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ACCELERATED BIOLOGICAL AGING: ENVIRONMENTAL, LIFESTYLE, AND SOCIAL DETERMINANTS OF EPIGENETIC AGE ACCELERATION: A SYSTEMATIC REVIEW

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ABSTRACT

Background: A gap between biological and chronological age known as "accelerated aging" has become a quantifiable phenomenon associated with modifiable features of modern urban and industrial environments. Two or more of the following factors linked to premature biological aging, independent of chronological age: environmental pollution and poor air quality, a sedentary lifestyle, circadian rhythm and sleep disorders, chronic psychosocial stressors, ultra-processed diet, and social or digital isolation.

Objective: To comprehensively explore the modifiable features of modern society that have been implicated in accelerated biological aging as measured by validated biomarkers (epigenetic clock, telomere length, allostatic load) and to discuss the implications for public health.

Methods: A structured narrative systematic literature review conducted in accordance with PRISMA guidelines. All sources identified using iterative keywords. Searches in peer-reviewed databases (PubMed, ScienceDirect, Frontiers, Nature journals and PMC/PubMed Central). Studies reporting associations between validated biological-aging biomarkers and modifiable environmental, behavioural, or psychosocial exposures prioritized for inclusion.

Results: The literature search identified six major domains of contemporary life consistently associated with accelerated biological aging, ambient and traffic-related air pollution, sedentary behaviour and physical inactivity, sleep deprivation and circadian disruption, chronic psychosocial stress and allostatic load, consumption of ultra-processed foods, and social isolation and loneliness or problematic digital media use. These exposures share common underlying biological mechanisms such as chronic low-grade inflammation, oxidative stress, telomere attrition, mitochondrial dysfunction and dysregulated DNA methylation patterns, which correspond to known hallmarks of aging. Emerging

intervention studies (including dietary, exercise and multi-domain lifestyle programs) suggest that epigenetic age acceleration is at least partially reversible.

Conclusion: Accelerated biological aging driven by modifiable aspects of contemporary society represents a novel and unrecognized public health challenge that extends beyond individual disease categories. Population level interventions aim to improving air quality, the built environment, food system and social connectivity could substantially reduce the overall age-related disease burden throughout life.

Keywords: Accelerated aging, Public health, Sedentary behaviour, Air pollution, Ultra-processed food.

1 INTRODUCTION

1.1 The Widening Gap between Lifespan and Health span

Global life expectancy at birth has experienced a dramatic upward trajectory over the past 100 years, and has showed a similar trajectory in the last 30 years from 1990 to 2023 when global life expectancy increased from 64.6 to 73.8 years [1]. However, this improvement in overall survival has not matched by a proportional increase in the number of years lived without disease or disability. Healthy life expectancy (HALE), which increased more slowly from 55.9 to 63.1 years over the same period, reflects this discrepancy. Consequently, the global morbidity gap expanded from 8.8 years spent in poor health in 1990 to 10.7 years in 2023, indicating that individuals now spend, on average, 14.5% of their lives in poor health [1]. A parallel study encompassing 183 WHO Member States demonstrated that the health span–lifespan gap widened from 8.5 years in 2000 to 9.6 years in 2019 a 13% increase over two decades. This gap exhibited notable disparities across biological sex and geographic regions, with non-communicable diseases (NCDs) serving as the primary contributor [2]. Crucially, this expanded morbidity is not restricted to the final years of life is observed across the entire adult lifespan, resulting in a growing proportion of adulthood spent managing chronic illnesses [1]. In response to this trend, the United Nations declared 2021-2030 as the Decade of Healthy Aging, shifting global policy focus from merely extending lifespan toward optimizing health span [3].

The widening gap is the population signature of a phenomenon that happens at the level of the individual too, the difference between chronological age (how many years the person has lived) and how old tissues and cells are (biological age). The present review focuses on this individual variation and its implications for various aspects of contemporary life.

1.2 From Chronological Age to Biological Age

Biological age based on a combination of molecular, cellular and functional markers of physiological decline, can significantly differ from chronological years lived. When biological age exceed chronological age, an individual considered to be experiencing accelerated aging which increased the risk of cardiovascular disease, neurodegeneration, cancer, multimorbidity and all-cause mortality, independent of chronological age. [4, 5].

The development of the instruments to measure this divergence has progressed rapidly over last decade. There are now several DNA-methylation-based 'epigenetic clocks' (such as the Horvath, Hannum, PhenoAge, GrimAge, and the Dunedin PACE pace-of-aging assay,) and various metrics measuring telomere lengths. These tool can reliably determine whether a particular exposure accelerate and deaccelerate a population aging trajectory [6, 7].

These biomarkers were identified in parallel with the support of the "hallmarks of aging" framework, a list of interconnected a molecular and cellular processes that underlying aging. Initially defined as nine hallmarks in 2013 and expanded to twelve in 2023, the framework includes genomic instability, telomere attrition, epigenetic alterations, loss of proteostasis, disabled macroautophagy, deregulated nutrient-sensing, mitochondrial dysfunction, cellular senescence, stem cell exhaustion, altered intercellular communication, chronic inflammation, and dysbiosis [8, 9]. Most importantly, each hallmark satisfies three key criteria: It emerges over time and experimentally worsened by factors that accelerate the aging, and can targeted by interventions that modify, slow, or reverse aging process. This establishes a clear biological basis, showing that every day, modifiable exposures might meaningfully accelerate or decelerate aging.

1.3 Modern Society as an Accelerant of Biological Aging

These findings highlight that a key feature of contemporary, industrialized, and urban lifestyles, including ambient air pollution, traffic related pollution, sedentary screen-based job and leisure activities, chronic sleep deprivation, circadian disruption, constant exposure to psychosocial stressors, ultra-processed diets, and declining face-to-face social interactions are independently and synergistically linked to accelerated biological aging as measured by these biomarker [10]. Rather than being accidental by-products of modern life, these exposures stem directly from major structural changes over recent decades across urban design, labour markets, food systems, the built environment, and

digital infrastructure. Given the ubiquitous nature of these exposures, their social gradients, and their potential for modification, accelerated biological aging recognized as a critical public health issue. This challenge has direct implications for health equity, healthcare expenditure, workforce productivity, and the long-term sustainability of health systems serving aging populations.

This means that the same public health risk factors that were discussed in this framing (inactivity, poor sleep, chronic stress, poor diet, social disconnection, and pollution) are already on the top lists for disease burden worldwide. What the accelerated-aging literature brings in is a single outcome measure of biology that quantifies their cumulative and multi-system effects and, potentially, interaction prior to a clinical disease presentation, affording a unique perspective on population health before disease observed, rather than an incidence-based view. The accelerated-aging literature adds a unifying biological outcome measure that quantifies the cumulative, multi-system, potentially synergistic effects of these before the onset of clinically manifest disease, providing an earlier and more sensitive view of population health than the incidence of disease alone [1].

1.4 Rationale and Significance of This Review

While primary research has established, relationship between modern exposures and individual aging biomarkers, evidence specific to each type of exposure remains scattered across the environmental health, nutrition, sleep medicine, behavioural science and social epidemiology literature. Few systematic or integrative analyses have occurred that examined all these types of exposures together, or the downstream impacts and synergies of such exposures and their related effects. A synthesis that brings these threads together is valuable to synthesize: (1) convergent biological pathways that might be useful for multi-domain interventions and (2) a biomarker level phenomenon into language that is relevant to public health policy and practice.

1.5 Aim and Structure of This Review

The purpose of this review is to systematically compile published, peer-reviewed evidence for modifiable aspects of contemporary society that are associated with accelerated biological aging; to explore the biological mechanisms underlying the link between these exposures and the hallmarks of aging; and to reflect on implications for public health policy.

2 METHODS

2.1 Review Approach

This review conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [11]. Due to substantial heterogeneity across study designs, exposures, and biological aging biomarkers, a narrative systematic synthesis performed rather than a quantitative meta-analysis.

2.2 Search Strategy

Literature searches conducted iteratively across PubMed/MEDLINE, PubMed Central (PMC), ScienceDirect, Frontiers, and Oxford Academic (Age and Ageing). Search strategies combined terms for biological age (e.g., biological age, epigenetic clock, telomere length, allostatic load) with modifiable environmental, behavioural, and psychosocial exposures (e.g., air pollution, sedentary behaviour, sleep deprivation, circadian disruption, chronic stress, ultra-processed food, social isolation, and screen time).

2.3 Eligibility Criteria

Inclusion was prioritized for peer-reviewed systematic reviews, meta-analyses, prospective cohort studies, Mendelian randomization analyses, and randomized controlled trials (RCTs) reporting quantifiable associations between modifiable exposures and validated biological-aging biomarkers. Mechanistic studies and narrative reviews were selectively included to provide biological context. Non-peer-reviewed sources and anecdotal reports excluded.

2.4 Data Synthesis

Data were synthesize narratively across six primary exposure domains. Representative studies, populations, exposures, biomarkers, and key findings are summarize in **Table 1**.

3 RESULTS

The literature that was gathered and reviewed on the lifestyle domains associated with accelerated biological aging synthesized into six interrelated themes. A domain specific narrative synthesis provided after summarizing representative studies in Table 1.

3.1 Ambient and Traffic-Related Air Pollution

In multiple cohort studies, strong relationships were seen between epigenetic age acceleration and long-term exposure to particulate matter (PM2.5, PM10), black carbon, and traffic-related air pollutants, as well as between telomere attrition and exposure to particulate matter (PM2.5, PM10), black carbon, and traffic-related air pollutants [12-14]. In the German KORA cohort, increases in PM2.5 for the interquartile range accompanied by measurable increases in EEA, with the highest EEA related to current smokers and air pollutants consistently correlated with decreased DNA-meTL [12, 13]. In addition, toxicogenomic studies show that occupational exposures to volatile chemical carcinogens like benzene, trichloroethylene and formaldehyde increase the rate of the epigenetic clock [7]. Moreover, particulate exposure has been associated with increased cardiovascular reactivity seen after exposure in cohorts from the United States [15]. Evidence on air pollution and ageing effects showed that air pollution exposure accelerates the ageing process across all population groups, and revealed significant air pollution exposure differences between racial groups, which highlights the environmental justice element of the accelerated ageing due to air pollution exposure [16].

3.2 Sedentary Behavior and Physical Inactivity

The prevalence of sedentary lifestyle has been drastically rising due to contemporary lifestyle and occupation, and population surveys suggest the majority of older adults are sedentary for 4-8 hours a day. Short periods of exercise do not completely reverse the correlation with accelerated aging as measured by multiple epigenetic clocks (Hannum, Horvath, PhenoAge, and GrimAge) seen in genome-wide association study (GWAS) data [18]. In the Lothian Birth Cohort 1936, objective accelerometry measurement used to confirm the link between sedentary time and epigenetic age acceleration, which was independent of overall movements [19]. To support these conclusions, the Bed rest, limb immobilization and space flight models have shown that prolonged physical inactivity directly induces molecular markers of cellular ageing regardless of caloric consumption [17].

3.3 Sleep Deprivation and Circadian Disruption

Circadian disruption caused by shift work, artificial night lighting, and digitally mediated evening activity leads to an increase in allostatic load, a composite measure of the physiological wear and tear. A meta-analysis supports the relationship between sleep disturbances, defined as too little and too much sleep, with higher levels of allostatic load [20]. Biologically, sleep loss disrupts the normal rhythm of cortisol, raises the level of pro-inflammatory cytokines, impairs lymphatic clearance of neurotoxic waste, and shortens the window for tissue repair (when growth hormone is highest), which presents a clear mechanistic connection between the effect of sleep on aging and its disruption.

3.4 Chronic Psychosocial Stress and Allostatic Load

The "weathering hypothesis" suggests that individuals who are socioeconomically disadvantaged are more likely to experience chronic exposure to social adversity that lead to molecular ageing [10]. Overstimulation of hypothalamic–pituitary–adrenal (HPA) axis and chronic hypercortisolemia are associated with accelerated shortening of telomeres, reduced DNA repair, and decreased autophagy [9]. Early adverse events seem to be setting up a neuro-inflammatory overdrive response that’s characterized by delayed recovery, loading up allostatic load over the lifespan, and exacerbating epigenetic age acceleration in older age [10].

3.5 Ultra-Processed Food Consumption

Ultra-processed foods (UPFs) diets have quickly become a trend worldwide, according to the NOVA classification system. Within a larger population analysis of NHANES, UFP intake was found to be linearly correlated with increased phenological Age Acceleration (biological age) for every 10% increase in UFP intake, in the range of 27 months greater than chronological age [21]. The overall diet quality explained only 52.2% of this association indicating that industrial food processing itself and not only its nutrient composition is responsible for biological aging [21]. Chronic low-grade inflammation (increased level of IL-6, TNF-alpha and C-reactive protein), gut microbiome imbalance, damage to the epithelial barrier and endocrine disruption due to contaminants in packaging are proposed mechanisms. Although the data on the epigenetic clock in older ages is still preliminary, systemic reviews have consistently found that higher UPF consumption is associated with higher risk of incident frailty, dyslipidaemia, kidney disease and cognitive impairment [23].

3.6 Social Isolation, Loneliness, and Digital Media Use

Chronic loneliness and social isolation are equal to other established risk factors including smoking and obesity, and have been associated with systemic inflammation, immune dysfunction, cardiovascular disease and higher cancer mortality rate [25]. For the mobility challenged elderly, the advent of modern digital communication creates a paradox -being able to connect with others, and sometimes feeling cut off in groups of young people that use social media in excess [25]. On the other hand, the longitudinal cohort studies show that being digitally excluded (lacking access to digital tools) per se can be a risk factor for depression in older people [26, 32], there are clearly psychosocial risks related to digital overuse and to digital exclusion.

3.7 Convergent Biological Mechanisms

Across all six environmental and behavioural domains, activated physiological pathways converge on core hallmarks of aging: chronic low-grade inflammation, oxidative stress, telomere attrition, mitochondrial dysfunction, epigenetic drift, loss of proteostasis, and cellular senescence [9]. This mechanistic convergence indicates that the modern exposome does not act through isolated biological cascades, but rather operates as an interconnected, multi-factorial network with synergistic pathological effects presenting direct targets for multi-domain preventive interventions.

3.8 Urban–Rural and Developed–Developing Country Disparities in Accelerated Aging

3.8.1 Developed versus Developing Country Disparities

The differences between developed and developing countries not evenly distributed globally and a considerable body of cross-national evidence has emerged that supports the idea that the differences in age gaps between countries is independent of economic development. A large-scale exposome study, based on 40 countries and over 160,000 individuals, revealed a gradient of increased ageing in low income countries versus high income countries, with the fastest ageing occurring in Africa and Latin America compared to Europe and Asia, and within Europe, eastern and southern countries versus northern and western [27, 28]. A complementary global measure of societal adaptation to aging across 143 countries also revealed that indicators designed for high income countries are not directly applicable to low and middle-income countries (LMICs) and that most of the gender gaps in aging-related wellbeing are found in LMICs [29]. This trend confirmed by a 2026 meta-analysis based on data from 140 studies and nearly 66,000 people in 23 countries, which found that exposure to socioeconomic disadvantage and discrimination is consistently link with accelerated epigenetic ageing, regardless of chronological age [30].

3.8.2 Urban versus Rural Disparities

The urban rural gap is a layer of disparity that exists over and above the country disparity, and is not as uniform. A nationally representative sample of U.S. adults demonstrated that several epigenetic clocks were associated with greater aging in adults living in rural regions than urban regions, and that there were regional differences with the Northeast showing greater aging than the South and Midwest [31]. This is not a straightforward transposition of the developed versus developing differential: In high-income countries, rural residents can experience multiple disadvantages such as decreased access to health care, younger population out-migration, and concentrated poverty that can result in accelerated aging profiles that can be as severe as (or more severe than) those observed in low-resource urban areas. Neighbourhood level analysis at a finer spatial resolution has revealed that low scores on the childhood opportunity scale (poor education, environment and economic conditions) are an independent predictor of a more rapid rate of biological aging even when controlling for individual race, income and education [35].

3.8.3 Underlying Mechanisms

The accelerated aging gradients observed across both global economic levels (LMICs vs. high-income countries) and rural–urban divides, likely related to two overlapping but distinct factors. First, populations in low- and middle-income countries (LMIC) are exposed to childhood stressors like famine, conflict, and natural disaster, that are associated with accelerated aging in children as young as those exposed in utero [36]; and there are fewer resources for healthcare delivery and less access to biomarker technologies that are required to identify and monitor accelerated aging.

In contrast, high-income city populations more frequently exhibit accelerated aging through the various pollution, sedentary, and psychosocial stress pathways described in Sections 3.1-3.4. The notion that 'modern society' does not age everyone in the same way and in the same manner but in a development-stage-specific and geographically patterned manner has direct implications for the nature of public health interventions delivered at the national and subnational levels.

Table 1: Representative studies linking modifiable features of modern society to accelerated biological aging.

Study (Author, Year)	Population / Design	Exposure	Aging Biomarker	Key Finding
Feng et al., 2025	13 observational studies; meta-analysis	N/A (predictor analysis)	DNAm epigenetic age acceleration (EAA)	EAA significantly associated with increased stroke risk (OR 1.16, 95% CI 1.13–1.19)
Nwanaji-Enwerem et al. / KORA cohort studies (Oncotarget 2016; Env. Health Persp. follow-ups)	German KORA F4/FF4 population cohort, ~1,600-4,100 obs.	Long-term PM2.5, PM10, black carbon, NOx	DNAmAge acceleration; telomere-length age (TeloAA)	IQR increase in PM2.5 associated with faster extrinsic epigenetic aging; air pollutants inversely related to DNAm telomere length
McGuinn et al. / CATHGEN cohort studies	Urban US cardiovascular cohort, ~1,000 participants	Traffic-related air pollution (residential roadway proximity)	Horvath and PhenoAge acceleration; transcriptomic age	Epigenetic age acceleration amplified the association between traffic pollution and peripheral arterial disease
Lawrence et al. (ScienceDirect, 2023)	US women, Black and White (n≈ 4,126)	Ambient PM2.5, PM10, NO2	DunedinPACE, GrimAge, PhenoAge acceleration	Air pollution exposure associated with faster pace-of-aging biomarkers, with disparities by race
Wang et al., 2025 (Leisure-Time PA/MR study)	GWAS meta-analysis, 34,710 participants of European ancestry	Leisure screen time vs. moderate-to-vigorous physical activity (Mendelian randomization)	Hannum, Horvath, PhenoAge, GrimAge acceleration	Genetically predicted sedentary screen time causally accelerated epigenetic aging; effect not fully offset by short exercise bouts
Marioni et al. / Lothian Birth Cohort 1936	248 adults, age 79, accelerometer-measured	Objectively measured sedentary time and steps	Extrinsic and intrinsic epigenetic age acceleration	More sedentary time and fewer sit-to-stand transitions associated with greater epigenetic age acceleration
Sabatino et al., 2022 (narrative review)	Bed-rest, limb-suspension, and spaceflight human models	Extreme sedentary behavior / immobilization	Telomere attrition, genomic instability, mitochondrial dysfunction	Prolonged immobilization reproduces multiple hallmarks of cellular aging independent of physical activity level
Carroll et al. (meta-analysis, 2022)	Multiple cohorts; systematic review and meta-analysis	Sleep disturbance (short/long sleep, poor quality)	Allostatic load composite biomarkers	Sleep disturbance significantly associated with higher allostatic load; long sleep (≥9h) also elevated risk
Cardoso et al., 2024 (Age and Aging)	NHANES 2003–2010, USA, n = 16,055, adults 20–79y	Ultra-processed food (UPF) intake (NOVA classification)	PhenoAge acceleration	Each 10% increase in UPF intake widened biological–chronological age gap by ≈ 2.4 months; association partly independent of overall diet quality
Frontiers in Nutrition, 2026	Synthesis of cohort and	UPF consumption	Inflammatory markers (IL-6, TNF-	UPF intake linked to chronic low-grade inflammation and

(narrative review)	mechanistic studies		α, CRP), gut dysbiosis markers	microbiome disruption proposed as mediators of accelerated aging
Fitzgerald et al., 2021 (pilot RCT)	43 healthy men, aged 50-72	8-week diet, sleep, exercise, and relaxation program	Horvath DNAmAge (saliva)	Treatment group showed 3.23-year lower DNAmAge versus controls (p = 0.018)
Fitzgerald et al., 2023 (case series)	6 women completing 8-week lifestyle program	Methylation-supportive diet and lifestyle intervention	Horvath DNAmAge (blood)	5 of 6 participants reduced biological age by 1.2–11.0 years; mean reduction 4.6 years
Kuo et al., 2022 (RCT secondary analysis)	Older adults with obesity, 12-month trial	Caloric restriction ± exercise	KDM biological age, PhenoAge	Diet and diet-exercise arms showed continued reduction in biological age versus control/exercise-alone

4 DISCUSSION

4.1 Air Quality and Climate Change

This reviewed here provides a new way of thinking about several existing public health issues, including those related to air quality, physical inactivity, sleep disruption, chronic stress, poor nutrition and social disconnect as chief drivers of a common and measurable biological process accelerated biological aging. This strategic shift is very useful in practice. Aging biomarkers represent the cumulative physiological damage and are a global indicator of aging, associated with commonalities along multiple disease pathways before clinical outcomes occur. Contrary to the markers of disease (e.g., attributing obesity to the consumption of ultra-processed foods), biological age markers used in a wide range of disease processes. [33, 37].

4.2 Health Equity and Environmental Justice

There are significant socioeconomic and racial differences on several domains observed, such as exposure to traffic-related air pollution, chronic psychosocial stress (weathering hypothesis), and lack of access to healthy food environments. Using accelerated aging as a physiological proxy, structural inequality is observable as health inequality. This highlights the importance of shifting towards structural rather than individual level public health policy interventions, which must designed to create equitable changes.

This inequity issue is not limited to racial and socioeconomic differences within countries. Indeed, the fact that biological aging is steeper in low-income countries and in under-resourced rural areas compared to high-income cities indicates that accelerated aging is not simply a consequence of "modern" over-consumption, but also a hallmark of the under-resourced environment of rural areas, characterized by low socioeconomic status and limited access to protective measures. This twofold dynamic makes any one narrative of policy especially difficult: interventions aimed at reducing the effects of pollution- and sedentary-behaviour-induced age among wealthier and urban populations would not help lower-income populations who are afflicted by a phenomenon of aging from famine, conflict, and infrastructure. Therefore, public health strategy should not be singular, based on a "modern lifestyle" approach, but be geographically and economically differentiated.

4.3 Reversibility and Intervention Potential

Preliminary clinical trial and pilot study evidence suggests that epigenetic “age acceleration” is not reversible. Multiple small-scale cohort studies have shown that multi-domain lifestyle changes that include dietary optimization, sleep hygiene, physical activity, and stress management can significantly decrease epigenetic age in just 8 weeks or less [34, 38, 39]. In addition, interventions focused on diet and exercise have demonstrated long-term epigenetic aging reversibility for older adults with obesity after 12 months [30], and specific metabolic optimized interventions have shown reversibility [41]. These initial interventional findings suggest a public health message of change and transformation over biological inevitability, but will need to replicate in a larger, more diverse population.

4.4 Policy Implications

- Air Quality Standards: Implement strict emission standards and traffic restrictions in urban areas, considering the dose-response relationship between PM exposure and epigenetic age acceleration.

- Workplace & Urban Design: Use policies to encourage breaking up long hours of sitting in workplaces and built environments outside of campaigns that promote general physical activity.
- Sleep Health Advocacy: include sleep hygiene and circadian alignment in public health messages and clinical screening as clinically relevant behaviours and contributors to aging beyond perceived as ancillary comfort initiatives.
- Food System Reform: Implement food-system and front-of-pack food labelling policies that take a more holistic approach than macronutrient profiles to encourage reduce ultra-processed foods consumption.
- Social Infrastructure: Support and fund social programs, intergenerational programs, and fair access to digital space as separate pathways to risk.
- Biomarker Integration: Use biological age biomarker as tool for population based health surveillance to identify vulnerable individuals before clinical disease.
- International capacity building and technology-transfer initiatives to expand access to biological-age biomarker assays in low- and middle-income countries, paired with geographically differentiated public health strategies that separately address urban-industrialized and rural/low-resource drivers of accelerated aging.

Limitations and future directions

- The present review identifies various critical connections between contemporary exposome stressors and faster aging of the biological system; however, there remain some important limitations in the literature that could suggest key future directions for research:
- Observational Reliance and Causality: The existing body of literatures are predominant based on methods such as cross sectional and observational cohort studies, limiting the generalizability of causal inferences.
- Prospective longitudinal cohorts, Mendelian randomization studies, and randomized controlled trials (RCTs) needed to conclude causality and should be priorities in future research.
- The direct cross-study comparison is also complicated because of the methodological variation between epigenetic clock algorithms (first generation vs. second/third generation clocks, tissue choice and CpG panels) [6]. The harmonisation of the biological ageing panels in epidemiological studies is essential for clinical translation.
- Global Diversity and Generalizability: Existing cohort studies tend to be limited to North American and European populations. In order to be applicable to public health on global level, biological aging processes need to expand under-represented groups from sub-Saharan Africa, South Asia, and Latin America.
- Implementation and Scalability of Interventions: The feasibility, adherence and cost-effectiveness of multi-domain lifestyle intervention in the population level remains unclear and needs further evaluation, however, by larger, multi-center pragmatic trials that show the promise of reversing epigenetic age acceleration in pilot studies [37-41].

5 CONCLUSION

Biological aging distance is constantly associated with modifiable characteristics of modern industrialized surroundings, air pollution, physical inactivity, sleep and circadian disruption, chronic psychosocial stress, UP diet and social or digital isolation. These exposures converge on shared biological hallmarks such as systemic inflammation, oxidative stress, telomere attrition and epigenetic drift. Accelerated aging poses a biomedical challenge and a great health equity issue, since these environmental burdens are borne by disadvantaged socio-economic groups. Data from interventional studies suggest a possible way partially reverse epigenetic age acceleration, thereby making biological aging a public health target. To take these insights and advance into scalable public health policies requires attention to the current gaps in method, prospective designs, standardized biomarker panels, and globally representative cohorts.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Disclosure of conflict of interest

The authors declare no conflicts of interest regarding this manuscript.

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