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**SHORT COMMUNICATION**

# Stress-Induced Accelerated Aging: A Challenge for Modern Biomedicine

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**ABSTRACT**

**Introduction:**
Ageing is a complex process influenced by various factors, such as chronic psychological stress, which accelerates biological ageing through persistent activation of stress-response pathways. This leads to molecular and cellular damage, increasing vulnerability to age-related diseases. Chronic stress increases cortisol levels, disrupts immune regulation, and promotes telomere shortening, contributing to cellular senescence. Recent epigenetic clock developments showed that stress can accelerate biological age beyond chronological age. Healthy lifestyles and stress reduction may alleviate biological ageing effects, although more research is needed to explore the relationship between specific stressors and ageing pathways.

**Conclusion:**
Stress significantly accelerates ageing, necessitating further interdisciplinary research for effective prevention and therapy.

**Keywords**
Accelerating, ageing, biological, chronic, stress

## Introduction

Ageing is an inevitable chronological process intricately associated with multiple factors. Biological ageing is a dynamic process and is influenced by multiple factors. Chronic psychological stress is the most powerful non-genetic determinant which accelerates the process of biological ageing. The other potential ageing factor is stress, which causes persistent activation of stress-response pathways that intensify molecular and cellular damage, increasing susceptibility to age-related diseases long before chronological ageing. Recent advances in molecular biology, psychoneuroimmunology, and epigenetics have shown this [1].

Eustress is essential for survival. Activation of the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system helps us adapt to environmental threats through the release of glucocorticoids and catecholamines. However, chronic or repetitive stimulation of these pathways results in allostatic loads, involves different organ systems, and affects their normal function. So, stress-induced increased levels of cortisol, continuous sympathetic impulses, and chronic low-grade inflammation interfere with immune regulation, metabolic homeostasis, mitochondrial function, and vascular integrity. Researchers reported that prolonged occupational stress, adverse childhood experiences, and poor socioeconomic status accelerate the biological ageing process [1,2].

At the cellular level, multiple interconnected mechanisms explain how chronic stress facilitates ageing. Excessive glucocorticoids increase oxidative stress, which causes DNA damage, impairs DNA repair mechanisms, and contributes to the ageing process. Reactive oxygen species interfere with mitochondrial function, reduce cellular energy production, and augment inflammatory signalling. Chronic stress causes telomere shortening, a well-established biomarker of biological ageing. Multiple genetic and environmental factors, including persistent psychosocial stress, increase telomere attrition, which in turn promotes cellular senescence and limits regenerative capacity. Stress-induced activation of pro-inflammatory cytokines also contributes to the senescence-associated secretory phenotype (SASP), creating a self-perpetuating inflammatory environment which causes tissue malfunction. [1,3, 4]

In recent years, an exciting development in ageing research has occurred after the emergence of epigenetic clocks, which are based on DNA methylation profiles. These biomarkers indicate chronic psychological stress, where an individual's epigenetic age exceeds their chronological age, indicating measurable acceleration of biological ageing [4, 5]. Unlike chronological age, epigenetic age reflects cumulative molecular damage and predicts future cardiovascular disease, diabetes, neurodegenerative disorders, frailty, and premature mortality. So, stress does not merely influence disease causation indirectly but directly changes the molecular architecture governing ageing itself.

The clinical implications are significant in this context, where a common biological pathway is used for different physiological processes. Metabolic syndrome, cardiovascular diseases, depression, cognitive deterioration, immunological dysfunction, and certain malignant conditions use the same biological pathways involving chronic inflammation, mitochondrial dysfunction, genomic instability, and cellular senescence. These closely associated mechanisms support the geroscience hypothesis that interventions targeting biological aging may simultaneously reduce the burden of multiple chronic diseases. [6]

The biological ageing process is not completely irreversible. Leading a healthy lifestyle, regular physical activity, adequate sleep, proper nutrition, less stress, quitting smoking, and strong social support help to reduce profoundly allostatic load and partly normalize biomarkers of biological ageing [7]. Although pharmacological geroprotective therapies are still under research, behavioural interventions are the safest and most accessible approaches to preserve biological resilience. Simultaneously, the use of precision medicine, integrating multi-omics technologies, artificial intelligence, and personalized biological age assessment, will allow future clinicians to recognize vulnerability before irreversible damage occurs. [8]

Although there is a remarkable advancement, multiple important questions need to be answered. Currently available biological ageing biomarkers vary in sensitivity, specificity, and predictive value. Relationships between specific stressors and molecular ageing pathways need more clarity. Genomic, epigenomic, metabolomic, and psychosocial research data will be useful in this context. Future research is required to determine whether reducing chronic stress can genuinely reverse biological age acceleration rather than merely slowing its progression.

## Conclusion

Stress accelerating the ageing process is one of the most persuasive paradigms in current biology. Psychoneuroimmunology, molecular biology, and geroscience research show that chronic psychological stress extends far beyond mental health, accelerating the ageing process. Continued interdisciplinary research is required for further insights into preventive and therapeutic strategies.

## Abbreviations

hypothalamic-pituitary-adrenal (HPA) axis, senescence-associated secretory phenotype (SASP)

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