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Editorial Comment

New Directions in Geroscience: Integrating Social and Behavioral Drivers of Biological Aging

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Abstract

The “geroscience hypothesis” posits that slowing the physiological processes of aging would lead to delayed disease onset and longer healthspan and lifespan. This shift from a focus on solely treating existing disease to slowing the aging process is a shift toward prevention, including a focus on risk factors found in the social environment. While geroscience traditionally has focused on the molecular and cellular drivers of biological aging, more fundamental causes of aging may be found in the social exposome - the complex array of human social environmental exposures that shape health and disease. The social exposome may interact with physiological processes to accelerate aging biology. In this commentary, we review the potential of these insights to shape the emerging field of translational geroscience. The articles in this special issue highlight how social stress and social determinants of health are associated with biomarkers of aging such as inflammation, epigenetic clocks, and telomeres, and spotlight promising interventions to mitigate stress-related inflammation. For geroscience to incorporate the social exposome into its translational agenda, studies are needed that elucidate and quantify the effects of social exposures on aging and that consider social exposures as intervention targets. The life course perspective allows us to measure both exposures and aging biology over time including sensitive periods of development and major social transitions. In addition, given rapid changes in the measurement of aging biology, which include machine learning techniques, multisystem phenotypes of aging are being developed to better reflect whole body aging, replacing reliance on single system biomarkers. In this expanded and more integrated field of translational geroscience, strategies targeting factors in the social exposome hold promise for achieving aging health equity and extending healthy longevity.

Introduction:

The National Institute on Aging defines geroscience as research that seeks "to understand the genetic, molecular, and cellular mechanisms that make aging a major risk factor and driver of common chronic conditions and diseases of older people" (1). The major thrust of this work is to test "[the geroscience hypothesis", which "posits that since aging physiology plays a role in many – if not all – chronic diseases, addressing aging physiology will allow a reduction or delay in the appearance of multiple chronic diseases" (2).

Environmental and social exposures are fundamental causes of aging. Though the roots of geroscience lie in the biology of aging, emerging research and transdisciplinary dialogue have illustrated ways that social and behavioral science can enhance geroscience and in fact are critical to help us understand and predict a large part of the variance in aging trajectories. Notable examples include presentations at the 2019 Geroscience Summit (3), a 2021 meeting of the Academy of Behavioral Medicine Research on *Optimal Longevity: Mechanisms, Reducing Health Disparities, and Increasing Healthspan,* and a series of essays published in *Aging Research Reviews* (4–6). These contributions highlight the role of the social exposome (7) - environmental exposures, social determinants, and psychosocial stress - as causal drivers of individual differences and population subgroup disparities in biological aging trajectories. These insights can inform an expanded Translational Geroscience, the study of understanding how to alter fundamental processes of human aging in vivo across the lifecourse (8,9).

Aging biology is a mediator between exposures and disease. This recent work in geroscience seeks to identify processes at the environmental, sociocultural, and behavioral/psychological

level that interact with aging biology to shape trajectories of physical aging and disease and sustain or undermine physiological resilience. An increasing number of publications examine aging biomarkers, including DNA methylation (epigenetic) clocks, inflammation, and multi-system phenotypic aging, in the context of longitudinal surveys of life course development and aging (10–14). Linking behavioral patterns, psychological states, and social environmental exposures to the molecular and cellular mechanisms of biological aging may identify critical behavioral or social/environmental targets for interventions that delay biological aging, extend healthspan, promote optimal functioning, and reduce health disparities at the population level. These efforts directly address the National Institute on Aging's (NIA) mission to "understand the nature of aging and the aging process, and diseases and conditions associated with growing older, in order to extend the healthy, active years of life" (15).

Health disparities are partly explained by an expanded Translational Geroscience--incorporating environmental, social, and individual factors. Unfortunately, opportunities for optimal health and functioning are not equally distributed in our society. Health disparities, those "preventable or avoidable health difference[s] that [are] closely linked with social, economic, and/or other environmental disadvantage" persist (16). In the NIA Health Disparities Research Framework, Hill et al (17) illustrate the multiple interacting levels of analysis needed to understand the causal drivers of health disparities and provide examples of malleable (preventable/avoidable) mechanistic processes at the environmental, sociocultural, behavioral, and biological levels that could be targets for intervention. Environmental causes include racial segregation and exposure to environmental toxins; sociocultural causes include features of cultural institutions and interpersonal processes like prejudice or norms that may result in social

exclusion; behavioral causes include health behaviors or psychological processes that operate as transducers of the aforementioned exposures. The framework also points to potential biological pathways through which these exposures manifest. "Ultimately," the framework's authors note, "we are interested in the pathways that environmental and sociocultural factors – often transduced through psychological and behavioral processes – become biologically embedded" (17). The inclusion of several hallmarks of biological aging in this framework incorporates the assumption that exposures and behavioral processes are causal drivers of biological aging. For maximal impact on population aging, interventions to ameliorate health disparities may need to target more fundamental environmental and sociocultural causes; those processes operating further upstream in the causal continuum.

The framework also emphasizes the need for a **life course perspective** (18). This takes into account structural factors -- how social identity and status lead to higher risk and lower resources, as well as developmental factors – where socially patterned exposures during sensitive biological periods increase disease risk (19). To answer many of these questions, we need longitudinal data from well-characterized, diverse samples to understand when aging processes begin, the timing and cumulative impact of exposures on health, periods of enhanced susceptibility or resilience, and when and how biological dysregulation associated with aging manifests. Existing deeply-phenotyped longitudinal social and behavioral science studies (See, for example Reference (20), with their rich assessments of stressors, lifestyle factors, and environmental exposures, alongside biomeasures and functional and health outcomes, are poised to help us answer these questions. The issue of timing is critical. Because the exposures and experiences that drive accelerated aging operate over the full life course, interventions may need

to occur in earlier life phases, when declines may still be reversible or can be compensated for; that is, when it is possible to build resilience or sustain resilience and slow the pace of aging. Lifecourse perspectives allow us to understand sensitive reproductive periods for females that impact aging, puberty, pregnancy, and menopause (21). Evidence also suggests that exposure to adversity during childhood has a stronger link to accelerated cellular aging throughout the life course (6,22,23). There may be other key developmental or social transitions that create opportunities for behavioral plasticity, serving as periods of potential risk or health enhancement, such as starting school or jobs, job loss and retirement, new parenting, marriage and divorce, and bereavement.

Progress in geroscience will take a "village" of collaborative interdisciplinary researchers and methods. Researchers in social and behavioral science fields aligned with psychosomatic medicine, such as life course epidemiology, sociology, public health, psychology, psychoneuroimmunology, behavioral medicine, and biological psychiatry are poised to offer insights. Social and behavioral scientists develop and refine measures of the exposures that predict aging. They examine their malleability and test hypotheses about the time scales, life stages, and magnitudes at which they shape health. They apply these approaches to study health disparities across and within populations and examine individual differences in trajectories of aging. Answering these questions can offer insights into when one should start anti-aging interventions, and who is most likely to benefit.

A trend toward increased early mortality. A recent National Academies report (24) starkly documents how the conditions that drive accelerated aging and premature mortality, including

mental distress and heightened cardiovascular risk, are driving increases in mortality among working age groups (25 to 64) in our society, with deaths especially high in black men and at rates unprecedented in comparable societies in the 21st century. The report serves as a timely reminder that not everyone in the United States gets to age optimally, or even live to the age of 50, and that research is needed to get at the causal drivers of these trends and to develop approaches to ameliorate them. A translational geroscience informed by behavioral and social science, conducted within a robust health equity framework, holds potential for discoveries in this field to lead to optimal health span for all. **This new science would expand our focus on biological manifestations of aging to include attention to the fundamental drivers of accelerated aging in the environments in which people live.**

Translational Geroscience. More work is needed to draw parallels between the complementary frameworks of social/behavioral science and geroscience, within a life course perspective. We can foster and explore their complementarity and bridge existing disciplinary silos to encourage development of more holistic frameworks for thinking about aging. This will involve behavioral and social studies adopting some of the exciting new biomarkers of aging. But full incorporation of "social hallmarks" (4) in geroscience studies in diverse populations and across the entire life course should also be a goal.

To date, Translational Geroscience has focused on trials to alter the aging biology that has been the focus of geroscience. As shown in Figure 1, an expanded Translational Geroscience focuses on examining biological aging through an environmental, social, psychological, and behavioral lens. Foundational drivers of human aging at these levels of analysis may interact with cellular

and molecular hallmarks of aging - particularly during sensitive periods across the life course, such as during early development, childhood, puberty, pregnancy, and menopause/andropause - to alter aging trajectories and contribute to health disparities.

Recent NIA-sponsored workshops (25,26) have sought to foster transdisciplinary dialogue among biological, social, and behavioral scientists to advance research on how behavioral processes and social and environmental exposures "get under the skin" to cause changes in biological processes responsible for accelerating or slowing aging and accelerating or delaying the onset of disease and frailty. The opportunity to discuss work across disciplines has been a highly generative way to bring attention to a broader range of potential intervention targets for translational geroscience.

Highlights of this issue

The current issue of *Psychosomatic Medicine* includes eleven articles examining evidence that psychological, sociocultural/environmental, and behavioral factors relate to molecular, cellular, and systemic markers of biological aging and thus may contribute to health risk and resilience across the lifespan. Six of the articles shed light on the association of **psychological factors with markers of aging**. These studies include a mouse model showing that exposure to chronic stress negatively impacts hippocampal-related cognitive function but does not impact tauopathies that are thought to contribute to the etiology of Alzheimer's Disease (27). Five studies focus on the impact of psychological factors, especially social stress, on markers of aging in human populations. Madison et al., examine circulating inflammatory markers, established biomarkers of aging that may play a role in the pathophysiology of comorbid aging-related diseases (28).

They find that among older, but not younger, adults, use of aggressive conflict resolution tactics in long-term romantic relationships relates to increased systemic inflammation whereas use of constructive tactics relates to lower levels of a primary proinflammatory marker. Wilson et al (29) also focus on the impact of relationship stress among older adults. Using data from The Health and Retirement Study (HRS) and the Mexican Health and Aging Study, they show that strained relationships are particularly detrimental for health outcomes, relating to risk for age-related functional limitations, poor self-rated health, and associated comorbidities. Together, these two studies highlight the health significance of social relationships and suggest the protective benefits of supportive relationships for markers of aging and health outcomes among older adults.

The microbiome has been understudied with respect to well-being. The article by Guimond et al (30) examines eudaimonic well-being (purpose in life and a sense of mastery), a psychological factor that may promote healthy aging. They find high eudamonia relates to a lower abundance of bacterial species known to associate with poor health outcomes in the gut microbiome of older adults. Telomere shortening is an accepted marker of cellular senescence and organismal aging, supporting intergenerational transmission of accelerated biological aging. Rinne et al (31) examines the possibility that post-traumatic stress among mothers impacts the biological aging of their offspring. They show that preconception symptoms of maternal stress and levels of systemic inflammation during the second trimester of pregnancy relate to shorter telomere length in 4-year-old offspring. Taken together, these six studies (maybe cite here) point to psychological risk and resilience factors that may impact trajectories of aging, contribute to the prediction of health span, and be modifiable by intervention.

While much of the extant literature focuses on factors that associate with aging among older populations, recent attention has begun to take a lifespan perspective and shows that socio-environmental exposures earlier in the life span contribute to trajectories of biological aging. Three articles in the current issue focus on these influences. De la Rosa et al (32) shows a cross-sectional inverse association between residential exposure to air pollution across a 12-month period and telomere length among children aged 1-11 years. A dose-response relationship is suggested, as associations are stronger in children who were exposed to more adverse social contexts. Two other studies in this issue focus more broadly on the impact of individual and neighborhood socioeconomic circumstances on markers of biological aging. Surachman et al (33) examine SES of individuals in the Midlife in the United States (MIDUS) cohort and show an association with age -related decrements in glomerular filtration rate, a marker of kidney function. Reed et al (34) also examine socioeconomic exposure, but in this case at the level of the community in which people resided in childhood and across adulthood. Independently of individual SES, the relative wealth and quality of the community in which an individual resided across childhood associates with cellular markers of immune aging among older adults, an effect that was mediated by cytomegalovirus (CMV) seropositivity. Reed et al. propose that socioeconomic contexts in childhood may increase risk for CMV infection and thus negatively impact trajectories of immune aging across the lifespan. Interestingly, among older adults who were seronegative for CMV, exposure to higher socioeconomic environments across adulthood was protective against immune aging. Taken together, these studies focus attention on the importance of considering socioeconomic exposures across the lifespan for understanding when and how biological aging trajectories become embedded.

In addition to psychological factors and environmental exposures, converging evidence shows that behavioral factors impact biological aging. In this issue, the article by Kusters et al (35) focuses on the impact of short sleep and insomnia on biological aging using DNA methylation (epigenetic) clocks. Among the HRS cohort, regularly sleeping less than 6 hours, and/or having insomnia is associated with epigenetic age acceleration. Health behaviors that contribute to interindividual differences in biological aging trajectories provide key targets for intervention to delay biological aging and extend the healthspan.

The final two studies in the current issue relate to the possibility of interventions designed to modify the impact of psychological factors on biological aging. Lindsay et al. (36) conducted a randomized controlled trial of a mindfulness-based stress reduction (MBSR) intervention delivered to older adults who endorsed loneliness. When compared to a health-enhancing control condition, the MBSR group shows intervention-related decreases in proinflammatory gene expression. These results are not reflected in circulating markers of inflammatory mediators, suggesting that MBSR may down-regulate the expression of inflammatory genes, but not the release of proinflammatory cytokines into circulation. To the extent that proinflammatory gene expression is a biomarker of biological aging and health risk, results suggest the health benefit of a psychosocial intervention for a high-risk group of older adults. Syed et al (37), examine whether metformin, an antidiabetic drug hypothesized to delay aging and protect against the progression of aging-related diseases, buffers the association of depressive symptoms with markers of inflammation among older adults. This was not a clinical trial, but compared individuals in MIDUS and the Sacramento Area Latino Study on Aging who were taking metformin with those who were not. Among non-users of metformin, the expected positive

association of depressive symptoms with inflammation was observed; however, in MIDUS, this association was not observed among those taking metformin, supporting that metformin may decrease the impact of psychological symptoms on biological aging. However, the same pattern of results was not observed in the Latino Study on Aging raising questions about the generalizability of the findings.

These two studies identify a psychosocial and a pharmacological intervention that may modify the impact of psychosocial factors on inflammatory markers of biological aging, possibly extending health span. In addition to replicating the improvements in inflammation, future longitudinal studies should examine the durability of the findings and whether the changes in biological indices of aging mediate improvements in health outcomes.

Summary

There has been tremendous progress in understanding how social exposures shape disparities in aging and population health. Evidence continues to accrue that factors in the social exposome influence the rate of biological aging, independent of chronological age. Geroscience interventions have been focused – to date - on how we can slow aging biology at the individual level, using behavioral and pharmacological means. By widening the scope of Translational Geroscience to include environmental, social, and psychological causes of aging, we expand our potential targets of intervention. Social and public health measures (e.g., improvements in air quality, education reform) could lead to better aging outcomes and propel us closer to health equity (9,10). Intervention focused on targets in the social exposome may decelerate aging at the cellular level and so interrupt a potentially bidirectional cycle between cellular aging and aging-

related outcomes (dementia, physical disability, healthspan). While social stress is a commonly studied predictor, there is also a critical need to understand both individual and social/structural factors that promote well-being and physiological resilience, including community or psychological assets that may buffer some of the negative sequelae of the social exposome and lead to better aging outcomes for individuals and population subgroups (38).

In addition to the recommendations throughout this commentary, we suggest four approaches that will deepen our understanding of how the social exposome shapes biological aging, to inform an expanded translational research agenda.

- **Include measures of environmental and social exposures**: Whenever possible, studies should include measurement of environmental exposures, social determinants, and psychosocial stress and assets. Well-validated measures that capture the timing, duration, intensity, and chronicity of exposures are essential (See: Stress Measurement Toolbox, www.stressmeasurement.org). In clinical trials, attending to these factors will lead to better findings with more external validity (5).
- **Take a life course approach**: Longitudinal studies in diverse, representative samples, spanning the full life course, can reveal divergence in trajectories of biological aging, uncover malleable targets, and identify windows of biological or behavioral plasticity to optimize the timing of interventions (e.g. sensitive periods in childhood, reproductive transitions, developmental and life stage transitions).
- **Advance causal understanding**: Adopt modern causal inference methods to exploit rich, longitudinal data, including quasi-experimental studies, and leverage natural experiments

(when possible) to study the impact of the social exposome on aging. Consider short-term experiments embedded in deeply-phenotyped longitudinal studies to probe physiological resilience.

- **Promote innovation in biological aging assays for population studies:** The most informative designs will include longitudinal tracking of biological aging hallmarks at multiple timepoints in diverse populations. Single biomarker studies are limited and there is a need to integrate multiple biomarkers of aging. Given advances in technology, machine learning methods, and ability to examine multi-omics, it is increasingly possible to examine multisystem phenotypes of aging. It will remain important to measure functional and clinical markers of aging and resilience to validate pace of aging metrics and whether they predict disease outcomes over time. These will be critical metrics for measuring meaningful change in rate of aging in shorter term interventions.

Ultimately, to determine whether preventing or reducing deleterious effects of the social exposome impacts aging, we will need longer observations and larger trials conducted over longer time frames in populations that are representative of individuals with different levels of financial, educational, social, and health resources.

Research will progress rapidly with the use of shared data and publicly available resources (39). Transdisciplinary collaboration will also be essential. The National Institute on Aging (NIA) and the National Institute of Environmental Health Sciences (NIEHS) have funded a Telomere Research Network to identify gold-standard measurement protocols for inclusion of telomere

assays in population studies (22). This network provides a model of how other fields can promote collaboration to improve the reliability and accuracy of complex biological measures.

The field of geroscience, in a short period, has already been successful in creating collaborative transdisciplinary synergy, catalyzing new research networks and new lines of biosocial and biobehavioral scientific inquiry. Accelerating progress in translational geroscience will come through deeper appreciation of the social exposome as a fundamental driver of biological aging, thereby broadening the reach and impact of the field.

Author Note

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Disclaimer: *The perspectives represented in this editorial should not be interpreted as representing the official viewpoint of the U.S. Department of Health and Human Services, the National Institutes of Health or the National Institute on Aging, except where noted.*

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FIGURE CAPTION

Figure 1: An Expanded Translational Geroscience spans from social to genomic factors across the life course.

An individual's aging biology is influenced by the social exposome, from environmental to individual factors (top triangle) and multisystem biological functioning (from physical function to genome) (lower triangle). Examining both social and biological factors, as well as interactions between them, should enable the most complete prediction of an individual's risk for early disease and mortality. An ideal social exposome will foster slower aging biology over the lifecourse, and ultimately healthy aging trajectories with a compressed diseasespan. The social factors offer additional intervention targets for slowing aging. This Figure is adapted from one that Dr. Nancy Adler used in talks to emphasize that precision medicine needed to incorporate the social factors to be of greater utility in public health.

Figure 1