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Anti-aging technoscience & the biologization of cumulative inequality: Affinities in the biopolitics of successful aging

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ABSTRACT

This paper charts the emergence of under-remarked affinities between contemporary anti-aging technoscience and some social scientific work on biological aging. Both have recently sought to develop increasingly sophisticated operationalizations of age, aging and agedness as biological phenomena, in response to traditional notions of normal and chronological aging. Rather than being an interesting coincidence, these affinities indicate the influence of a biopolitics of successful aging on government, industry and social science. This biopolitics construes aging as a personal project that is mastered through specific forms of entrepreneurial individual action, especially consumption practices. Social scientists must remain alert to this biopolitics and its influence on their own work, because the individualization of cumulative inequalities provides intellectual and moral justifications for anti-aging interventions that exploit those inequalities.

Introduction

Over recent decades, anti-aging technoscience and a body of social scientific work on aging have, separately but simultaneously, expanded intellectually and institutionally in pursuit of a common aim. Both have sought to develop sophisticated operationalizations of age, aging and agedness in a more molecular manner. Though highly distinct, these two enterprises are remarkably aligned in ways that are currently under-explicated. This paper unpacks the affinities between anti-aging technoscience and social scientific engagements with biological aging, particularly as they relate to the observation that inequality is cumulative, increasing exponentially across the life course. Doing so draws attention to the potential for innovation within social scientific research on aging to conform to a broader biopolitics of successful aging as a personal project that is mastered through specific forms of entrepreneurial individual action. In this context, social scientific appeals to the badness of unequal biological aging risk providing justifications for technoscientific interventions that exacerbate inequalities in later life.

The paper proceeds in four parts. First, I outline contemporary anti-aging technoscience's need to pathologize aging as a means of gaining regulatory status and legitimizing its activities. Second, I explicate the remarkable, yet largely unremarked upon, affinities between anti-aging attempts to pathologize age and the attempts of some social science, principally at the intersections of demography and functionalist social

gerontology, to biologize age, both in response to problems with more traditional notions of age. Third, I note that social scientific innovations regarding biological age hold considerable potential for extending the longstanding tradition of sociological work on cumulative (dis)advantage, revealing the potential for inequality to age us unequally. Finally, I argue that the individualization of social thought within a biopolitics of successful aging provides a cautionary tale for bio- and social-gerontology. While increasingly sophisticated operationalizations of biological aging draw our attention to biosocial inequalities, they are simultaneously conducive to justifications for technoscientific interventions that are already increasing those inequalities.

Anti-aging technoscience

Over the past three decades, public, media and research interest in anti-aging therapeutics has increased substantially (Vaiserman & Lushchak, 2017). The contemporary growth of anti-aging technoscience, representing the latest iteration of millennia-long interest in artificial healthspan and lifespan extension, can be traced back to the 1990s (Fishman, Binstock, & Lambrix, 2008; Iparraguirre, 2018; Le Bourg, 2017). It emerged out of growing insight into molecular aging, coupled with rising political alarm regarding global population aging and associated welfare expenditure (Vaiserman & Lushchak, 2017). Over subsequent decades, a combination of demographic concern, animal

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experimentation and promissory science has transformed anti-aging technoscience from a matter of *if* to a matter of *when*, symbolically at least (Mykytyn, 2010).

While numerous animal-model studies over the past three decades have suggested that aging is biologically malleable (Vaiserman & Lushchak, 2017), it is largely the institutionalization of anti-aging technoscience that has contributed to the field's development (Mykytyn, 2010). The American Academy of Anti-Aging Medicine (A4M) was founded in 1992 to promote the anti-aging technoscience agenda (A4M, 2020). Today, it convenes two annual world congresses with several thousand attendees, offers research fellowships and two masters programmes, and boasts over 26,000 members, 85% of whom are physicians (A4M, 2020). The American Federation for Aging Research (AFAR) was established in 1981 to encourage scientists to pursue careers in aging research. It has awarded over \$181million in grants to projects working on molecular aging (AFAR, 2020a) and is now worth over \$20million (AFAR, 2019). The Strategies for Engineered Negligible Senescence Research Foundation (SRF) was founded in 2009 to promote anti-aging research. It offers research grants, internships and post-baccalaureate programmes, and organizes numerous conferences (SRF, 2020), while its charismatic founder, Aubrey De Grey, has become an anti-aging figurehead. The field boasts several dedicated peer-review journals and special issues in other notable scientific publications (Dumas & Turner, 2007). Anti-aging technoscience has hence accrued considerable institutional weight (Mykytyn, 2010; Petersen & Seear, 2009). That said, a substantial field of anti-aging medical practice has also emerged (Fishman, Settersten Jr, & Flatt, 2010).

It is important to partially define contemporary anti-aging technoscience, because the anti-aging field is vast and diverse, ranging from popular skin creams (Searing & Zeilig, 2017; Smirnova, 2012) to cryotherapies based on exposure to extreme cold (Farberg, Donohue, Farberg, Teplitz, & Rigel, 2017). While acknowledging that the boundaries between purported categories of anti-aging are vague and porous (Vincent, 2009), this paper focuses on institutionalized endeavors that explicitly seek to technologically intervene in the molecular processes of biological aging to address various age-associated disorders and extend healthspan (the period of life lived in good health). Its advocates are therefore generally more concerned with reducing later life morbidity and disability than extending lifespans indefinitely (Mackey, 2003). This healthspan focus deliberately distinguishes contemporary anti-aging technoscience from pseudo-scientific historic efforts to bolster its scientific legitimacy (Moreira, 2015). It also reflects the emergence of societal concerns regarding the continued extension of global life expectancy, and fears that growing populations of older people are spending more time in (economically undesirable) ill-health (Crimmins, 2015). As a social entity, anti-aging technoscience is intriguingly ambiguous, on the one hand resembling a social movement advocating utopian societal transformation, and on the other representing a form of biotech venture capitalism chasing a rapidly growing market (Neilson, 2006).

Intellectually, contemporary anti-aging technoscience exploits tensions between clinical and molecular gerontology. While age-related conditions are approached clinically as distinct diseases with discrete aetiologies, molecular evidence suggests that they are instead co-rooted in "senescence" (Lees, Walters, & Cox, 2016; Tchkonja & Kirkland, 2018). Senescence is a broad biological process characterized by progressive physiological degeneration and functional decline over time (Comfort, 1979). It entails decreasing DNA repair and increasing mutation, and the accumulation of damaged proteins (Kirkwood, 2005; Tchkonja & Kirkland, 2018). The effects of this process are manifest in many familiar forms – tooth enamel wears away, arteries harden, bone density diminishes – and it also leads to various notable conditions (Rattan, 2005; Tchkonja & Kirkland, 2018). For example, the pathological pathways of neurodegenerative conditions such as Parkinson's, Huntington's and Alzheimer's disease can be traced to senescent processes (Abrahams, Haylett, Johnson, Carr, & Bardiën, 2019). Indeed,

age is the strongest risk factor for Alzheimer's disease and is the only significant risk factor for the oldest-old (Ganguli & Rodriguez 2011; Guerreiro & Bras, 2015). However, despite such associations, caution is required here. The mechanisms involved remain poorly understood, and uncertainties persist regarding whether the quasi-pathophysiological phenomena associated with aging are pathogenic, pathognomonic or protective (Castellani et al., 2006; Nelson, Braak, & Markesbery, 2009).

Senescence sits within a wider bio-gerontological history of attempts to explain the aging process, spanning over 300 different theories across more than two centuries of scholarship (Jin, 2010; Medvedev, 1990). These theories of aging can broadly be categorized as extrinsic (damage is done to the organism) and intrinsic (degeneration is preprogrammed in the organism). The *wear-and-tear* theory, attributed to influential 19th century bio-gerontologist August Weismann, attributes aging to the random accrual of damage resulting from the organic processes of life, wherein cells eventually degrade through repeated use. *Wear-and-tear* has long been contested by proponents of intrinsic aging models. For example, the *aging-clock* theory contends that the aging process, through conception, development and senescence, is an evolutionary legacy that is genetically and hormonally managed, potentially benefiting a species by minimizing the competition faced by new cohorts. This is based on the observation that specific hormones are typically produced at specific points in the lifecourse to optimise the organism for certain requirements, particularly with regards to reproduction (Moody & Sasser, 2018). Another popular intrinsic theory of aging is *cellular senescence*, not to be confused with senescence, which describes the observation that cells are generally limited to a certain number of divisions (the "Hayflick limit", see (Hayflick, 1965)), after which they die.

While some advocates of singular foundational causes remain, most bio-gerontologists now reject the likelihood of a grand universal theory of aging. Indeed, the major theories that have been put forward, e.g. *wear-and-tear* and *aging-clock*, are not mutually exclusive. Hence, the long search for a fundamental driver of aging has gradually given way to recognition that the aging process is characterized by the complex interplay of both extrinsic and intrinsic factors (Jin, 2010; Weinert & Timiras, 2003). Nonetheless, various attempts at theorisation typically approach senescence as the result of the hypothesized processes (Moody & Sasser, 2018). Importantly for this paper, these bio-gerontological debates have traditionally shunned disease-based accounts of aging and have refuted anti-aging claims. It is instead molecular biologists who have emphasized the similarities between aging and disease, inspiring anti-aging aspirations (Moody & Hayflick, 2003).

Irrespective of fundamental causes of aging, anti-aging technoscience seeks to intervene in senescence to address numerous complex conditions, in a manner that blurs aging and disease (Vaiserman & Lushchak, 2017). It thus centres on a type of boundary work, reimagining aging, disease and intervention in molecular terms, in opposition to clinical convention (Fishman et al., 2008). Historically, clinical medicine has successfully addressed several acute illnesses stemming from discrete pathologies, and efforts to cure chronic illnesses often emulate this approach, targeting distinct processes, albeit with less success (Rosenberg, 2002). The specialization of medicine focuses interventions on single components of health issues, but the physiological complexity of aging undermines the isolation of discrete processes (Childs, Durik, Baker, & Van Deursen, 2015; Vincent, 2006). Proponents of a molecular approach argue: i) that curing any single condition may have little impact on healthspan due to the abundance of comorbidities in older populations, ii) that the interrelatedness of comorbidities means that targeting one often produces unintended influences on another, and iii) that intervening in each condition individually is more resource-intensive than targeting shared fundamental processes (Vaiserman & Lushchak, 2017; Vincent, 2006).

These contested notions of aging and disease catalyse traditional sociological concerns of normality and abnormality (Dumas & Turner, 2007). Canguilhem (1998) famously described the modern concept of disease as being founded on the idea of a spectrum of quantified

normality and abnormality, wherein pathology is an extreme variation of a possible range of physiology, deviating from a healthy average. This quantified approach is an attempt to develop value-neutral definitions of (ab)normality. The ontological status of pathology is central to contemporary anti-aging technoscience's challenge to clinical medicine's normalization of aging as a natural physiological process that does not warrant intervention (Janac, Clarke, & Gems, 2017). The public and professional positioning of aging beyond the scope of technological intervention aligns with fears regarding the broader socioeconomic implications of "curing" aging. Normalization thus precludes both the possibility and the rightfulness of intervention (Dumas & Turner, 2007; Fishman et al., 2008; Mackey, 2003; Vincent, 2006). A crucial institutional manifestation of normalization is the Food and Drug Administration's (FDA) refusal to define aging as an indication (an officially sanctioned target for intervention). Regulatory bodies therefore will not review agents that explicitly target aging, inhibiting pharmaceutical development (Fleming, Zhao, Seoh, & Barzilai, 2019; Newman et al., 2016; Stambler, 2017). In this context, anti-aging appeals to molecular science are attempts to pathologize aging in order to encourage regulation (Vaiserman & Lushchak, 2017; Vincent, 2006).

A revealing example of the regulatory vacuum is young blood transfusion. Since the early-2000s, experiments based on linking the cardiovascular systems of young and old mice – a procedure named *parabiosis* – have shown that the blood of young mice can rejuvenate older mice and reverse age-associated conditions (Kaiser, 2014). In 2017, Californian start-up "Ambrosia" began selling plasma from young human donors as an anti-aging therapy, based on parabiosis mouse-models. They did not require dedicated clinical trials because human plasma is already transfused for other medical purposes. Though rarely entering the anti-aging field, in 2019 the FDA issued a warning against anti-aging blood transfusion. Ambrosia ceased trading shortly after, but young blood research continues, anti-aging stakeholders continue to advocate it, and plasma remains available to consumers (Pandika, 2019). Ambrosia represents an interesting juncture in anti-aging technoscience, firstly because the company functioned for two years selling products based on molecular science, and secondly because its activities moved the FDA to intervene. Despite distancing itself, the FDA is central to anti-aging technoscience because, although the field has recently accrued various forms of capital, the regulatory position on aging limits its core claims to animal-based conjecture about human potentials (Vaiserman & Lushchak, 2017).

A major initiative to address the regulation vacuum is AFAR's Targeting Aging with Metformin (TAME) clinical trial. TAME hopes to test Metformin, a first-line diabetes treatment, as an anti-aging intervention to treat various age-associated conditions (AFAR, 2020b). TAME is based on experiments in which Metformin has extended the lifespans and healthspans of nematodes and rodents (Barzilai, Crandall, Kritchevsky, & Espeland, 2016). Importantly, TAME also aims to achieve the symbolic and political goal of providing proof-of-concept of "aging" as a practicable indication (AFAR, 2020b). The TAME team have successfully negotiated a new indication with the FDA for the purposes of measuring aging-related outcomes. This indication resembles multimorbidity, being comprised of several conventional age-associated chronic diseases that are intended to collectively represent aging, albeit in a manner that satisfies FDA conventions and circumvents the difficulties of explicitly defining aging. This composite indication, including cancer, cardiovascular disease and Alzheimer's disease, will measure a range of biomarkers associated with senescence, creating a blueprint for future anti-aging trials (de Grey, 2019; Fleming et al., 2019). Thus, institutionalizing a redefined molecular aging is central to the progression of anti-aging technoscience both symbolically and pragmatically.

Biological age

While contemporary anti-aging technoscience has been gathering momentum over recent decades, social scientists have separately been

developing biological operationalizations of aging (Jylhävä, Pedersen, & Hägg, 2017). These operationalizations, remarkably similar to those desired by the anti-aging project, have emerged from a longer tradition of American biogerontology (Moreira, 2015) and have inspired a marketplace of companies selling personal biological age measurements (Moreira, Hansen, & Lassen, 2020). However, this paper focuses on contemporary social scientific iterations, especially evident at the intersections of functionalist social gerontology and demography. Notions of "biological age" center on measuring physiological and functional attributes that typically change over time (e.g. declining lung capacity) and assigning a population-based numerical "age" scale to those attributes (Iparraguirre, 2018). Anti-aging technoscience and certain social scientific approaches to aging are hence intriguingly aligned in their positioning of age, aging and agedness as molecular matters. Indeed, the development of biological age in the social sciences represents something of an epistemological and methodological progression of TAME's multimorbidity indication, providing an accessible and quantifiable molecular metaphor for aging.

Understanding contemporary efforts to operationalize aging in terms of biology requires some historical perspective on the evolution of concepts of age in the social sciences. "Social" age is the version of age that has traditionally sufficed for everyday purposes (Rose, 1972). In practice, we often deem people to be young or old without needing to ask for their ages directly. Instead, we draw on a range of indicators, such as appearance, behavior and the broader context of the interaction, to interpretively ascribe fitting age-identities. This social age is arguably the most experientially significant type of age in everyday life (Bytheway, 2005; Macnicol, 2008). Beyond social age, social scientists typically approach age as a continuous variable represented by yearly gradients (Baars, 1991). The resulting figure, denoting age as a number of years, is "chronological" age. It is mostly an empty variable and is rarely the entity of primary analytic interest, but provides an accessible proxy for assumptions regarding various biological, psychological and social states (Settersten Jr & Mayer, 1997). That said, chronological age is heavily naturalized in our everyday lives. If one asks another person for his/her age, that person will likely automatically reply with the number of years since his/her birth (Iparraguirre, 2018).

As with much quantification of human life, the current importance of chronological age is partly a modernist legacy (Cruz, 2017). Chronological age became a powerful operationalization of age through the 19th and 20th century institutionalization of the modern lifecourse. The spread of institutionalized age-based education, work and retirement practices required numerical standardization and exactitude that was not satisfied by the vagueness of social age (Anderson, 1985; Moreira, 2015). The need for a more generalizable and precise version of age was facilitated through the 19th century proliferation of record-keeping across industrializing states. These new records granted large populations personal chronological ages, and chronological age became an increasingly usable means of conceptualizing the age of oneself, others and populations (Bytheway, 2005). Within rapidly aging and industrializing states, chronological age gained power as a means of institutional and personal organization (Hacking, 1990).

While chronological age is often preferred as an indicator of age, it is not necessarily aligned with different types of age (Pickard, 2016). Consider the example of John and Kevin. John, a wealthy 70-year-old who plays tennis at his local club and organizes fundraisers for a local charity, is unlikely to be the same social or biological age as Kevin, a poor 70-year-old who is limited to his flat by morbidity and rarely socializes beyond 15-min visits from carers. John and Kevin share a chronological age, but their biological and social ages are dissimilar. As a result, the respective influences of age, broadly conceived, within their lives are also dissimilar. Kevin is more likely to be the subject of negative age-based appraisals because he seemingly personifies negative imaginings of dependency, abjectness and decrepitude in *old* age (Gilleard & Higgs, 2013). Kevin is an *aged* subject in ways that John is not.

Biological and social heterogeneity among older people means that it

is not always analytically useful to treat John and Kevin as members of the same category, despite their shared chronological status, especially if our primary interest is health outcomes. The utility of chronological age as a social variable has therefore been criticized since the mid-20th century, when several scholars argued that the broad heterogeneity within older populations meant that chronological age categories contained too much to be analytically useful (e.g. [Atchley & George, 1973](#); [Heron & Chown, 1967](#); [Murray, 1951](#)). Indeed, some scholars began to view chronological age as a hindrance to aging research ([Katz, 2006](#)). Instead, they argued for a broader range of constellation of ages, such as the social age discussed above. Among the new imaginings of age being pursued were functional variables to stratify a type of physio-functional age. The 1950s saw the first attempts to distinguish, measure and compare the functional and chronological ages of individuals, of which biological considerations were an important component ([Murray, 1951](#)). Over recent decades, work on biological age has emerged as an important component of the broader turn to diversified functional measurements of aging and is now an important facet of social scientific aging research. While functional age does engage with physiology under the guise of phenotypes (particularly behavioral), it pursues a broader approach to age in terms of psychological and social considerations. Biological age is principally concerned with biomarkers that denote senescence rather than questions of functionality *per se* and is thus indebted to Comfort's aforementioned work ([Moreira, 2016](#)). The turn to biology over chronology was evident in the British Society of Gerontology's recent statement on Covid-19, which "urge[d] the Government to reject the formulation and implementation of policy based on the simple application of chronological age" because population-level associations between chronological age and mortality "[wou]ld not be the case for all individuals, amongst whom biological age and immune responses vary greatly" ([BSG, 2020](#)).

As with anti-aging technoscience, social science's biological aging enterprise has relied on notions of senescence ([Iparraguirre, 2018](#); [Jylhävä et al., 2017](#)). A wide range of senescent biomarkers have been proposed as reliably and accurately documenting biological age. The most popular of these is telomere length, the focus of over 1000 studies of aging biomarkers ([Jylhävä et al., 2017](#)). Telomeres are found at the ends of chromosomes and shorten during cell division, meaning that telomere length decreases over time ([Lai, Wright, & Shay, 2018](#)). Another popular aging biomarker is DNA methylation, often referred to as the "epigenetic clock", a form of epigenetic modification that accumulates over time and can therefore be correlated with chronological age ([Chen et al., 2016](#); [Jylhävä, Jiang, Foebel, Pedersen, & Hägg, 2019](#)). Another notable approach to biological aging is the search for composite biomarkers, combining several factors, such as cholesterol and blood pressure, to produce weighted operationalizations of biological age ([Levine, 2013](#); [Levine & Crimmins, 2014a](#)). This composite approach is currently the strongest predictor of biological age but is far less tested than single-biomarker alternatives ([Jylhävä et al., 2017](#)). Ultimately, such research is moving toward evermore statistically sophisticated operationalizations of age, aging and agedness as essentially biological.

Tellingly, one of the key actors in the contemporary biological aging space has been AFAR, having proposed widely accepted criteria for a biological age biomarker. This biomarker must: 1) predict lifespan more effectively than chronological age; 2) measure a fundamental aging process rather than disease effects; 3) be easily, unobtrusively and repeatedly measurable; and 4) be translatable to animal models ([Jylhävä et al., 2017](#)). AFAR has a significant interest in the development of aging biomarkers because success could facilitate an aging indication. Recent operationalizations of biological age have had some success, and are generally much better than chronological age at predicting morbidity, disability and mortality ([Iparraguirre, 2018](#)). However, a biomarker is yet to be discovered that satisfies AFAR's criteria. Given the apparent inseparability of disease from aging, some researchers have questioned whether it is possible to satisfy the second criterion ([Jylhävä et al., 2017](#)). Indeed, if biological aging is defined in reference to molecular

characteristics of chronic disease, it follows that biological age is a strong predictor of morbidity. Thus, social scientific work on biological aging inadvertently substantiates anti-aging technoscience's pathologization of aging through the circular conflation of aging and disease.

Cumulative inequality

A major sociological implication of the recent biologization of age is that it reveals strong socioeconomic associations. Several biomarkers commonly used to denote biological age, e.g. telomere length and DNA methylation, are inversely associated with measures of socioeconomic status, e.g. education level, social class and parental income ([Hughes et al., 2018](#); [Iparraguirre, 2018](#)). Such findings suggest that social disadvantage ages people, or rather leads people to senescence, faster than their more privileged counterparts. For example, the financial hardship faced by African American women can accelerate their biological aging, and the greater average biological age of African Americans accounts entirely for their comparatively high mortality rate ([Levine & Crimmins, 2014b](#); [Simons et al., 2016](#)). In this manner, biological age reveals health inequalities that chronological age does not, making possible new analyses of the potential for disadvantage to worsen health outcomes ([Ferraro & Shippee, 2009](#); [Levine & Crimmins, 2018](#)).

This linking of social and biological inequalities in aging contributes to, and extends, one of the most influential areas of social scientific aging research – cumulative (dis)advantage (CAD). CAD denotes the tendency for inequality to increase over the lifecourse as intra-cohort trajectories diverge exponentially ([Crystal & Shea, 1990](#); [Dannefer, 2003](#); [Platt, 2019](#)). CAD stems from [Merton's \(1968\)](#) research on the "Matthew effect" in institutional science practices of communication and reward, whereby individuals with initial small advantages accrue greater advantage over time ([Bask & Bask, 2015](#)). In the late-20th century, sociologists of aging began to explore similar mechanisms in relation to lifecourse inequality ([Crystal & Shea, 1990](#); [Dannefer, 1987](#)). These early applications have subsequently developed, theoretically and empirically, into a strong CAD tradition within social scientific work on aging ([Dannefer, 2018](#)).

Today, a sizeable body of scholarship explicates CAD in various contexts ([Crystal, Shea, & Reyes, 2016](#); [Dannefer, 2018](#)). Studies have principally focused on the relationships between socioeconomic factors and later life physiological and psychological outcomes. For example, [Damaske and Frech \(2016\)](#) have used the National Longitudinal Survey of Youth to reveal that the employment trajectories of women in the United States (US) are significantly constrained by their early life socioeconomic circumstances. Focusing on health outcomes, [Ferraro, Schafer, and Wilkinson \(2016\)](#) have used the US' Midlife Development dataset to show that socioeconomic disadvantage in childhood is associated with greater lifestyle risk factors in adulthood and subsequently increased morbidity. Similarly, [Shrira and Litwin's \(2014\)](#) analysis of data from the Survey of Health, Aging and Retirement in Europe has revealed that increases in depressive symptoms and functional decline over time are larger for those exposed to greater adversity and hardship. In sum, a broad body of social research indicates that initial advantage generally begets greater advantage, and vice versa, though it should be noted that CAD is not without contestation (see [Kim & Durden, 2007](#)).

CAD has been important in revealing "aged heterogeneity", the tendency for a range of inequalities to be greater among people of older ages than among people of different ages ([Dannefer, 1987](#)). As with John and Kevin, the differences between two 70-year-olds may be far greater than the differences between a 70-year-old and a 45-year-old. Historically, the preponderance of chronological age within social scientific aging research, particularly in the functionalist social-gerontological and demographic veins, concealed this intra-age diversity, but since the late 20th century CAD has successfully drawn greater attention to aged heterogeneity ([Stone, Lin, Dannefer, & Kelley-Moore, 2017](#)). Moreover, contemporary research suggests that inequalities within chronological age groups are increasing, rendering CAD evermore

pertinent to social scientific engagements with aging (Grenier et al., 2019). This growing recognition of intra-age diversity is intimately bound up with the aforementioned problems of using chronological age as a social variable, potentially concealing more than it reveals. CAD's problematization of chronological age therefore provides further justification for the use of biological age as a worthwhile analytic route into aging and later life.

CAD also highlights the dependence of biological age on chronological age, because it is in reference to chronological age that the biologic nature of cumulative inequality is revealed. For example, to be biologically 40 is rather meaningless by itself, but to be biologically 40 and chronologically 50 may be normatively appraised as a success (e.g. Levine & Crimmins, 2018). There is, of course, an irony here in that biological age is intended to be a challenge to chronological age, and yet it relies on chronological age to generate meaning (Katz, 2006; Moreira, 2015; Moreira et al., 2020). The biologization of age as an alternative to chronology has facilitated the application of CAD to the molecular and phenotypic characteristics that are employed as aging biomarkers. There is limited support for claims to the effect that being biologically 40 and chronologically 50 is more likely the more socioeconomically advantaged one is, but this is nonetheless an increasingly popular trope of biosocial research (Kelly-Irving & Vineis, 2019; Robertson et al., 2013; Stringhini & Vineis, 2018; Thyagarajan & Levine, 2019). In this manner, parallel social scientific work on CAD and biological age over the past few decades has converged to reveal novel biologic entanglements of aging and (dis)advantageous exposure. To an extent, this work adopts a social determinants of health approach to aging, wherein 'health' is directly substituted with 'age', so that age is recast as a type of negative outcome. This would not be so problematic if, as will be discussed further, those social determinants were not similarly substituted with personal responsibilities. There are hence two simultaneous conceptual substitutions here.

Finally, biology has itself become a popular variable, or set of variables, within CAD research. Ferraro and Shippee (2009) have promoted a model of cumulative inequality, based on CAD, that aims to unpack lifecourse determinants of health in later life with particular reference to biological factors. Their proposals are based on the observation that sociological research on CAD is remarkably aligned with molecular research on senescence. Both delineate long-term effects of the lifelong accumulation of various exposures. This alignment is ultimately echoed in cumulative inequality's focus on uniting sociological and biological aging research, or perhaps translating the former into the latter. To this end, their foundational text states:

We seek to develop meaningful links between [cumulative inequality] theory and selected findings and models of biologic processes associated with aging... we hope to illustrate how [cumulative inequality] leads to biologic changes in humans that are commonly associated with the process of growing older. In this sense, inequality may well lead to the accumulation of biologic materials under the skin that are markers of aging and predictive of senescence (Ferraro & Shippee, 2009: 334).

From this initial outline, cumulative inequality has gained popularity within social scientific aging research and has been used to frame various correlations between early life exposures and later life health outcomes (e.g. Ferraro et al., 2016; Kemp, Ferraro, Morton, & Mustillo, 2018). Its pursuit of the intersections of biological age and CAD has generated imaginings of later life in terms of unequally accumulated senescence. This senescence-centric use of lifecourse cumulation transforms the original CAD treatment of social (dis)advantage as an analytic focal point into a precondition of more important molecular mechanisms. Thus, the social inequalities that a biology-sensitive CAD can explicate are themselves biologized, so that ethnicity or income differences become DNA methylation or telomere differences. That said, cumulative inequality should not be read as a biological theory. It is

principally indebted to sociology, and particularly demography (though as will be discussed shortly, biological/sociological boundaries are increasingly blurred), and the biologization of aging is not an explicit aim.

The reimagining of later life inequalities as molecular matters is part of a broader history of scholarship seeking to integrate CAD and biological phenomena. In his foundational paper on CAD, Dannefer (1987) suggested the potential for future research to study relationships between social conditions and later life heterogeneity across blood pressure, immune function and testosterone levels. Though fleeting, this observation does reveal that notions of biology have always been at stake in gerontological scholarship on longitudinal inequality. This style of work has flourished into the substantial tradition of CAD research, discussed above, and has always been particularly conducive to extrinsic theories of senescence (Alkema & Alley, 2006). What is different in cumulative inequality is the move toward depictions of aging as a set of biomarkers, with the implication that inequality determines aging and agedness, rather than influencing issues associated with later life. For example, Simons et al. (2020) have recently used the aforementioned epigenetic clock to show that social adversity and discrimination faced by African Americans accelerates their aging. Aging is here defined as DNA methylation. They conclude: "social disadvantages commonly experienced by Black Americans exhibited both unique and combined effects on accelerated aging" (Simons et al., 2020: 7). This builds on Levine and Crimmins' (2014b, 30) earlier study, which concluded that: "racial differences in the pace of aging—as signified by biological age—may be a central mechanism for the earlier overall and disease-specific mortality of black individuals." Aging here is a composite of ten biomarkers.

The biologization of CAD sits within a broader biosocial turn in the social sciences since the 1990s, with work on issues such as local biologies, epigenetics and the microbiome revitalizing social scientific relations with biology (Fitzgerald, Rose, & Singh, 2016; Landecker & Panofsky, 2013; Meloni & Testa, 2014). At the same time, similar developments in the biological science, such as epigenetics and distributed neurocognition, have rendered those sciences increasingly receptive to traditional social scientific concerns (Meloni, 2014). Across these fields, the question of *how the social gets under the skin* has become a central concern (Manning, 2019). Thus, the biologization of later life inequalities must be read within a broader recent history of a blurring of boundaries between historically compartmentalized, if not hostile, social and biological sciences. A striking example of this biosocial turn was a special issue of the American Journal of Sociology dedicated to "exploring genetics and social structure", which deftly contested the designation of certain types of research as either biological or sociological (see Bearman, 2008). Celebrating this development, Rose (2013) has suggested that biology is the future of sociology, and vice versa. However, Fletcher and Birk (2019, 2020) have cautioned against the biologization of social phenomena within this novel biosocial landscape, noting how complex psychosocial and socio-economic considerations are switched into epistemically convenient molecular characteristics. This creates the risk that those molecular characteristics are successfully ameliorated, but that such molecular amelioration has no impact on the problematic phenomenon that said molecules are purported to represent. Moreover, the biosocial turn is characterized by an emerging replicability problem, and should be approached with caution (Das, 2019).

In biologically-inclined work on cumulative inequality, the potentials of CAD are transformed in a similar fashion, from a means of uncovering underlying social causes of biological inequalities, into a means of delineating simplistic molecular solutions to the problems wrought by social phenomena that either preclude intervention, or for which interventions are met with political impediments. This is a major transformation because there are evidently considerable differences between addressing the effects of racism by tackling racism, and addressing the effects of racism by tackling DNA methylation. An

important corpus of critical scholarship in the CAD tradition engages with biology to emphasize the negative effects of social pathologies. For example, [Leopold \(2016\)](#) has shown how unequal education provision in early life put poorer Swedes at greater risk of unemployment during the 1990s leading to worse health outcomes in the 2000s. [Crystal \(2018\)](#) has charted the detrimental influence of growing economic inequality during the late 20th century on the later life health statuses of Americans born between 1956 and 1975. These studies point to the political economy as a progenitor of biological inequality, and thus as a site for potential intervention. However, there is always a risk that by attempting to engage with biology on its own terms, such work inadvertently facilitates the biologization of social inequalities. It is important to note that the critical tradition of CAD scholarship is not necessarily motivated by such biologization, and indeed largely rejects it, but that its insights are vulnerable to exploitation in the service of biologization. Ultimately, there is a fine line between, on the one hand, an approach to inequality that highlights its potential pathophysiological implications and explicates the ramifications of those pathophysiologicals for aging and later life, and on the other hand, an approach to inequality and aging as pathophysiological phenomena. Scholars should be alert to this distinction.

The biopolitics of successful aging

The affinities between contemporary anti-aging technoscience and certain social scientific approaches to aging are remarkable, yet rarely remarked upon. Over recent decades, both have pursued increasingly sophisticated operationalizations of aging in terms of biology in response to problems with more traditional approaches. Anti-agers have challenged the clinical normalization of aging as being distinct from discrete diseases, proposing in its place a more pathological molecular account of aging. Social scientists have criticized chronological age as an empty variable that conceals aged heterogeneity and inequalities, advocating biological operationalizations of age that help to reveal the extent of those inequalities. Under the guise of cumulative inequality, biological aging has gained greater attention as a type of inequality problem in its own right. This biologization of aged heterogeneity indicates the potential for social scientific engagements with aging to evolve in accordance with biopolitical ends that simultaneously guide anti-aging technoscience. In this final section, I argue that the biologization of age can be read within a broader history of social scientific insights on aging being reformulated to serve a biopolitics of successful aging that constrains our relations with age, aging and agedness ([Lamb, 2014](#)).

Several scholars have noted that anti-aging, social science and international governance manifest a biopolitics of successful aging ([Lamb, 2014](#); [Neilson, 2006, 2012](#); [Otto, 2013](#)). This biopolitics (a Foucauldian term denoting the governance of human life) encourages individuals to achieve desirable forms of aging through the right kinds of personal conduct. Intellectually, it is rooted in a longstanding “activity” tradition within social gerontology, positing that a good later life is dependent on maintaining certain kinds of activities, e.g. volunteering after retirement ([Havighurst, 1961](#)). Today, this prescription of the right kinds of later life, heavily contingent on a rationally self-governed able body, inspires the policies of several international institutions, variably articulated as “active aging”, “productive aging”, “healthy aging”, etc. (e.g. [EC, n.d.](#); [UN, 2018](#); [WHO, 2018a](#)). Most notable among these is “successful aging”, which defines a successful later life as being characterized by: 1) low probability of disease and disability; 2) high cognitive and physical function; and 3) active engagement with life ([Rowe & Kahn, 1998](#)). These measures of success are satisfied through appropriate individual action and their attainment is a personal responsibility. Critics have argued that this approach ignores social determinants and attributes deterioration in older age to personal failure ([Katz & Calasanti, 2015](#); [Martin et al., 2015](#); [Peterson & Martin, 2015](#)). Successful aging has also been criticized for universalizing experiences of aging based on

exclusionary white, middle-class, ableist values that diminish all other varieties of aging and agedness as comparatively ‘unsuccessful’ ([Baker, Buchanan, Mingo, Roker, & Brown, 2015](#); [Holstein & Minkler, 2003](#); [Minkler & Fadem, 2002](#); [Pace & Grenier, 2017](#)). Nevertheless, successful aging permeates global governance and social scientific engagement with aging ([Pruchno, 2015](#); [Pruchno & Carr, 2017](#)).

The biopolitics of successful aging responds to and reflects what [Moreira \(2017\)](#) has termed the epistemic assemblage of the aging society as a meaningful political entity. Here, a confluence of specific uses of demographic and economic data and analysis has rendered population aging a stark political issue through measurement, depiction, and especially the dramatic elucidation of its potential economic ramifications. Measurement is an essential apparatus of this assemblage, particularly as it relates to populations through the epistemological assumption that large human collectives manifest regularities that are empirically knowable. Population aging can also imperil such assumptions through undermining the actuarial knowledge-base upon which redistributive social security is predicated, in terms of the shifting proportionalities of age-groups, as well as uncertainties regarding the relations between life expectancy, health expectancy and morbidity, particularly given the possibility of longer lives lived with greater disability. What are states, organizations and individuals to do when the numbers paying in are fewer, and the numbers taking out are greater, than has been predicted and accounted for in welfare calculations?

Concerns regarding the implications of population aging, and especially morbid population aging, echo Malthusian population dispositifs that have long inspired imaginings of aging as an economic *problem*. In this vein, population aging is commonly presented across various fora as leading to scarcity and associated ills, e.g. conflict. Indeed, demography has been instrumental in generating representations of aging as a societal issue through placing its measurements alongside appeals to undesirable economic consequences. Such dispositifs are bound up with wider eugenic propositions relating bad biologies to bad economies, for instance, via claims that medicine’s artificial maintenance of the biologically inferior poor inadvertently undermines the economic prosperity of society at large ([Moreira, 2017](#)). As a population trait, aging comes to pose a significant societal risk, at the intersections of economics and biology. A biopolitics of successful aging is thus a response to the aging society, cast as a means of ameliorating the promissory economic ruin of demographic forecasting.

Foucault’s explication of biopolitics was famously short-lived, and biopolitical thought has since broadly split into two categories ([Neilson, 2012](#)) – that focused on macro political developments, espoused by scholars such as [Agamben \(1998\)](#), and that focused on molecular technoscience, knowledge and values, popularized in the works of scholars such as [Rose \(2007\)](#). This paper is principally embedded in the former tradition given its focus on a range of efforts to render age a molecular matter at an institutional level, amidst the familiar concerns of welfare, the state and the market. [Neilson \(2012\)](#) has attempted to intersect the two categories, uniting macro political concerns with the influence on molecular technoscience and associated values on contemporary manifestations of aging. His work is principally concerned with introducing the experience of aging into biopolitical scholarship. I draw on his work here where relevant, yet I am less concerned with the implications for experience, than I am with disciplinary epistemological machinations, specifically those at stake in certain social scientific developments. That said, such concerns are never entirely extricable ([Neilson, 2012](#)).

The ascendancy of a successful aging biopolitics has been attributed to its concordance with neoliberal capitalism ([Katz, 2006, 2013](#); [Rubinstein & de Medeiros, 2014](#)). It portends that proactive individuals can defeat the evils of aging through correct styles of consumption, underpinning a rapidly growing “third-age” marketplace offering products that facilitate a successful old age, or even the abolition of *old* age ([Gilleard & Higgs, 2000, 2005](#); [Kampf & Botelho, 2009](#); [Katz & Marshall, 2018](#); [Neilson, 2006](#)). Depictions of successful aging in the manner highlighted above are particularly conducive to a style of

biopolitics that relies on “technologies of the self”, wherein “the capacities of rational autonomy and self-determination – the *homo economicus* – are generated in entanglement with enactments of liberal, soft power” (Moreira, 2017: 37). The conviction that biological aging is dependent on individual action, and by extension consumption, is part of a biopolitics of aging to which certain sociologies have contributed (Gilleard & Higgs, 2000, 2005). CAD’s aforementioned recognition and explication of aged heterogeneity has diverted attention away from the social structuring of age, and age as a structuring social force, facilitating the individualization of biological aging as discrete exposures, encompassing products such as gym memberships and health foods (Hendricks & Hatch, 2009). Recognizing these affinities, critics have cautioned that the biologization of aging facilitates a biopolitics wherein older people are made “tractable in the interests of the state, commerce and other powerful elites” (Vincent, 2009: 202).

Noting the biopolitical entanglements of government, capitalism and gerontology, Neilson (2012, 51) argues that we must pay greater critical attention to “the intersection of financially driven rejuvenation medicine and policy discourses of ‘healthy’ or ‘positive aging’”. He notes that a dominant politics of individual responsibility simultaneously substantiates the erosion of welfare provision in later life and the flourishing of rejuvenation consumption possibilities and ethical imperatives, within which those consumers consider their practices to be agentic (Neilson, 2012). The booming third-age market offers considerable growth potential for longevity products, while healthspan extension is conducive to state initiatives to “extend working lives” (Dumas & Turner, 2007; Petersen & Seear, 2009). The contemporary expansion of anti-aging technoscience is particularly reliant upon venture capital and consumer demand for promissory biotech, and has hence become more amenable to the conditions of neoliberal capitalism (Neilson, 2012). Thus, anti-aging both replicates and responds to a broader biopolitics (Fishman et al., 2010). As an example, parabiosis was first developed in early-20th century Russia as a type of egalitarian blood-sharing or “physiological collectivism” (Bernstein, 2019: 69). Ambrosia’s recent foray into young blood transfusion is hence a reimagining of an originally cosmist endeavor to benefit the more liberal ideals of Silicon Valley. There is no obvious reason that these entanglements of government, capitalism and gerontology will change in the immediate future, and therefore no reason that the biologization of aging will not be further bent to the biopolitics of successful aging.

Such bending is not novel and is echoed in social science. Various strands of social thought have been molded to the biopolitics of successful aging under the guise of social scientific research. Katz’s (2013) account of “lifestyle” provides a good example, detailing the concept’s adoption and adaption by the more functionalist sections of mainstream social gerontology. Sociological work on lifestyle stems from Simmel and Weber. Simmel (1978) claimed that new lifestyles of social individualization, differentiation and alienation were created by modern financial economies. Lifestyle was not an agentic choice, but rather an attempt to solidify an identity amidst the tumult of social fragmentation. Weber (1978) considered lifestyle to be an overt manifestation of one’s desired social status, albeit constrained by one’s material circumstances. For each author, lifestyle was a compromise between choices and constraints, both of which were socially determined. Katz (2013) identifies the continuation of this approach to lifestyle through the more recent sociologies of Bourdieu (1984) and Giddens (1991, 1999), each centering on syntheses of agency and structure. However, the concept of lifestyle has been individualized within the sociology of aging and stripped of its ability to unpack structural social determinants (Hendricks & Hatch, 2009). Far from the structural constraints articulated by Weber, lifestyle in social scientific aging research has become a composite of moralized personal decisions, typically centering on smoking, diet and exercise (Katz, 2013).

Intuitively, a concept of aging as cumulative biological inequality might appear well-aligned with critical social scientific perspectives on structural inequalities, providing molecular justifications for equality-

enhancing social interventions. However, as with lifestyle, notions of biological aging have generally been individualized in social scientific work in the more functionalist and demographic sections of social gerontology, presenting biological age as a personal responsibility, of which lifestyle is an important component (Kampf & Botelho, 2009; Katz & Marshall, 2018). This is echoed in individualized anti-aging, under the guise of superior *personalized* medicine (Fishman et al., 2010). Industries, researchers and governments attempt to map out the behaviors of optimal later life and devise strategies for encouraging their uptake by aging citizens. Such interventions are often well-intentioned efforts to reduce human suffering, albeit articulated as individual problems of dependency and societal problems of welfare expenditure (Katz, 2006, 2013). Moreira (2015) has identified the turn to biological age as a core infrastructural process supporting this individualization of the life-course. Such biological individualization reflects earlier functionalist sociologies of aging, wherein aging is envisaged as an intrinsic and impervious molecular universal, upon which secondary social phenomena play out.

This biological individualization of age generates new justifications for risk and intervention that are conducive to anti-aging (Katz, 2006). Contemporary anti-aging technoscience centers on notions of aging as individualized cumulative biological deterioration and associated imaginings of our aging molecular selves as projects to be improved. Through individualizing biological age, it promises personalized solutions to unsuccessful aging that avoid the kinds of largescale social interventions of which neoliberal governments are wary (Neilson, 2006). Social scientific engagements with biological aging perpetuate this individualization of inequality. As an example, Levine and Crimmins’ (2018, 400) analysis of racial and socioeconomic inequalities in biological aging “used race/ethnicity and education as covariates. ... due to their known associations with health behaviors.” Here we find social scientists explicitly repurposing complex social variables as proxies for undesirable activities. The aging subject is transformed, from a member of a minority ethnic group, to a smoker, to an unsuccessful ager.

Such operationalizations of biological age lay fertile ontological ground for anti-aging technoscience. First, age becomes a familiar type of technical challenge that is surmountable with sufficient technoscientific advancement. Second, the ascription of badness to biological aging, in reference to associated inequalities, furnishes moral justifications for technoscientific intervention, righteously providing those poorly educated ethnic minority agers (recast as unsuccessful agers) with the means to save themselves. Given that the recent development of contemporary anti-aging technoscience has relied on accruing conceptual legitimacy (Mykytyn, 2010), the social conceptualization of aging as unfair molecular deterioration is a powerful justification. Aging becomes a problem that is not only amenable to, but that also morally compels, technoscientific action.

These syntheses of technical and moral imaginings of biological aging are disconnected from real-world observations. Though infrequently articulated as such, recent human history resembles an anti-aging intervention, characterized by dramatic successes in the moderation of biological aging (Vaiserman & Lushchak, 2017). During the 19th and 20th century, global human life expectancy increased from below 30 to almost 67 years (Riley, 2001), and Levine and Crimmins (2018) have shown steady decreases in biological age, relative to chronological age, in recent decades. This real-world anti-aging enterprise is marked by substantial inequality. For example, international life expectancy ranges from 52.9 in Lesotho to 84.2 in Japan (WHO, 2018b). The means already exist to dramatically increase the lifespan and healthspan of Lesotho’s population, and yet Ambrosia and A4M are not championing such initiatives. This highlights a core anti-aging paradox: “On the one hand, there is a wish to prolong life and on the other, an indifference to the means to prevent premature death” (Dumas & Turner, 2007: 12). This example reveals that contemporary anti-aging technoscience, repeatedly presented as a straightforward problem of technoscientific progress, is not solely limited by value-neutral technical

feasibility. It is also constrained by a specific biopolitics of successful aging and the types of life, and types of person, that fit into its imaginary.

This observation that anti-aging is a fundamentally value-laden enterprise leads to a final question regarding to what extent a hypothetical fully functioning anti-aging technoscience might be able to address cumulative biological inequalities if it were not under the influence of successful aging. For example, could the original cosmist rationales of parabiosis be realized? First, it is important to reiterate that the successes of anti-aging to date are principally its founding of institutions and accrual of resources, rather than its production of any clinically effective interventions. Furthermore, its dissolution of boundaries between “serious” science and promissory populism undermines any attempt to ascertain its practical potentials. Putting technical feasibility to one side, it also seems unlikely that the biologization of inequality could ever furnish sincere solutions, because it attends to effects rather than causes, and is hence inherently more palliative than curative. Moreover, biologization distracts from social causes, undermining the case for interventions to target those causes. Even a cosmist approach would hence likely preclude the effective amelioration of the political and economic pathologies so deftly explicated by the critical social sciences. Can the affinities discussed herein be exploited for critical ends? Perhaps not.

Conclusion

The emergence of affinities between anti-aging technoscience and social scientific aging research is remarkable, yet rarely remarked upon. This paper has sought to address this deficit as a means of provoking more critical thought on social scientific engagements with biological aging. Both endeavors share a rejection of chronological aging as a form of falsely homogenizing temporal determinism that obscures more than it reveals. Instead, both favor sophisticated biological operationalizations that are intended to generate more legitimate, or perhaps more useful, depictions of age, aging and agedness. This is not simply an interesting parallelism; it is an indication of specific imaginings of age that propagate within, and contribute to, a broader biopolitics of successful aging. Ultimately, this biopolitics begets a blurring of boundaries (Neilson, 2012), whereby separate enterprises of biotech start-ups and methodological innovation converge to produce new intellectual infrastructures for governing later life.

Social scientists must remain alert to the ways in which their engagements with biological age reflect and extend specific biopolitics, firstly by individualizing age, aging and agedness in a manner that dismisses structural determinants (or at least makes them more culturally palatable), secondly by presenting age as a technoscientific problem, and thirdly by moralizing that problem as compelling technoscientific intervention. Insights into biological age have enhanced social scientific work on aging and health, and the links between the two, but they are vulnerable to corruption in the service of unintended, or even perverse, consequences. This is especially true in relation to CAD and cumulative inequality, which highlight problematic aspects of aging and inspire sincere desires to intervene and improve the lives of older people. Very different types of intervention can be justified in reference to biosocial insights into cumulative inequality, ranging from redistributive public pensions to the marketing of personal training regimes. Scholars must remain alert to the important similarities and differences between such initiatives.

Though long associated with the individual in mainstream gerontology (Moreira, 2015), biological age need not be an essentially individual concern. It becomes so within a biopolitics of successful aging (Neilson, 2006). The biologization of cumulative inequality offers considerable potential for drawing our attention to new and profound manifestations of structural inequalities. Yet within an influential portion of the contemporary sociology of aging, biological age is progressing down a theoretical route similar to that of lifestyle, tending

away from a complex structure-agency nexus and toward a simplified matter of personal action, and, poignantly, consumption. This microfication of biological aging is framed as a progressive endeavor. It facilitates better recognition of the great diversity of later life and draws attention to those for whom aging is a more detrimental phenomenon, yet it is also used to promote the sale of young plasma for \$8000 a liter to consumers who will go to extreme lengths to be less “old” (Pandika, 2019). Social science has the potential to either challenge or facilitate such scenarios.

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