addition to DNA repair, cells have systems to remove damaged proteins, where misfolded or damaged proteins are degraded and replaced, thus preventing the accumulation of dysfunctional components.

Autophagy is a vital repair process that removes and recycles damaged cellular structures, such as mitochondria. It maintains cellular health, especially in young cells where repair systems detect and fix damage efficiently. With age, this balance shifts as repair efficiency declines gradually, leading to persistent damage and accumulation of damaged molecules. Although minor damage may not cause immediate issues, over time, it can lead to a significant decline. This damage-repair imbalance is a key early step in ageing, impacting cell signalling and function. Ageing results from continuous, gradual processes, with the balance between damage and repair central to maintaining health. When balanced, function is preserved; when shifted, ageing begins.

Cellular Senescence: Protection That Becomes Persistent. One of the most important discoveries in modern ageing biology is the concept of cellular senescence. Cells divide to support growth, repair, and renewal, but they do not do so endlessly. Under specific conditions, a cell can enter a state called cellular senescence, where it permanently stops

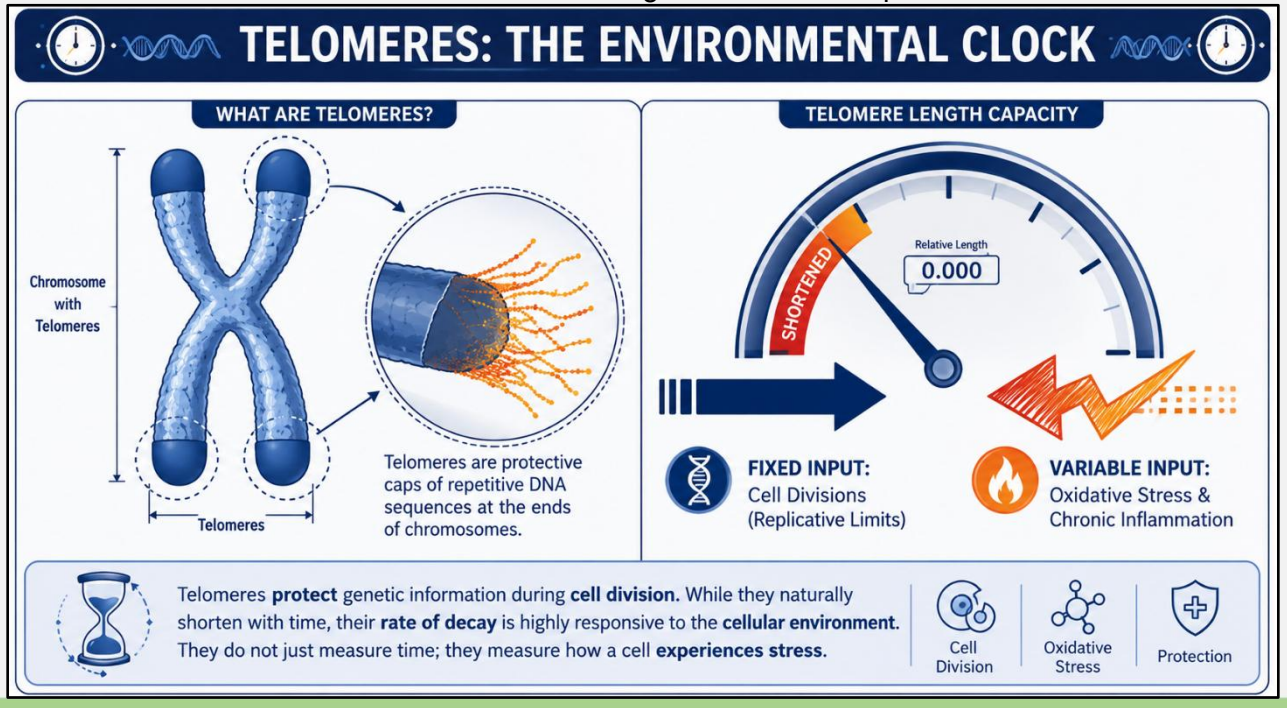


dividing. Cells enter senescence in response to different types of stress, with the most well-studied trigger being DNA damage. When DNA is damaged beyond a certain threshold, the cell activates internal pathways that halt division. This prevents the damaged DNA from being passed on to daughter cells. In this sense, senescence acts as a safety mechanism. Another trigger is telomere shortening. As telomeres become critically short, cells interpret this as a signal of instability and stop dividing. This is often referred to as replicative senescence.

Experimental evidence for senescence comes from early studies of human cells grown in culture. Researchers observed that normal human cells divide a limited number of times before entering a non-dividing state. This phenomenon, known as the Hayflick limit, was one of the first indications that cells have built-in mechanisms to limit division. Further research has shown that senescence is not simply a passive state. Senescent cells remain metabolically active. They undergo significant changes in gene expression and begin to secrete a wide range of signalling molecules. This collection of secreted factors is known as the senescence-associated secretory phenotype, or SASP. SASP includes molecules such as inflammatory cytokines, chemokines, growth factors, and enzymes that modify the surrounding tissue. These molecules influence nearby cells and the local environment. In the short term, this response can be beneficial. For example, during wound healing, senescent cells can help recruit immune cells to the injury site. These immune cells remove damaged cells and support tissue repair. In this context, senescence contributes to recovery.

Similarly, senescence plays a role in preventing cancer. Stopping the division of cells with damaged DNA reduces the likelihood of uncontrolled growth. However, the situation changes over time. As organisms age, senescent cells begin to accumulate. This accumulation occurs because the immune system, which normally clears senescent cells, becomes less efficient with age. As a result, senescent cells persist in tissues. When senescent cells are present in small numbers, their effects are limited. When they accumulate, their continuous secretion of SASP factors creates a persistent inflammatory environment. This chronic, low-level inflammation is often referred to as inflammaging. It is not the same as acute inflammation, which occurs in response to injury or infection. Instead, it is a subtle and ongoing state that affects tissue function over time. Experimental studies in animals have provided strong evidence for the role of senescent cells in ageing. In mouse models, researchers have developed methods to selectively remove senescent cells. These studies have shown that removing senescent cells can improve physical function, reduce inflammation, and delay the onset of age-related conditions. These findings suggest that senescence has a dual role. In early life, it protects the organism by preventing the spread of damaged cells and supporting repair. In later life, its accumulation contributes to dysfunction. A useful analogy is that of a safety system in a building. Imagine a system designed to shut down a machine when it detects a fault. In the short term, this prevents damage. However, if too many machines are shut down and not repaired or removed, the building's overall functioning is affected. Senescent cells function similarly. They prevent immediate harm but create long-term challenges when they accumulate.

Clinical observations also support this concept. Increased markers of senescence have been detected in tissues from older individuals. These markers are associated with inflammation and functional decline. It is important to note that senescence does not act alone. It interacts with other processes such as DNA damage and inflammation. These interactions amplify its effects. Understanding senescence helps explain why ageing is not simply a process of loss but also one of altered function. Cells do not just stop working.



They change their behaviour, and these changes influence the surrounding environment. In summary, cellular senescence is a protective mechanism that becomes persistent over time. Its short-term benefits are clear, but its long-term accumulation contributes to chronic inflammation and tissue dysfunction.

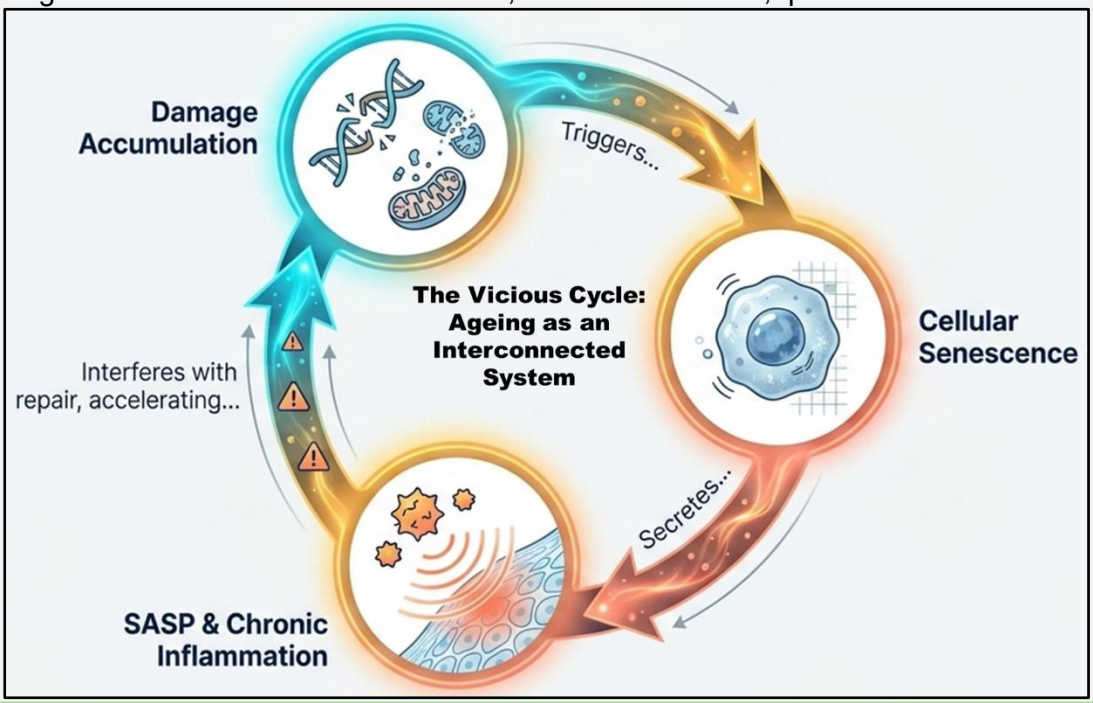
Telomeres: Replicative Limits and Environmental Influence. Telomeres are among the most widely studied structures in ageing biology and provide a clear example of how cellular processes are linked to both time and environmental factors. Telomeres are repetitive DNA sequences located at the ends of chromosomes. Their primary function is to protect the chromosome during cell division, and without telomeres, important genetic information could be lost during DNA replication. Each time a cell divides, its DNA must be replicated. However, the replication machinery cannot fully copy the very ends of chromosomes. As a result, a small portion of the telomere is lost with each division. Over time, this leads to the gradual shortening of telomeres. When telomeres become critically short, the cell detects this as a signal of instability. In response, it may enter senescence and stop dividing. This mechanism limits the number of times a cell can divide.

The relationship between telomere length and cell division has been demonstrated in laboratory studies. Human cells grown in culture show progressive telomere shortening with each division. When telomeres reach a certain length, the cells stop dividing. This has led to the idea of telomeres as a biological clock. According to this idea, telomere length reflects a cell's replicative history. However, this analogy is incomplete since telomere shortening is influenced not only by the number of cell divisions but also by other factors, including oxidative stress. Reactive molecules produced during metabolism can damage telomeric DNA, accelerating its shortening. Inflammation also plays a role since chronic inflammation increases cellular turnover and stress, both of which can contribute to faster telomere shortening. Clinical studies in humans support these observations. Research has shown that individuals exposed to chronic psychological stress may have shorter telomeres compared to less stressed individuals. For example, studies of caregivers of chronically ill patients have demonstrated associations between stress and reduced telomere length. Other studies have examined the relationship between lifestyle factors and telomere length. Factors such as smoking, obesity, and lack of physical activity have been associated with shorter telomeres. These findings suggest that telomere dynamics are influenced by environmental conditions.

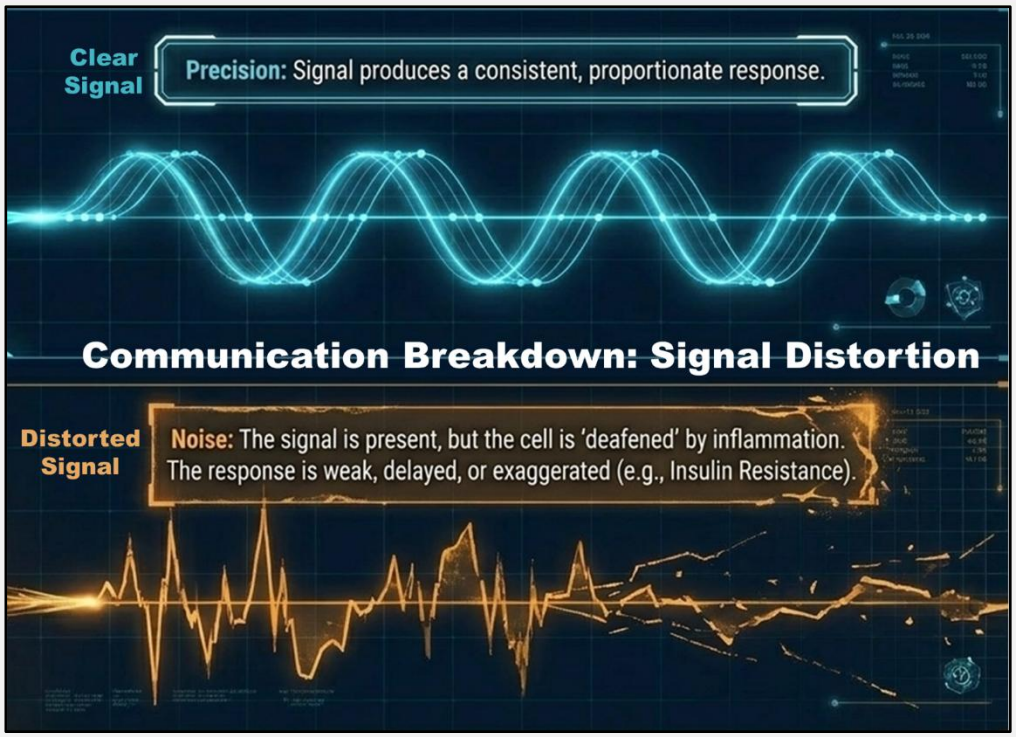
At the same time, it is important to interpret these findings carefully. Telomere length varies between individuals and across different cell types. It is not a single measure that fully determines ageing. A helpful way to understand telomeres is to think of them as protective caps that become shorter each time a cell divides. When it becomes too short, the cell can no longer divide safely. However, the rate at which the cap shortens depends on factors beyond the number of divisions, such as the amount of stress the cell experiences. This means that telomeres reflect both time and environment.

Telomeres also interact with other aspects of ageing. Short telomeres can trigger senescence, which contributes to inflammation. Inflammation, in turn, can accelerate telomere shortening. This creates a cycle in which different processes reinforce each other. In summary, telomeres provide an important link between cellular division, environmental stress, and ageing. They demonstrate that ageing is not determined solely by time but is influenced by how cells experience their environment.

Ageing as an Interconnected System. One of the most important insights from ageing research is that ageing does not arise from a single cause, nor is it driven by a single isolated pathway; rather, it emerges from the interaction of multiple biological processes that influence one another over time. Among the most central of these are damage accumulation, cellular senescence, and inflammation, processes linked in



self-reinforcing cycles that shape the progression of ageing.

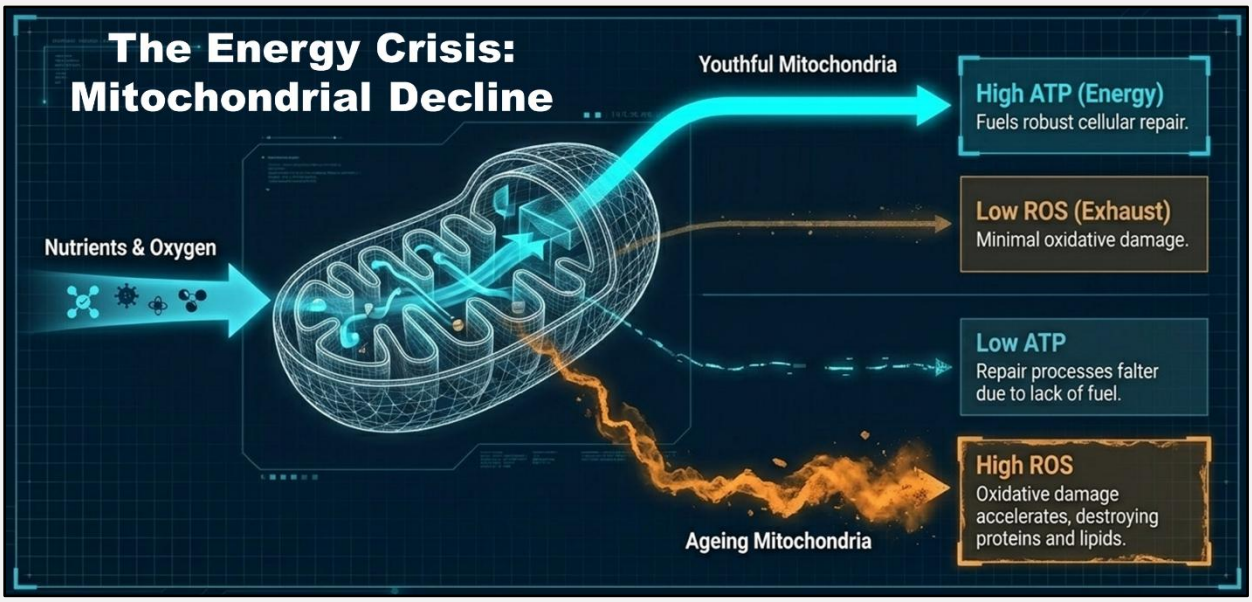


Cells continuously experience damage, including damage to DNA, proteins, and cellular structures. When this damage exceeds a certain threshold, it can trigger cellular senescence. Senescent cells, while no longer dividing, remain active and release a range of inflammatory molecules, altering the surrounding tissue environment and promoting chronic low-grade inflammation. Inflammation, in turn, increases cellular stress, interferes with repair mechanisms, and disrupts signalling pathways, thereby accelerating further damage. This creates a cycle in which damage promotes senescence, senescence promotes inflammation, and inflammation feeds back to increase damage. This interconnected model is supported by experimental and clinical evidence. Ageing tissues often show the simultaneous presence of increased DNA damage, accumulation of senescent cells, and elevated inflammatory markers, all of which are associated with functional decline. In animal studies, interventions that reduce inflammation or selectively remove senescent cells have been shown to improve tissue function and extend healthy lifespan, indicating that modifying one component can influence the entire system. This interconnectedness also helps explain why ageing progresses gradually. Small initial changes do not remain isolated but interact and accumulate, leading to larger functional consequences.

This framework also provides insight into variability in ageing. Different individuals may experience different patterns of interaction among these processes, which can influence how ageing manifests. For example, some individuals may show more pronounced metabolic changes, while others may exhibit greater vulnerability in neural or immune systems. These differences likely reflect variations in the evolution of interconnected pathways over time. Understanding ageing as an interconnected system shifts the focus from individual mechanisms to their relationships. It highlights that ageing is not the result of a single failure but a network of interacting processes that gradually reduce biological stability and function.

Ageing and Cellular Signalling: Key Impacts on Physiological Responses. Ageing brings significant changes in cellular signalling, affecting how cells interpret and respond to signals. In healthy organisms, signals trigger precise cellular responses, but as they age, this precision declines. This leads to unreliable responses, in which the same signal can result in weaker, exaggerated, or delayed actions, as seen in insulin resistance. Here, insulin levels may be normal or elevated, yet cells fail to respond appropriately. This showcases a critical principle of ageing: while signals remain, their meanings can become distorted, indicating a loss of reliability in cellular functioning. Several factors contribute to this decline. The number of receptors may decrease, and intracellular pathways can become less efficient over time. Additionally, increased biological variability or "noise" leads to greater differences in how cells respond, diminishing overall predictability and coherence. Cellular signalling involves complex processes like hormone production and receptor detection. Ageing disrupts these stages, resulting in receptor dysfunction, impaired signalling, and reduced communication due to chronic inflammation. Although cells continue to receive signals, they do so with less precision, contributing to conditions like type 2 diabetes. Feedback mechanisms are crucial for maintaining physiological stability. While these mechanisms don't entirely fail with age, their timing and regulation often become compromised, leading to delayed or inadequate responses. For example, in glucose regulation, ageing can delay insulin release, resulting in prolonged elevated blood glucose levels. Similar declines occur in blood pressure regulation, where the body's ability to respond to changes becomes less effective. Overall, ageing affects both the timing and regulation of feedback loops, resulting in misaligned responses that undermine physiological stability and overall health.

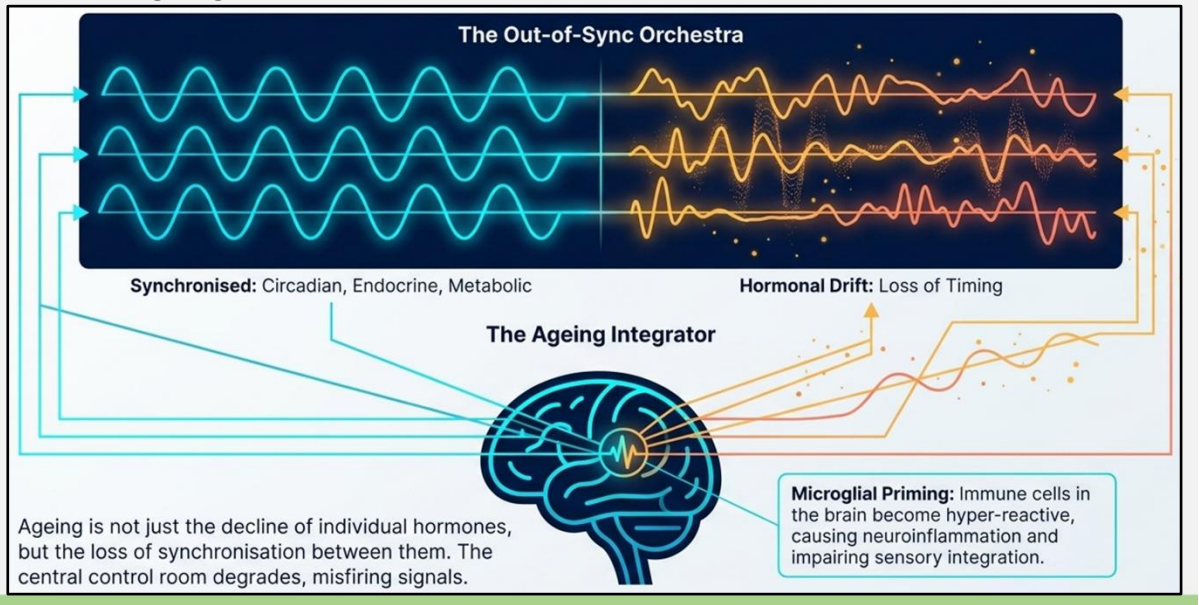
Energy Decline: Mitochondria and Biological Efficiency. Energy is essential for all biological processes, and cells primarily generate it through mitochondria, known as the powerhouses of the cell.

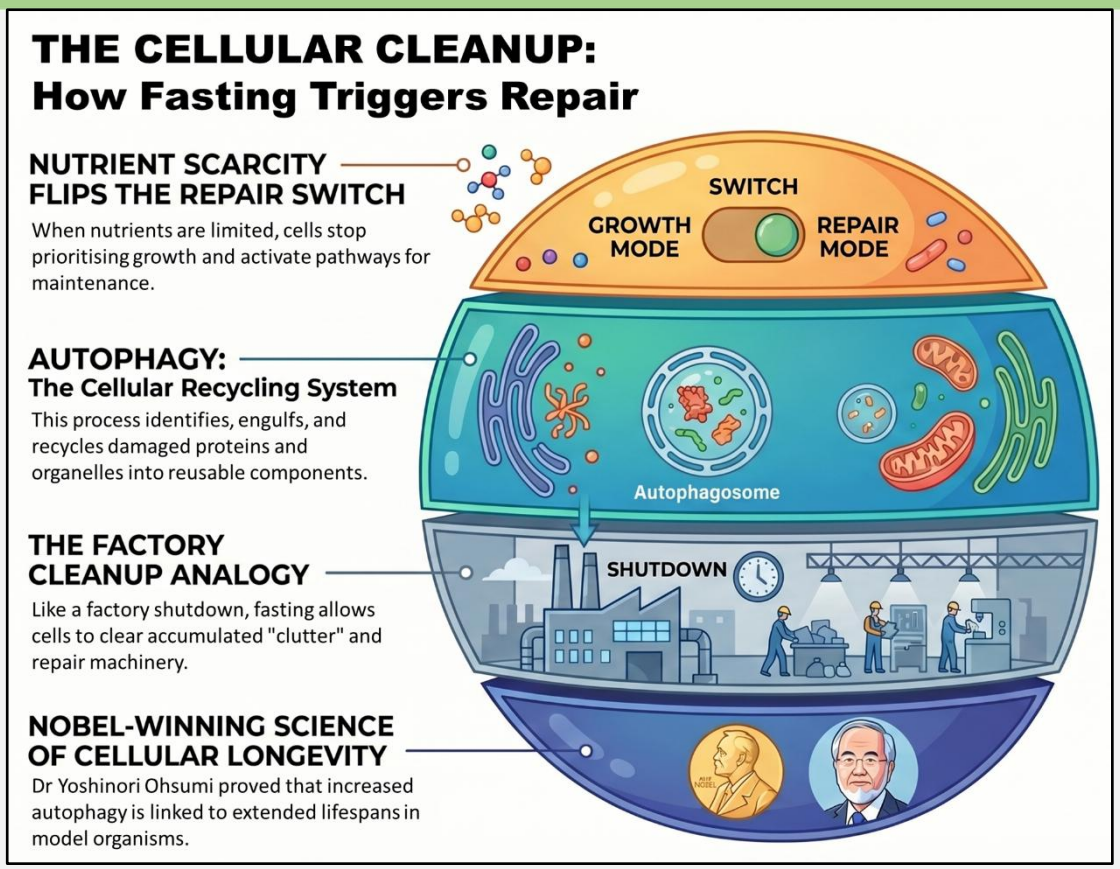


Mitochondrial dysfunction is closely linked to ageing: as we age, mitochondrial efficiency declines, leading to reduced energy production and increased generation of reactive oxygen species. If not controlled, these byproducts can damage cellular components, leading to increased oxidative stress and energy deficits. In healthy young cells, energy production is well-regulated, and antioxidants manage reactive molecules. However, as mitochondrial function deteriorates with age, the ability to repair and maintain cells diminishes. Damaged mitochondria accumulate, and although cells employ mechanisms such as mitophagy to remove them, these mechanisms become less effective over time. Mitochondrial decline is interconnected with other ageing processes, such as inflammation. For instance, mitochondrial dysfunction can exacerbate inflammation, underscoring the complexity of ageing. In summary, mitochondria are crucial for energy supply and cellular function, and their age-related decline disrupts repair and signalling mechanisms, contributing to the overall loss of biological precision seen in ageing.

The Brain as an Integrator Under Ageing. The brain is essential for coordinating the body's activities by processing information from our senses, hormones, and immune responses. It acts like a central control system, helping different parts of the body work together effectively. However, as we age, the brain's ability to coordinate these activities declines, which makes communication and coordination less precise. One significant change in the brain as it ages involves microglial cells, the brain's immune cells. In a healthy brain, microglia monitor the environment and support nerve function. As we age, these cells become more active and produce more inflammatory substances, leading to neuroinflammation and further disruption of normal brain function.

Research indicates that ageing also affects synaptic function, which is how brain cells communicate. Changes in synaptic density and neurotransmitter levels reduce the precision of communication. While these changes don't immediately cause cognitive decline, they do reduce the brain's ability to communicate, affecting memory, attention, and decision-making. Clinical observations show that age-related changes in cognition result from gradual shifts in how the brain processes information. Because the brain integrates various systems, these functional changes can impact hormonal balance, immune responses, and behaviours. Disruptions in brain function can also affect sleep and metabolism. There is variability in how each person's brain ages, influenced by genetics and lifestyle. However, the general pattern is a slow decrease in precision and coordination. In summary, changes in microglial activity and synaptic function due to ageing impair the brain's ability to integrate multiple systems, leading to





the coordination challenges commonly seen in older adults.

Fasting, Autophagy, and Cellular Maintenance. One of the most extensively studied ways in which cellular behaviour can be influenced is through nutrient availability. When nutrients are abundant, cells prioritise growth and proliferation. When nutrients are limited, cells activate pathways that promote maintenance and repair. This shift is central to the concept of fasting and its biological effects. A key process activated during nutrient scarcity is autophagy. Autophagy is a cellular mechanism that removes and recycles damaged components. It involves the formation of structures that engulf damaged proteins and organelles, which are then broken down and reused. This process helps maintain cellular quality and efficiency.

The importance of autophagy has been demonstrated in numerous experimental studies. Research in model organisms such as yeast, worms, and mice has shown that increased autophagy is associated with improved cellular function and extended lifespan. These findings were recognised with the awarding of the Nobel Prize to Yoshinori Ohsumi for his work on the mechanisms of autophagy. In humans, evidence suggests that fasting and caloric restriction can activate similar pathways. Studies have shown that short-term fasting can influence metabolic markers and activate processes related to cellular maintenance. However, it is important to note that long-term effects on ageing in humans are still under investigation, and results from animal studies cannot be directly applied without caution. A useful analogy is a factory cleanup process. When production is continuous, damaged components may accumulate. Periodic shutdown allows for cleaning, repair, and maintenance. In cells, fasting creates conditions that favour such maintenance processes.

It is important to maintain a balanced perspective. While the biological mechanisms are well established, the extent to which fasting influences human ageing remains an active area of research. Clinical studies are ongoing to better understand these effects. In summary, fasting and autophagy illustrate how cells shift from growth to maintenance in response to environmental signals. This process supports cellular health by removing damaged components and improving efficiency, but its long-term impact on human ageing remains under study.

Stress, Ageing, and the Possibility of Recovery. Stress is a fundamental biological response that helps the body adapt to challenges. In its short-term form, known as acute stress, it is beneficial. It activates coordinated systems involving the brain, hormones, and immune pathways, particularly the hypothalamic–pituitary–adrenal axis, leading to the release of cortisol. This response mobilises energy, sharpens attention, and supports survival. Experimental studies have shown that brief stress can enhance immune function and cognitive performance when followed by adequate recovery.

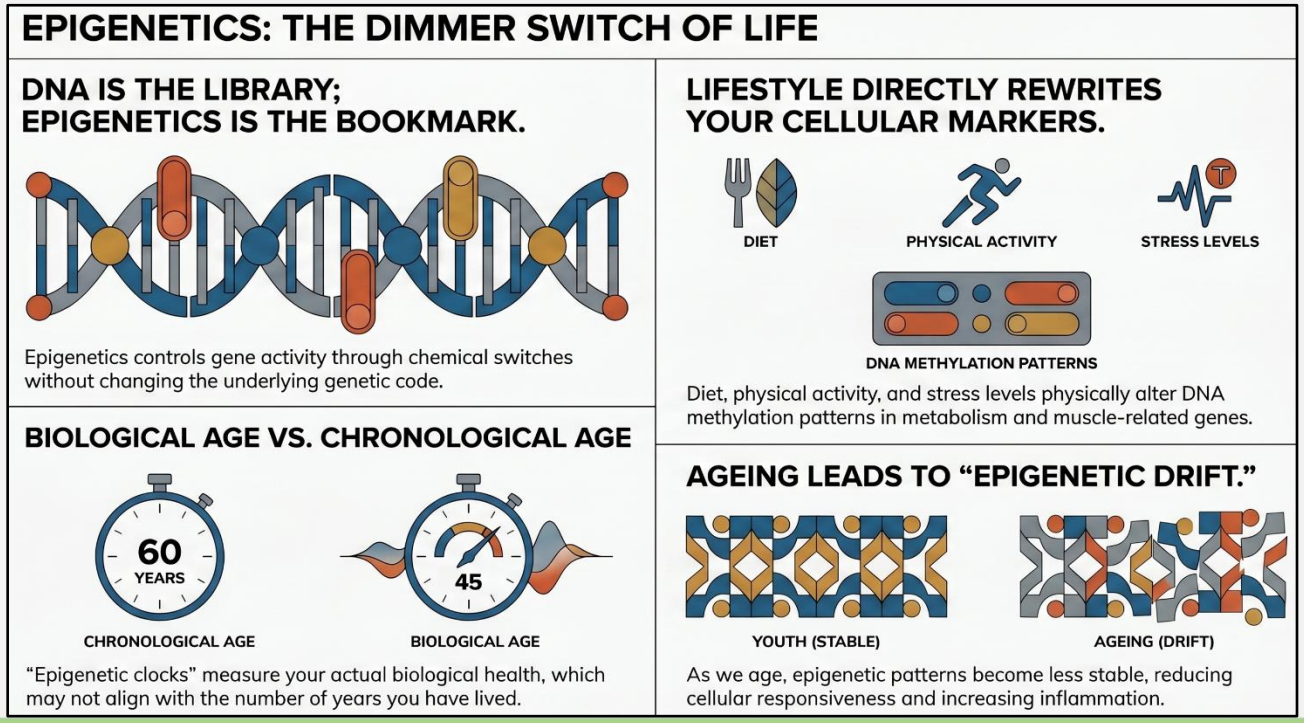
The problem arises when stress becomes chronic. Chronic stress refers to a prolonged state in which stress pathways remain active rather than returning to baseline. Clinical and experimental research has consistently shown that this sustained activation leads to measurable biological changes. One key effect is increased production of reactive oxygen species, which can damage DNA, proteins, and lipids. Studies measuring oxidative stress markers have found higher levels in individuals exposed to long-term psychological stress, indicating that chronic stress contributes to molecular damage. Chronic stress also promotes inflammation. Research has shown that

prolonged activation of stress pathways is associated with elevated inflammatory markers such as interleukin-6 and C-reactive protein. Large population studies have linked chronic stress to persistent low-grade inflammation, which interferes with cellular signalling and contributes to ageing-related changes. This inflammatory state affects multiple systems, including metabolism, cardiovascular function, and the brain. Another important link between stress and ageing involves telomeres. Studies in human populations have shown that individuals experiencing chronic stress may have shorter telomeres compared to less stressed individuals. For example, research on caregivers of chronically ill patients has demonstrated associations between high perceived stress and reduced telomere length. While such findings are observational and do not establish direct causation, they consistently support a relationship between stress and markers of biological ageing. Chronic stress also alters cellular function. Prolonged exposure to cortisol can disrupt signalling pathways and affect metabolic regulation. This has been linked to insulin resistance, a well-documented feature of ageing and metabolic disease. In this way, stress not only increases damage but also reduces the precision of cellular responses. A useful analogy is to think of stress as an emergency system designed for short-term use. When activated briefly, it resolves a challenge and shuts down. If it remains active continuously, it begins to interfere with normal operations. Resources are diverted away from maintenance and repair, leading to a gradual imbalance.

This raises an important question. If chronic stress contributes to ageing, can reducing stress improve biological function? Research on practices such as meditation and mindfulness has shown changes in physiological markers. Studies have reported reductions in inflammatory markers and improvements in stress-related measures following such interventions. Some research has also observed increased telomerase activity, the enzyme involved in maintaining telomere length, in individuals undergoing stress-reduction programs. However, these findings must be interpreted carefully. Many studies are observational or involve small sample sizes. Individuals who practice meditation may also engage in other healthy behaviours, making it difficult to isolate specific effects. Even in controlled studies, results can vary depending on the population and methodology.

Despite these limitations, the overall evidence suggests a consistent pattern. Chronic stress is associated with biological changes linked to ageing, including damage, inflammation, and altered signalling. Reducing stress appears to improve markers of physiological stability, even though its long-term impact on ageing remains under investigation. A helpful way to view this is as a balance between activation and recovery. Stress represents activation, while rest and recovery restore equilibrium. When this balance is maintained, the system remains stable. When activation dominates, strain accumulates over time. In summary, stress has a dual role in biology. Acute stress is adaptive, while chronic stress contributes to damage and reduced cellular precision. Evidence supports the benefits of stress reduction for improving biological stability, but careful interpretation is essential when considering its role in long-term ageing.

Epigenetics: Linking Lifestyle to Cellular Function. A key question in biology is how the same DNA can produce different outcomes in various cells and life stages. Despite nearly identical genetic codes, nerve cells behave differently from liver cells, and younger cells differ from older ones. This variation comes from epigenetics, which controls gene activity without changing the DNA sequence. Think of DNA as a library of instructions, with epigenetic mechanisms acting like bookmarks and switches that determine

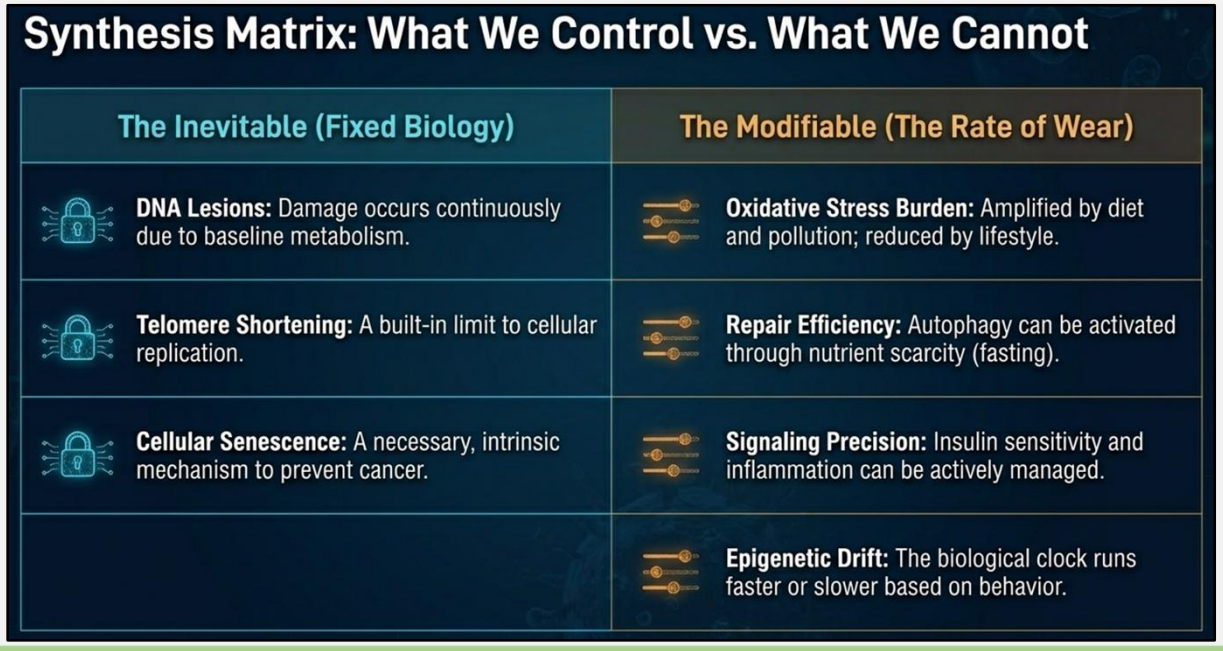


which genes are active.

Recent research shows that epigenetic patterns can change over time and are influenced by environmental factors. For instance, as we age, these patterns become less stable, a phenomenon known as epigenetic drift. Studies reveal that older individuals have distinct DNA methylation patterns compared to younger ones, linking these changes to inflammation, metabolism, and cellular repair. Notably, epigenetic clocks can estimate biological age, which may not always align with chronological age, affecting health outcomes. Moreover, lifestyle factors such as diet, exercise, and stress can alter epigenetic markers. Research indicates specific eating habits can change DNA methylation in metabolism-related genes, while physical activity and stress influence gene expression related to muscle function and inflammation. In essence, epigenetics acts like a dimmer switch, allowing genes to vary in activity. As we age, this responsiveness diminishes, leading to altered gene expression. While external factors can influence epigenetic changes, some may be permanent, making these mechanisms crucial to understanding health and disease.

What Can Be Influenced and What Cannot: Understanding Ageing in a Changing World. Understanding ageing involves distinguishing between fundamental biological processes and those that can be modified through environmental and lifestyle factors. Some aspects of ageing are intrinsic to living systems and cannot be completely prevented. For instance, DNA damage is a continuous process; cells are regularly exposed to factors that can harm their DNA, such as metabolic byproducts and environmental stressors. Despite repair mechanisms, some DNA damage is inevitable. Experimental studies have shown that cells inevitably accumulate DNA damage over time, even under controlled conditions. Another intrinsic process is telomere shortening, which follows a predictable pattern and undergoes shortening each time a cell divides due to limitations in DNA replication. While enzymes such as telomerase can extend telomeres in certain cells, most somatic cells lack this capacity, leading to progressive telomere shortening with age. This shortening constrains cellular division and contributes to ageing. Additionally, cellular senescence occurs when cells experience significant damage or reach replicative limits, causing them to enter a non-dividing state. This serves as a protective mechanism against uncontrolled cell growth. Although the accumulation of senescent cells contributes to the ageing process, it is essential for maintaining overall stability in biological systems.

While these processes are fixed aspects of ageing, research indicates that the rate at which they occur can be influenced. This distinction is important for understanding how lifestyle choices and environmental factors can modulate the effects of ageing. For instance, DNA damage rates can be significantly affected by environmental exposures such as smoking, pollution, and poor dietary habits, all of which increase oxidative stress. Conversely, reducing such exposures can reduce the damage burden. Clinical studies further corroborate that lifestyle factors are associated with markers of oxidative stress and inflammation. In addition to damage accumulation, the efficiency of cellular repair mechanisms is also modifiable. Although these repair systems naturally decline with age, their activity can still be influenced by environmental and metabolic conditions. Research on caloric restriction and metabolic pathways has shown that these interventions can activate cellular maintenance processes, including DNA repair and autophagy. These findings suggest that cellular systems retain the capacity to adapt, even in the face of ageing. Signalling precision within cells is another area where external factors can affect it. Inflammation and metabolic stress can negatively affect signalling pathways, impairing cellular responses. By reducing these stressors, we can enhance the fidelity of cellular functions. Clinical evidence supports this, showing that interventions to improve metabolic health can increase insulin sensitivity and reduce inflammatory



markers. Analogously, one can think of the ageing process as akin to the wear and tear on a machine. While some degree of wear is unavoidable, the rate and effects of this wear can be influenced by maintenance and operational practices. Proper care can slow progression and enhance overall performance, highlighting the importance of moderating lifestyle choices. However, it is also important to avoid oversimplifying the issue. Lifestyle factors do impact biological ageing, but they don't give us complete control over how we age. The interplay among genetics, environment, and random factors is complex. Scientific evidence shows that we need to understand ageing in a balanced way, recognising both the unchangeable and the changeable aspects of the ageing process. Furthermore, the concept of ageing is not confined to biological definitions; it also reflects changes in lifestyle and environment. Recent years have seen significant lifestyle shifts, particularly in rapidly urbanising countries like India. Traditional habits involving regular physical activity, structured meal times, and consistent sleep patterns are being replaced by sedentary behaviours, irregular eating habits, and disturbed sleep. Clinical and epidemiological research in Indian populations indicates rising rates of metabolic conditions such as type 2 diabetes, obesity, and cardiovascular diseases, often manifesting at younger ages than previously observed. This suggests that modern lifestyle changes are influencing the early stages of biological ageing, particularly in metabolic health-related systems.

In summary, ageing is a complex interplay of fixed biological processes, such as DNA damage, telomere shortening, and cellular senescence, alongside modifiable factors where lifestyle and environmental influences can have substantial effects. Understanding this delicate balance provides a realistic perspective on ageing, empowering individuals to make informed choices that may mitigate some of the processes accelerated by modern lifestyles.

We cannot stop time.
But we may influence how precisely our biology keeps up with it.

Suggested Reading

1. López-Otín, C., Blasco, M. A., Partridge, L., Serrano, M., & Kroemer, G. (2013). The hallmarks of aging. Cell, 153(6), 1194–1217.
2. López-Otín, C., Galluzzi, L., Freije, J. M. P., Madeo, F., & Kroemer, G. (2023). Hallmarks of aging: An expanding universe. Cell, 186(2), 243–278.
3. Hayflick, L., & Moorhead, P. S. (1961). The serial cultivation of human diploid cell strains. Experimental Cell Research, 25(3), 585–621.
4. Campisi, J. (2013). Aging, cellular senescence, and cancer. Annual Review of Physiology, 75, 685–705.
5. Coppé, J. P., Desprez, P. Y., Krtolica, A., & Campisi, J. (2010). The senescence-associated secretory phenotype: The dark side of tumor suppression. Annual Review of Pathology, 5, 99–118.
6. Baker, D. J., Wijshake, T., Tchkonia, T., et al. (2011). Clearance of p16Ink4a-positive senescent cells delays ageing-associated disorders. Nature, 479(7372), 232–236.
7. Blackburn, E. H. (2000). Telomere states and cell fates. Nature, 408(6808), 53–56.
8. Epel, E. S., Blackburn, E. H., Lin, J., et al. (2004). Accelerated telomere shortening in response to life stress. Proceedings of the National Academy of Sciences USA, 101(49), 17312–17315.
9. Ohsumi, Y. (2014). Historical landmarks of autophagy research. Cell Research, 24(1), 9–23.
10. Mizushima, N., & Komatsu, M. (2011). Autophagy: Renovation of cells and tissues. Cell, 147(4), 728–741.
11. Levine, B., & Kroemer, G. (2008). Autophagy in the pathogenesis of disease. Cell, 132(1), 27–42.
12. Harman, D. (1956). Aging: A theory based on free radical and radiation chemistry. Journal of Gerontology, 11(3), 298–300.
13. Finkel, T., & Holbrook, N. J. (2000). Oxidants, oxidative stress and the biology of ageing. Nature, 408(6809), 239–247.
14. Franceschi, C., Bonafè, M., Valensin, S., et al. (2000). Inflamm-aging: An evolutionary perspective on immunosenescence. Annals of the New York Academy of Sciences, 908, 244–254.
15. Ferrucci, L., & Fabbri, E. (2018). Inflammageing: Chronic inflammation in ageing, cardiovascular disease, and frailty. Nature Reviews Cardiology, 15(9), 505–522.
16. Mattson, M. P., Longo, V. D., & Harvie, M. (2017). Impact of intermittent fasting on health and disease processes. Ageing Research Reviews, 39, 46–58.
17. Longo, V. D., & Panda, S. (2016). Fasting, circadian rhythms, and time-restricted feeding in healthy lifespan. Cell Metabolism, 23(6), 1048–1059.
18. Kennedy, B. K., Berger, S. L., Brunet, A., et al. (2014). Geroscience: Linking aging to chronic disease. Cell, 159(4), 709–713.
19. Horvath, S. (2013). DNA methylation age of human tissues and cell types. Genome Biology, 14(10), R115.
20. Sen, P., Shah, P. P., Nativio, R., & Berger, S. L. (2016). Epigenetic mechanisms of longevity and aging. Cell, 166(4), 822–839.
21. López-Lluch, G., & Navas, P. (2016). Calorie restriction as an intervention in ageing. The Journal of Physiology, 594(8), 2043–2060.
22. Sun, N., Youle, R. J., & Finkel, T. (2016). The mitochondrial basis of aging. Molecular Cell, 61(5), 654–666.

23. Wyss-Coray, T. (2016). Ageing, neurodegeneration and brain rejuvenation. Nature, 539(7628), 180–186.
24. Dilman, V. M. (1994). The neuroendocrine theory of aging and age-related pathology. Experimental Gerontology, 29(1), 11–20.
25. McEwen, B. S. (1998). Protective and damaging effects of stress mediators. New England Journal of Medicine, 338(3), 171–179.
26. Campisi, J., & d'Adda di Fagagna, F. (2007). Cellular senescence: When bad things happen to good cells. Nature Reviews Molecular Cell Biology, 8(9), 729–740.
27. Gladyshev, V. N. (2016). Aging: Progressive decline in fitness due to the rising deleteriome adjusted by genetic, environmental, and stochastic processes. Aging Cell, 15(4), 594–602.
28. Kirkwood, T. B. L. (2005). Understanding the odd science of aging. Cell, 120(4), 437–447.
29. Partridge, L., Deelen, J., & Slagboom, P. E. (2018). Facing up to the global challenges of ageing. Nature, 561(7721), 45–56.

SCIENCE SPECTRUM

"Changing the Narrative on Suicide" Series. - Dr. Malini Srivastava

9. Lived Experience is Expertise: Listening to Suicide Survivors

Suicide is often discussed through statistics, diagnoses, and prevention strategies, but behind every number is a deeply personal human experience. This article explores why listening to people who have lived through suicidal crises or loss is essential for truly understanding suicide. It reminds us that empathy, human connection, and the courage to listen without judgment can sometimes become the first step toward healing and hope.

One of the most important conversations we need to strengthen today is about **suicide survivors**, those who have lived through suicidal thoughts, attempts, or the loss of a loved one to suicide. Too often, their voices are unheard, their experiences overlooked, and their insights underused. But the truth is simple: *If we truly want to understand suicide, we must listen to those who have lived it.*

A Moment That Changes Understanding

During a clinical meeting in a hospital, a group of professionals gathered to discuss suicide prevention strategies. There were presentations on risk factors, statistics, and intervention models. Everything was structured, evidence-based, and carefully discussed. Then, a suicide survivor was invited to speak. She did not speak in clinical terms. She spoke about what it felt like to wake up with overwhelming hopelessness, about the silence she experienced when she tried to express her pain, and about how one moment of genuine human connection made her choose to stay. The room changed. What was earlier a discussion about "risk" became a deeper understanding of human experience.

Why Suicide Survivors' Voices Matter.

Traditionally, suicide has been studied through data such as rates, causes, and treatment outcomes. While this research is important, it does not fully capture what individuals actually go through. The World Health Organisation (2021) emphasises that effective mental health care must include the voices of people with lived experience. Similarly, research by Larry Davidson (2006) shows that recovery is deeply personal and shaped by meaning, identity, and connection, not just by symptom reduction. Suicide survivors help us understand:

- What thoughts feel like from the inside
- What triggers emotional crisis
- What actually helps in moments of distress

Their insights are not just emotional but are practical and life-saving.

Understanding Suicide Beyond Symptoms
Clinical models often focus on warning signs and diagnostic categories. But suicide survivors describe something more complex, such as feelings of isolation, worthlessness, emotional pain, and sometimes even numbness. Trauma expert Judith Herman (1992) explains that survivors often experience deep emotional disconnection and loss of control with experiences that go beyond what clinical labels can explain.

In India, this becomes even more complex. Many individuals do not express suicidal distress openly. Instead, they report physical symptoms or general distress. Research by Vikram Patel shows that a large number of individuals express emotional pain through physical complaints, especially in primary care settings. This means that unless we listen carefully to lived experiences, we may miss the signs entirely.

The Indian Context: What Survivors Teach Us
In India, suicide survivors often face multiple challenges:

- Strong social stigma
- Fear of judgment
- Limited access to mental health services
- Family pressures and expectations

The National Mental Health Survey of India reports a **treatment gap of up to 92%**, meaning most people do not receive the help they need. Survivors often share that:

- They hesitate to seek help due to shame
- They fear being misunderstood
- They struggle to find safe spaces to talk

These are not just personal struggles, but system-level gaps.

From Silence to Participation
Earlier, suicide survivors were rarely included in discussions about prevention. Today, this is beginning to change. Research approaches, such as community participation, supported by experts such as Barbara Israel (1998), show that involving people with lived experience improves outcomes. In India, organisations like Sangath have demonstrated that community-based approaches can be highly effective. The MANAS study showed improved recovery outcomes when care was delivered through community-linked support systems (Patel et al., 2010). This shows that survivors are not just recipients of care but can also be partners in creating solutions.

What Survivors Tell Us About Care
One of the most important lessons from suicide survivors is this: **What helps is not always what we assume.** Many survivors say that:

- Being listened to without judgment matters more than advice
- Small acts of kindness can have a big impact
- Feeling understood can reduce suicidal thoughts

Research by Graham Thornicroft (2016) highlights how stigma prevents people from seeking help. In India, studies from the National Institute of Mental Health and Neurosciences show that many individuals discontinue treatment due to distance, cost, or lack of family support. These insights remind us that prevention is not only about systems but also about human connection.

The Role of Peer Support
Peer support or help from someone who has gone through similar experiences is becoming an important approach in suicide prevention. In Gujarat, the Atmiyata program trained community volunteers to support people in distress. Research (Jain & Jadhav, 2008) showed clear improvements in mental health outcomes. The reason it works is that survivors often feel understood and not alone, and this sense of connection can significantly alleviate emotional pain.

Moving Beyond Tokenism
While we are beginning to include suicide survivors in discussions, we must ensure that this inclusion is meaningful. Research by Peter Beresford (2013) warns against tokenism, where survivors are invited to speak but not included in decision-making. In India, studies (Semrau et al., 2016) show that survivor involvement is still limited in policy and planning. True inclusion means:

- Giving survivors a voice in decisions
- Respecting their knowledge
- Creating safe spaces for participation

The Role of Family and Culture
In India, family plays a major role in a survivor's journey. Families can be supportive, but sometimes stigma or lack of awareness can delay help. Findings from the National Mental Health Survey of India show that family attitudes strongly influence whether individuals seek help. Understanding these dynamics is important for effective suicide prevention.

The Way Forward
Policies like the Mental Healthcare Act (2017) emphasise dignity, rights, and care. However, to make these policies truly effective, we must include suicide survivors in shaping them. Research suggests that without lived experience, policies remain incomplete (Duffy & Kelly, 2019).

Conclusion: Listening Can Save Lives
At the end of any discussion on suicide prevention, one truth remains clear: **Data informs us, but lived experience transforms our understanding.** When suicide survivors are heard:

- Prevention becomes more realistic

- Care becomes more compassionate
- Systems become more responsive

For professionals and researchers, this is not just an addition, but an essential step. The future of suicide prevention is not only based on evidence. It is based on listening, understanding, and learning from those who have lived through it. And perhaps the most important lesson survivors teach us is this: **Sometimes, what saves a life is not a system or a theory, but it is a simple act of being heard, understood, and not left alone.**

References

1. Beresford, P. (2013). From “other” to involved: User involvement in research: An emerging paradigm. Nordic Social Work Research, 3(2), 139–148.
2. Davidson, L., O’Connell, M. J., Tondora, J., Lawless, M., & Evans, A. C. (2006). Recovery in serious mental illness: A new wine or just a new bottle? Professional Psychology: Research and Practice, 37(5), 480–487.
3. Duffy, R. M., & Kelly, B. D. (2019). India’s Mental Healthcare Act, 2017: Content, context, controversy. International Journal of Law and Psychiatry, 62, 169–178.
4. Gururaj, G., Varghese, M., Benegal, V., Rao, G. N., Pathak, K., Singh, L. K., … NMHS Collaborators Group. (2016). National Mental Health Survey of India, 2015–16: Summary. NIMHANS.
5. Herman, J. L. (1992). Trauma and recovery: The aftermath of violence—from domestic abuse to political terror. Basic Books.
6. Israel, B. A., Schulz, A. J., Parker, E. A., & Becker, A. B. (1998). Review of community-based participatory research. Annual Review of Public Health, 19, 173–202.
7. Jain, N., & Jadhav, S. (2018). Community-based mental health care in India: The Atmiyata intervention. Global Mental Health, 5, e36.
8. Kumar, S., et al. (2019). Incorporating patient perspectives in psychiatric training: Impact on empathy and clinical skills. Indian Journal of Psychiatry, 61(Suppl 4), S789–S795.
9. McPherson, H. L., et al. (2005). A review of qualitative studies of recovery from mental illness. International Journal of Social Psychiatry, 51(3), 249–259.
10. Murthy, R. S., & Kumar, K. V. (2011). Treatment gap in mental health care: Issues and challenges in India. Indian Journal of Psychiatry, 53(1), 1–2.
11. Patel, V., Pereira, J., & Mann, A. (2001). Somatic and psychological models of common mental disorders in India. Psychological Medicine, 31(2), 213–223.
12. Patel, V. (2010). Mental health in low- and middle-income countries. British Medical Bulletin, 81–82(1), 81–96.
13. Patel, V., Weiss, H. A., Chowdhary, N., Naik, S., Pednekar, S., Chatterjee, S., … Kirkwood, B. R. (2010). MANAS trial. The Lancet, 376(9758), 2086–2095.
14. Rose, D. (2014). Patient and public involvement in health research. Journal of Health Psychology, 19(1), 149–158.
15. Semrau, M., Evans-Lacko, S., Alem, A., et al. (2016). Strengthening mental health systems: The Emerald programme. BMC Medicine, 14, 79.
16. Thornicroft, G., Mehta, N., Clement, S., et al. (2016). Reducing mental-health-related stigma. The Lancet, 387(10023), 1123–1132.
17. World Health Organization. (2021). Guidance on community mental health services. WHO.

SCIENCE SHAPERS

Dr Yoshinori Ohsumi

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Dr Yoshinori Ohsumi is a pioneering Japanese cell biologist whose discoveries transformed modern understanding of how cells maintain health, survive stress, and adapt during ageing. He is internationally recognised for uncovering the fundamental mechanisms of autophagy, a cellular self-recycling process through which cells identify, degrade, and reuse damaged or unnecessary components. His work fundamentally changed how scientists view ageing, disease, and cellular resilience.

Awarded the 2016 Nobel Prize in Physiology or Medicine, Dr Ohsumi demonstrated that autophagy is not merely a passive waste-disposal system, but a highly regulated biological process essential for maintaining cellular balance and survival. Using simple yeast cells, he identified key genes and molecular pathways that govern autophagy, laying the foundation for one of the most rapidly expanding areas of biomedical research.

His discoveries revealed that cells continuously monitor internal damage and metabolic stress, activating autophagy to restore order and preserve function. This process becomes especially important during starvation, nutrient deprivation, and ageing, when cells must efficiently recycle internal resources to survive. The decline of autophagy with age is now linked to the accumulation of damaged proteins, dysfunctional organelles, inflammation, neurodegeneration, metabolic disorders, and loss of cellular precision.

Dr Ohsumi's work also helped explain why processes such as fasting and caloric restriction can influence health and longevity. By showing how nutrient sensing and cellular recycling are interconnected, his research provided critical insights into how cells respond to stress and maintain biological stability over time.

Trained at the University of Tokyo, Dr Ohsumi pursued a research path driven by curiosity, persistence, and fundamental biological questions rather than immediate clinical applications. His work stands as a powerful example of how basic science can ultimately reshape medicine and human health.

Over the course of his career, he has received numerous international honours, including the Kyoto Prize, the Canada Gairdner International Award, the Breakthrough Prize in Life Sciences, and the Nobel Prize. His discoveries continue to influence diverse fields ranging from cancer biology and neuroscience to immunology and ageing research.

In this May issue of Science Prism, Dr Yoshinori Ohsumi represents a defining scientific voice in understanding how cells preserve order amidst stress and time. His work reveals that ageing is not simply the passage of years, but also a gradual decline in the cell's ability to maintain clarity, balance, and internal renewal.

ABOUT THE TEAM:


Dr. Kaushik P Sharma


Dr. Kanchan Bisht


Dr. Shreesh Raj Sammi


Dr. Manju Tewari

***Dr Kaushik P. Sharma and Dr Kanchan Bisht** are neuroscientists at the Department of Neurology, Himalayan Institute of Medical Sciences, Swami Rama Himalayan University, Dehradun. With over a decade of extensive research experience at renowned institutions in India, Europe, Canada, and the USA, they are dedicated to making science accessible and inspiring the next generation of thinkers, innovators, and problem solvers.*

***Dr Shreesh Raj Sammi**, an Assistant Professor in the Department of Translational Neuroscience at Michigan State University, USA, brings his expertise in neurotoxicology and neurodegenerative disorders to the team. In addition to his research pursuits, Dr Sammi is deeply committed to training the next generation of researchers and making science accessible to diverse audiences.*

***Dr Manju Tewari,** an Assistant Professor at Kumaun University, is a neuroscientist specialising in neuron–glial interactions, microglial biology, and neuroinflammation. With training at NBRC, India, and postdoctoral research at Saint Louis University, USA, she brings strong expertise in cellular and molecular neuroscience and is dedicated to advancing research and mentorship in Uttarakhand.*

This newsletter is our humble effort to empower readers with insights into scientific breakthroughs and foster a foundational understanding of nature, health, and science. We hope it sparks curiosity and supports students, educators, and young researchers in their journeys.

We'd truly love to hear from you. Your thoughts, questions, and reflections are greatly appreciated. Your feedback not only helps us grow but also motivates us to keep moving forward with these small but heartfelt efforts.

*Let's make **Science Prism** a two-way conversation where curiosity flows both ways.*

Stay curious, stay connected.

– The Science Prism Team.

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