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I, Peter T Tanksley, hereby submit this original work as part of the requirements for the degree of Doctor of Philosophy in Criminal Justice.

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From age to aging: Biological age and its role in the criminal career.

Student's name: **Peter T Tanksley**

This work and its defense approved by:

Committee chair: J.C. Barnes, Ph.D.

Committee member: Daniel Belsky, Ph.D.

Committee member: Hexuan Liu, Ph.D.

Committee member: Christopher Sullivan, Ph.D.



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From Age to Aging: Biological Age and its Role in the Criminal Career

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By: Peter T. Tanksley

M.S., University of Texas, San Antonio, 2016

B.A., University of Texas, San Antonio, 2014

July 13, 2020

Dissertation Committee:

J.C. Barnes, Ph.D. (Chair)
Hexuan Liu, Ph.D.
Christopher J. Sullivan, Ph.D.
Daniel W. Belsky, Ph.D.

Abstract

The age-crime relationship is one of the most canonical and law-like relationships that criminology has observed; however, the age-crime relationship is more complicated than criminological theories have yet appreciated. Criminologists generally view the influence of age in one of two ways: (1) invariant, inexplicable, and non-interactive—and thus scientifically uninteresting—or (2) as signifying nothing so much as the timing of certain developmental and societal events that are actually responsible for explaining the variation in crime. Both approaches view the age-crime relationship in a unidirectional manner (i.e., age impacts crime), never considering the opposite possibility. This dissertation reconsiders the age-crime relationship by applying a new conceptualization of age that is rooted in the physiological integrity of the human organism: biological age. By viewing age as a biological construct that indicates one's progress in the process of birth, maturation, senescence, and ultimately death, I am able to reverse the causal arrow and ask the question: how does crime influence (biological) age?

In order to explore the relationship between biological age and crime, I conduct three studies in which I leverage two recently developed methods for quantifying biological age and use data from two longitudinal cohort studies—the Health and Retirement Study (HRS; United States) and the Dunedin Longitudinal Study (DLS; New Zealand). In Chapter 1, I provide a brief introduction to three relationships that make up a tripartite structure: (1) age and crime, (2) age and health, and (3) crime and health. These interrelated literatures provide the theoretical and empirical premises for the rest of the dissertation. In Chapter 2, I examine the impact of lifetime incarceration on the biological age in the HRS and find that individuals with a history of incarceration tend to experience faster biological aging, but only among non-Black respondents.

In Chapter 3, I use the DLS to explore how early offending and criminal justice contact impacts biological aging throughout middle adulthood and find that criminal justice involvement (criminal conviction) predicts accelerated biological aging even after accounting for prior offending behavior. In Chapter 4, I again use the DLS to examine whether individuals who persist in offending longer than others also have a more accelerated pace of biological aging and find that the evidence is, at most, only suggestive. In Chapter 5, I summarize the overall findings of the study and describe how concepts like biological age may be situated in the current theories of age and crime in the criminological literature.

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Dedication

For my girls: Anna and Darwin. Nothing else needs be said, y'all are my world.

Acknowledgement

There is no end to the list of friends and family who have helped me throughout the years and throughout my time working on this dissertation. Beyond my wife, Anna, no one has helped and supported me during this process more than my advisor, Dr. J.C. Barnes. Thanks for everything, dude.

I have already made this paper too long, for which I must crave pardon, not having now time to make it shorter.

-Benjamin Franklin

Table of Contents

<i>Abstract</i>	<i>ii</i>
Dedication.....	v
Acknowledgement.....	v
List of Figures	ix
List of Tables.....	x
Chapter 1 —From Age to Aging: Introduction	1
Introduction.....	2
Crime ↔ Age	3
Age ↔ Health	4
Crime ↔ Health	6
Area 1—The Health Consequences of Criminal Justice Contact	6
Area 2—The Long-Term Health Effects of Early Offending Behavior	8
Area 3—Biological Age and the (Non-Normative) Desistance Process	10
Structure of the Dissertation.....	12
Chapter 2 —Biological Age and the Iatrogenic Effects of Incarceration	13
Introduction.....	14
Literature Review	16
Incarceration and Later Health Outcomes	17
Deprivation or Importation?.....	19
Physiological Stress, Organ Integrity, and Aging: Mechanisms of the Deprivation Model.....	24
Biological Age—Mechanism of The Incarceration-Health Relationship?.....	25
Measuring Biological Age.....	27
Methods	29
Data.....	29
Measures	30
Analytic Sample.....	33
Analytical Strategy	35
Results.....	39
Preliminary Analysis.....	39
Regression Analysis.....	42
Propensity Score Analysis	48
Discussion	57
Limitations	59
Conclusion.....	62
Chapter 3 —A Young Man’s Game: Early Offending Behavior and its Influence on Biological Age	64
Introduction.....	65
Literature Review	66
Offending and Health Outcomes Across the Life Course	69
Biological Age: Theoretical Construct, Quantification, and its Association with Health	73
The Current Study	79

Methods	80
Data.....	80
Measures	80
Analytic Strategy.....	86
Results.....	89
Preliminary Analysis.....	89
Regression Analysis.....	93
Inverse Probability of Treatment Weighting (IPTW) Analysis.....	94
Is Receipt of a Criminal Conviction Before Age 26 Associated with Pace of Aging?	99
Sensitivity Analysis.....	100
Discussion	102
Limitations	104
Future Directions.....	105
Conclusion.....	110
<i>Chapter 4 —Aging Out: Biological Age and its Role in the Desistance Process.....</i>	<i>111</i>
Introduction.....	112
Literature Review	114
Normative Desistance.....	115
Non-Normative Desistance	117
Aging Out.....	121
The Reason for Aging	124
Biological Age	126
Quantifying Biological Age.....	127
The Current Study	129
Methods	129
Data.....	129
Measures	130
Analytic Plan.....	135
Results.....	136
Group-Based Trajectory Modeling Analysis	138
A Four-Group Model of Desistance.....	141
Predicting Trajectory Group Membership—Does Pace of Aging Differ Across Trajectory Groups?...	142
Conclusion.....	149
<i>Chapter 5 —From Age to Aging: Discussion and Conclusions</i>	<i>150</i>
Discussion	151
Area 1—The Health Consequences of Criminal Justice Contact	152
Area 2—The Long-Term Health Effects of Offending Behavior	154
Area 3—Biological Age and the (Non-Normative) Desistance Process	155
Theoretical Considerations.....	156
An Integrated Vision of Biological Age and Developmental/Life-Course Theory	158
The Limitations of Biological Age.....	160
Conclusion	161
<i>References</i>	<i>164</i>
<i>Appendix.....</i>	<i>174</i>

List of Figures

Figure 1.1. The crime-health-age triad.....	3
Figure 2.1. Biological age as the general factor of chronological age, environment factors, and genetic factors.	26
Figure 2.2. Kernel density plot showing the distribution of PhenoAge Acceleration for males and females.	40
Figure 2.3. Bar graphs of PhenoAge Acceleration (z-score) by Lifetime Incarceration (A) in the analytical sample and broken down by (B) sex and (C) race/ethnicity.	44
Figure 2.4. Bar graphs of PhenoAge Acceleration (z-score) by Lifetime Incarceration in the analytical sample and broken down by race/ethnicity and stratified by sex.....	47
Figure 2.5. Absolute standardized mean differences across childhood covariates between treated and control cases. Note: mean=arithmetic.....	52
Figure 2.6. Variance ratios across childhood covariates between treated and control cases (Note: mean=geometric).	53
Figure 2.7. Kernel density plots of propensity scores for Lifetime Incarceration across treated and control cases.....	54
Figure 3.2. Histogram of Pace of Aging from phase 26-45, stratified by sex.	91
Figure 3.3. Box plots of Self-Reported Offending Variety across phases 15-26 and Average Offending Involvement, stratified by sex.	92
Figure 3.4. Absolute standardized mean differences across all covariates, before and after weighting with inverse probability of treatment weights.	97
Figure 3.5. Variance ratios across all covariates, before and after weighting with inverse probability of treatment weights.	99
Figure 4.1. Self-Reported Offending Variety across phases 26-45, stratified by sex.....	131
Figure 4.2. Histogram of Pace of Aging from phase 26-45, stratified by sex	132
Figure 4.3. Four-group trajectory model of Self-Reported Offending Variety from age 26-45.....	142
Figure 4.4. Group-specific Averages of Pace of Aging.....	146

List of Tables

Table 2.1. Childhood Covariates for Propensity Score Model of Lifetime Incarceration.	37
Table 2.2. Descriptive Statistics of the Health and Retirement Study Analytical Sample (N=7606).....	40
Table 2.3. Cross-tabulation of Lifetime Incarceration and Race/Ethnicity Across Sex.	41
Table 2.4. Ordinary Least Squares Regression of PhenoAge Acceleration on Lifetime Incarceration, Sex, and Race/Ethnicity, and Their Interactions.....	43
Table 2.5. Childhood Covariates Missingness, Counts and Sample Percentage.	49
Table 2.6. Covariate Balance Across Childhood Covariates.	51
Table 2.7. The Average Treatment Effect on The Treated (ATT) of Lifetime Incarceration on PhenoAge Acceleration (N=3036).....	55
Table 3.1. Constituent Biomarkers of Pace of Aging.	81
Table 3.2. Distribution of key variables across analytic (N=808) and non-analytic samples (N=123-229).	90
Table 3.3. Univariate statistics of key variables and t-test/chi-square test comparisons across male (N=407) and female (N=401) respondents (total N=808).	91
Table 3.4. Multiple Linear Regression of Pace of Aging on Average Offending Involvement and Developmental Trajectories.....	93
Table 3.5. Covariate balance metrics across before and after IPTW weighting.	97
Table 3.6. Average Treatment Effect Analysis of Conviction Status on Pace of Aging Using Inverse Probability of Treatment Weights and Sensitivity Analysis.	100
Table 3.7. Criminal Lifestyle Factors Implicated in the Acceleration of Biological Age.	107
Table 4.1. Univariate Statistics of Key Variables and T-Test/Chi-Square of Group Mean Differences Across Male (N=412) and Female (N=423) Respondents (Total=835).....	137
Table 4.2. Results from The Model Fitting Process of The GBTM Analysis.....	139
Table 4.3. Prediction of Offending Trajectory Group Membership among Males (N=384) with Pace of Aging and Covariates Using Multinomial Logistic Regression.	143
Table 5.1. Glossary.....	163

Chapter 1 —From Age to Aging: Introduction

Introduction

The relationship between age and crime is more complicated than is acknowledged by criminological theories. Traditionally, age has been viewed as a temporal construct. Developmental/life-course criminology has argued about where to place age in theories of crime causation; however, these debates were typically argued by a) those who saw age as scientifically uninteresting or b) those who saw age as little more than proxy for the timing of socially important events in the lives of offenders. Age was rarely thought to be more than a temporal status indicator; a mile marker. But age is more than that. Age is the ticking of a biological clock that determines many aspects of our lives. And it is this aging process that partly determines our health and well-being. To fully appreciate age and its role in the criminal career, then, criminological theories will need to embrace it from a different perspective.

This dissertation examines issues at the intersection of criminology, public health, and geroscience. My specific focus will be the integration of theoretical perspectives from these disciplines that have previously been considered in isolation but are all composed of interconnections between three common factors: crime, health, and age or the aging process. Three distinct literatures have grown up around these concepts; however, recent developments point to the possibility for consilience between them. This dissertation represents a first attempt at such an integration. In the sections that follow, I will briefly describe the focal relationships that make up the three sides of the triangle presented in Figure 1.1. In so doing, I will identify three key research questions that need to be addressed in order to facilitate an integrated model of crime, health, and age/aging. These three relationships will form the body of this dissertation and each will be the focal topic of a specific chapter and analysis.

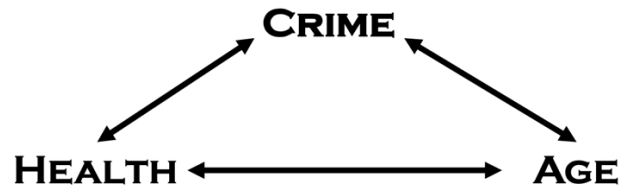


Figure 1.1. *The crime-health-age triad.*

Crime ↔ Age

Criminologists have known for almost two centuries that age exerts some influence on crime (see, e.g., Quetelet, [1831] 1984). The age-crime relationship has always been intriguing to criminologists; however, popular developmental/life-course perspectives in criminology have been somewhat limited in their explanation of the role of age in the criminal career. For instance, developmental/life-course criminology has historically viewed the age-crime relationship in one of two ways. The first view is that age is a purely exogenous influence on crime, rendering it practically inert, scientifically uninteresting, and plausibly ignorable (e.g., Hirschi & Gottfredson, 1983).

The other view of age is that it represents a temporal mile marker in the life course, demarcating the onset of various developmental (e.g., puberty, maturation; Glueck & Glueck, 1950; Moffitt, 1993) or societal events (e.g., marriage, joining the labor force, military service; Sampson & Laub, 1993) that shape the life course. According to this perspective, it is the events signaled by age—not age itself—that is of theoretical and practical import in the age-crime relationship. Both of these perspectives agree that age exerts *some* influence on offending, be it ‘direct but uninteresting’ or ‘mediated by the real difference makers’. What these perspectives do not consider, however, is that offending might influence age in return.

As a purely temporal construct, age is truly exogenous and cannot be influenced by lifestyle factors. But recent work out of the field of geroscience offers a potential third view of

the age-crime relationship by changing how scientists think of age. Rather than simply a temporal construct, age is perhaps better understood as a biological state that is dynamic and responsive to lifestyle factors (Belsky et al., 2017; Quach et al., 2017). This conceptualization of age is referred to as “biological age”, which can be broadly defined as the integrity of the various organ systems of the body at a given point in time. Viewing age in this way lends criminology the ability to more fully integrate *aging* into its life-course paradigms as a factor that both affects and is affected by the criminal lifestyle. The major consequence of a biological approach is that age and health become conjoined. Researchers will be unable to explore the association between biological age and crime without also considering health.

Age ↔ Health

During the past century, human life expectancy (i.e., the average lifespan in a population) has dramatically increased in the developed world. Owing mostly to improvements in the treatment of infectious diseases and the reduction of infant mortality, life expectancy in the United States has risen from 47.3 to 78.7 years between 1900 and 2010 (Arias, 2012; Murphy et al., 2013). Modern limitations on lifespan are now predominantly chronic, age-related conditions like cardiovascular diseases and cancers. The focus on lifespan extension has produced a number of unanticipated results, however, because individuals who live *longer* do not necessarily live *better*. The oldest population strata are characterized as having lower quality of life due to the prevalence of age-related conditions. By focusing on mortality without addressing rates of underlying morbidity, the upper age groups of our population are 1) living increasing numbers of unhealthy years and are 2) actually reducing overall population health (Crimmins, Hayward, & Saito, 1994).

In an effort to distinguish between the quantity and quality of years lived, geroscientists began emphasizing the idea of the “healthspan”, defined as “the period of life spent in good health, free from chronic disease and the disabilities of aging” (Kaeberlein, 2018, p. 361). Unlike lifespan—measured as the number of chronological years lived from birth to death—healthspan has no obvious metric. However, recent years have seen improvements in the measurement of a concept that may provide much needed insight in the healthspan: biological age. Measurements of biological age probe the underlying health and functional status of an individual. Specifically, biological age refers to the “declining integrity of multiple organ systems” (Belsky et al., 2015a). Because organ integrity is intimately related to functionality, biological age is highly predictive of age-related morbidities (Belsky et al., 2018; Liu et al., 2018).

Despite the fact that age has consistently been the strongest predictor of morbidity and mortality for centuries (e.g., Costa & McCrae, 1980), measures of biological age offer several distinct advantages over the classical chronological measurement of age. First, biological age is rooted in the biology of the individual and is therefore sensitive to idiosyncratic changes in the pace of aging due to genetic or lifestyle factors (Belsky et al., 2017). This is a major departure from the chronological conception of age because it suggests the ability to differentiate between *age* as a designation and *aging* as a physiological process. For instance, consider two individuals who share a birthday. These individuals will always be the same chronological age; however, their pace of aging will likely display some degree of difference, especially if their lifestyles are dissimilar with regard to their health behaviors (e.g., diet, exercise, regular doctor visits). Biological age offers researchers the ability to distinguish between individuals who are aging at different rates—and thus have different risks of morbidity and mortality—where chronological age would have cast them as equivalent.

Second, because biological age is sensitive to changes of the internal environment of the body, biological age is, at least theoretically, manipulable (Austad, 2016). This is referred to as the “geroscience hypothesis” (Austad, 2016) and it suggests that if the integrity of the body’s organ systems can be improved through lifestyle changes or medical interventions, then biological aging can, in principle, be slowed down (Barzilai et al., 2018) or even reversed (Sinclair & LaPlante, 2019). Third, and finally, just as the slowing of biological age may offer insights into geroprotective factors, so too might the acceleration of biological aging help identify risk factors for premature onset of morbidity and mortality. Viewed through the lens of criminology, these points have a number of immediate implications. The first and most salient of these being that any “age effect” on crime may be more fully understood if the context of aging and declining health is also considered.

Crime ↔ Health

How might biological age provide new insights into existing areas of criminological research? While the possibilities are no doubt numerous, this dissertation will explore three areas wherein the use of biological age may offer novel insights for criminological research.

Area 1—The Health Consequences of Criminal Justice Contact

Marked health disparities have been observed among the current and previously incarcerated individuals when compared to the general population. While this literature is largely based on comparisons of morbidity prevalence rates, some quasi-experimental evidence has shown that having a history of incarceration is bad for your health in a variety of ways, including psychological symptoms, cardiovascular problems, and pulmonary diseases (Massoglia, 2008). Given the large number of morbidities associated with spending time behind bars, this

dissertation will probe whether the incarceration experience may actually be associated with something much more general: the acceleration of biological age.

Biological age is a global indicator of organ system integrity in the body. Thus, if biological age becomes accelerated due to environmental insult or endogenous stress response (both are commonly associated with being incarcerated), then the body may become more vulnerable to morbidities, as well as early mortality. In this way, biological age may be thought of as a general latent factor that informs the levels of specific morbidities. Such an explanation, if supported, would provide a common etiology for the many disparate health conditions disproportionately observed among the current and formally incarcerated.

Although criminal justice contact comes in a variety of forms (i.e., arrest, conviction, probation, incarceration, parole), incarceration is the most logical starting point for the search for age-related collateral consequences because incarceration represents the most extreme form of criminal justice contact. Thus, the first hypothesis examined in this dissertation is the following:

H1—Experiencing incarceration at some point in the life course will be associated with an accelerated biological age.

In chapter 2, I test this hypothesis using data from the Health and Retirement Study (HRS), a nationally representative sample of elderly Americans. Using a self-reported lifetime incarceration measure and a measure of biological age called “PhenoAge” (Levine et al., 2018) and self-reported lifetime incarceration, I examine how biological age differs across individuals with and without a history of incarceration. This analysis comes in two parts: 1) an unadjusted comparison between those with and without a history of incarceration on levels of PhenoAge, with the focus of testing for differential patterns across sex and demographic groups; and 2) a

propensity score matching (PSM) analysis of the male subsample wherein I attempt to adjust for childhood factors that might confound the focal association.

The findings of these analyses suggest that 1) unique patterns of association between incarceration and biological age exist across racial/ethnic groups, but not across sex, and 2) an association between incarceration and biological age was observable among males even after adjusting for childhood confounders. These analyses were limited in scope, due in large part to the current dearth of data sources with both biomedical and criminal justice data reflected in the HRS. Having found modest support for the association between criminal justice contact and biological age, however, the dissertation then turned to examining the behaviors that come before incarceration: offending behavior and criminal convictions. The relationship between early offending and receiving criminal sanctions (though of a lesser form than incarceration) and biological age is the topic of the next area.

Area 2—The Long-Term Health Effects of Early Offending Behavior

Criminologists have long hypothesized that criminal behavior is associated with negative health outcomes. Research in this area has been slow to develop, however, especially with regard to the physical health (compared to mental health) morbidities associated with offending. The emerging field of health criminology has begun to demonstrate that crime is related to a wide array of physical morbidities, including respiratory symptoms (Shepherd et al., 2002), cardiometabolic problems (Schwartz et al., 2020), infectious diseases (Massoglia, 2008), various minor symptomatology (Stogner, Gibson, & Miller, 2014), as well as early mortality (Piquero et al., 2011; Skinner & Farrington, 2020).

There is, however, one fact that is not well accounted for by the above literature: based on the age-crime curve and public health literatures, offending largely subsides long before the

onset of major health morbidities. This calls into question a direct and contemporaneous relationship between offending and health. This dissertation suggests a possible explanation for this apparent discrepancy: early offending behavior contributes to a change in the more general process of aging, which may predispose offenders to developing morbidities later on. This proposition represents that second hypothesis of this dissertation:

H2—Offending behavior early in the life course influences the aging process later in the life course.

In chapter 3, I test *H2* using data from the Dunedin Longitudinal Study, a prospective birth cohort study of New Zealanders ($N=1037$) from birth to age 45. Biological aging is operationalized using a longitudinal measure called “Pace of Aging” (Belsky et al., 2015a), which was estimated using biomedical data collected at age 26-45. Early offending behavior was operationalized in three ways that captured offending variety, pattern of offending, and criminal justice contact (i.e., criminal conviction), each measured up to the age of 26. The analysis proceeded in two parts. First, multiple linear regression analysis was used to test for associations between offending frequency and offending pattern during the adolescent/early adulthood years with aging in middle adulthood. Second, inverse probability of treatment weighting (IPTW) was used in an attempt to estimate the average treatment effect of criminal justice contact above and beyond other offending characteristics on aging later in the life course.

The results of this analysis revealed that both offending variety and offending patterns independently predicted aging in middle adulthood. In the IPTW analysis, receiving a criminal conviction was found to also significantly predict later aging. All told, these results point to a long-lasting effect of early offending behavior on the aging process experienced later on in adulthood. These findings offer a possible explanation of the results from the tests of *H1* that

linked offending behavior and health by suggesting that offending behavior may be operating through the more general process of aging. These results also lend themselves to another question: given that early offending behavior is associated with more advanced aging, might aging influence the criminal career in return? This is the topic of the third and final area of this dissertation.

Area 3—Biological Age and the (Non-Normative) Desistance Process

Within life-course/developmental criminology, many theories posit explanations for the onset and maintenance of offending behavior. Fewer theories deal directly with the desistance process, however, and most that do focus on what might be referred to as “normative” desistance. For those offenders who persist in offending long into adulthood—and thus experience “non-normative” desistance—explanations are fewer still, but most suggest some type of affirmative mental change experienced by the non-normative offender. For instance, offenders may experience identity transformation (Bushway & Paternoster, 2014), develop a sense of agency (Laub & Sampson, 2003), change their appreciation of risk/reward (Wilson & Herrnstein, 1985), or finally attain some level of psychosocial maturity (Monahan et al., 2009). What none of these explanations consider is that non-normative desistance may be an involuntary process driven by the functional limitations brought on by “the inexorable aging of the [offender]” (Gottfredson & Hirschi, 1990, p. 141).

The “aging out” explanation of non-normative desistance has gained little traction within life-course/developmental criminology. One reason for this is that, like other explanations of offending that are ontogenetic (i.e., naturally occurring), the aging out explanation fails to offer any targets for intervention (Dannefer, 1984). Another reason for the lack of attention given to the aging out explanation is that the construct of aging (as it is understood in the health sciences

literature) has not been integrated into criminological research. To these critiques, I would add that the aging out explanation of desistance, as posited by Gottfredson and Hirschi (1990), is too broad in its scope and should be constrained to those individuals who make up the non-normative group of desisters. In other words, there is survivor-bias in who lives long enough to age out of crime. In order to overcome this limitation, I will assess biological aging in middle adulthood. The third and final hypothesis of this dissertation is as follows:

H3—Non-normative desistance will be associated with more advanced aging throughout middle adulthood.

In chapter 4, I explore this hypothesis by again using data from the Dunedin Longitudinal Study. I again rely on the longitudinal measure of biological aging, “Pace of Aging” (Belsky et al., 2015a) to assess individual differences in aging. I examine non-normative desistance by employing group-based trajectory modeling (GBTM; Nagin, 2005) to identify a group of male respondents who exhibited elevated levels of self-reported offending throughout middle adulthood (i.e., from age 26-45). After identifying non-normative desisters, I compare groups across biological age.

Results revealed a three-group model as the best-fitting solution to the data, which I labelled “normative”, “non-normative”, and “abstainer”. Compared to the other two groups, non-normative desisters demonstrated the fastest pace of biological aging, lending support to the aging-out explanation. Further analysis revealed that prior offending and health behaviors like smoking also play a role in the relationship. Interestingly, biological aging was not associated with being in the normative desistance group. This suggests that other explanations of desistance likely account for desistance that occurs at the “normal” time and that “aging out” is most appropriately aimed at non-normative desistance.

Structure of the Dissertation

This dissertation deals with three major elements of human life: crime, health, and age/aging. Thus far, I have described a basic schematic for aligning these three elements into a series of analyses. Using these analyses, I hope to address long-standing issues at the intersection of criminology and public health. The following three chapters will be dedicated to expanding on each of the intersections mentioned above. Each chapter is structured to stand alone from the others by having its own introduction, literature review, methods, results, and discussion sections. But the topics covered in each chapter are interrelated in many ways. Thus, I have endeavored to order the chapters in a fashion that will best facilitate the progression of a discussion on the topics of crime, health, and age/aging.

I will conclude this dissertation with an overarching review and discussion of all three studies, tying together the common threads and highlighting directions for future work in this nascent area of criminology. The aim of the fifth chapter will, therefore, be three-fold. First, I will describe and synthesize the findings from each of the preceding chapters into a single narrative concerning the role of bodily health and aging for the criminal career. In this way, I hope to make plain how the results of chapters 2-4 contribute to the fields of criminology, public health, and geroscience. Second, I will describe the limitations of this research and attempt to situate the findings alongside the subareas that constitute the larger life-course paradigm in criminology. Third, I will discuss the primary policy implications that may be derived from the analyses carried out in chapters 2-4.

Chapter 2 —Biological Age and the Iatrogenic Effects of Incarceration

Introduction

Incarceration, in the form of jail or prison, represents a major life event that consistently signals the presence, or nascent onset, of negative health outcomes. Prior research has documented marked health disparities between inmates and the general population on most conventional health metrics. These health disparities include both chronic and infectious diseases, as well as a host of psychiatric disorders and substance abuse problems (Kirk & Wakefield, 2018; Massoglia & Pridemore, 2015). Many of these issues are detectable while individuals are still incarcerated, while other outcomes do not manifest until sometime after release.

Health care in the criminal justice system is a complicated issue as, for instance, inmates represent one of the only populations in the US for which health care is a right (e.g., see *Estelle v Gamble*, 1976). Despite this unique situation, the health status of inmates is consistently shown to be below that of the general population. Individuals with histories of incarceration have been shown, when compared to the general population, to have disproportionate rates of hypertension (Wang et al., 2009), asthma (Wang & Green, 2010), infectious diseases (Massoglia, 2008), and health-related functional limitations (Schnittker & John, 2007). Although the incarceration-health relationship is well-documented, recent developments in the area of geroscience provide reasons to believe that the effect of incarceration may go beyond specific morbidities to affect a general latent liability for disease. Put in the context of this dissertation, there is reason to believe incarceration will accelerate the biological age of individuals who experience it, putting them at increased risk for all age-related morbidities and mortality.

Biological age represents the bodily integrity of the many organ systems in the body, and it is the loss of integrity in these systems that eventually gives rise to age-related deterioration of

the body and its functions. Biological age has been shown to predict morbidity and mortality in healthy as well as diseased individuals (Belsky et al., 2015a; Liu et al., 2018); however, biological age has never been assessed among individuals with incarceration histories. With an eye towards the health disparities experienced by current/former inmates, this study asks the question: is incarceration associated with an accelerated biological age? The implication of this possibility is that incarceration, by accelerating biological aging, may be extracting more than *chronological* time from inmates; it may also be extracting *biological* time.

To assess the relationship between incarceration and biological age, I examine data from the Health and Retirement Study (HRS), a large nationally representative sample of elderly Americans that features retrospective data that covers most of the life course. I leverage respondents' self-reported contact with the criminal justice system to assess how biological age differs according to lifetime incarceration history.

In the sections that follow, I describe the literature documenting the many negative health outcomes associated with the incarceration experience. As I show, this literature has paid little attention to the possibility that incarceration may be aggravating a single latent factor, biological age, that is then responsible for the many negative health associations I observe among current/former inmates. The results demonstrate that biological age does indeed vary according to incarceration experience, though the patterns of association are complicated by important factors like race/ethnicity. Finally, I close by considering the implications of these findings for the US correctional system, but also for how we think about incarceration generally. I will also consider how these results inform the larger discussion surrounding the causal association between criminal justice contact and later health outcomes.

Literature Review

Incarceration is the most severe form of criminal justice contact. Compared to more invasive forms of contact like arrest, conviction, and probation, incarceration represents the most physically and psychologically taxing experience the criminal justice system can mete out. Prisons and jails not only represent the deprivation of many liberties but also the constant threat of personal harm. Prolonged psychological strain, physical injuries experienced, and, once released, the stigma associated with having been behind bars, are detrimental to a host of later outcomes. Yet, the incarceration experience is not an uncommon one. According to a 2018 report from the Bureau of Justice Statistics, 6.6 million US adults (i.e., ~1 in 38 adults) were under some form of correctional supervision in 2016, with a little over 2 million in jail or prison (Kaeble & Cowhig, 2018). Compounding this fact is the very real concern of overrepresentation of minorities and the poor in the carceral population.

It should be of little surprise, then, that current and former inmates experience particularly poor health outcomes. The incarceration experience is associated with a wide array of morbidities including chronic disorders and early mortality (Fazel & Baillargeon, 2011). The incarceration-health association is likely mediated by a variety of mechanisms present during periods of incarceration, such as stress, infectious disease, physical altercations, and exposure to illicit substances. Many of these mechanisms follow inmates after their incarceration period has ended and, in addition to other post-imprisonment factors like stigma, continue to negatively affect health and contribute to health disparities over the life course. In the sections that follow, I outline the current appraisals of health outcomes among current and formerly incarcerated individuals, as well as how these association may be mediated by inmates' biological age.

Incarceration and Later Health Outcomes

Incarceration is associated with a wide variety of negative health outcomes, including chronic health conditions, infectious diseases, psychiatric problems. When compared to the general population, national reports show that inmates have higher prevalence of many chronic diseases. These include cancer, cardiovascular conditions (e.g., high blood pressure/hypertension, stroke), metabolic disorders (e.g., diabetes), respiratory conditions (e.g., asthma, chronic obstructive pulmonary disorder), and arthritis (see Binswanger et al., 2009; Maruschak & Berzofsky, 2015; Wilper et al., 2009).

Along with stress-related chronic diseases, incarcerated individuals also suffer disproportionately from infectious diseases like HIV/AIDS, hepatitis, and tuberculosis (Baillargeon et al., 2004). Infectious diseases have the opportunity to spread rapidly in inmate populations through a number of mechanisms such as sharing needles, consensual/non-consensual sexual contact, overcrowding, and exposure to blood or other bodily fluids through violence (Jurgens, Nowak, & Day, 2011). In a report prepared by the Bureau of Justice Statistics, state/federal prisoners and local jail inmates were compared to the general population and were found to have significantly higher rates of infectious diseases (Maruschak et al., 2015). Specifically, both prisoners and jail inmates had higher prevalence rates of tuberculosis, hepatitis (all types), HIV/AIDSs, and STDs broadly.

But the incarceration-disease relationship is unlikely to run in just one direction. Instead, it is likely bidirectional as a large number of individuals are incarcerated every year that are infected prior to admission to a correctional facility. For instance, estimates for the US suggest that 17% of the estimated AIDS-infected population, 13-19% of the US HIV-positive population, 12-15% of the hepatitis B (HBV)-infected population, 29-32% of the HCV-infected population,

and 35% of the active TB population would serve time in a correctional facility in a single year (National Commission on Correctional Health Care [NCCHC], 2002). Thus, a contributing factor to the high-risk of disease transmission in prisons and jails is the high rates of infectious diseases that are introduced to the carceral population every year.

In addition to chronic and infectious diseases, psychiatric conditions are also overrepresented in correctional facilities. With limited variation across studies, prevalence rates of major psychiatric disorders like schizophrenia/psychosis, major depression, bipolar (manic), dysthymia, post-traumatic stress disorder, and anxiety are consistently and substantively higher among state/federal prisoners, as well as local jail inmates, compared to the general population (NCCHC, 2002). According to one report, the estimated size of the state and federal inmate population with serious mental illness was 10 times larger than the population of individuals with serious mental illness in state mental health hospitals (Torrey et al., 2010). For this reason, some have concluded that the criminal justice system is now the largest mental health facility in the world (Al-Rousan et al., 2017).

Correctional facilities play host to a significant percentage of the country's mentally ill; however, some research suggests that prisons and jails are likely not the origin of many cases of psychiatric disorders (though they almost certainly exacerbate symptoms). Using data from the National Comorbidity Survey Replication (NCS-R), Schnittker and colleagues (2012) demonstrated that individuals with a history of incarceration had significantly higher odds of mental disorders, but these relationships were very sensitive to childhood factors. The authors concluded that, with the exception of mood disorders, psychiatric disorders manifesting early in childhood and adolescence (especially substance use disorders) were responsible for much of the apparent incarceration-mental health relationship.

One curious note in the incarceration-health literature is the recurrent and paradoxical findings regarding minorities. While minorities experience dual disparities in economic disadvantage and contact with the criminal justice system that might otherwise suggest heavy selection bias, studies on the health effects of incarceration have repeatedly failed to identify any unique effects for racial minorities (e.g., see Massoglia, 2008; Schnittker & John, 2007). When considering mortality, certain minority groups actually appear to experience a protective effect from incarceration. For instance, some studies have observed that incarcerated Black males have a lower mortality rate than the race-specific mortality rates in the population (Mumola, 2007; Patterson, 2010; Rosen et al., 2011).

These findings suggest that incarceration may reduce mortality risk among minority inmates by reducing exposure to risks that are disproportionately high in minority communities (e.g., violence, vehicle accidents) and increasing exposure to protective factors that are more scarce in minority communities (e.g., food/housing security, access to healthcare) (Massoglia & Pridemore, 2015). Despite the lack of evidence for differential health effects across racial/ethnic groups, researchers still suggest that incarceration (and the criminal justice system more broadly) is a major source of health disparities overall due to the differential levels of criminal justice contact experienced in those communities.

Deprivation or Importation?

The existing research attempting to connect incarceration with inmate physical/mental health suffers from one major validity threat—selection bias (see Kirk & Wakefield, 2018; Massoglia & Pridemore, 2015). While it is true that the burden of disease among current and formerly incarcerated individuals is disproportionately large compared to the population, it is also true that many newly incarcerated individuals come into the correctional system already

possessing physical/mental symptoms (NCCHC, 2002; Schnittker et al., 2012). The corrections literature describes the contention between incarceration effects and selection effects as “deprivation” and “importation,” respectively.

Writing in 1958, Sykes suggested that the “pains of imprisonment” (i.e., the *deprivation* of personal liberties) were the key factors driving the development of oppositional subcultures in prisons/jails that resist the efforts of the correctional administration. In contrast, Irwin and Cressey (1962) asserted that inmates’ behavior was largely a product of personal background factors (e.g., personality, experiences, beliefs) and it was the *importation* of sufficient numbers of individuals with negative personal factors that drove prison misconduct. Though initially aimed at prison/jail misconduct and the development of inmate subcultures, this line of reasoning can be seamlessly applied to the current discussion surrounding inmate health.

The above research on the health outcomes of current/previously incarcerated individuals paints a compelling picture of the health disparities suffered by those individuals who experience life behind bars (i.e., supporting the deprivation model). However, most of that work is descriptive and/or correlational in nature, with no real means of adjusting for confounding factors like childhood socioeconomic status (SES), health status prior to incarceration, and drug use. The few studies that use rigorous statistical models with the ability to adjust for the influence of these factors often find mixed results or, in some cases, demonstrate that prison is most likely a correlate, rather than cause, of these poor health outcomes (i.e., supporting the importation model) (NCCHC, 2002; Schnittker et al., 2012).

Recently, however, a number of quasi-experimental methods have been employed in an attempt to adjudicate between the impacts of deprivation and importation. One such method involves the use longitudinal data within a fixed-effects framework. With this approach, all time-

stable variables (i.e., observed or not) are conditioned out of the regression equation, thereby removing all stable confounding influences on the association between incarceration and later health (e.g., family upbringing, personality, childhood health). By eliminating the influence of all time-stable factors, the fixed-effects model essentially makes each individual his or her own control, modeling only the intraindividual change occurring within the observation period (Wooldridge, 2016).

Using the fixed-effects approach, Schnittker and John (2007) found that, among respondents of the National Longitudinal Survey of Youth (1979; NLSY79), being incarcerated did indeed exert a substantive negative effect on later physical health (i.e., measured by severe physical limitations). Interestingly, whether or not an individual was incarcerated seemed to be the most salient factor for predicting later health outcomes—the duration of incarceration did not appear to have an effect on later physical health outcomes. In another fixed effects analysis, Sugie and Turney (2017) used the 1997 version of the NLSY (NLSY97) to examine the effects of criminal justice contact on later mental health outcomes (e.g., mood and anxiety disorders). The authors found that contact with the criminal justice system (especially incarceration) exerted deleterious effects on later mental health, and that arrest accounted for about half of the incarceration effect. The authors interpreted this finding from a stress process perspective (Pearlin, 1989), in that arrest and incarceration are two of the most visible stages of criminal justice contact and thus incur the highest rates of secondary stressors (i.e., criminal label/stigma).

Propensity score matching (PSM) is another quasi-experimental design that has been used to examine the effects of incarceration on later health outcomes. The full logic of this approach will be formally presented below, as it will be employed in the current analysis. Briefly, however, PSM is a method of constructing pseudo-controls for individuals exposed to a

treatment condition. PSM does this by 1) modeling the propensity of each individual to experience the treatment (e.g., incarceration) based on their observed characteristics and then 2) matching the “treated” cases (i.e., those who were incarcerated) with “control” cases who shared a similar propensity score but who were not exposed to the treatment. In this way, PSM simulates experimental conditions with observational data and is able to derive treatment effects by evaluating differences in the outcome of interest (e.g., physical/mental health) between “treatment” and “control” cases.

In 2008, Massoglia used PSM on data from the NLSY79 to estimate the effect of incarceration on later health symptoms and diagnoses. The analysis revealed that incarceration predicted a variety of health outcomes, but specifically those that were strongly linked to either stress (e.g., psychiatric disorders, cardiovascular conditions, respiratory conditions) or were infectious diseases (e.g., urinary tract infection, hepatitis, tuberculosis).

In another PSM analysis, Porter (2014) added nuance to the incarceration-health discussion by demonstrating that incarceration is associated with poor health behaviors that are themselves predictive of later health problems. Porter’s viewpoint is similar to the one that inspired the present study: if we assume incarceration is bad for your health, then the next question is *why*. With data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), respondents who were incarcerated were matched to respondents who were similarly situated (i.e., convicted of a crime), but who were not incarcerated. The analysis revealed that being incarcerated was associated with higher rates of cigarette smoking and fast food consumption. Though health outcomes were not analyzed in the study, the findings suggest that the incarceration-health relationship may be the result of changes across many different domains of social life brought about by the incarceration experience (e.g., stress proliferation).

One final method that has been used to demonstrate the effect of incarceration on health is the natural experiment. Briefly, natural experiments leverage variation in an exposure (e.g., incarceration) that is brought about by an exogenous (i.e., random) event (e.g., the implementation of a new policy). Because of the exogenous nature of the variation, treatment effects can be estimated by comparing the outcomes of individuals who experienced the exposure before a random event (i.e., the “controls”) to those who experienced it after (i.e., the “treated”) (see generally, Morgan & Winship, 2015).

Taking advantage of a 1994 policy change in Denmark that allowed Danish citizens to become incarcerated at younger ages, Baćak and colleagues (2019) were able to estimate the treatment effect for timing of incarceration on later mental health outcomes. Using data from the Danish population registry, the authors found that Danes who were incarcerated after the policy went into effect (i.e., at younger ages) experienced an increased likelihood of having contact with the mental health system, receiving psychiatric treatment, and later being charged with possession of drugs. This analysis not only provides support for the deprivation model, it also adds nuance to the incarceration-health discussion by incorporating social/developmental theory (e.g., sensitive periods; see Dannefer et al., 2016).

Based on the quasi-experimental evidence above, it is appropriate to say that the deprivation model of incarceration and health appears to be supported. The high likelihood of selection effects notwithstanding, spending time behind bars appears to be bad for your health. Yet, current evidence for the deprivation model is still preliminary because research has been unable to formally establish a set of processes that explain how incarceration influences health. The present study will contribute to this part of the literature by offering insight into a potential mechanism that could—at least partially—explain the link. That mechanism is biological age.

Physiological Stress, Organ Integrity, and Aging: Mechanisms of the Deprivation Model

A key mechanism in the incarceration-health literature is stress (Massoglia, 2008; Pearlin, 1989; Schnittker & John, 2007). Both acute and chronic stress are associated with the onset or exacerbation of many chronic conditions (Chrousos, 2009), as well as the susceptibility to infectious diseases (Glaser et al., 1999) and the acquisition of psychiatric conditions (Carr et al., 2013). Contact with the criminal justice system in general, and incarceration in particular, is stressful both in the immediate sense and in terms of the stigma that follows afterward. Pearlin (1989), in his pioneering work on the stress process paradigm, refers to these sources of stress as primary (e.g., incarceration) and secondary stressors (e.g., post-incarceration stigma). Secondary stressors like stigma are thought to play an outsized role in the disparities in morbidity and mortality found among former inmates because of their stress-proliferation properties (Massoglia, 2008). These influences are simultaneously stressful while also acting to erode the individual's ability to cope with the stress (Pearlin et al., 2005; Schnittker & John, 2007).

In line with the prior literature, this study posits that the stress (direct or indirect) associated with incarceration is the key linking mechanism with later health outcomes. The challenge associated with this causal logic is one of measurement—how does one measure stress? Prolonged exposure to stress results in the degradation of organ system integrity, sometimes referred to as “allostatic load” (McEwen & Stellar, 1993). Thus, stress exposure is in some part measurable by assessing the organ system integrity of the individual.

Recent developments in the field of geroscience have produced methods for measuring a construct called “biological age” that captures this integrity loss. In acknowledgement of the fact that integrity loss is normative (i.e., everyone ages and experiences integrity loss), measures of biological age capture more information than simply the gross level of physiological

deterioration. Measures of biological age also convey how much integrity loss has occurred relative to the levels expected at a specific age (Kirkwood, 2005). Biological age thus captures the pathological integrity loss (i.e., accelerated aging) that one would be the result of prolonged stress exposure (Belsky et al., 2017). The ability of biological age measures to distinguish between normative and pathological integrity loss in the body provides the tool necessary to explore the deprivation model in a more mechanistic fashion than has been possible previously. The following sections will discuss the theory, methods, and implications behind the concept of biological age, as well as its possible utility in explaining the incarceration-health relationship.

Biological Age—Mechanism of The Incarceration-Health Relationship?

Biological age refers to the integrity of all of the various organ systems across the body. The integrity of an organ system directly relates to its functional status. If the system is whole and in a conducive environment (i.e., it is surrounded by other integral organ systems), the likelihood of dysfunction is low. If the integrity of a system is low, however, dysfunction can become more common. If not addressed through intervention or by one of the body's many mechanisms for maintaining homeostasis, prolonged dysfunction may result in a more permanent condition and, eventually, to the collapse of the system (i.e., total integrity loss—death). Thus, biological age tracks the decline of organ system integrity of the body and is strongly associated with morbidity and mortality.

The onset and progression of most morbidities is heavily influenced by the physiological integrity of the affected organs systems. In this way, biological age may be thought of as a general latent construct representing the general liability for disease. Supporting this view are the observations that measures of biological age are 1) able to predict morbidities across many different organ systems (e.g., Belsky et al., 2017; Horvath & Raj, 2018; Liu et al., 2018) and 2)

are predictive in diseased as well as non-diseased samples (e.g., Belsky et al., 2015a; Liu et al., 2018). Together, these findings suggest that biological age is predictive of many, if not most, of the major morbidities observed in the population. But how might biological age be linked to the incarceration experience?

Biological age can be thought of as a measure of *aging* rather than age. This distinction is important because individuals' chronological age, although fairly predictive, does not directly account for the variation in the underlying mechanisms that drive risk of morbidity and mortality. In contrast, biological age is sensitive to a number of factors that greatly impact how much individuals age, such as environmental exposures or lifestyle factors (see Figure 2.1). In the current study, the environmental exposure of incarceration is hypothesized to have a substantive impact on biological age, representing an elevated general risk for disease. To better understand why measures of biological age are able to capture the influence of environmental exposures like incarceration, I now turn to a discussion of recent methods used to construct measures of biological age

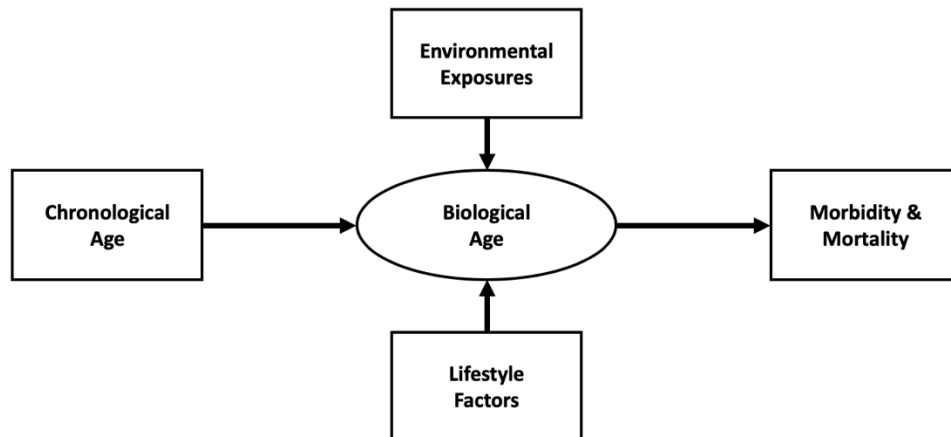


Figure 2.1. *Biological age as the general factor of chronological age, environment factors, and genetic factors.*

Measuring Biological Age

Measuring biological age can be analogized to measuring IQ: there are many methods of producing IQ scores and each method captures a slightly different (though largely overlapping) aspect of the latent construct of general intelligence, or *g*. For the current case, aging represents *g* (i.e., the latent construct that is hopefully being captured), biological age represents IQ (i.e., a useful, if imperfect, metric that captures the latent construct), and the various IQ tests are represented by what are called “biomarkers of aging”. Writing in 1988, Baker and Sprott defined biomarkers of aging as “a biological parameter of an organism that either alone or in some multivariate composite will, in the absence of disease, better predict functional capability at some late age than will chronological age” (p. 223). Identification biomarkers of aging is the first step to in measuring biological age and in developing our understanding of the general construct of aging.

Recent work by geroscientists has focused primarily on the “composite” approach to assessing biomarkers of aging. This approach typically involves 1) estimating the associations between multiple biomarkers and a target outcome (e.g., chronological age, mortality), 2) computing an index of these associations, and 3) standardizing this index so as to be interpretable like chronological years. To illustrate this last point, imagine a pair of twins who are dramatically different in terms of health (i.e., one healthy, the other sickly). Despite their shared birthday, a measure of biological age would likely give the healthier twin a younger biological age and the sickly twin an older biological age, with their shared chronological age being somewhere in the middle.

The types of biomarkers used vary widely—some are physical (e.g., BMI), biometric (e.g., heart rate, blood pressure), or blood-based (e.g., cholesterol, glucose, c-reactive protein).

Others rely on biological substrates that reside at the subcellular level, like epigenetic tags that sit atop DNA. Most approaches estimate biological age rely on a multivariate composite approach (i.e., not individual biological factors; e.g., leukocyte telomere length) that is measured in a cross-sectional fashion.

For centuries, chronological age has been the strongest single predictor of major morbidities and all-cause mortality (Costa & McCrae, 1980). With the use of biomarker indices, however, researchers are finally able to estimate an individual's global bodily integrity and outperform chronological age in the prediction of health outcomes. For instance, a recently developed biomarker index called phenotypic age (PhenoAge) has been able to outperform chronological age in the prediction of morbidity and mortality (Levine et al., 2018).

Using individuals ($N=9926$) from the National Health and Nutrition Examination Survey III (NHANES III), Levine and colleagues (2018) constructed a multivariate composite biomarker, PhenoAge, by training 9 individual biomarkers and chronological age on a 10-year hazard of age-related mortality (i.e., mortality from age-related disease) (note: more details will be provided in the methods section as PhenoAge will be used in the current analysis). Using PhenoAge in an independent validation sample (i.e., the NHANES IV; $N=11432$), Liu and colleagues (2018) demonstrated that PhenoAge was predictive of disease count, even among young adults. The researchers also found that phenotypic age acceleration (i.e., PhenoAge adjusted for chronological age) significantly predicted all-cause mortality and cause-specific mortality (i.e., death due to heart disease, cancer, chronic lower respiratory disease, diabetes, influenza/pneumonia, nephritis/nephrosis).

Later, these same researchers used data from the Health and Retirement Study (HRS; i.e., the same data used in the current analysis) to explore the sources of variation in PhenoAge (see

Liu et al., 2019). The largest explanatory domain was behavior (e.g., smoking, alcohol consumption, and exercise), closely followed by adversity in adulthood (e.g., military service, homelessness). These findings suggest two very important things for the current study: measures of biological age, specifically the multivariate composite biomarker index PhenoAge, are 1) able to predict a large diversity of morbidities and mortality and 2) are sensitive to environmental exposures like incarceration that are at the heart of the current study.

The purpose of the current study is to test the following hypothesis:

H1—Experiencing incarceration at some point in the life course will be associated with an accelerated biological age.

To test this possibility, I rely on data from the Health and Retirement Study (HRS), a large nationally representative study of elderly Americans. As I describe in the next section, the HRS has many advantageous characteristics that allow me to examine the complicated interplay between incarceration experiences and biological age. Furthermore, I seek to add to the burgeoning quasi-experimental literature on the incarceration-health relationship by using PSM to explore the association between incarceration and biological age after adjusting for childhood confounders. Finally, I rely on a novel theoretical and methodological approach for capturing variation in health in the form of physiological integrity (i.e., biological age), an approach that is yet unexamined in the incarceration-health literature.

Methods

Data

The Health and Retirement Study (HRS) is a nationally representative study of Americans aged 50 years and older (mean age is ~69 for the analytic sample). Begun in 1992, the HRS has a rolling cohort design wherein new cohorts are recruited as older cohorts diminish

in size due to mortality attrition—currently there are seven cohorts in the HRS sample. The HRS surveys its participants every two years (since 1998) across a broad range of demographic, behavioral, economic, and health related fields of inquiry. The HRS possesses a number of qualities that make it a good choice for this analysis: 1) it contains information related to adversities experienced in adulthood, including incarceration experiences; 2) it has an extensive panel of blood-based biomarkers (i.e., necessary for assessing biological aging); 3) it focuses on older Americans when differences in biological aging should be most pronounced; and 4) it contains enough racial and ethnic heterogeneity to allow for stratification across minority populations that experience disproportionate levels of contact with the criminal justice system and bear most of the national health burden.

Measures

Biological age. In 2016, the HRS conducted a biospecimen study, the Venous Blood Study (VBS), in which they collected venous blood samples from participants ($N \approx 9000$) with the goal of profiling levels of blood-based biomarkers of internal health. Data from this sub-study of the HRS has been compared with nationally representative health studies in the United States (e.g., the National Health and Nutrition Examination Survey [NHANES]) and has been found to be comparable (see VBS documentation, 2016). These data are well suited for assessing biological age and have already been used to construct a measure of *PhenoAge* (e.g., Liu et al., 2019), the method of estimating biological age originally proposed and validated by Levine and colleagues (2018) using data from the NHANES III.

PhenoAge was originally constructed using nine blood-based biomarkers (i.e., albumin, creatinine, glucose, [log] C-reactive protein, lymphocyte percent, mean cell volume, red cell distribution width, alkaline phosphatase, and white blood cell count) plus chronological age. A

parametric proportional hazards model (based on a Gompertz distribution) was used to predict a 10-year mortality risk in the NHANES III sample using the biomarkers and chronological age and the resulting regression coefficients were used for scoring. The outcome, which can be thought of as a mortality risk score, was originally calibrated with reference to months but was converted into units of years for interpretability. The following equation describes the computation of *PhenoAge* (Levine et al., 2018):

$$PhenoAge = 141.50 + \frac{\ln[-0.00553 \times \ln(1 - xb)]}{0.09165} \quad (1)$$

Where,

$$\begin{aligned}
 xb = & -19.907 \\
 & -0.0336 \times albumin \\
 & +0.0095 \times creatinine \\
 & +0.0195 \times glucose \\
 & +0.0954 \times \ln(CRP) \\
 & -0.0120 \times lymphocyte\ percent \\
 & +0.0268 \times mean\ cell\ volume \\
 & +0.3356 \times red\ blood\ cell\ distribution\ width \\
 & +0.00188 \times alkaline\ phosphate \\
 & +0.0554 \times white\ blood\ cell\ count \\
 & +0.0804 \times chronological\ age
 \end{aligned}$$

The resulting variable is interpretable as the phenotypic age of HRS respondents and is conveyed in scores that are directly comparable to chronological years.

In order to assess departures of biological age from chronological age (i.e., accelerated/decelerated aging), chronological age will be subtracted from participants' *PhenoAge* scores to produce *PhenoAge Acceleration*. Values >0 on *PhenoAge Acceleration* indicate an accelerated biological age compared to chronological age (i.e., premature aging), while values <0 suggest the opposite. As the sample contains individuals of many different ages, the use of

PhenoAge Acceleration allows for a focus on aberrant aging in the sample, rather than aging in general.

Lifetime Incarceration. The HRS asks a number of questions concerning respondents' experiences with adversities in adulthood. One question concerns incarceration experiences and reads as follows: "Have you ever been an inmate in a jail, prison, juvenile detention center, or other correctional facility?". Respondents received a score of 1 if they answered "Yes" and a score of 0 if they answered "No". This question was asked in two waves of data collection (i.e., 2012 and 2014) and answers were pooled across waves so that if an individual answered "Yes" to either wave they received a score of 1 on the *Lifetime Incarceration* measure. Cases were only categorized as "missing" if they did not provide responses on both waves of data collection. The resulting variable, *Lifetime Incarceration*, indicated that 7.68 percent of the analytic sample ($N=7606$) had some history of incarceration. This level of incarceration prevalence is in keeping with population estimates of US imprisonment rates (see e.g., Bonczar, 2003).

This retrospective measure of *Lifetime Incarceration* may be subject to certain threats to validity, including recall bias and social desirability bias. Indeed, research has demonstrated that prospective and retrospective accounts of delinquency only bear "moderately good" rates of concordance (Henry et al., 1994, p. 98). In contrast, however, self-reported contact with the criminal justice system (e.g., arrests and court appearances) has demonstrated much higher concordance rates (Henry et al., 1994, p. 99), likely because incarceration represents the deepest and most impactful level of contact one can have with the criminal justice system.

Matching Covariates. Because the timing of *Lifetime Incarceration* (i.e., the treatment) could not be ascertained, I restricted propensity score covariates to only those variables that specifically relate to participants' early life (i.e., before ages 16-18). This approach rests on the

assumption that, while HRS participants may have had criminal justice contact by age 18, few will have experienced incarceration by that time. By focusing on factors early in life, this approach helps to establish an appropriate temporal order between predictors and treatment and helps avoid issues of simultaneity (Apel & Sweeten, 2010; Wooldridge, 2016).

The variables used to estimate the propensity score for experiencing incarceration (i.e., the treatment) included retrospective measures tapping into HRS participants' childhood experiences (i.e., before age 16-18). The variable selection process was supported by a number of criminological perspectives and the final selection included 18 variables across three broad domains: childhood disadvantage and parenting (i.e., as emphasized by control theories; Hirschi, 1969; Sampson & Laub, 1993), as well as personal difficulties (i.e., as emphasized by strain theories; e.g., Merton, 1938; Agnew, 1993). See Table 2.1 for a list of all 18 childhood matching variables. These variables were compiled using repeated measures across waves of the HRS from 1998-2016 in an attempt to maximize sample coverage and minimize data loss.

Analytic Sample

The HRS sample was restricted based on the availability of data for the key variables in the analysis: *Lifetime Incarceration* and *PhenoAge Acceleration*. Information on *Lifetime Incarceration* was originally obtained across two waves of data collection occurring in 2012 and 2014. For the 2012 wave (total $N=20554$), a one-half random sample of the participants were asked to provide the information in a leave-behind questionnaire (72.7% completed the questionnaire). The following year, in 2014 (total $N=18747$), the samples were reversed (77.8% completed the questionnaire). A total $N=12262$ participants provided information on *Lifetime Incarceration* across the 2012 and 2014 waves of data collection. Given the use of randomization in the collection data from the leave-behind questionnaires and the high overall response rates

from both waves, the drop in case count is not anticipated to have a substantive impact on the current analysis.

During the 2016 wave (total $N=15283$), participants were asked at the end of the data collection interview if they would consent to participating in an HRS sub-study, the Venous Blood Study (VBS), that would involve a blood draw. Ultimately, 65% of the eligible respondents ($N=9934$) were enrolled and completed data collection/processing for the VBS. Participation rates were very similar (i.e., within 1-2%) across strata of age and education; however, participation did vary more across racial groups with Whites and Hispanics participating at the highest rates (i.e., 67.9% and 65.2%, respectively) and Blacks at the lowest (i.e., 57.3%). Racial disparities in participation mean that representativeness of the VBS may be somewhat reduced Blacks and that results of the current analysis may be less generalizable to Black populations.

After listwise deletion of cases without valid information on both of the key variables, the analytical sample was $N=7606$ participants. Complete case analysis in the current study has major two implications. First, statistical power is reduced, making the detection of small effects more difficult. Given the overall size, the analytic sample appears to be adequately powered for a straightforward analysis of incarceration effects; however, stratification by sex and racial/ethnic categories quickly reduces statistic power (this issue is mentioned below in the results section) and possibly reduces the ability to contextualize findings across these dimensions. Second, issues of selection bias may be introduced (or exacerbated) by the removal of cases with incomplete information if those cases do not constitute a random draw from the larger study sample. Given these concerns—and especially in light of the aforementioned racial disparities in participation in the VBS—it is important use caution when interpreting the findings below.

Analytical Strategy

My analysis of *Lifetime Incarceration* and its effects on biological age in the HRS will unfold in three steps. First, I will thoroughly describe the distribution of focal variables across the sample, with special emphasis given to particular demographic differences. For instance, I will calculate crosstabulations of sex and race/ethnicity in order to observe the rates of *Lifetime Incarceration* in the sample. This will help confirm that the incarceration rates in the HRS reflect those of the general population in terms of demographic break down. Having representative rates of incarceration is important for this analysis because, based on the prior literature, it is expected that some differences across racial/ethnic groups will emerge (Mumola, 2007; Patterson, 2010; Rosen et al., 2011).

Second, I will assess the association between *Lifetime Incarceration* and biological age by ordinary least squares (OLS) regression. I will also test for moderation of the incarceration effect examining the interactions between incarceration, sex, and race/ethnicity. The purpose of this second step in the analysis is more descriptive than inferential. Because incarceration is rare in the general population—and HRS is a nationally representative study—the raw numbers of previously incarcerated individuals is expected to be relatively low. By observing the unadjusted levels of biological age in the HRS sample across the dimensions of *Lifetime Incarceration*, sex, and race/ethnicity, I hope to provide a roadmap for the final step in the analysis (described below) and a starting point for future studies.

The third step will employ PSM to estimate the treatment effect of *Lifetime Incarceration* on biological age. PSM is useful for simulating experimental conditions in observational data. PSM accomplishes this by comparing a “treatment group” (in this study, HRS participants who reported being incarcerated at some point) with a “control group” (i.e., HRS participants with no

history of incarceration) that differs only in terms of their exposure to the treatment. In effect, the treated and non-treated members of a matched pair represent statistical control cases in that, their probability (i.e., propensity) of receiving the treatment, as estimated with the set of covariates, is identical or very similar. When matched on their propensity for the treatment, actual exposure to the treatment may be considered random, conditional on an assumption of no unobserved confounding (Guo & Fraser, 2014; Morgan & Winship, 2015).

Table 2.1. Childhood Covariates for Propensity Score Model of Lifetime Incarceration.

Financial Situation		
Before age 16...		
1.	<i>Would you say your family during that time was pretty well off financially, about average, or poor?</i>	1=Pretty well off financially 2=About average 3=Poor
2.	<i>Did financial difficulties ever cause you or your family to move to a different place?</i>	1=Yes 0=No
3.	<i>Was there a time when you or your family received help from relatives because of financial difficulties?</i>	1=Yes 0=No
Employment		
Before age 16...		
4.	<i>Was there a time of several months or more when your father had no job?</i>	1=Yes 0=No
5.	<i>What was your father's occupation?*</i>	1=Managerial/Professional 2=Sales 3=Clerical 4=Service 5=Manual/Operators 6=Armed Forces
6.	<i>What portion of the time did your mother work outside the home when you were growing up?</i>	1=All of the time 2=Some of the time 3=Not at all
Education Attainment		
7.	<i>What is the highest grade of school your father completed?</i>	0-17 (years)
8.	<i>What is the highest grade of school your mother completed?</i>	0-17 (years)
Parenting		
The next statements are about people's relationships with their parents early in life (before age 18). Please tell us how much you agree or disagree with each statement for you personally.		
9.	<i>I had a good relationship with my father.</i>	1=Strongly disagree : 5=Strongly agree
10.	<i>I had a good relationship with my mother.</i>	1=Strongly disagree : 5=Strongly agree
11.	<i>How much time and attention did your mother give you when you needed it?</i>	1=A lot : 4=Not at all
12.	<i>How much effort did your mother put into watching over you and making sure you had a good upbringing?</i>	1=A lot : 4=Not at all
13.	<i>How much did you mother teach you about life?</i>	1=A lot : 4=Not at all

Table 2.1. Cont.

Personal Difficulties		
Before you were 18 years old...		
14.	<i>Did you have to do a year of school over again?</i>	1=Yes 0=No
15.	<i>Were you ever in trouble with the police?</i>	1=Yes 0=No
16.	<i>Did either of your parents drink or use drugs so often that it caused problems in the family?</i>	1=Yes 0=No
17.	<i>Were you ever physically abused by either of your parents?</i>	1=Yes 0=No
Before you were 17 years old...		
18.	<i>Did you ever live in the same household with a grandparent for a year or more?</i>	1=Yes 0=No
*The variable for father's occupation was recoded as five dummy variables wherein each dummy received a score of 1 for a specific category of paternal occupation and 0 otherwise. The category of "managerial/professional" served as the reference category.		

The PSM analysis proceeded in four steps. First, propensity scores were estimated by regressing the *Lifetime Incarceration* variable on the 18 childhood predictors and demographic variables (see Table 2.2) by logistic regression. Second, individuals from the treatment group (i.e., those with a history of incarceration) were matched with individuals from the control group (i.e., those with no history of incarceration) according to their propensity score. The matching analysis was restricted to the region of common support (i.e., propensity score values of the treatment group that do not go beyond the minimum or maximum propensities score values of the control group) and kernel estimators were used for the matching procedure (kernel=Epanechnikov; bandwidth=0.03). The advantage of the kernel approach is that it utilizes all of the control cases within a certain bandwidth of a given treatment case and weights each matched control according to its distance from the treatment case (i.e., more similar cases are given more weight). Matching was accomplished with use of the *psmatch2* suite (Leuven & Sianesi, 2003) in Stata version 14 (StataCorp, 2015).

Third, the treatment and control groups were compared for balance and bias reduction. After kernel matching, sample balance was examined by comparing standardized mean

differences and variance ratios between treated and control cases, both before and after matching. Fourth, and finally, a matched sample *t*-test was used to estimate the average treatment effect *Lifetime Incarceration on PhenoAge Acceleration* among those with a history of incarceration (i.e., the average treatment effect on the treated [ATT]).

Results

Preliminary Analysis

I will begin by describing the Health and Retirement Study (HRS) sample in terms of the focal variables for the current analysis (refer to Table 2.3). *PhenoAge* ($M=70.04$; $SD=14.04$) was normally distributed in the analytical sample and was strongly correlated ($r=0.77$) with chronological age ($M=69.37$; $SD=9.98$). *PhenoAge Acceleration* (i.e., *PhenoAge – Chronological Age*) was thus centered on zero ($M=0.67$; $SD=8.90$), with a larger range of aging deceleration/acceleration (min-max=-19.38 to 52.12). Figure 2.2 displays that the biological age for most HRS participants was relatively concordant with their chronological age (indicated by the dashed line). For some participants, however, their biological age was discordant with *PhenoAge Acceleration* being positive (i.e., indicating an acceleration of aging beyond chronological age) or negative (indicating a deceleration of aging below chronological age).

Table 2.2. Descriptive Statistics of the Health and Retirement Study Analytical Sample (N=7606).

	Mean (%)	SD	Min	Max
PhenoAge	70.04	14.04	22.67	115.04
Chronological Age	69.37	9.98	21.00	107.00
PhenoAge Acceleration	0.67	8.90	-19.38	52.12
Lifetime Incarceration	(7.68)	-	0	1
Male	(39.92)	-	0	1
Race/Ethnicity				
<i>White</i>	(69.68)	-	0	1
<i>Black</i>	(15.04)	-	0	1
<i>Hispanic</i>	(12.32)	-	0	1
<i>Other</i>	(2.96)	-	0	1

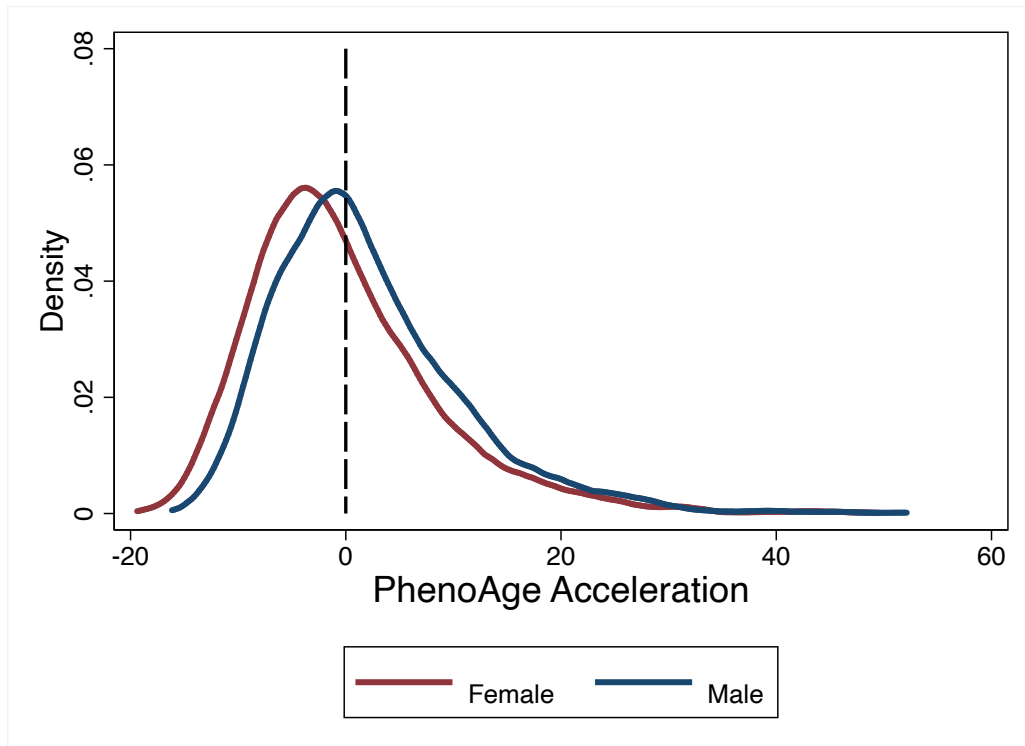


Figure 2.2. Kernel density plot showing the distribution of PhenoAge Acceleration for males and females.

The sample reported a total *Lifetime Incarceration* rate of 7.68%, which is in keeping with population estimates for the US (e.g., 6.6% in 2001; Bonczar, 2003). Additionally, the racial/ethnic composition of the HRS participants who indicated being incarcerated at some point was reflective of the race/ethnicity-specific rates reported for the overall US jail population (e.g., Zeng, 2020)—although, it was less reflective of the US prison population (e.g., Carson, 2020). For instance, in 2018 the US male jail population was 59% White (compared to 54% in the HRS), 38% Black (compared to 27% in the HRS), and 17% Hispanic (compared to 15% in the HRS) (Zeng, 2020). Cross-tabulations of *Lifetime Incarceration* across racial/ethnic groups are provided in Table 2.3 for both sexes. The sample was ~40% male, with a racial/ethnic breakdown of 69.68% White, 15.04% Black, 12.32% Hispanic, and 2.96% other groups.

Table 2.3. *Cross-tabulation of Lifetime Incarceration and Race/Ethnicity Across Sex.*

Males	Ever Spent Time in Jail/Prison?				Total
	No	(%)	Yes	(%)	
White	1965	(76)	244	(54)	2209
Black	263	(10)	119	(27)	382
Hispanic	282	(11)	68	(15)	350
Other	78	(3)	17	(4)	95
Total	2588	(100)	448	(100)	3036

Females	Ever Spent Time in Jail/Prison?				Total
	No	(%)	Yes	(%)	
White	3028	(68)	63	(46)	3091
Black	710	(16)	52	(38)	762
Hispanic	572	(13)	15	(11)	587
Other	124	(3)	6	(4)	130
Total	4434	(100)	136	(100)	4570

Regression Analysis

I now turn to the primary analysis. The goal is to explore the relationship between *PhenoAge Acceleration* and *Lifetime Incarceration*, with a special emphasis on how that relationship may differ across the dimensions of sex and race/ethnicity. I take a three-step approach to the analysis by: 1) examining the overall differences in *PhenoAge Acceleration* across incarcerated and non-incarcerated respondents; 2) disaggregating the sample by sex and race/ethnicity; and finally, 3) disaggregating the sample by sex and race/ethnicity sequentially (i.e., first by sex, then by race/ethnicity). At each step, I examine the effect of *Lifetime Incarceration* on *PhenoAge Acceleration*. The results are presented in Table 2.4. Results are also presented visually as histograms (Figures 2.3 and 2.4) for ease of interpretation. Histograms display the marginal effects of *Lifetime Incarceration* on *PhenoAge Acceleration* across the various demographic breakdowns examined in Table 2.4. *PhenoAge Acceleration* was standardized (*z*-scores) in Figures 2.3 and 2.4.

Is a History of Lifetime Incarceration Associated with Higher Levels of PhenoAge Acceleration? Turning to model 1 of Table 2.5 (panel A of Figure 2.3), a clear and substantive increase in *PhenoAge Acceleration* is visible for respondents who reported being incarcerated at some point in their lives. The non-incarcerated sample demonstrated a biological age of about ½ year in excess of their chronological age (constant=0.51; $p<0.001$), but the incarcerated sample demonstrated an additional 2 years of *PhenoAge Acceleration* ($b=2.10$; $p<0.001$). In total, these results mean that HRS participants with a history of incarceration are living with a biological age that is more than 2 ½ years in excess of their actual chronological age. Non-incarcerated participants are only experiencing a biological age acceleration of about ½ year. It appears, from this preliminary model at least, that incarceration is associated with accelerated biological aging.

Table 2.4. Ordinary Least Squares Regression of PhenoAge Acceleration on Lifetime Incarceration, Sex, and Race/Ethnicity, and Their Interactions.

	Model 1		Model 2		Model 3		Model 4 (males)		Model 5 (females)	
	<i>b</i>	(SE)	<i>b</i>	(SE)	<i>b</i>	(SE)	<i>b</i>	(SE)	<i>b</i>	(SE)
Lifetime Incarceration	2.10***	(0.38)	1.26	(0.77)	2.55***	(0.52)	1.79**	(0.59)	1.22	(1.11)
Male			2.13***	(0.22)						
Race/Ethnicity										
<i>Black</i>					3.20***	(0.31)	3.42***	(0.57)	3.49***	(0.37)
<i>Hispanic</i>					0.93**	(0.33)	1.98***	(0.56)	0.62	(0.40)
<i>Other</i>					-1.25*	(0.63)	-1.00	(1.01)	-1.38	(0.80)
Lifetime Incarceration ×										
Male			-0.01	(0.89)						
Race/Ethnicity										
<i>Black</i>					-2.80**	(0.90)	-2.91*	(1.13)	-2.43	(1.68)
<i>Hispanic</i>					-1.24	(1.14)	-3.04*	(1.32)	2.10	(2.54)
<i>Other</i>					1.09	(2.01)	1.55	(2.42)	-0.20	(3.82)
Constant	0.51***	(0.11)	-0.28*	(0.13)	-0.01	(0.13)	1.32***	(0.20)	-0.88***	(0.16)
<i>N</i>	7606		7606		7606		3036		4570	

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

Does the Association Between Lifetime Incarceration and PhenoAge Acceleration Vary Across Sex? Moving to model 2 of Table 2.5 (panel B, Figure 2.3), a similar pattern to what we saw in the overall sample was observed across male and female respondents. Females who did not report experiencing *Lifetime Incarceration* possessed lower than average *PhenoAge Acceleration* (constant=-0.28; $p < 0.05$) while females who were incarcerated possessed a *PhenoAge Acceleration* that was near zero (1.26 - 0.28=0.98; $p > 0.05$). The findings for female HRS respondents suggest that, on average, females with a history of incarceration have a higher biological age than females who do not have a history of incarceration.

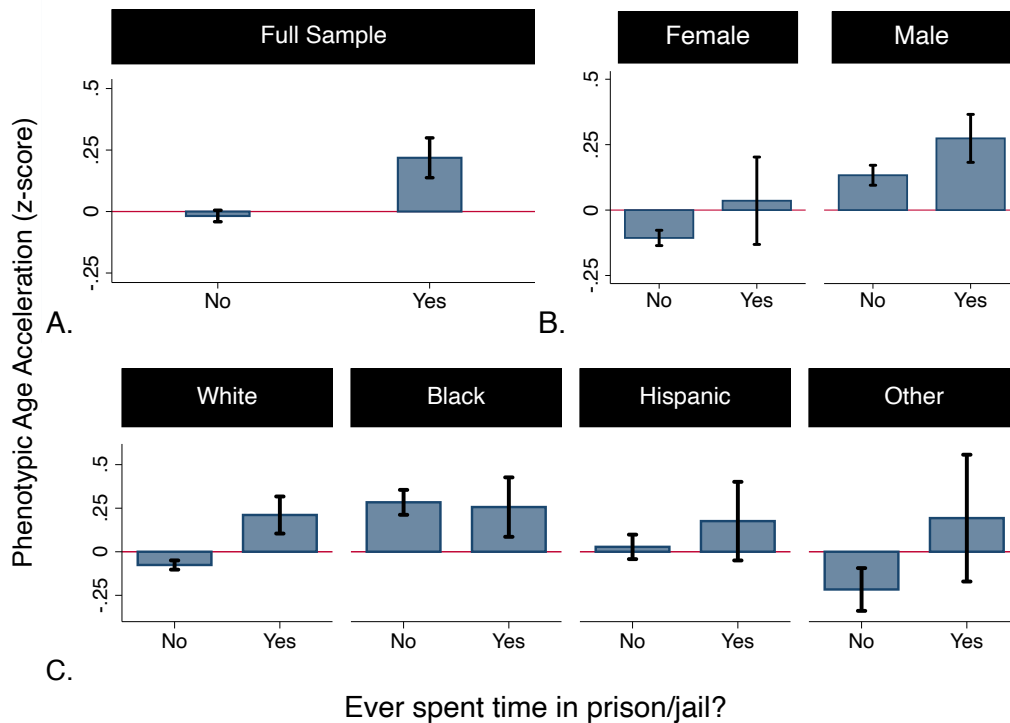


Figure 2.3. Bar graphs of *PhenoAge Acceleration* (z-score) by *Lifetime Incarceration* (A) in the analytical sample and broken down by (B) sex and (C) race/ethnicity.

Males presented with higher *PhenoAge Acceleration* than females, regardless of *Lifetime Incarceration*. Males with no history of *Lifetime Incarceration* demonstrated *PhenoAge Acceleration* that was more than two years in excess of non-incarcerated females ($b=2.13$; $p<0.001$). Males who had been incarcerated experienced, on average, more than an additional year of biological aging ($b=1.26$; $p=0.10$)—although the interaction was not statistically significant ($b_{Male \times Lifetime\ Incarceration} = -0.01$; $p=0.99$). These findings indicate that males with no history of *Lifetime Incarceration* demonstrated an average acceleration of phenotypic age of almost 2 years, while incarcerated males averaged more than 3 years ($t=-2.77$; $p<0.01$). These results suggest that *Lifetime Incarceration* is especially impactful for males, translating into an acceleration of biological age by more than a year.

Does the Association Between Lifetime Incarceration and PhenoAge Acceleration Vary Across Race/Ethnicity? In model 3 of Table 2.5 (panel C, Figure 2.3), racial/ethnic differences in the association between *Lifetime Incarceration* and *PhenoAge Acceleration* are displayed. Within racial/ethnic groups, *Lifetime Incarceration* was generally associated with higher *PhenoAge Acceleration*, although the only increase that associated with *Lifetime Incarceration* that was statistically significant was for White participants ($b=2.55$; $p<0.001$). An exception to this pattern was observed for Black participants, where we see a slightly lower *PhenoAge Acceleration* for those with a history of *Lifetime Incarceration* (marginal effects: $b=3.19$ vs. $b=2.95$; both $p<0.001$). Although the observed mean difference in *PhenoAge Acceleration* between the incarcerated and non-incarcerated Black participants was not statistically significant ($t=0.29$; $p>0.05$), both groups presented with a higher level of *PhenoAge Acceleration* than any other racial/ethnic group.

Looking now at the between-group effects, each racial/ethnic group significantly differed from White participants in terms of *PhenoAge Acceleration*, with Black and Hispanic participants demonstrating higher levels ($b=3.20$; $p<0.001$; and $b=0.93$; $p<0.01$, respectively) and members of the Other group demonstrating lower levels ($b=-1.25$; $p<0.05$).

For incarcerated individuals, both White ($b=2.55$; $p<0.001$) and Black participants ($b_{Lifetime\ Incarceration \times Black}=-2.80$; $p<0.01$) demonstrated a statistically significant increase from the non-incarcerated White participants (the reference category). This finding is consistent with previous literature on mortality that noted differential effects across race (Mumola, 2007; Patterson, 2010; Rosen et al., 2011). None of the other interaction terms were statistically significant.

Does the Association Between Lifetime Incarceration and PhenoAge Acceleration Vary Across the Intersection of Sex and Race/Ethnicity? The final regression analysis attempts to parse the HRS sample across three highly impactful dimensions: *Lifetime Incarceration*, sex, and race/ethnicity. Before describing the results of the final analysis, however, it is important to draw attention back to Table 1 where the cross tabulations of the HRS sample are provided. As shown in Table 1, some of the categories pertinent to the current analysis contain very small case counts and any results derived from said categories must be interpreted with caution. In particular, the estimates for incarcerated females and minorities should be interpreted carefully in light of the low statistical power in those analyses. With these cautions in mind, I approach this final analysis as a descriptive analysis wherein I focus on substantive trends, giving less weight to statistical significance.

Average values of *PhenoAge Acceleration* across categories of *Lifetime Incarceration*, race/ethnicity, and sex are presented in models 4 (males) and 5 (females) of Table 2.5. These values are also shown visually in Figure 2.4. Looking across the panels for both males and females, three trends become apparent. First, *Lifetime Incarceration* was generally associated with an increased average *PhenoAge Acceleration*. The two exceptions to the pattern were observed for Black respondents and Hispanic males, among whom the opposite trend was observed. Second, males consistently displayed higher average levels of *PhenoAge Acceleration* than females, regardless of race/ethnicity or *Lifetime Incarceration*. The only exception to this pattern was for incarcerated Hispanic females ($N=6$) compared to incarcerated Hispanic males ($N=17$). Note, though, that these two categories have very small case counts so the pattern of findings should be interpreted cautiously.

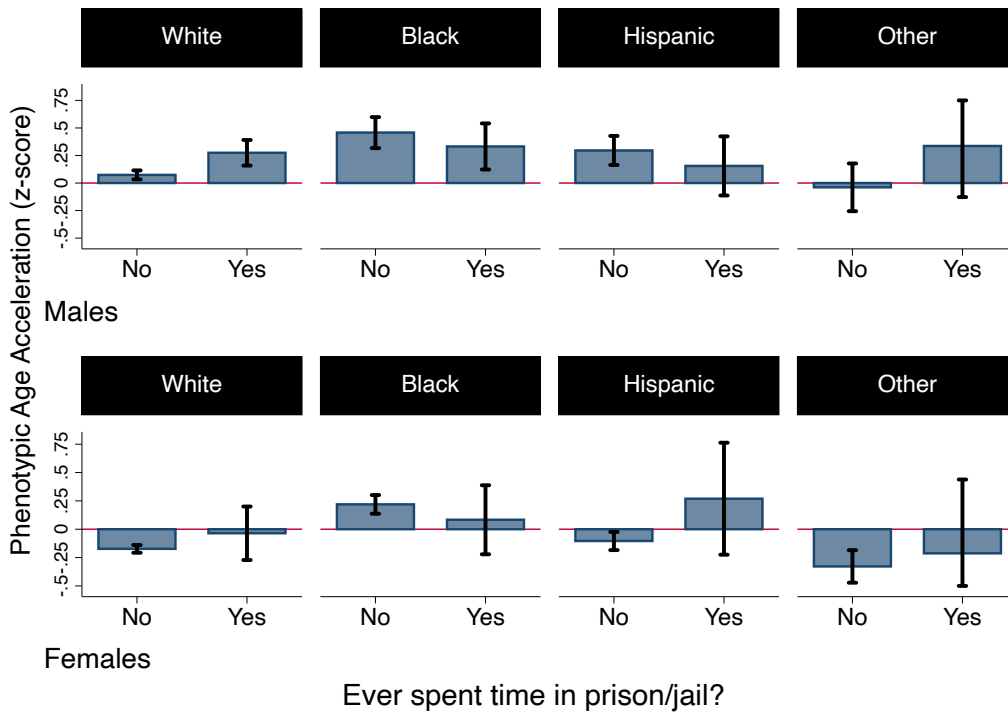


Figure 2.4. Bar graphs of *PhenoAge Acceleration (z-score)* by *Lifetime Incarceration* in the analytical sample and broken down by race/ethnicity and stratified by sex.

Third, racial differences in the association between *Lifetime Incarceration* and *PhenoAge Acceleration* was reproduced when stratified by sex (i.e., within-race/between-sex). For example, *Lifetime Incarceration* was associated with higher average *PhenoAge Acceleration* in both male and female White respondents, while *Lifetime Incarceration* was associated with lower *PhenoAge Acceleration* among both male and female Black respondents. The one exception to this pattern was among Hispanics. Hispanic males shared the same pattern as Black respondents (i.e., *Lifetime Incarceration* was associated with lower *PhenoAge Acceleration*), while Hispanic females showed the pattern established by White respondents (i.e., *Lifetime Incarceration* was associated with high average *PhenoAge Acceleration*).

Propensity Score Analysis

The previous analysis was meant to offer a description of the patterns of association, meaning it did not adjust for potential sources of confounding in the association between *Lifetime Incarceration* and *PhenoAge Acceleration*. I now turn to a propensity score matching analysis to adjust for selection bias for exposure to *Lifetime Incarceration* (i.e., the treatment). Moving forward, I restrict the analytic sample to males for two reasons. First, as demonstrated in the above regression analyses, most of the variation in *PhenoAge Acceleration* is concentrated among males. Second, consistent with population estimates of incarceration rates (Zeng, 2020) males made up the majority (~75%) of the reported incarcerations in the HRS sample. The all-male analytic sample was $N=3036$.

Among the male-only analytic sample, 31% ($N=964$) had some level of missingness on the childhood covariates. Thus, multiple imputation was used to estimate values for these missing pieces of information. As multiple imputation assumes that the missingness in a sample is random, the childhood covariates were subjected to Little's test (Little, 1988) for missing completely at random (MCAR) and the results suggested that the childhood covariates were not MCAR. Upon further investigation, all of the childhood covariates had low levels of individual missingness (i.e., all were <1.9% missing; see Table 2.5) and most patterns of missingness were found to comprise a trivial portion of the sample (e.g., most patterns comprised <1% of the sample). Given these findings from the MCAR analysis, the proceeding results from the propensity score matching analysis should be interpreted with caution.

Table 2.5. Childhood Covariates Missingness, Counts and Sample Percentage.

Childhood Predictors	Count	%
Family financial situation	32	0.2
Move due to financial difficulty	54	0.3
Received financial help	17	0.1
Father unemployed	32	0.2
Father occupation	393	1.9
Mother work outside home	359	1.7
Father's educational attainment	263	1.3
Mother's educational attainment	11	0.1
Relationship quality with father	399	1.9
Relationship quality with mother	312	1.5
Attention from mother	236	1.1
Effort from mother	236	1.1
Life lessons from mother	238	1.1
Do over school year	174	0.8
Trouble with the police	236	1.1
Parental substance use	175	0.8
Parental physical abuse	179	0.9
Life with grandparents	11	0.1

Missing data on the childhood predictors were imputed using chained equations available in the *mi* suite in Stata 14 (StataCorp, 2015; $m=10$ imputations). All childhood variables were used in specifying the imputation model, as well as variables for HRS cohort membership and race/ethnicity. All male respondents (i.e., with and without data on the treatment) were used in the imputation model (Graham, 2009). Estimates derived from imputed datasets were combined using Rubin's rules (1987).

Following imputation, the propensity score analysis proceeded in four steps. First, a logistic model was estimated predicting *Lifetime Incarceration* with all childhood predictors, as well as race/ethnicity and cohort indicators. The predicted logit values from this analysis ranged from 0-1 and represented each individual's propensity score for experiencing *Lifetime Incarceration*, given their levels on the observed predictors. Second, the propensity scores were

used to match treated individuals (i.e., previously incarcerated males; $N=448$) in the sample to untreated individuals (i.e., never incarcerated males; $N=2588$) using kernel matching. During this process, 7 treated cases were dropped because they were outside the region of common support, leaving a new analytic sample of $N=3029$. (Note: a visual representation of the region of common support can be found in Figure 2.7).

Third, having matched the sample on the propensity score for *Lifetime Incarceration*, covariate and propensity score balance across treatment and control groups was assessed. Covariate balance was assessed in terms of both mean values and distributional variance. Differences in mean values between treated and control cases was assessed using standardized mean differences (SMDs). Balanced samples should have SMDs close to zero, and, while no consensus currently exists in the literature, an upper limit of 0.2 indicates a cutoff for adequate balance (Linden & Samuels, 2008). This cutoff is generally used because SMDs are a form of Cohen's d and a value of 0.2 is recommended by Cohen (1988) as the lower threshold of a small effect. Table 2.6 displays the covariate balance across treated and control cases, both before and after matching. Visual presentation of these statistics can be found with Figure 2.5, with the numbers along the y-axis correspond to the numbered childhood covariates listed in Table 2.1 (note: the occupational categories of father's usual occupation [variable 5] were coded as dummy variables, with "managerial/professional" excluded as the reference category). The arithmetic mean of SMDs was also calculated (a global metric suggested by Linden & Samuels, 2008) and is presented at the bottom of the figure as a triangle. As can be seen in Figure 2.5, matching substantially improved the covariate balance in the sample, with no variables demonstrating an absolute SMD greater than 0.2. This indicates that balance of mean values was achieved in the matched sample.

Table 2.6. Covariate Balance Across Childhood Covariates.

	Unweighted		Weighted	
	SMD	VR	SMD	VR
Childhood Predictors				
Family financial situation	0.19	1.10	0.04	1.02
Move due to financial difficulty	0.21*	1.35	0.01	1.01
Received financial help	0.23*	1.47	0.04	0.95
Father unemployed	0.17	1.17	0.03	1.02
Father occupation ^a				
Sales	0.11	0.70	0.06	0.82
Clerical	0.03	0.86	0.05	0.76
Service	0.03	1.15	0.07	0.79
Manual/Operators	0.12	0.93	0.10	0.94
Armed forces	0.09	1.85	0.02	1.13
Mother work outside home	0.12	1.06	0.03	0.99
Father's educational attainment	0.13	1.12	0.03	1.00
Mother's educational attainment	0.11	1.38	0.01	1.24
Relationship quality with father	0.25*	1.18	0.02	0.92
Relationship quality with mother	0.19	1.23	0.02	0.89
Attention from mother	0.18	1.26	0.05	0.93
Effort from mother	0.19	1.31	0.02	0.98
Life lessons from mother	0.19	1.30	0.03	1.09
Do over school year	0.23*	1.32	0.05	1.06
Trouble with the police	0.68*	2.58 [^]	0.01	1.00
Parental substance use	0.34*	1.61	0.03	1.03
Parental physical abuse	0.21*	1.97	0.06	0.86
Life with grandparents	0.08	1.10	0.07	0.94
Arithmetic Mean	0.19		0.04	
Geometric Mean		1.27		0.97

*SMD \geq 0.02; [^]VR \neq 0.05-2; SMD=(absolute) standardized mean difference; VR=variance ratio; ^aRef. Group=managerial/professional.

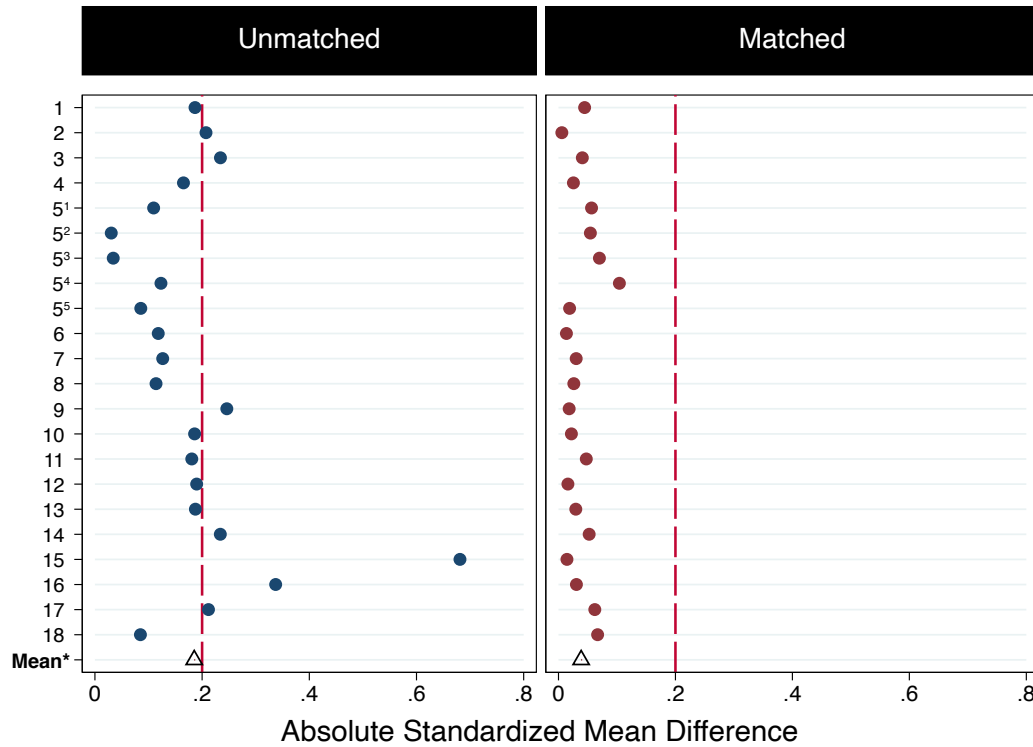


Figure 2.5. Absolute standardized mean differences across childhood covariates between treated and control cases. Note: mean=arithmetic.

Distributional differences were assessed with variance ratios (VRs). As VRs approach 1, the balance between two distributions improves. With a central limit of 1 being ideal, lower and upper bounds of acceptable distributional balance are 0.5 and 2, respectively (i.e., as suggested by Rubin, 2001). Table 2.5 also displays the VRs of the childhood covariates across the treated and control cases. Visual presentation of these statistics is provided with Figure 2.6, with the numbers along the y-axis correspond to the numbered childhood covariates listed in Table 2.1. Looking at Figure 2.6, only one covariate (i.e., early trouble with the police) was out of range before matching, although a few other covariates also approached the limits. The geometric mean (a metric suggested by Linden & Samuels, 2008) was also estimated to represent the overall distributional balance among covariates and is presented at the bottom of the figure. After

matching, all covariates (and the geometric mean) were tightly clustered around 1, indicating distributional balance in the matched sample was achieved.

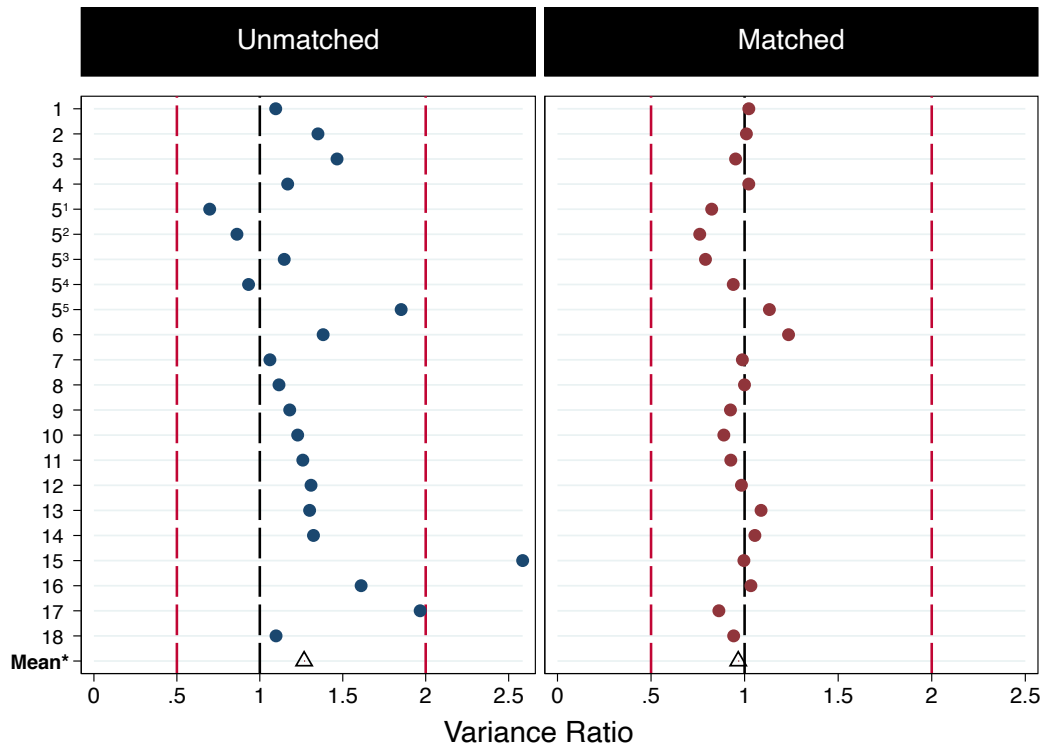


Figure 2.6. Variance ratios across childhood covariates between treated and control cases (Note: mean=geometric).

Fourth, propensity score balance across treated and control cases was assessed visually before and after matching with a kernel-density plot (see Figure 2.7). As seen in Figure 2.7, before matching the treated and control cases were divergent on the propensity score, particularly on the low end of the propensity score distribution. After matching, however, only a small departure of the control cases on the low end of the propensity distribution was apparent. This difference indicates that treated cases with a lower propensity score were outnumbered by the control cases.

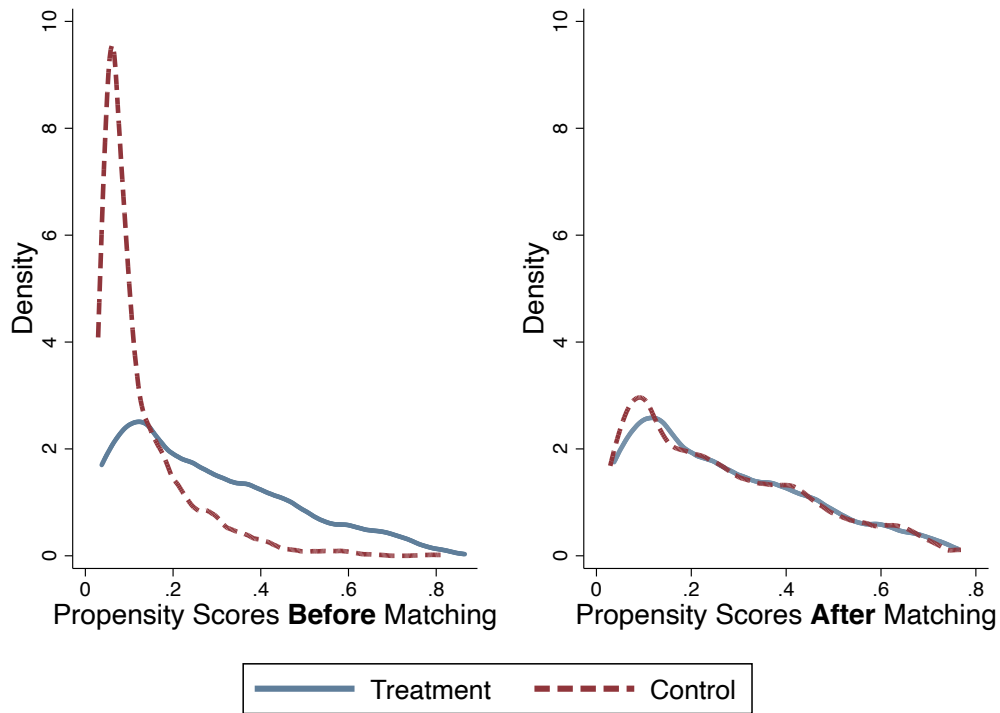


Figure 2.7. Kernel density plots of propensity scores for Lifetime Incarceration across treated and control cases.

The fifth step to the propensity score analysis, having achieved covariate balance, was to estimate the treatment effect of the *Lifetime Incarceration on PhenoAge Acceleration* by comparing the treated cases and matched controls. Specifically, the average treatment effect on the treated (ATT) was estimated. The ATT is defined as:

$$ATT = E(Y_1 - Y_0) | A = 1 \quad (2)$$

where E is the expected value for differences in potential outcomes of receiving the treatment A (i.e., Y_1) and not receiving the treatment (i.e., Y_0) for individuals who in fact received the treatment (i.e., $A=1$). We have observed values for Y_1 (i.e., Y for those who experienced incarceration) but values for Y_0 are unobserved and must be estimated. The PSM model will estimate Y_0 by observing Y among the matched controls, allowing for an estimate of the ATT.

Is the Association Between Lifetime Incarceration and PhenoAge Acceleration

Robust to Childhood Selection Factors? Looking at the model for the full sample in Table 2.7, we see that the ATT for the *Lifetime Incarceration on PhenoAge Acceleration* was nonsignificant. Specifically, *Lifetime Incarceration* was associated with almost a year’s worth of *PhenoAge Acceleration* (ATT=0.91; bootstrapped CI=-0.03-1.85; reps=50), but the effect was nonsignificant. The effect size (SMD) was 0.01, which indicates a trivial effect (Cohen, 1988). Finally, the variance ratio between the treated cases and their matched controls was 1.11 suggesting that distributional differences did not play a role in skewing the results. All told, the PSM analysis revealed that in the HRS sample males with a history of *Lifetime Incarceration* did not reliably demonstrate a biological age that surpassed their chronological age (i.e., after adjusting for selection due to childhood factors).

Table 2.7. The Average Treatment Effect on The Treated (ATT) of Lifetime Incarceration on PhenoAge Acceleration (N=3036).

	$\bar{Y}_{Treated}$ (N)	$\bar{Y}_{Control}$ (N)	ATT	Bootstrap 95% CI (Reps=50)	SMD	VR
Full	3.00 (441)	1.95 (2588)	0.91	[-0.03, 1.85]	0.01	1.11
Full (minus Black)	2.96 (324)	1.09 (2325)	1.87**	[0.48, 3.26]	0.21	1.26
Black	3.27 (119)	4.53 (263)	-1.76	[-4.48, 0.95]	0.16	0.99
White	3.09 (242)	0.75 (1965)	2.31**	[0.76, 3.85]	0.26	1.46

** $P < 0.01$. ATT=average treatment effect on the treated; CI=confidence interval; SMD=standardized mean difference; VR=variance ratio; \bar{Y} =group-specific mean for PhenoAge Acceleration.

Sensitivity Analysis

As indicated by the regression analysis, Black HRS participants presented with a different pattern of association between *Lifetime Incarceration* and *PhenoAge Acceleration* than was observed among the rest of the sample. It is possible, then, that the Black subsample may be suppressing the effects for the rest of the sample. In order to assess this possibility, the PSM

model was stratified in three ways: 1) with Black male participants removed, 2) with only Black male participants, and 3) with only White male participants included. (Note: due to the small group sizes and low numbers of incarcerated individuals, I did not stratify by Hispanic ethnicity or by those of other race/ethnicity groups).

Turning to the second model of Table 2.6, the sample of HRS males with Black participants removed demonstrated a statistically significant effect of *Lifetime Incarceration* on *PhenoAge Acceleration* (ATT=1.87, bootstrapped CI=0.48, 3.26). This finding suggests that being a non-Black male who has experienced incarceration at some point during the life course was associated with an almost 2-year acceleration of biological age over chronological age. The effect size of this result is 0.21, meaning that it is a “small effect” (Cohen, 1988). In contrast, Black male HRS participants demonstrated an ATT of -1.76 (bootstrapped CI=-4.48, 0.95; reps=50), but confidence intervals of the ATT overlap zero by wide margins meaning we should avoid placing any emphasis on this result.

In the final model, White male HRS participants demonstrated an ATT of 2.31 (bootstrapped CI=0.76, 3.85; reps=50), suggesting that white males with a history of incarceration can expect for have a biological age in excess of their chronological age by ~2.3 years. The effect size of the result is 0.26, meaning that effect is still considered a “small effect” (Cohen, 1988). Based on the above results, it appears that the Black subsample of the HRS was suppressing the association between *Lifetime Incarceration* and *PhenoAge Acceleration*. This finding suggests that some process unique to Black males may be occurring that alters the effect of incarceration on their biological aging later on. For the majority of males, however, the hypothesized relationship between incarceration and biological age appeared to be supported (although the size of the effect was small-to-modest).

Discussion

The current study represents a preliminary investigation into the association between incarceration and biological aging. While previous work has highlighted the marked health disparities among the current and formerly incarcerated (Massoglia & Pridemore, 2015), no studies have examined whether incarceration might actually be associated with the more general phenomenon of aging (i.e., the primary driver of morbidity and mortality later in life; Kennedy et al., 2014). This is an important distinction to make because incarceration has been linked to a long list of later morbidities and mortality, but few potential mechanistic pathway(s) that link the experience of incarceration to the many diverse health outcomes it impacts have been uncovered. The current study serves to highlight the possibility that biological aging is part of the mechanism(s) that links incarceration to poor health.

The novel component of this study is that it examined biological aging, which can be thought of as a general latent factor of morbidity and mortality. This proposition aligns with much of the prior work examining the health effects of incarceration that rely on Pearlin's (1989) stress process paradigm. The stress process paradigm suggests that events like incarceration influence health in at least two ways: 1) as primary stressors, which are the direct experiences associated with the event that evoke a stress response (e.g., prison crowding, social isolation, inmate altercations); and/or 2) as secondary stressors, which are the result of the initiating event and also elicit a stress response (i.e., a criminal label/stigma). Applying the stress process model to the current study, the primary and secondary stressors association with the becoming incarcerated are believed to accelerate biological age, which then places the former inmate at a higher risk of developing not one but many different morbidities.

Though not fully tested in the current study, the above results provide suggestive evidence for this perspective. Incarceration was associated with an increase in biological age beyond one's chronological age. This pattern emerged across sex (but not race/ethnicity) and was robust to adjustment for potential confounding influences. When viewed through the lens of sex, expected patterns of aging differences appear. Males were generally worse off, and incarcerated males were worst of all in terms of biological age. Racial/ethnic differences acted more as a prism than a lens, however, as group dynamics varied widely in terms of the magnitude, and even direction, of the association between incarceration and biological aging. For instance, White, Hispanic, and participants of other backgrounds all reported higher average levels of biological aging among those individuals with a history of incarceration.

The findings for Black participants were unique in that they did not demonstrate an association between incarceration and biological age, and what little indication there was of a possible trend was in the direction opposite that of the rest of the sample. This finding suggests that incarceration may not be a meaningful dimension across which Black males vary in terms of their biological age.

One major source of confounding for both health and criminal justice contact is the childhood environment one grew up in. After adjusting for a host of childhood covariates using propensity score matching, the effect of incarceration on biological age did not remain for the full sample of males. Sensitivity analyses revealed that, due to the abovementioned unique patterns of association for Black participants, the effect was being suppressed for the other groups. After removing Black participants, the hypothesized relationship between incarceration and biological age reappeared (i.e., after adjusting for childhood confounders). The general trend

in the observed effects was that non-Black males with a history of incarceration could expect their biological age to acceleration by about two years.

Though substantively small, the effects observed here suggest there may be something about the incarceration experience itself that affects the way in which individuals age. It is important to remind the reader that the PSM adjustment for confounding only adjusted for childhood covariates. Thus, any confounders more contemporaneous to the incarceration experienced were not adjusted for, making the results reported here tentative and in need of replication and extension. Future studies attempting to examine the incarceration-biological age relationship are encouraged to use data that include the timing of incarceration, thus allowing for the adjustment of additional sources of confounding.

These findings also suggest future research should “look backward” at the criminal career to determine whether the “incarceration effect” observed in this study can be attributed to the incarceration experience itself, or if perhaps the “incarceration effect” is instead a proxy indicator of criminal lifestyle and its influence on biological aging. The current analysis adjusted for some of the structural factors that may predispose individuals to come into contact with the criminal justice system, but it could not adjust for criminal behavior/offending directly due to the lack of available data. Because the current analysis was unable to address the latter possibility, exploring the relationship between early criminal lifestyle factors and biological age will be the focus of the next chapter.

Limitations

The current analysis was intended as a preliminary assessment of whether the most severe form of criminal justice contact, namely incarceration, is associated with variation in biological age later in life. In order to make such a demonstration, I turned to one of the few datasets in world—the HRS—that contained the requisite data, including comprehensive

biomedical data on blood-based biomarkers and information on incarceration experiences. The HRS proved to be a uniquely useful data source; however, the analysis is not without limitations that future studies should seek to overcome.

First, the crudeness of measurement of incarceration in the HRS is something that should be addressed by future work. The incarceration measure groups together several major forms of incarceration (i.e., jail, prison, juvenile detention, other). These forms of incarceration are vastly different, and each has different implications in terms of the character of the incarceration experience, as well as the type of offenders who will be exposed to it. For instance, whether an individual spent time in a juvenile detention facility or a jail/prison points to their likely age of admission (i.e., juvenile vs legal adult). Whether an individual experienced jail or prison gives some indication of the length of their sentence (e.g., <1 year for jails, >1 year for prisons). Additionally, the incarceration measure does not distinguish between state and federal prisons.

Second is a related issue concerning the lack of specificity with regard to the number of incarceration terms served and their lengths, the security level and condition of the correctional facility, as well as their experience during sentence. The dichotomous approach to measuring incarceration in the HRS is not unique in the incarceration-health literature, although researchers have begun to call for improvements in this approach as it limits the ability to identify the vast heterogeneity within to incarceration experience (Kirk & Wakefield, 2018; Massoglia & Pridemore, 2015; Porter & DeMarco, 2019). However, criminal justice contact measures are rarely included in large national health surveys like the HRS. Some large longitudinal studies have begun to include in-depth measurement of criminal justice contact (e.g., the National Longitudinal Study of Adolescent to Adult Health [Add Health], the Fragile Families and Child Wellbeing Study), but these studies focus on cohorts that are still too young for an appreciable

number of participants to have 1) come into contact with the criminal justice system, 2) spent time in a correctional facility, and 3) begun to demonstrate age-related symptomatology.

Third, the advanced age of the HRS sample (i.e., 50+) does provide a means of observing a group of individuals with little fear of right-censoring of the incarceration experience (i.e., most individuals will have spent all of the time behind bars that they will in their lifetime by age 50). Given that the HRS only commenced in 1992, this also means that all of the data on incarceration and childhood covariates were retrospective and may suffer from recall bias. Prospective data on early risk factors would provide a more reliable means of adjusting for selection for coming into contact with the criminal justice system.

Fourth, the age of the sample also suggests the possibility of survivor bias (i.e., differential attrition), the effect being that the sickest individuals were not included when estimating the association between incarceration and biological age. It is important to pause and consider how this concern may have impacted the results. Recall that the overall pattern uncovered in this study was that incarceration experiences are correlated with advanced biological aging (with the exception of the Black subsample). If these patterns are indicative of actual causal directions (i.e., if we assume the direction of association is correct), then the impact of survivor bias would mean that the estimates presented above are conservative (i.e., closer to zero than they would be if the bias were not present). But caution is encouraged here because this explanation assumes the direction of association has been estimated correctly. If this assumption fails, then the biasing influence would be the opposite.

Fifth, lifetime incarceration in this analysis may actually be acting as a proxy for individuals who lived a “hard” lifestyle or who were exposed to a “hard” environment (e.g., areas of concentrated disadvantage). This could mean that the “incarceration effect” identified in

this study is not a true causal effect, but rather an indicator of much broader lifestyle factors that puts these individuals at generalized risk for later morbidity and early mortality.

Conclusion

At its core, incarceration is a temporal sanction that is measured in years or months and experienced by the inmate second by second. Whether paired with the goals of punishment or rehabilitation, incarceration is always a means of achieving incapacitation—an irrevocable loss of time. What the criminal justice system and the public have not yet realized is that incarceration may be taking away biological time at a faster rate than it takes away chronological time. As inmates suffer the disconnection from the social world, they may also be aging prematurely, a process that signals the early onset and accelerated progression of age-related morbidities and mortality. In essence, inmates might be doing more “biological time” than their sentence prescribed.

But such conclusions must be made cautiously because this is the first study of its kind. Noting the limitations outlined above, it would be premature to declare a discovery of “the incarceration effect on aging.” Some of the complications that arose in this study must be overcome before we can be sure we have identified the true impact that incarceration has on aging. Recognizing these points inspired the study that comes in the next chapter. In order to assess whether incarceration has a causal influence on biological aging, we need to gain a better sense of whether incarceration has its own influence, or if it is simply a proxy for a “hard”, criminal lifestyle. Acknowledging that incarceration is, in many ways, the endpoint of a long process of offending and criminal justice contact, prior offending and criminal justice contact should also be investigated. The next chapter takes this next step in exploring the association between the criminal lifestyle and biological age. As this analysis focuses on behaviors and

outcomes earlier in the criminal career (i.e., at least prior to incarceration), chapter 3 will change datasets to a prospective birth cohort that covers the first 45 years of life for a birth cohort from New Zealand—the Dunedin Longitudinal Study.

**Chapter 3 —A Young Man’s Game: Early Offending Behavior and its Influence on
Biological Age**

Introduction

Age is one of the most long-standing and universally acknowledged correlates of crime. Going as far back as the 1800s, Quetelet, a French mathematician, wrote that: “Among all the causes which have an influence for developing or halting the propensity for crime, the most vigorous is, without contradiction, age” ([1831] 1984, pp. 54-56). Modern criminological research on the “age-crime curve” has observed that, with very little variation, offending rates consistently adhere to a singular pattern: sharp increases during early adolescence, a peak in late adolescence, and an asymptotic decline throughout adulthood. The age-crime curve has been said to be universal, in so far as it does not vary across time, space, or crime type (Hirschi & Gottfredson, 1983)—though some research contests the full validity of these claims (e.g., Steffensmeier et al., 1989).

As a result, criminology has incorporated age into several of its theoretical paradigms. There are two main criminological explanations of the age-crime relationship. First, age is assumed to exert an exogenous influence on offending rates. In this case, “exogenous” stresses the idea that age will have an influence on crime and that influence is 1) invariant, 2) non-interactive (i.e., it is not moderated), and 3) inexplicable (i.e., it cannot be explained away through mediation) (Hirschi & Gottfredson, 1983). This perspective ultimately casts age as something that is unnecessary to study because it is purely exogenous, meaning it is something that “just happens” to all of us. Age, therefore, cannot be subject to intervention, which makes it uninteresting from a scientific perspective. This perspective even draws into question whether age can be viewed as a causal factor (see, generally, Holland, 1986, 2003)

The second criminological explanation of the age effect is that age is a temporal mile marker that signals the onset of various developmental (e.g., puberty, maturation) (Moffitt, 1993)

and societal events (e.g., entry into the labor force, marriage) (Sampson & Laub, 1993) that, in turn, exert their influence on crime rates. Both of these perspectives align in assuming age exerts *some* effect on offending (age→offending). What both perspectives fail to consider, though, is the reverse possibility (age←offending).

The purpose of this study is to propose and investigate a novel way of conceptualizing age—one that does not view age as an exogenous influence, nor does it view age as a simple indicator of factors that may have taken place in the social world. Instead, I propose that age plays an active role in that it both affects, and is affected by, the criminal lifestyle. This novel approach to studying the age-crime relationship coincides with recent developments in the field of geroscience, which is the study of biological aging. Using measurement strategies developed by geroscience scholars, I examine the relationship between a concept known as biological age and offending over the life course using the Dunedin Longitudinal Study, a prospective cohort study of $N = 1,037$ New Zealanders. In the sections that follow, I review the literature linking offending behavior to health outcomes and describe how these associations may be substantively extended by considering biological age as a mediator—rather than an exogenous cause—of the relationship between chronological age and offending.

Literature Review

The central focus of life-course criminology is on the “effects of life events and life transitions on offending...” (Farrington, 2003). Life events and transitions like marriage, entering the workforce, and military service have been the primary focus of much of the life-course literature—the assumption being that such events presage changes in social ties (Sampson & Laub, 1993) or cognitive transformations (Giordano et al., 2002) that facilitate desistance from crime. Interestingly, the backdrop that exists behind each of these life events/transitions—

age/aging—has received relatively little direct scrutiny from developmental/life-course criminological research. The classic assumption among mainstream criminological theories is that age is largely a passive force, mostly useful for marking the passage of time or the arrival of age-related events (e.g., puberty, maturation, legal majority). The purely temporal conception of age is limiting, however, as it keeps researchers' sights set on those things associated with age rather than asking the most basic question: what is age?

The use of chronological time (i.e., number of years lived since birth) to mark the progression of age has proven useful as much of the human life course is associated with time in systematic ways (Costa & McCrae, 1980). But the concept of age evokes much more than simply the number of years lived since birth. Age also serves as a crude tracker for many factors such as knowledge accumulation, maturity, independence, physical/mental capabilities, and health. This complex interplay between age and other factors can be seen in the notion of “lifespan development” out of lifespan psychology (Baltes & Nesselrode, 1984). Lifespan development is conceptualized as a lifelong process of continual growth, maintenance, and decline, and it is influenced by three key factors: age-graded factors (i.e., biological and/or social events that occur with statistical regularity at certain ages), history-graded factors (i.e., the cultural/historical factors that shape environments and the social behaviors acted out within them), and non-normative factors (i.e., the stochastic events that impact an individual's behavior) (Baltes & Smith, 2004). These three factors come together to shape behavior over time and lead to the gradual decline in physical and mental capacity as individuals navigate the latter part of the life course.

The monotonicity of age-related declines in cognitive function, and health more broadly, means that chronological age (i.e., a linear construct by definition) is seen as a consistent and

powerful predictor in studies of health and aging (Costa & McCrae, 1980). There is variation in the onset and progression of age-related morbidities, however, and it is this variation that chronological age is insensitive towards. Life-span developmental psychologists conceptualize this variation as the result of “ontogenesis”, or the gradual unfolding of development (Baltes & Nesselrode, 1984). As individuals age, the variability of any given trait is known to “fan out” due to the interactions of these three factors, accentuating individual differences and producing “aged heterogeneity” in outcomes (Dannefer, 1987). The upshot of this is that, while most physical, mental, and behavioral traits of interest to social scientists decline monotonically with age, there is wide and increasing variation within those traits of interest that is not explained by chronological age.

Recently, the field of geroscience has endeavored to move beyond chronological age and situate the process of aging within the body by mapping the biological “hallmarks of aging” (López-Otín et al., 2013). Because virtually every age-related process is traceable to physiological changes in the body, geroscience has placed special emphasis on biological age—which captures the overall integrity of the body’s many organ systems—rather than chronological age as the primary determinant of health and longevity. It is not enough to attribute declining health to chronological age, a construct that gives little information with which to pursue interventions. Rather, biological age asserts that age-related morbidities have biological roots, the identification and quantification of which facilitates the study of aging as a biological process, as well as provide substantive grounds for intervention.

The current study seeks to uncover whether the effect of biological age extends beyond health-related outcomes and into the realm of behavior. There is reason to believe that biological age may play a particularly important role for behaviors like offending, owing to the large body

of evidence suggesting a strong age-crime association (Hirschi & Gottfredson, 1983). The logic being that biological age, which may be seen as a biological means of quantifying one's place in the course of lifespan development (i.e., arising from the age-normative, history-graded, and non-normative factors; Baltes & Smith, 2004), may be impacted by highly taxing behaviors like crime. In the sections that follow, I motivate an examination of offending and biological age across the life course by: 1) examining the literature linking offending with health outcomes—the focus on health, as opposed to chronological age, is necessary as biological age is primarily a measure of subclinical variations in health-related biomarkers; 2) reviewing the recent developments around the quantification of biological age and its association with health outcomes, and 3) outlining a theoretical framework that incorporates biological age into a life-course model of offending.

Offending and Health Outcomes Across the Life Course

Criminologists are often victim-centric in their investigations of the crime-health relationship, focusing on the public health costs of crime (e.g., Shepherd & Farrington, 1993), as well as the physical, mental, and emotional burdens experienced by victims of crime (Tan & Haining, 2016). Less attention is generally paid to the health costs experienced by the offenders themselves. One study, an examination of 411 males from the Cambridge Study of Delinquent Development (Farrington, 1995), found that earlier offending (i.e., self-reported and official convictions) was predictive of poorer health outcomes in the form of hospital visits for injury and illness, as well as road accident and fighting-related injuries. What is more, these relationships remained consistent across ages 18-32. A later analysis of the same sample found that offenders who were characterized as being high-rate, chronic offenders had the highest risk of hospitalization and being registered as disabled later in life compared to other types of

offenders in the sample (Piquero et al., 2011). These findings support the most apparent explanation for a crime-health association: crime is dangerous, and criminals get injured.

A number of studies have gone beyond looking at injuries to test for associations between offending and physical/mental health disorders. Relying on Moffitt's (1993) taxonomic theory, one study examined 526 males from the Dunedin Longitudinal Study (i.e., the same data as the current study) and identified four groups of offenders using trajectory analysis: 1) life-course persistent (LCP), 2) adolescent onset, 3) childhood limited, and 4) low (Odgers et al., 2007). Comparing health outcomes across these groups, the analysis revealed that physical and mental health burdens increased across groups with severity of offending (i.e., LCP > adolescent onset > childhood limited > low). Of the fourteen physical health outcomes measured (e.g., respiratory function, periodontal disease, cardiovascular disease risk), respondents in the LCP group experienced significantly worse physical health in 12 outcomes compared to the low group, 8 compared to the childhood limited group, and 4 compared to the adolescent onset group. Although they made up a small proportion of the overall sample (i.e., 10.5%), members of the LCP group were responsible for 17.5% of the traffic injuries and 29.4% of the days spent in psychiatric hospitals experienced by all of the respondents.

Another study using data from the National Collaborative Perinatal Project also observed that LCP offenders (i.e., defined as the top 5% of individuals in terms of arrest frequency) experienced a disproportionate number of adverse health conditions compared to the rest of the study sample (Piquero et al., 2007). Specifically, LCPs were nearly two times more likely to report suffering at least one major health condition, including heart trouble, hypertension, kidney problems, diabetes, and ulcers compared to the rest of the sample. Thus, evidence seems to be

consistent with a model of crime-health association wherein criminals are not only more susceptible to injury but also to additional or accelerated onset of multiple disease morbidities.

The health-crime literature has also found that when offending behavior brings individuals into contact with the criminal justice system, additional health costs can be incurred (e.g., Kirk & Wakefield, 2018; Massoglia & Pridemore, 2015). Typically measured as incarceration, criminal justice contact has been found to be associated with severe health-related impairments (Schnittker & John, 2007), mental health symptoms (Massoglia, 2008; Sugie & Turney, 2017), and greater prevalence rates of infectious diseases (Massoglia, 2008). While some see incarceration as the most impactful stage of criminal justice contact, some scholars have noted the impact of lesser forms of criminal justice contact. For instance, using data from the National Longitudinal Survey of Youth (NLSY97), Sugie and Turney (2017) found that nearly half of the association between incarceration and poor mental health was explained by arrest experiences.

The above literature has demonstrated that offending, offending trajectories, and criminal justice contact are all associated with many different negative health outcomes. For instance, most analyses of crime include some combination of subjective health ratings (e.g., Farrington, 1995; Odgers et al., 2007; Semenza et al., 2020), chronic diseases (e.g., Odgers et al., 2007; Piquero et al., 2007), gum disease (Odgers et al., 2007; Testa & Fahmy, 2019), minor health conditions (Semenza et al., 2020; Stogner et al., 2014), accidents/injuries/hospitalizations (Farrington, 1995; Odgers et al., 2007; Piquero et al., 2007; Piquero et al., 2011), time off work due to illness/injury (Farrington, 1995; Piquero et al., 2011), or death (Piquero et al., 2011; Skinner & Farrington, 2020). The large number of associated health outcomes may actually be a

limitation of the crime-health literature, however, because it complicates the mechanistic pathway between crime and health.

The Crime-Health Relationship: A Question of Mechanisms

The health criminology literature, though still emerging, has identified the criminal lifestyle as substantive risk factor for later morbidity and mortality. A key concern, however, is explaining the mechanistic pathway from crime to health. Two possible explanations of the relationship are possible. First, the crime-health relationship is confounded by lifestyle factors. This “crime-as-proxy” explanation suggests that offenders live particularly “hard” lives (both in terms of circumstances and behavior) and that, short of these other factors, crime would fail to predict differences in health outcomes. A second explanation draws on the stress literature and suggests that offending is mechanistically linked to health through physiological stress. This explanation, built on the work of Pearlin (1989), suggests that stress is generated from two types of stressors: 1) primary stressors, which are the specific stress-causing events (e.g., the commission of a crime); 2) and secondary stressors, which are the results of the initial event but elicit stress-responses in their own rite (e.g., criminal labels/stigma). The accumulation of physiological stress from both primary and secondary stressors are hypothesized to produce a number of negative health outcomes if exposure is sustained.

The proxy explanation of the crime-health relationship is intuitive and aligns with much of the literature exploring the lifestyles of offenders. For instance, individuals high in offending tend to be of lower socioeconomic class (Skarðhamar, 2003), engage in higher levels of substance use (Mulvey et al., 2010), and have lower access to health care (Hawkins et al. 2010). What is not known, however, is whether the criminal lifestyle goes beyond these factors to produce negative health outcomes that are not explainable by lifestyle factors alone.

This dissertation explores a stress-based explanation of the crime-health relationship by attempting to adjust for lifestyle factors. Given the wide variety of negative health outcomes that appear to be associated with a life of crime, this dissertation posits that offending influences the physiological integrity of a body through stress, which then increases the general susceptibility of offenders to morbidity and mortality. Recent developments in the field of geroscience have produced methods for quantifying this physiological deterioration, which they refer to as “biological age”. I now turn to a discussion of “biological age”, highlighting its possible role in the crime-health relationship.

Biological Age: Theoretical Construct, Quantification, and its Association with Health

Geroscience research shows that aging is a biological *process* (Baltes, 1987; Baltes & Nesselrode, 1984) marked by declining integrity of multiple organ systems throughout the body (López-Otín et al., 2013). The integrity of an organ system directly relates to its functional status. If the organ system is whole and in a conducive environment (i.e., it is surrounded by other integral systems), the likelihood of dysfunction is low. If the integrity of a system is low, however, dysfunction can become more common. If not addressed through intervention or by one of the body’s many mechanisms for maintaining homeostasis, the dysfunction may result in a more permanent condition and, eventually, to the collapse of the system (i.e., total integrity loss). Thus, biological age tracks the decline of integrity of the body and is highly associated with morbidity and mortality.

The operationalization of biological age has received increased attention in recent years (e.g., Belsky et al., 2018; Jylhävä, Pedersen, & Hägg, 2017; Levine, 2013). The process of operationalization begins with the identification of “biomarker[s] of aging,” which are, as Baker and Sprott (1988) defined them, “biological parameter[s] of an organism that either alone or in

some multivariate composite will, in the absence of disease, better predict functional capability at some late age than will chronological age” (p. 223). While singular biomarkers of aging have been identified previously (e.g., telomere length), these biomarkers have only demonstrated moderate predictive validity (for a review, see Jylhävä, Pedersen, & Hägg, 2017). In recent years, the focus has shifted to a ‘multivariate composite’ approach to identifying biomarkers of aging. Algorithms combine information from multiple biospecimens across the body in order to predict aging outcomes (e.g., chronological age, mortality).

The multivariate composite biomarkers, in contrast to the single biomarkers of aging, have proven to be powerful tools in the study of aging, even outperforming chronological age in the prediction of morbidity and mortality (Levine, 2013)—a long-held goal of geroscientists (Costa & McCrae, 1985). For instance, after adjusting for chronological age, multivariate composite biomarkers of aging have been able to differentially predict all-cause and cause-specific mortality (Levine, 2013; Liu et al., 2018), in both healthy and diseased individuals (Liu et al., 2018), as well as cause-specific morbidities (e.g., stroke, cancer, diabetes; Waziry et al., 2019) and disease count (Liu et al., 2018). Overall, the use of multivariate composite biomarkers of aging allows health researchers to tap into the individual differences in aging that make up an individual’s biological age and are undetectable with the use of chronological age.

An example may prove helpful for drawing out the distinction between chronological and biological age (see to figure 3.1). Consider an individual named Achilles who is 60 years old according to his chronological age (i.e., years since birth). If we assume Achilles has engaged in an unhealthy lifestyle (e.g., sedentary, poor diet, heavy alcohol use) from age twenty until now, a measure of biological age may give him a score of 70 (see the dashed red line in Figure 3.1). Having a higher biological age than his chronological age suggests that Achilles’s aging process

has become accelerated and he can expect to experience the health and functionality of a man 10 years his senior (e.g., early onset of geriatric conditions). It should be noted that poor lifestyle choices are not the only means by which biological age may become accelerated, however; individuals who possess genetic liability to premature aging, are exposed to adverse life events, or experience chronic health conditions are also likely to have an accelerated biological age.

Now picture a scenario in which Achilles engaged in a healthy lifestyle (e.g., regular exercise, good diet, adequate sleep) from age twenty onward. A measure of biological age may give him a score of 50 (see the dashed blue line in figure 3.1), indicating that his biological age has become decelerated compared to his chronological age. Accordingly, Achilles will likely experience the health and functionality of a man 10 years his junior (e.g., delaying the onset of geriatric conditions).

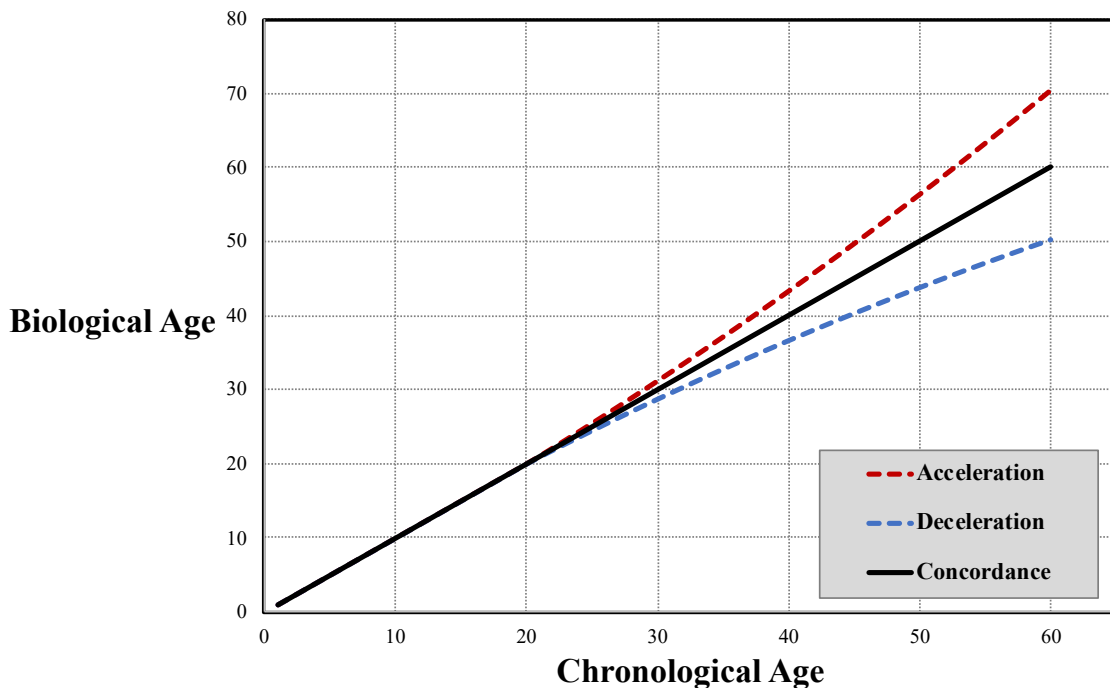


Figure 3.1. Biological age concordance, acceleration, and deceleration from chronological age.

We now return to the discussion of how biological age is operationalized. A multivariate composite biomarker of aging typically includes information from a variety of biological measurements including, anthropometric (e.g., body mass index [BMI], blood pressure, heart rate) and blood-based biomarkers (e.g., cholesterol, glucose, c-reactive protein, interleukin-6). With the use of different algorithms, these biomarkers have been used to produce many metrics for biological age (e.g., Belsky et al., 2015a; Cohen et al., 2013; Klemera & Doubal, 2006; Levine et al., 2018). The multivariate composite biomarker of aging method is a powerful approach to measuring biological age because of its breadth. By assessing multiple biomarkers that signal the integrity of many different organ systems, a multivariate composite biomarker of aging can be thought of as a means of gaining global estimates of biological age.

One commonality among virtually all methods for quantifying biological age is that they are cross-sectional approaches. Multivariate composite biomarkers of aging represent a snapshot of the internal environment of an individual's body at a single point in time. While predictive of later morbidities, the body's internal environment can vary for a large number of reasons that are not substantively related to aging more broadly. In acknowledgement of this, a new longitudinal multivariate composite biomarker of aging has been developed using the same data used in the current study (i.e., the Dunedin Longitudinal Study). "Pace of Aging", as it is called, was calculated by estimating the longitudinal changes in 19 biomarkers across 12 chronological years (i.e., age 26-38) for each person in the sample (more details are provided in the methods section; but see Belsky et al., 2015a). Pace of Aging thus captures the rate of decline in a person's physiological state and is comparable to the rate of physiological change expected of a single chronological year. Going back to the example used above, this means that if Achilles received a Pace of Aging score of 1, then his biological age has been increasing (i.e., physiological integrity

has been declining) with an exact year-to-year correspondence with his chronological age. If, however, he received a score of 1.5m, then Achilles's biological age would have increased at 1½ times the rate expected for a single chronological year increase.

Research into the predictive utility of biological age measures has produced a number of important results. First, though typically highly correlated with chronological age (Costa & McCrea, 1985), measures of biological age are able to predict health outcomes independent of the influence of chronological age (e.g., Levine, 2013). For instance, Levine (2013) used an algorithm developed by Klemera and Doubal (2006) to estimate biological age in the NHANES-III data and found that, after adjusting for chronological age, participants could expect a 9% increase in their risk of mortality over a 12-18 year period for each year increase in biological age (Hazard Ratio=1.09; CI=1.08-1.09). This reveals that biological age is tapping into something more than just number of years since birth.

Second, changes in biological age have been shown to substantively differ from individuals' chronological ages and these differences have been predictive of later morbidity (Belsky et al., 2015a; Levine, 2013) and mortality (Chen et al., 2016; Levine et al., 2018). Third, differences between biological and chronological age appear relatively early in life (e.g., age 26; Belsky et al., 2015a). Fourth, measures of biological age predict health outcomes among individuals with and without disease diagnoses (i.e., they are able to detect subclinical variation in health-related biomarkers) (Belsky et al., 2015a). For example, Belsky and colleagues (2015, p. 4107) observed that their longitudinal measure of aging, "Pace of Aging", was normally distributed in their study sample despite only 1.1% of participants being diagnosed with a chronic disease by the end of the observation period (i.e., from age 26-38).

Fifth, though all are attempting to capture the internal state of the body, many commonly used measures of biological age appear to predict different health outcomes and are only moderately correlated with one another (Belsky et al., 2018). For instance, Belsky and colleagues (2018) compared eleven different biomarkers of aging and found that, while each predicted some aspect of aging (e.g., physical functioning, cognitive performance, subjective aging), the individual biomarkers were only moderately associated with one another (highest $|r|=0.56$; lowest $|r|=0.01$). This point suggests individual measures of biological age are not exhaustive but are instead overlapping and/or complementary.

Sixth, and finally, research has shown that biological age is sensitive to environmental stressors and lifestyle factors (Belsky et al., 2017; Quach et al., 2017). This final point provides the impetus for the current study's investigation of biological age—if biological age is responsive to lifestyle factors, then life-course criminology may need to incorporate such findings into contemporary theories and research into the impact of age on crime.

The research reviewed above points to a consistent, though complicated, relationship between offending and later health outcomes. This association was observed, both in terms of injury and disease morbidities. Support for the crime-health association is a necessary prerequisite for reconceptualizing age from a chronological abstraction into a biological state. As biological age is now considered the preeminent predictor of morbidity and mortality (i.e., beyond that of chronological age), I am able to postulate that offending, rather than simply being affected by the passage of chronological time, is in a dynamic relationship with the internal biological states experienced by offenders.

The Current Study

This study brings together life-course/developmental criminology and modern geroscience in an attempt to reconceptualize a classic criminological relationship: the age-crime relationship. To accomplish this, I shift focus away from “age” as a temporal abstraction and instead emphasize “aging” as a biological process. One product of “biologizing” age, as I advocate, is that it can now be conceptualized as playing an active role in the lives of offenders—age affects, and is affected by, the criminal lifestyle. While the prior age-crime literature has thoroughly documented the effect of age on offending, little research has studied the reverse (though, it is important to note that some scholars [e.g., Shover & Thompson, 1992] have acknowledged that the role of age is more complicated, especially its impact on/with emotion and other subjective factors). Aided by a biological reconceptualization of age/aging, the current study provides the first evidence of a possible impact of offending on the aging process.

The current study will test the following hypothesis:

H2—Offending behavior early in the life course influences the aging process later in the life course.

I test this hypothesis using data from the Dunedin Longitudinal Study to test if offending early in life (i.e., from age 15-26) is associated with accelerated Pace of Aging throughout middle adulthood (i.e., from age 26-45). In order to test this model, we examine offending in three different ways: 1) average amount of offending, 2) pattern of offending behavior, and 3) contact with the criminal justice system (i.e., criminal conviction). We capture biological aging in middle adulthood with use of the “Pace of Aging” measure developed by Belsky and colleagues (2015).

Methods

Data

To analyze the relationship between crime and biological aging, I will use data from a prospective birth cohort study, the Dunedin Multidisciplinary Health and Development (Dunedin Longitudinal) Study. The Dunedin Longitudinal Study includes prospective data collection on $N = 1,037$ individuals born in Dunedin, New Zealand, between 1972-73. The Dunedin study has conducted 13 phases of data collection at ages 3, 5, 7, 9, 11, 13, 15, 18, 21, 26, 32, 38, and 45. At the most recent phase (phase 45, collected in 2018-19), $N = 938$ individuals participated (i.e., 94% of the original sample still living).

The Dunedin Longitudinal Study is unique for its extensive collection of information on criminal (i.e., both self-reported and official) and deviant behavior (i.e., multisource, from self, parent, and teachers) throughout the life course. Additionally, the Dunedin Study has been a pioneering force in the incorporation of biological/physiological data into sociological study designs. It was among the first studies to collect candidate gene data in the early 2000s (e.g., see Caspi et al., 2002) and it remains at the cutting edge of biomedical research. The above factors make the Dunedin Study an ideal resource for analyzing the role of biological aging in the criminal career across the life course.

Measures

Pace of Aging. The “Pace of Aging” measure of biological age was created using data from phases 26, 32, 38, and 45 (see Belsky et al., 2015a). *Pace of Aging* was calculated in three steps. First, data on 19 anthropometric/blood-based biomarkers were collected at each phase and then standardized using z -scores ($M=0$, $SD=1$). The 19 biomarkers were (listed in Table 3.1): body mass index, waist-hip ratio, glycated hemoglobin (HbA1C), leptin, blood pressure (mean

arterial pressure), cardiorespiratory fitness (VO2Max), forced expiratory volume in one second (FEV1), forced vital capacity ratio (FEV1/FVC), total cholesterol, triglycerides, high-density lipoprotein (HDL) cholesterol, apolipoprotein B100/A1 ratio, lipoprotein(a), creatinine clearance, urea nitrogen, C-reactive protein, white blood cell count, gum health, and caries-affected tooth surfaces. Some variables were reverse coded so as to ensure that higher scores always indicated greater physiological deterioration (i.e., more advanced age). Second, longitudinal mixed-effects growth models were used to estimate random slopes of all 19 biomarkers from phase 26 to 45.

Table 3.1. Constituent Biomarkers of Pace of Aging.

1.	Apolipoprotein B100 & Apolipoprotein A1 Ratio	
2.	Arterial Pressure (Mean)	
3.	Body Mass Index	
4.	C-Reactive Protein	
5.	Cardiorespiratory Fitness	(Rev)
6.	Caries-affected tooth surfaces	
7.	Cholesterol (Total)	
8.	Creatinine Clearance	(Rev)
9.	Forced Expiratory Volume	(Rev)
10.	Forced Expiratory Volume & Forced Vital Capacity Ratio	(Rev)
11.	Glycated Hemoglobin Level	
12.	Gum Health (Combined Attachment Loss)	
13.	High-Density Lipoprotein	(Rev)
14.	Leukocyte Telomere Length	(Rev)
15.	Lipoprotein (A)	
16.	Triglycerides (Non-Fasting)	
17.	Urea Nitrogen	
18.	Waist-Hip Ratio	
19.	White Blood Cell Count	

Rev=reverse coded.

Finally, the individual slopes for each participant were summarized into a single *Pace of Aging* score. The resulting *Pace of Aging* measure was normally distributed in the sample,

suggesting that some individuals age faster and some slower than their chronological age would suggest. To aid interpretation, the *Pace of Aging* measure was scaled so that the central tendency of the measure reflected the physiological change expected over the course of a single chronological year (rescaling occurred separately for male and female participants). After rescaling, the sample ranged in the *Pace of Aging* from nearly 0 to almost 3 years of physiological change per chronological year. To illustrate the interpretation of *Pace of Aging*, imagine Achilles has a *Pace of Aging* score of 1.2. This would indicate that he will have aged (physiologically) the equivalent of 1.2 years for every year between ages 26 and 45 (i.e., the phases across which *Pace of Aging* is estimated). At age 45 then, Achilles can expect to have a biological age of ~49.

Self-Reported Offending Variety. Measures of self-reported offending were collected during phases 15, 18, 21, and 26. Participants were asked how many times in the past year they had committed 12 different deviant/criminal behaviors, including: running away from home, carrying a hidden weapon, destroying property, setting fire to property, braking into a building to steal, stealing less than \$100, stealing more than \$100, stealing from a store, stealing a motor vehicle, using force to rob, using marijuana, and using a harder drug (than marijuana). In order to create a variety index of offending, all non-zero responses were given a value of 1 and the resulting items were summed together.

This produced an index wherein higher scores represent more diversity in the types of offending committed by study members. Variety scores typically correlate with frequency (Monahan & Piquero, 2009), but the variety score is preferred over frequency because variety is less skewed, does not overweight trivial offenses (Sweeten, 2012), and is less affected by recall errors (e.g., “Have you shoplifted?” is more accurately recalled than “How many times have you

shoplifted?") (Moffitt et al., 2001). Finally, these measures of offending were averaged across all four phases to produce an indicator of *Average Offending Involvement* during the key years of offending (i.e., 15-26).

Developmental Trajectories. I rely on the conduct disorder (CD) trajectory groups identified in the Dunedin Longitudinal Study sample by Odgers and colleagues (2007). Based on Moffitt's (1993) taxonomic theory, Odgers and colleagues used general growth mixture modeling to assess the different trajectories of conduct disorder reported by participants across phases 7, 9, 11, 13, 15, 18, 21, and 26. Four trajectory classes were identified as the best-fitting solution for the data. The trajectory classes were identified as follows: Low, Childhood Limited, Adolescence Onset, and Life-Course Persistent. These *Developmental Trajectories* correspond to the offending groups proposed by the taxonomic theory (Moffitt, 1993) and provide theoretically and empirically supported groups that meaningfully differ in their levels of offending.

Developmental Trajectories provide more information on offending beyond that of *Average Offending Involvement*, as they also describe the patterning of offending behaviors throughout development.

Given the marked differences in the *Developmental Trajectories*—as well as their various lifestyle implications—analyzing trajectory groups provides an additional level of depth to the analysis of early offending behavior. It is important to note, however, that the *Developmental Trajectories* were estimated from data and not observed. As with any group-based trajectory framework, this runs the risk of grouping individuals together that are not substantively similar except in terms of the variables of interest (i.e., conduct disorder symptoms/offending, in this case). Analyzing group differences on variables not used in the construction of trajectory groups (e.g., Pace of Aging) should thus be approached cautiously.

In this scenario, Clark and Muthén (2009) recommend that group comparisons only be used for models with an entropy score of at least 0.8 (p. 32)—entropy being the average classification accuracy when assigning individuals to a trajectory group, with values closer to 1 indicating higher accuracy. According to this threshold, group comparisons were considered appropriate as the four-group solution originally estimated by Odgers and colleagues (2007) reported an entropy=0.8 (p. 479). Clark and Muthén (2009) additionally suggest that the method can deflate standard errors, which should be countered by adopting an alpha level that is more stringent than $p<0.05$. In accordance with their suggestion, an alpha level of $p<0.01$ is adopted for *Developmental Trajectories* in the analysis below.

Conviction Status. Criminal convictions were included in the analysis for phases 15, 18, 21, and 26. Official conviction records were obtained through a search of the central computer system of the New Zealand police that provides details of all New Zealand convictions and Australian convictions communicated to the New Zealand police. Searches for all convictions occurring from the age from which conviction was permissible (14 years) were conducted after each assessment at ages 21, 26, 38, and 45. *Conviction Status* was coded so that if a participant was convicted of a crime by phase 26, they would receive a 1 and 0 otherwise. While inevitably losing some information with dichotomization (i.e., 36% of those with convictions had more than one), this approach better maintained the interpretability of the variable compared to other approaches for dealing with the skew of most count variables of offending (e.g., using logarithms) and was more appropriate for the inverse probability of treatment weighting analysis (described below) that was used to adjust for selection.

Covariates. A number of covariates were included in the analysis to adjust for lifestyle factors that might confound associations between criminal behavior/trajectory and biological

age. These covariates included sex as well as three childhood covariates: IQ, SES, and poor health. *Childhood IQ* was assessed with the Weschler Intelligence Scale for Children-Revised (WISC-R [Wechsler, 1974]) and was administered to the study members at ages 7, 9, and 11 years. IQ scores for the three ages were averaged and standardized ($M=100$, $SD=15$). *Childhood SES* was measured by assessing the cohort members' families on a six-point scale that assessed parents' occupational statuses, defined based on average income and educational levels derived from the New Zealand Census (Poulton et al., 2002). Parents' occupational statuses were assessed when participants were born and again at subsequent assessments up to age 15 years. The highest occupational status of either parent was averaged across the childhood assessment.

Childhood Poor Health was measured from a panel of biomarkers and clinical ratings taken at assessments from birth to age 11 years (Belsky, Caspi, Israel, et al., 2015), including motor development (at ages 3, 5, 7, and 9 years), overall health (at ages 3, 5, 7, 9, and 11 years; rated by two Unit staff members based on review of birth records and assessment dossiers including clinical assessments and reports of infections, diseases, injuries, hospitalizations, and other health problems collected from children's mothers during standardized interviews), body mass index (at ages 5, 7, 9, and 11 years), tricep and subscapular skinfold thicknesses (at ages 7 and 9 years), and, finally, forced expiratory volume in one second (FEV1) and the ratio of FEV1 to forced vital capacity (FEV1/FVC; at ages 9 and 11 years). To calculate the *Childhood Poor Health* measure, assessments were standardized using z-scores ($M=0$; $SD=1$) within age- and sex-specific groups. Cross-age scores for each measure were then computed by averaging standardized scores across measurement ages. The final *Childhood Poor Health* score was calculated by taking the natural log of the average score across all measures, resulting in a

normally distributed childhood health index. High scores indicate poorer average health in childhood.

Tobacco Pack-Years. Cumulative tobacco exposure was calculated from the reported number of cigarettes smoked per day at each assessment divided by 20 and multiplied by the number of years smoked at that rate through age 45 years. One pack-year reflects the equivalent of 20 cigarettes a day for 1 year. The average number of pack-years for the analytic sample was 6.81 ($SD=10.13$).

Analytic Strategy

The analysis in this study will begin by describing in detail the distribution of key variables across the analytic sample. Having characterized the sample, I will then proceed to examine the association between *Average Offending Involvement* and *Developmental Trajectories* and *Pace of Aging* using a multiple linear regression framework. This analysis will allow me to assess how two key aspects of the offending lifestyle (i.e., the level and pattern of offending) are associated with aging in middle adulthood after adjusting for confounders.

Next, I assess the association between contact with the criminal justice system (measured as convictions) and *Pace of Aging* through middle adulthood. For this analysis it is important to acknowledge that becoming involved in the criminal justice system to the point of receiving a criminal conviction is a highly selected process. Individuals who receive criminal convictions are disproportionately likely to come from disadvantaged backgrounds. These individuals are also more likely to experience early morbidity and mortality, resulting in a more advanced *Pace of Aging*. Thus, selection into the criminal justice system is a likely source of confounding when attempting to examine the impact of that contact on later *Pace of Aging*. In order to adjust for

this selection bias, I employ a propensity score and inverse probability of treatment weighting approach.

Propensity score matching (PSM) is an analytical tool that can be used to estimate counterfactual scenarios in observational data. PSM is simple in principle: 1) estimate the probability of exposure to a treatment in a sample, 2) match treated individuals with non-treated controls on the probability of treatment (i.e., their propensity score), and 3) perform a *t*-test between the treated and matched controls on observed values of the outcome. In essence, PSM provides insight into the counterfactual outcome for the non-treatment condition in observational data. In a fully specified PSM model, the difference in the outcomes between treated and matched controls represent the causal treatment effect of a given treatment.

A related approach to estimating treatment effects is the inverse probability of treatment weighting (IPTW) method. Rather than using the propensity score to construct pseudo-experimental conditions with treatment and matched control groups, IPTW uses the propensity score to weight a study sample in order to produce a “pseudo-population” wherein the distribution of confounders is balanced between the treated and non-treated groups. IPTW accomplishes this by weighting cases according to the inverse of the probability of the treatment that they received. For instance, treated cases with a high probability of treatment would be downweighted while treated cases with a low probability of treatment would be upweighted (vice versa for the non-treated group). Using IPTW induces balance across the covariates between the treatment and control groups in a study, thus making the actual distribution of the treatment unbiased (i.e., random).

While PSM and IPTW share many similarities, the current analysis uses IPTW because it has been shown to be comparable to PSM in most cases (Austin, 2009), but it does not induce the

same amount of data loss. Depending on the matching approach used in the analysis (e.g., 1-to-1, k-nearest neighbor, kernel, Mahalanobis matching), PSM has the potential to drop portions of the sample that do not fall within a specified range of coverage. Given the moderate size of the Dunedin Longitudinal Study, the conservation of cases is a primary concern. Additionally, IPTW makes fewer distributional assumptions than PSM allowing the researcher to focus more on the ability of the IPTW weights to balance covariates rather than having a correctly specified propensity model (Austin & Stuart, 2015).

The main analysis of early criminal lifestyle and *Pace of Aging* will use an IPTW approach to estimate the treatment effect of contact with the criminal justice system (i.e., criminal conviction) of *Pace of Aging*. The analysis will begin by estimating the propensity score of receiving a criminal conviction by age 26 by using a logistic model to regress *Conviction Status* on the abovementioned covariates, as well as the key predictors from the prior analysis: *Average Offending Involvement* and *Developmental Trajectories*. The predicted probabilities produced by this analysis represents the propensity score for receiving a criminal conviction, which ranges between 0 and 1. These propensity scores will be used to inversely weight the sample according to their given treatment. After which, covariate balance will be assessed by comparing the standardized mean differences and variance ratios between the convicted and non-convicted subsample. Assuming adequate balance is achieved, the final analysis will take the form of a weighted multiple linear regression model of *Pace of Aging* on *Conviction Status*.

Finally, a series of sensitivity analyses will be conducted in order to assess the impact of simultaneity bias. The treatment window for the analysis ranges from 14-26 (i.e., the portion of the age-crime curve in which most variation occurs). The large treatment window makes it impossible to include criminal lifestyle factors that occur in the same time frame (e.g., *Average*

Offending Involvement and Developmental Trajectories) without incurring bias arising from simultaneity (i.e., when predictors and treatments are measured concurrently, and temporal order is ambiguous). Thus, the main analysis will be reanalyzed by selectively removing or adjusting variable coding in order to assess the likely impact of the bias on the results.

Results

Preliminary Analysis

I will begin by describing the Dunedin Longitudinal study sample in terms of the key variables for the present analysis. After listwise deletion, $N=808$ participants comprised the analytical sample. Compared to the omitted cases, the analytic sample had a lower average *Pace of Aging*, as well as lower average offending variety at Phases 15 and 21 and overall. The analytic sample also demonstrated higher average childhood IQ, childhood SES, and better childhood health (see Table 3.2). These differences highlight the fact that dropped cases were generally more disadvantaged and had worse behavioral and health profiles.

Table 3.2. Distribution of key variables across analytic (N=808) and non-analytic samples (N=123-229).

	<i>t</i> -value (<i>Chi</i> ²)
Pace of Aging	2.54*
Self-Reported Offending Variety	
<i>Phase 15</i>	3.45***
<i>Phase 18</i>	1.63
<i>Phase 21</i>	0.73
<i>Phase 26</i>	2.54*
Average Offending Involvement	2.01*
Developmental Trajectories	(5.18)
<i>Low</i>	
<i>Childhood Limited</i>	
<i>Adolescent Onset</i>	
<i>Life-course Persistent</i>	
Conviction Status	(0.74)
Childhood IQ	-5.20***
Childhood SES	-3.41***
Childhood Health (z-score)	2.08*
Sex	(2.18)

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Turning now to the distribution of key variables within the analytic sample, *Pace of Aging* scores ranged from 0.38 to 2.43, suggesting that some participants in the sample are aging appreciably slower and others much faster (almost 2.5 times faster) than would be expected according to their chronological age (see Table 3.3). Figure 2.2 displays the distribution of *Pace of Aging* scores for both males and females, with the red line indicating the reference score of *Pace of Aging*=1 (i.e., the rate of physiological decline expected for a single chronological year). The distribution of *Pace of Aging* for male and female participants was highly overlapping, and mean sex differences were not statistically significant ($t=0.72$; $p > .05$). These results suggest that participants are highly variable in their *Pace of Aging* from Phase 26-45, but not across sexes.

Table 3.3. Univariate statistics of key variables and t-test/chi-square test comparisons across male (N=407) and female (N=401) respondents (total N=808).

	Mean (Proportion)	SD	Min	Max	T-statistic (χ^2)
Pace of Aging	0.99	0.30	0.38	2.43	1.36**†
Self-Reported Offending Variety					
<i>Phase 15</i>	0.97	1.79	0	12	-2.69**
<i>Phase 18</i>	1.19	1.81	0	12	-6.29***
<i>Phase 21</i>	1.21	1.70	0	11	-6.51***
<i>Phase 26</i>	0.91	1.26	0	11	-7.45***
Average Offending Involvement	1.07	1.28	0	8.5	-7.23***
Developmental Trajectories					(7.28)
<i>Low</i>	(0.51)	-	0	1	-
<i>Childhood Limited</i>	(0.22)	-	0	1	-
<i>Adolescent Onset</i>	(0.18)	-	0	1	-
<i>Life-course Persistent</i>	(0.08)	-	0	1	-
Conviction Status	(0.24)	-	0	1	(64.33***)
Childhood IQ	100	15	39.01	143.40	-2.16*
Childhood SES	3.82	1.08	1	6	-0.04
Childhood Health (z-score)	-0.03	0.92	-2.5	2.5	-0.18

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. P-values correspond t-/chi-square tests across female and male respondents. Negative t-values indicated a higher group means for male participants. †*F*-statistic: *Pace of Aging* was scaled within sex so that both male and female participants would have a group mean of 1, thus a variance ratio test was employed to assess distributional instead of mean differences.

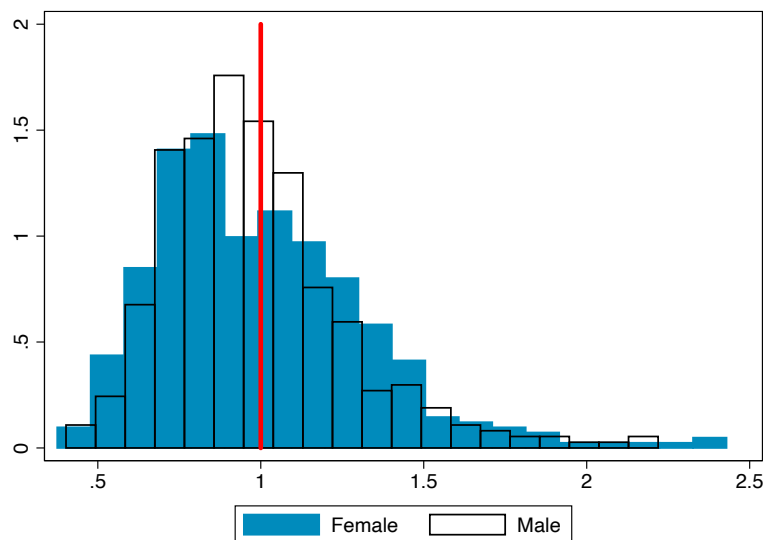


Figure 3.1. Histogram of Pace of Aging from phase 26-45, stratified by sex.

As expected, *Self-Reported Offending Variety* decreased from Phase 15-26 and statistically significant sex differences in favor of the male participants persisted across all four phases (p -values ranged from <0.01 to <0.001), as well as the *Average Offending Involvement* across all phases ($t=-7.23$; $p<0.001$). Figure 2.3 displays boxplots of the *Self-Reported Offending Variety* scores for males and females across Phase 15-26, as well as *Average Offending Involvement* across all phases. Among the participants, 24% were convicted of a criminal offense by the age of 26, with males receiving most of the convictions ($\chi^2=64.33$; $p<0.001$). Of the childhood covariates in the analysis, only *Childhood IQ* demonstrated a small but statistically significant sex difference ($t=-2.22$; $p<0.05$) in favor of males. The other covariates, *Childhood SES* and *Childhood Poor Health*, both demonstrated no statistically significant sex difference among participants.

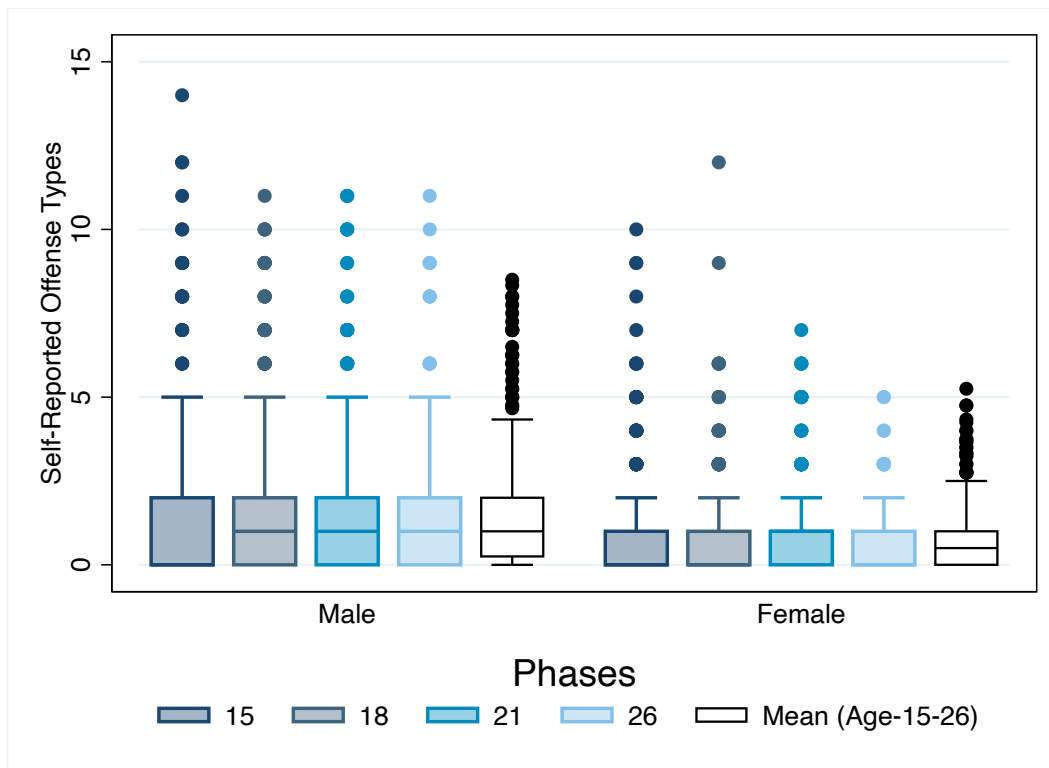


Figure 3.2. Box plots of *Self-Reported Offending Variety* across phases 15-26 and *Average Offending Involvement*, stratified by sex.

Regression Analysis

Is offending variety and pattern associated with the Pace of Aging in middle age?

Table 3.4 presents the results of the multiple regression analysis of *Pace of Aging* on *Average Offending Involvement* (i.e., from age 15-26) and *Developmental Trajectories*. Looking to model 1, *Average Offending Involvement* demonstrated a statistically significant positive association with Pace of Aging ($b=0.038$; $p<0.001$) after adjusting for covariates. This suggests that when the average number of crime types between ages 15-26 goes up by one, *Pace of Aging* throughout midlife (i.e., ages 26-45) is expected to accelerate by almost 4%. While statistically significant, the size of this effect is substantively small. Given the crudeness of *Average Offending Involvement*, however, it does provide grounds to continue our investigation and consider not only the average level of involvement but also the pattern.

Table 3.4. Multiple Linear Regression of Pace of Aging on Average Offending Involvement and Developmental Trajectories.

	Model 1		Model 2		Model 3	
	<i>b</i>	[SE]	<i>b</i>	[SE]	<i>b</i>	[SE]
Offending						
Average Offending Involvement	0.038***	[0.008]			0.029**	[0.010]
Developmental Trajectories ^a						
<i>Childhood Limited</i>			0.035	[0.026]	0.033	[0.026]
<i>Adolescent Onset</i>			0.068†	[0.028]	0.028	[0.031]
<i>Life-course Persistent</i>			0.159***	[0.039]	0.090†	[0.046]
Covariates						
Male ^b	-0.029	[0.021]	-0.013	[0.020]	-0.028	[0.021]
Childhood IQ	-0.004***	[0.001]	-0.004***	[0.001]	-0.004***	[0.001]
Childhood SES	-0.032**	[0.010]	-0.028**	[0.010]	-0.029**	[0.010]
Childhood Poor Health	0.051***	[0.011]	0.049***	[0.011]	0.050***	[0.011]
Constant	1.494***	[0.070]	1.454***	[0.074]	1.448***	[0.074]
N	808		808		808	

* $p<0.05$, ** $p<0.01$, *** $p<0.001$. †Significant at the $p<0.05$ level, but not at the adjusted level of $p<0.01$. ^aRef. group: Low. ^bRef. group: female.

Turning to model 2, we see that, after adjusting for covariates, the *Developmental Trajectories* identified by Odgers and colleagues (2007) do predict *Pace of Aging* later in life. Compared to the *Low* group, the *Life-Course Persistent* group (LCP; $b=0.159$; $p<0.001$) demonstrated statistically significant associations with *Pace of Aging*. The *Adolescent Onset* group did not demonstrate a statistically significant association with *Pace of Aging* at the adjusted alpha level of $p<0.01$ ($b=0.068$; $p=0.034$). The *Childhood Limited* group (i.e., those who began high in CD symptoms and then declined throughout adolescence) did not demonstrate an association with *Pace of Aging* that differed significantly from the *Low* group ($b=0.035$; $p=0.105$). These results suggest that *LCP* group (i.e., the trajectory group that emerged from adolescence with a higher level of conduct disordered behavior) experienced almost a 16% increase in their *Pace of Aging* throughout middle adulthood.

Finally, in model 3 of Table 3.4, we observe *Average Offending Variety* and *Developmental Trajectories* included in the same analysis as predictors of *Pace of Aging*. *Average Offending Variety* remained a statistically significant predictor of increased *Pace of Aging* ($b=0.029$; $p<0.01$) while the *LCP* trajectory group did not meet the adjusted significance threshold ($b=0.09$; $p=0.04$).

Inverse Probability of Treatment Weighting (IPTW) Analysis

I now turn to the analysis of criminal justice contact and its association with *Pace of Aging* using IPTW. Analyses using IPTW begin by estimating the propensity scores for the treatment using a logistic regression framework. When estimating propensity scores, however, it is important to acknowledge and evaluate the assumptions that underly the propensity score approach, including consistency, exchangeability, positivity, and no misspecification of the propensity score model (Cole & Hernan, 2008). Consistency suggests that an individual's

outcome under the treatment assignment that they actually received is equal to their observed outcome. Exchangeability refers to the state of having measured all of the relevant variables that affect the outcome. Positivity suggests that every subject has some none-zero probability of receiving each treatment.

Some of these assumptions can be evaluated objectively (e.g., positivity), while others are more subjective in that they rely on the subject matter expertise of the researcher (e.g., exchangeability). Austin and Stuart (2015), however, argue that the key concern when estimating a propensity score model for IPTW models is the resulting covariate balance. Thus, even misspecified propensity models that induce covariate balance in the sample can be useful for IPTW models. Thus, I focus on the balancing capability of the propensity score model. First, I estimated a logistic regression model of *Conviction Status* on *Average Offending Involvement*, *Developmental Trajectories*, *Sex*, *Childhood IQ*, *Childhood SES*, and *Childhood Poor Health*. This model explained over a fifth of the variation in *Conviction Status* ($R^2_{\text{pseudo}}=0.22$). The predicted values of this model were then used to create inverse probability weights, the standard formula is as follows (Thoemmes & Ong, 2016, p. 42):

$$w|Z = 1| = \frac{1}{P(Z = 1|X)} \quad (3)$$

for the treated case and

$$w|Z = 0| = \frac{1}{1 - P(Z = 1|X)} \quad (4)$$

for the non-treated case, where the inverse probability of treatment weight w is given according to the treatment status of some binary treatment variable Z (e.g., 1=conviction by age 26, 0=no convictions by age 26) with $P(Z=1)$ representing the probability of receiving the treatment Z given some vector of covariates X .

Research has found that IPTW weights can sometimes be very large or small (i.e., close to 1 or 0), which can introduce bias (Cole & Hernán, 2008). For instance, a treated case with a very small probability of receiving treatment would be dramatically upweighted with the use of IPTW, and thus might introduce bias into the analysis. One strategy for addressing this issue is to truncate weights according to a prespecified floor/ceiling (e.g., the 1st and 99th percentiles or the 5th and 95th percentiles)—some fields also refer to this procedure as trimming or winsorization. After computation of weights using equations 1 and 2, weights were truncated at the 1st and 99th percentiles and then normalized (i.e., dividing weights by the sum of all weights) ensuring that the normalized weights summed to 1.

Before using the normalized weights to estimate the treatment effect of receiving a *Conviction Status* on *Pace of Aging*, it is important to assess their influence on covariate balance. Austin and Stuart (2015) note that standardized mean differences (SMDs) between treated and non-treated study members is a useful method to assess the covariate balance in the sample before and after weighting. Table 3.5 presents the absolute SMDs across all covariates used in the propensity score model and compares them before and after weighting (Figure 3.4 presents the results visually). Those variables with SMDs beyond the threshold of 0.2 (indicated by the red vertical line) represent imbalances across treatment categories in the sample. Looking across the distributions of weighted and unweighted SMDs, it is clear that the normalized weights produce substantive improvements in the covariate balance in the sample as none of the variables' SMDs surpassed 0.2 after weighting. Overall, the distribution of covariate means appears to be well balanced after the application of normalized weights.

Table 3.5. Covariate balance metrics across before and after IPTW weighting.

	Unweighted		Weighted	
	SMD	VR	SMD	VR
Key Predictors				
Average Offending Involvement	0.86*	3.80^	0.10	0.74
Development Trajectories ^a				
<i>Childhood Limited</i>	0.16	1.22	0.05	1.07
<i>Adolescent Onset</i>	0.35*	1.63	0.06	1.10
<i>Life-course Persistent</i>	0.50*	3.95^	0.04	1.13
Covariates				
Male ^b	0.71*	0.76	0.16	0.97
Childhood IQ	0.32*	0.90	0.02	0.95
Childhood SES	0.00	1.02	0.04	0.99
Childhood Poor Health	0.23*	0.94	0.06	0.87
Arithmetic Mean	0.39*		0.07	
Geometric Mean	1.45		0.97	

*SMD \geq 0.02; ^VR \neq 0.05-2; SMD=(absolute) standardized mean difference; VR=variance ratio; ^aRef. group: Low; ^bRef. group: female.

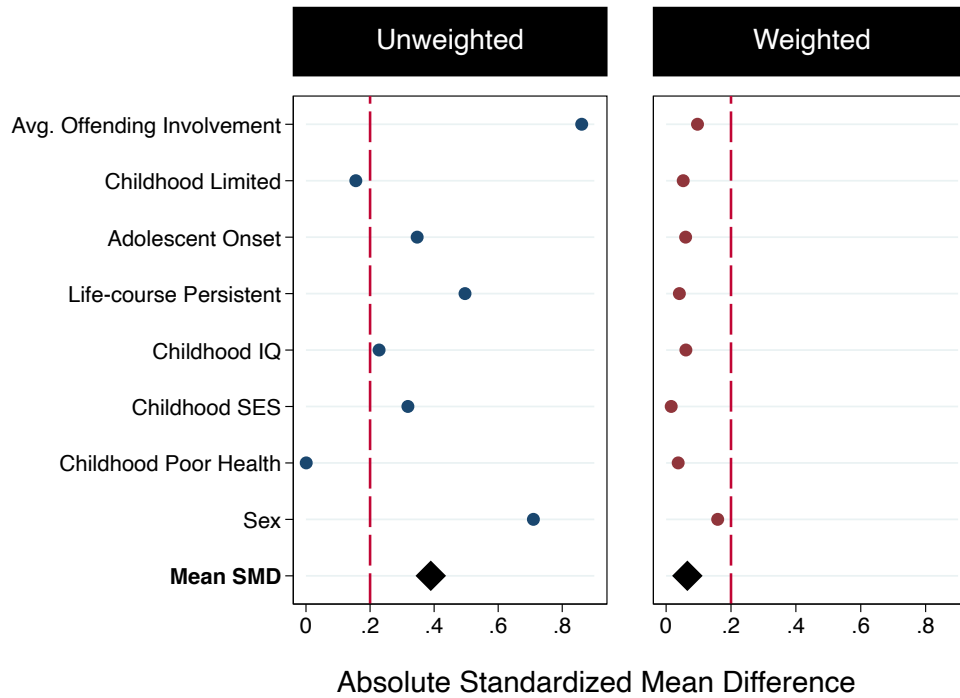


Figure 3.3. Absolute standardized mean differences across all covariates, before and after weighting with inverse probability of treatment weights.

Covariate balance is more than a property of sample means, however, as the overall distribution of each covariate should also show some parity between treated and non-treated cases within the weighted sample. Rubin (2001) suggested the use of the variance ratio as a distributional balance metric. While variance ratios with values close to 1 are ideal, Rubin suggested that variance ratios above 0.5 and below 2 were indicative of adequate balance. Table 3.4 displays the variance ratios of all covariates in the propensity score model, as well as the geometric mean variance ratio for the covariates (a metric suggested by Linden & Samuels, 2008; Figure 3.5 presents these results visually). Variance ratios of most covariates in the unweighted sample were balanced within the thresholds, although *Average Offending Involvement* and the proportion of *Life-Course Persistent* offenders was outside of the range. After weighting, however, all covariates were found to have variance ratios within the balance range. Having found evidence for adequate covariate balance in the sample after the application of normalized weights, I now proceed to the final analysis.

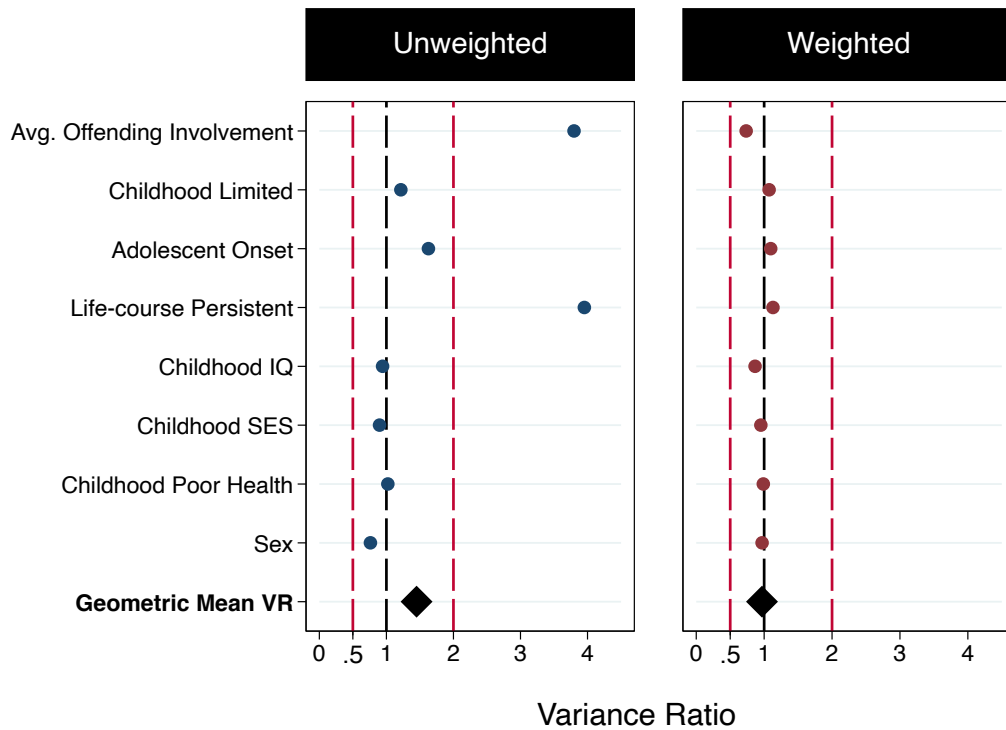


Figure 3.4. Variance ratios across all covariates, before and after weighting with inverse probability of treatment weights.

Is Receipt of a Criminal Conviction Before Age 26 Associated with Pace of Aging?

Table 3.6 presents the findings of the weighted multiple linear regression of *Pace of Aging* on *Conviction Status*. The primary model of interest is the model in the first row of the weighted column (i.e., the full sample weighted model). This model finds a statistically significant average treatment effect for receiving a conviction by age 26 ($b=0.08$; $p<0.01$). This suggests that *Conviction Status* is associated with an advanced *Pace of Aging* throughout middle adulthood, amounting to an 8% increase in speed of physiological deterioration above what is expected for normal aging. To put this finding into concrete terms, if a person’s chronological and biological age were identical at age 26 and they had a *Pace of Aging* of 1.08 then by age 45 their biological age will have advanced to 46.52 (i.e., a 1 ½ year difference).

Table 3.6. Average Treatment Effect Analysis of Conviction Status on Pace of Aging Using Inverse Probability of Treatment Weights and Sensitivity Analysis.

	Model 1	Model 2	Model 3	Model 4
	OLS	IPTW Adjusted	CJ Covariates Fully Removed	Temporally Adjusted CJ Variables
	<i>b</i> [SE]	<i>b</i> [SE]	<i>b</i> [SE]	<i>b</i> [SE]
Conviction by Age 26	0.109*** [0.026]	0.080** [0.030]	0.130*** [0.030]	
Conviction from Age 21-26				0.108** [0.035]
Observations	808	808	808	734

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Note: OLS model included all of the covariates used in the propensity score model used to construct the IPTW weights. Models 2-4 were bivariate models that used the IPTW weights.

Sensitivity Analysis

This study used a pooled logistic regression approach for estimating the propensity score used in the IPTW analysis. This means that the treatment period (i.e., the time frame within which a study member might have received a criminal conviction) includes time-varying covariates that might have been influenced by exposure to the treatment. For instance, if a study member received a conviction at age 14, it may have an effect on their offending variety later on. Thus, a variable used as a covariate in the propensity score model predicting convictions by age 26 may actually be influenced by prior exposures to the treatment. This problem is referred to as simultaneity and it is a form of endogeneity bias (see generally Morgan & Winship, 2015).

Typically, the solution to this issue is to only use variables that precede the entire treatment period in time, thus ensuring the correct temporal order. Given the long treatment period of the current study (the sample experienced criminal convictions as early as 14 years of age), this procedure would mean removing all of the observed variables related to the criminal lifestyle from the propensity score model. Thus, the main analysis incurred this bias by keeping

the criminal lifestyle variables in the IPTW analysis. Here I explore how sensitive the findings in the main analysis are when I attempt to adjust for the simultaneity bias in two different ways.

First, I estimate a model that keeps the original treatment period (i.e., ages 15-26) but removes the criminal lifestyle variables. This model removes the simultaneity bias but risks misspecification as receipt of a criminal conviction is likely a heavily selected process, making the inclusion of offending variables necessary for a fully specified model. Second, I split the treatment period so that some of the criminal lifestyle variables may still be included in the model without violating temporal ordering between predictor and treatment. To achieve this, I adjust the treatment period to only include convictions received after age 18 and up to age 26.

Additionally, I restrict the *Average Offending Involvement* variable to only include information from offending behaviors reported during Phases 15 and 18. To ensure that offending during these phases was not influenced by prior exposure to the treatment (i.e., criminal conviction), I removed all cases from the analysis that received a criminal conviction by age 18 ($N=74$). The Developmental Trajectories were also removed from this model as they were estimated using information from Phases 7-26 and would violate the restricted treatment period in this analysis. The IPTW procedure was repeated for both analyses and the results are reported in Table 3.6. (Note: both sensitivity analyses achieved an acceptable level of covariate balance—results are presented visually in the appendix).

Looking at model 3 of Table 3.6, we see that fully removing criminal lifestyle variables has had a predictable effect on the result observed in the main analysis. The effect of *Conviction Status* on later *Pace of Aging* has been inflated ($b=1.30$; $p<0.01$) beyond even that of the OLS estimate in model 1 ($b=1.09$; $p<0.001$). This finding should emphasize the importance of a fully specified model when considering a treatment like criminal convictions. The effect identified in

the main analysis, though likely biased by simultaneity, provides a more conservative estimate of the influence of *Conviction Status* on *Pace of Aging* than the simultaneity adjusted model—although both approaches demonstrate a level of association (i.e., between 1.08-1.13 increase in the *Pace of Aging*).

Turning to model 4 of Table 3.6, the reduced treatment period approach also produced a statistically significant association between *Conviction Status* and *Pace of Aging* ($b=0.11$; $p<0.01$). This effect was closer in size to the effect observed in the main analysis ($b=0.08$), which might suggest that even moderate adjustment for offending behavior can help specifying the model predicting an effect of criminal convictions. The results of these sensitivity analyses suggest that, while simultaneity may be a crucial conceptual issue to the main IPTW analysis reported above, it is unlikely that the findings are completely spurious as a result.

Discussion

With some notable exceptions (e.g., Shover & Thompson, 1992), theories of age and crime often cast the role of age as an exogenous influence on crime—unmanipulable and ultimately scientifically uninteresting (Hirschi & Gottfredson, 1983)—or as a temporal mile marker, signaling the timing of life events that bear the true association with crime (e.g., Moffitt, 1993; Sampson & Laub, 1993). Age has always been thought to influence crime in some fashion. Drawing on developments in the assessment of “biological age” from the field of geroscience and using data from the Dunedin Longitudinal Study, the paper has sought to explore the opposite possibility: could crime influence age in return?

The study examined three dimensions of the criminal lifestyle for associations with Pace of Aging in middle adulthood: mean level of involvement, pattern of involvement, and criminal conviction. The average level of offending from age 15-26 predicted a roughly 3-4% increase in

the Pace of Aging. This finding suggests that, despite the dramatic changes in offending rates that occur during the transition from adolescence to adulthood, those individuals who participated in more types of offending experienced marginally faster rates of physiological deterioration in middle adulthood. With regard to patterns of involvement, the life-course persistent trajectory group originally identified in the Dunedin Longitudinal Study by Odgers and colleagues (2007) did show an association with Pace of Aging (i.e., 16% increase), but that association disappeared after adjusting for average offending level in the full model.

Finally, this study examined the association between contact with the criminal justice system—in the form of receiving a criminal conviction—with Pace of Aging. An inverse probability of treatment weighting approach was used in an attempt to adjust for the influence of selection and estimate the average treatment effect of receiving a criminal conviction on later biological aging. The results of the analysis revealed that receiving a criminal conviction was indeed associated with a more advanced Pace of Aging, amounting to an 8% increase in total. This finding has a number of implications. First, these findings suggest that some higher order effects of offending (e.g., criminal sanctions) may be influencing aging outcomes for justice-involved individuals. Contact with the criminal justice is largely a function of one's level of involvement in a criminal lifestyle—the more illegal behaviors one engages in, the higher the likelihood that they will be subject to criminal sanctions. Thus, criminal justice contact is a highly selected process and not randomly distributed in the population. The findings of the IPTW analysis revealed that receiving a criminal conviction by age 26 was predictive of more advanced aging, even after adjusting for criminal lifestyle (i.e., level and pattern of involvement) and other factors.

Contact with the criminal justice system likely influences later aging through two mechanisms, what Pearlin (1989) refers to as primary and secondary stressors. Primary stressors are first order events that evoke a stress response in those who experience them (e.g., receiving a criminal conviction). Secondary stressors, in contrast, are the second order events that are brought about in response to primary stressors (e.g., criminal label, stigma). Research on incarceration and health has pointed to the crucial role played by stress as a linking mechanism. Stress-related health outcomes (e.g., chronic conditions, infectious diseases, psychiatric disorders) have all been shown to be more strongly associated with a history of incarceration than health conditions not strongly associated with stress. The findings from this study point to the need to examine other stages of contact with the criminal justice system (e.g., arrest, incarceration, probation, parole) for, not only an association with health, but with aging more generally.

Second, while adjusting for selection and identifying causal effects is crucial for advancing the understanding of the role the criminal lifestyle plays in processes like aging, it is important to remember why selection among justice-involved populations is so high. The level of disadvantage among those who come into contact with the criminal justice system is considerably higher than that of the general population. Evidence of negative health effects from receiving a criminal conviction—the goal of the current study—means that most justice-involved individuals are doubly compromised with regard to health and aging.

Limitations

This study used offending and biomedical data to examine the influence of involvement in a criminal lifestyle up to age 26 on aging throughout middle adulthood. Despite the strengths

of the data and methods used, this study has a number of limitations that should be considered when interpreting the results.

Another limitation of the current study is the reliance on a measure of criminal justice contact that does not distinguish between various levels of penetration into the criminal justice system. Research on the health effects of criminal justice contact has found that the first and last levels of exposure (i.e., arrest and incarceration) explain most of the relationship. Explanations of this pattern have relied mostly on the stress process paradigm (Pearlin, 1989), suggesting that arrest and incarceration are the most outwardly visible forms of criminal justice contact and thus induce the highest levels of secondary stressors (e.g., criminal labels, stigma) that then influence health.

The measure of criminal conviction used in the current study implicitly captures individuals who were also arrested; however, the number of people who receive a criminal conviction only represent a small fraction of those who get arrested. For instance, many arrested individuals never get formally charged, do not go to court, and thus cannot receive a conviction. Future research on the influence of criminal justice contact on health/aging outcomes should endeavor to distinguish between each level of penetration into the system. This will help future efforts to identify the most stressful levels of the system and establish policies aimed at mitigating the impact of contact with the criminal justice system on health outcomes.

Future Directions

Criminal behavior takes a heavy toll on society, but the criminal does not escape unmarked. The goal of the current study was to examine the association between the criminal lifestyle and age by relying on a biological conceptualization of age. But why might criminal behavior exert an influence on an offender's biological age? Criminal behavior and the criminal

justice system do not exist in a social vacuum; rather, these factors exist in a causal web of mediators and moderators that, by degrees, link the criminal lifestyle to health and biological age more generally. Identifying the precise mechanistic process will be crucial if interventions aimed at slowing biological age (and thus the onset/progression of age-related health conditions) can be achieved.

With the identification of causal mechanistic processes as the goal, I believe it will first be necessary to define the various types of effects one might expect to observe when investigating the association between the criminal lifestyle and aging (the points of the following discussion are also presented in table 4). To that end, I propose three distinct, yet highly interrelated, types of effects researchers should aim to identify.

Zeroth Order Effects— “Proxies”. Individuals who engage in a criminal lifestyle are disproportionately drawn from areas with concentrated disadvantage, a classification that typically includes high rates of poverty, unemployment, family instability, physical deterioration/dilapidation, and high crime. Individuals who live in areas of concentrated disadvantage disproportionately suffer from issues like food insecurity, reduced access to health care, and demonstrate poor health behaviors like tobacco and alcohol use. Any analysis that attempts to link the criminal lifestyle with health and aging outcomes will face the challenge of avoiding the identification of proxy effects. Without rigorous controls, criminal behavior runs the risk of simply being a proxy for factors related to concentrated disadvantage.

Table 3.7. Criminal Lifestyle Factors Implicated in the Acceleration of Biological Age.

Order	Source	Description
Zeroth “Proxies”	Concentrated disadvantage <ul style="list-style-type: none"> • Unemployment • Food insecurity • Residential instability • Urbanicity • Family structure 	These criminal lifestyle factors are considered zeroth order because they do not necessarily stem from a criminal lifestyle, but individuals who demonstrate a criminal lifestyle disproportionately experience these factors.
First “Direct Effects”	Criminal behavior <ul style="list-style-type: none"> • Illicit drug use • Violent altercations • Injury-prone activities Criminal sanction (incarceration) <ul style="list-style-type: none"> • Social isolation • Violent altercations 	These criminal lifestyle factors are the direct effects or “primary stressors” associated with either the criminal act itself or the criminal sanction.
Second “Indirect Effects”	Criminal label/stigma <ul style="list-style-type: none"> • No social support • Shaming • Unemployment 	These criminal lifestyle factors are the consequences of either criminal behavior or a criminal sanction (i.e., “secondary stressors”), and are generally elicited from community members upon learning of an offender’s past.

It should be emphasized that, while not causal, proxy effects are still important from a public health perspective. For instance, assume that incarceration behaved as nothing more than a proxy for other lifestyle factors (e.g., poverty, substance use) that causally impact biological aging. Incarceration could still be used to identify populations who disproportionately suffer from accelerated biological aging due to these lifestyle factors. In this scenario, incarceration itself should not be intervened on due to its lack of causal influence on biological aging. Crucially, however, the time spent in a correctional facility may provide the context within which interventions aimed at the improvement of inmate health could be employed (Hammet, 2001; Jürgens, Ball, & Verster, 2009). It is important to additionally note, however, that the effectiveness of interventions aimed at improving inmate health has been shown to be contingent on factors external to the prison itself, such as community support (Watson, Stimpson, &

Hostick, 2004), which highlights the importance of community partnerships for achieving real health improvements among inmates.

First Order Effects— “Direct Effects”. Criminal behavior is physically dangerous. Criminal behavior often involves the use of illicit substances, violent altercations, or the commission of injury-prone activities (e.g., burglaries). These direct effects of criminal behavior are perhaps of more interest to criminologists, as they represent the possible means of explaining the health disparities between offenders and the general population beyond that which is explainable with concentrated disadvantage. To this order of effects, the direct effects of criminal sanctions may be added. Especially in the case of incarceration, the experience of criminal justice contact exerts many direct effects on the offender. For instance, serving a sentence in prison or jail subjects the inmate to psychological stress from social isolation as well as physical stress in the form of crowding, cramped living conditions, and violent interactions with other inmates. All of these effects, whether from the criminal sanction or the behavior that evoked it, exert direct effects on the offender and run the risk of compromising health and accelerating aging later on.

Second Order Effects—“Indirect Effects”. Criminal behavior is social behavior, and social behavior has reputational consequences. When an individual’s criminal activities become known to the community, a criminal label can be applied to him or her. Criminal labels have been shown to influence social integration, employment opportunities, and family structure, among other things. These indirect effects of criminal behavior, whether evoked in direct response to criminal behavior or because of a formal sanction, expose the offender to chronic stress in a variety of domains including social, economic, psychological, and physical. What is more, criminal labels have been shown to actually increase the level of offending later on rather

than acting as a deterrent (Motz et al., 2019). Thus, criminal labels cause chronic stress across multiple domains and have the potential for establishing a positive feedback loop, all of which may translate into an acceleration of the aging process and contribute to the health disparities experienced by justice-involved individuals.

Together, the zeroth, first, and second order effects of the criminal lifestyle have the possibility to wreak havoc on the physiological integrity of the offender. In order for health criminologists, epidemiologists, and public health researchers to better understand the factors that influence biological aging among the offender population, the isolation and parameterization of each of these effects will be necessary. Having characterized each order of effect in isolation of the others, it will then be important to examine the effects of cross-order interactions and their implications for the aging process. For instance, concentrated disadvantage (zeroth order) may interact with a criminal sanction (first order) such that individuals from an impoverished area actually experience improvements in health/deceleration in aging during an incarceration sentence. Or, perhaps individuals from an impoverished area (zeroth order) actually experience reputational gains (second order) in response to a criminal sanction (e.g., viewing a jail sentence as a rite of passage; Anderson, 2000), thus reducing stress to some extent.

The difficulty will be identifying when order effects interact and compound upon one another—thus offering the most cost-effective targets for intervention—and when they operate in isolation to influence on aging and health. If health disparities among the justice involved population are to be addressed, however, aging must play a central role because it emphasizes the systems approach to health. Aging increases the likelihood of all major health conditions and it can be influenced by processes starting early in the life course.

Conclusion

As a temporal concept, age has specific social implications that relate to crime. As a biological state, crime has specific health implications that relate to biological age. This paper has attempted to show the biological side of aging as an entity that is not wholly exogenous to the criminal lifestyle but is instead impacted by it. Drawing on innovations from the field of geroscience, I propose that a new biological dimension be added to theories of age and crime. Specifically, I envision an updated life-course approach to the study of age and crime, wherein the social implications of (chronological) age predominate the early life-course (e.g., the maturity gap; Moffitt, 1993) and the health implications of (biological age) rise to prominence during the latter part of the life course (e.g., aging out/desistance) (i.e., among those whose criminal career lasts that long).

The analyses in this and the previous chapter provide evidence from two samples showing a connection between offending, criminal justice contact (i.e., criminal conviction and incarceration), and biological aging. In other words, crime is hard on the body. In the next chapter, I question whether the causal arrow can be reversed by exploring the possibility that biological age influences the criminal career in return. Specifically, I will examine the interplay between desistance (i.e., the latter part of the criminal career) and biological age. Given that most criminals desist before the physical effects of aging are felt, I propose that aging will play an outsized role in predicting desistance but only for those individuals who persist in offending long enough to encounter aging and offending concurrently.

Chapter 4 —Aging Out: Biological Age and its Role in the Desistance Process

Introduction

The age-crime curve is perhaps the most ubiquitous crime trend known to modern criminology. With a sharp increase in offending rates throughout childhood and adolescence, the age-crime curve quickly peaks in late adolescence/early adulthood and then declines asymptotically throughout the remainder of the life course. Although the notion of the age-crime curve is not a new one (e.g., see Quetelet, [1831] 1984), its etiology still resists criminologists' attempts at explanation (see Sweeten et al., 2013). There is one specific aspect of the age-crime curve that has intrigued criminologists for years: the process of desisting from crime. After initially offending, the majority of young offenders change their ways in favor of more prosocial behavior. Despite the phenomenon of "aging out" of crime being a nearly universal process, the relationship between age and crime remains, as Moffitt (1993) put it: "at once the most robust and least understood empirical observation in the field of criminology" (p. 675).

Part of the difficulty in explaining the latter part of the age-crime curve comes from the fact that individuals will desistance from crime at different times and for different reasons. What might be referred to as "normative" desistance occurs in early adulthood and is typically accompanied by socially-timed transitions to adult roles, the receipt of adult responsibilities, and the completion of physical development—all thought to be factors in the normative desistance process. A small contingent of offenders persist beyond this point and continue offending throughout their life course. Even these offenders desist eventually (Sampson & Laub, 2003), however, suggesting the existence of a second, prolonged, and qualitatively different desistance process—a "non-normative" desistance process.

The non-normative desistance process has been subject to little theorizing within criminology and even less empirical testing. Theories of cognitive transformations, agency

development, risk aversion, and maturational reform have all been submitted as possible explanations for the non-normative process. What none of these theories consider, however, is the possibility that, for the non-normative desister, desistance may not be a choice so much as an involuntary state brought on by the ravages of a lifetime of crime. In essence, the non-normative desister may be an unwilling traveler on the path of desistance, unable to wrest themselves from their course due to the physical toll of their own offending lifestyle.

This explanation of non-normative desistance has been proffered before (e.g., Gottfredson & Hirschi, 1990; Piquero & Moffitt, 2005), but it has seldom been pursued to the point of empirical testing. While criminologists have known about the *age-crime* relationship for almost two centuries, they have heretofore lacked the tools to explore the impact of physical aging itself. Geroscientists define aging as the “progressive, generalized impairment of function, resulting in an increasing vulnerability to environmental challenge and a growing risk of disease and death” (Kirkwood, 2005, p. 438). Using newly developed measures of “biological age” (i.e., proxies of physical aging), criminologists now have the opportunity to reevaluate the physical aging in the age-crime curve and (especially) non-normative desistance.

The current study focuses on the non-normative desistance process and its association with biological age. Leveraging newly developed methods for quantifying biological age from the field of geroscience, this study provides some of the first evidence that the aging out hypothesis may be more physical than previous theories have surmised. The current study uses group-based trajectory modeling (GBTM; Nagin, 2005) to identify three trajectories of desistance and compare them across a newly developed longitudinal measure of biological age called “Pace of Aging” (Belsky et al., 2015a). This study uses data from a longitudinal prospective birth cohort, the Dunedin Longitudinal Study (analytic $N=845$). The following

sections will describe the state of criminological theory devoted to distance and outline a need for new work emphasizing a biological aging approach to understanding non-normative desistance.

Literature Review

Defined as ceasing to do something, desistance has been remarked on as an “unusual dependent variable for criminologists because it is not an event that happens, but rather it is a sustained *absence* of a certain type of event (in this case, crime)” (Maruna, 2001, p. 17; emphasis in original). Desistance has obvious appeal as an area of focus because it offers the possibility of identifying factors that reduce crime on the individual level and in a sustained way.

Unfortunately, the study of desistance has not developed to the same degree as other aspects of the criminal career (e.g., onset, frequency, specialization). The impediments to the study of desistance stem largely from the lack of theoretical and methodological consensus. Desistance researchers continually bemoan the confusing tangle of definitions and operationalizations of “desistance” present in the literature (Laub & Sampson, 2001). Rocque (2017, p. 51), for instance, provides a list of 18 different operationalizations found in the literature.

In an effort to maximize consistency with the literature, I will draw from a seminal review of the topic written by Laub and Sampson (2001) for my definition of desistance. Fortunately, Laub and Sampson describe a meaningful distinction between two terms that are often conflated in the literature: termination, which is not the focus of the current study, and desistance, which is. First, termination is defined as “the time at which criminal activity stops” (Laub & Sampson, 2001, p. 11). In contrast, desistance is defined as “the causal process that supports the termination of offending” (p. 11). Borrowing these authors’ analogy, it can be helpful to think of termination/desistance by considering the institution of marriage. Marriages

are marked by specific beginning and end points (wedding and divorce, respectively). These points were not arrived at by chance alone, they were supported by an underlying causal process (i.e., the relationship) that gradually brought them about. In the same way, the onset and termination of the criminal career represent substantively important events; however, the underlying process that drove the individual to initially offend or cease altogether is much more subtle and continuous. Thus, desistance should be thought of as a *process of change* that is gradual and specific to the individual. With these definitions in mind, let us now turn to consider how desistance makes up the latter part of the age-crime curve.

Normative Desistance

Most individuals commit some amount of crime during their adolescent period. Moffitt (1994), remarking on the offending literature, noted that, “it is statistically aberrant to refrain from crime during adolescence” (p. 29). For this reason, it is unsurprising that most individuals also then go through the desistance process and eventually terminate their short-lived criminal careers. Desistance can then be considered a normative process, as most individuals in society experience it to some degree. Mainstream developmental/life-course criminology has offered several compelling explanations of this normative desistance process.

Writing in 1993, Sampson and Laub suggested that the desistance process was largely explained by age-graded transitions from adolescent to adult roles. As individuals aged into early adulthood, their adoption of adult roles and responsibilities presages the development of new social bonds. The events that brought about the opportunity for these new bonds (i.e., “turning points”) are the necessary—though perhaps not sufficient (cf. “hooks for change”; Giordano et al., 2002)—conditions for the process of desistance to occur. Also writing in 1993, Moffitt suggested that normative desistance was largely explained by what she termed “adolescence-

limited” offenders. Moffitt described offending during the adolescent period as a normative behavior pattern and adolescence-limited offenders are those individuals who engage in it, but then stop. Likewise, Moffitt described the process of desistance for these adolescence-limited offenders as an expression of healthy, adaptive behavior. As adolescence-limited offenders mature into adulthood, they take on adult responsibilities, establish new social bonds, and rarely feel the need to reoffend.

Much earlier, the Gluecks (1943) described a process of “maturation” that occurs for most offenders and drives the desistance process. For the Gluecks, maturation refers to a multidimensional construct including elements of an individual’s biology, psychology, and sociology. Maturation was not constrained to chronological age (i.e., some individual mature early and some late), but the two are highly correlated. Ultimately, it was the multidimensional development of individuals into fully adult roles, relationships, and cognitions that signaled desistance.

While the above perspectives on normative desistance invoke the influence of various normal biopsychosocial processes, Gottfredson and Hirschi (1990) posited a view of desistance that is more rigid. Specifically, they drew out the distinction between “crime” (i.e., a specific illegal behavior) and “criminality” (i.e., behavioral propensities; e.g., self-control). They suggest that criminality is relatively stable throughout the life-course. While crimes may decrease over time, the propensity for behaving in aggressive, exploitative, or selfish ways and commit behaviors analogous to crime (e.g., alcohol abuse, manipulation) does not decline. How then do Gottfredson and Hirschi reconcile 1) a drive to commit crime that does not diminish with time and 2) rates of offending that demonstrably do diminish? “[T]he inexorable aging of the organism” (Gottfredson & Hirschi, 1990, p. 141). Gottfredson and Hirschi suggest that offenders

“age out” of crime because they find themselves progressively less able to maintain high rates of offending as they age—crime is, after all, a young man’s game.

The primary thesis of the current study is that Gottfredson and Hirschi (1990) went too far in suggesting that age alone could explain variation in desistance. Research has found that almost two-thirds of the association between age and crime could be explained away using variables already identified in the criminological literature (Sweeten, 2013). Gottfredson and Hirschi’s “aging out” hypothesis (i.e., that physical decline leads to desistance) is particularly lacking when confronted with the fact that most of the desistance process occurs during early adulthood when virtually no signs of aging are yet apparent. Despite this, I believe that the “aging out” hypothesis can still be useful in exploring the desistance process.

The current study proposes a change to the original scope of the “aging out” hypothesis of desistance proposed by Gottfredson and Hirschi (1990). Specifically, I propose that physical aging only plays a role in the desistance process for those individuals who persist in their criminal career long enough to experience it. Just as for most of human history, disorders of advanced age (e.g., Alzheimer’s disease, cancer) were rare because few lived to advanced age, so too is the influence of “aging out” rare in the desistance process because few individuals persist in crime long enough to experience the two simultaneously. The focus of the “aging out” hypothesis of desistance should therefore be on non-normative desistance and the individuals who define the tail of the age-crime curve.

Non-Normative Desistance

Describing what they referred to as the “ground rules” for the study of desistance, Laub and Sampson (2001) suggested that “[b]ecause low-rate offending is normative, especially in adolescence, criminologists should [...] not spend much time or energy studying termination or

desistance of low-rate offenders” (p. 10). This should not be taken to mean that criminologists should not spend *any* time or energy studying why offending rates decrease with time (cf. Hirschi & Gottfredson, 1983), but that effort should be focused largely on those individuals for whom the trend of normative desistance does not hold. For those individuals who persist in offending after the normal age of desistance and termination (e.g., 18-25 years old), desistance may be a qualitatively different process. The goal of studying the non-normative desistance process is ultimately the identification of factors that exert a suppressing influence on offending among individuals for whom normative desistance factors had no/little effect. If such non-normative desistance factors are identified, it is hoped that impactful interventions may be developed to truncate the tenure of would-be career criminals.

The question then becomes, how does non-normative desistance substantively vary from normative desistance? The causal mechanisms suggested by the above theories may still apply. The sociogenic perspective of Sampson and Laub (1993) would posit that the prolonged criminal career of some individuals is due to a lack of convergence between offenders and turning points in space and time. Other theorists strike more of a balance between socially based events and individual differences by suggesting that turning points may be necessary but not sufficient for initiating desistance. Giordano, Cernkovich, and Rudolph (2002) suggest that a cognitive transformation (i.e., a “hook for change”) must precede the presentation of the turning point, or else the offender will fail to leverage the turning point into a new life trajectory.

Moffitt’s (1993) taxonomic theory, in contrast, suggests that it is not the simple lack of convergence with turning points that keeps some individuals offending longer—individual differences also contribute to the length of the criminal career. Moffitt’s life-course persistent offenders 1) carry with them a set of behavioral characteristics that continually predispose them

to criminality (i.e., contemporary continuity) and 2) are followed by the compounding consequences of earlier misbehaviors (i.e., cumulative continuities). These continuities in behavior facilitate the life-course persistent offending pattern in a criminal career that extends far beyond the normative termination point. By outlasting the adolescence-limited offenders, life-course persistent offenders define the non-normative desistance process according to Moffitt.

The above perspectives constitute two of the developmental/life-course perspectives that have hypothesized about the mechanisms that drive non-normative desistance. On one hand, a failure of the offender and turning points to converge in time and space. On the other hand, individuals with characteristics that exert negative effects on their behavior/decision-making, effectively ensnaring them in the criminal career. Having described the major criminological perspectives on the *why* of non-normative desistance, it is time to address the *how*. What specific causal mechanisms drive the desistance process in those individuals who did not go through the normative process earlier in life?

Views on mechanisms of desistance vary widely across the field of developmental/life-course criminology. Some suggest that the factors driving the desistance process are simply risk factors for offending, but in reverse. For instance, if lack of social bonds causes offending early in the life course then perhaps the presence of social bonds may bring about desistance later in the life course. The application of this logic to normative desistance has produced a great deal of research in criminology (Massoglia, 2006; Mulvey et al., 2004; Rocque, Beckley, & Piquero, 2019; Warr, 1998). Criminologists have also explored the idea that the factors affecting offending and desistance are not simply mirror images (e.g., Uggen & Piliavin, 1998). Farrington (1992) described the pattern of life-course offending as being comprised of shifts in social influence from parents to peers (i.e., onset) and then from peers to spouses (i.e., desistance). This

suggests an age-graded quality to all putative desistance factors (see especially, Sampson & Laub, 1993). Going one step further to consider non-normative desistance, what unique factors might affect desistance differentially in later adulthood compared to early adulthood/late adolescence? That is, which factors are peculiar to the non-normative desistance process?

Two major contributors have been repeatedly implicated in the non-normative desistance process: cognitive changes and aging. Changes in offenders' cognition have been hypothesized to indirectly influence offending behaviors. Cognitive changes linked to desistance include identity transformations (e.g., Giordano et al., 2002; Maruna, 2001; Paternoster & Bushway, 2009), the development of personal agency (e.g., Sampson & Laub, 2003), and the ability to assess risks and rewards (e.g., Shover & Thompson, 1992). A combination of qualitative and quantitative work has found that, indeed, many high-rate offenders who desist later in life experience remarkable personal cognitive change (Kazemian & Maruna, 2009).

The second mechanism invoked when discussing non-normative desistance is that of physical aging itself. Of course, many of the abovementioned cognitive changes may, to some degree, be considered indirect effects of physical aging. Although some have suggested that persistent offenders may experience the desistance-promoting effects of aging (e.g., physical limitations) with no accompanying cognitive changes. As described by Gottfredson and Hirschi (1990), crime declines because of the aging of the organism, but the criminality of the individual offender (i.e., their propensity for antisocial behavior) does not decline with time. In agreement, Piquero and Moffitt (2005) acknowledge that life-course persistent offenders do indeed desist eventually; however, this process is marked more by physical limitation than cognitive transformation. They wrote that (p. 61):

Although virtually everyone gives up street crime as they age, [life-course persistent offenders] would be expected to maintain antisocial attitudes as long as they live, and to take advantage of even meager opportunities for antisocial activity late in life, such as hitting their wives, cheating at cards, kicking the dog, or falling over drunk.

Nonetheless, physical aging still exerts an effect on the latter part of the criminal career—it plays some role in the desistance process leading up to termination. This brings to bear the possibility that desistance may not be a voluntary decision (see Laub & Sampson, 2001; Reiss, 1989). The involuntary act of aging is the aspect of the desistance process that has received the least amount of attention in the empirical literature. Aging, its effects on the body, and on the criminal career will be the subject of the remainder of this section.

Aging Out

The process of physically “aging out” of crime due to the accrual of physical limitations has not been extensively investigated in the life-course literature, although it has always had a part to play theoretically. Writing about recidivism risk, Hoffman and Beck (1984) described a “burnout” point for prisoners as the “age after which recidivism rates diminish significantly, even for previously recidivistic offenders” (p. 617). Le Blanc and Loeber (1998) admonished desistance researchers to remember that desistance is a process embedded in “developmental contexts, such as a decrease in physical strength and fitness with age” (p. 166). Gove (1985) highlighted the utility of considering the biological components of aging (i.e., declining physical strength, energy, and drive) in combination with social behaviors in order to better understand the desistance process. For instance, Gove noted that virtually all criminological theories were theories of “amplification” (i.e., they predict ever-increasing levels of criminality in individuals) with no theoretical means of predicting reductions in offending over time. By considering

biological variables as well, a natural suppression of offending behavior may be incorporated into criminological theories that adequately explains desistance later in the life course.

Perhaps the most explicit use of aging in the criminological literature is the concept of “maturational reform” promulgated by the Gluecks (1937, 1940; see also Rocque, 2015, 2017). The maturational reform perspective asserted that individuals would desist from crime gradually, as they mature. Becoming mature, in this case, meant that an individual had come to the realization that “crime does not lead to satisfaction” (Glueck & Glueck, 1974, p. 170). They argued that maturation was a process that differed across individuals and that maturity could be reached at different ages depending on various social, environmental, psychological, and biological factors.

Maturational reform has traditionally been overlooked because, among other criticisms, it was seen as being tautological (e.g., Wootton, 1959; Laub & Sampson, 2001). When has a person reached maturity? When they have desisted from crime. When will a person desist from crime? When they have reached maturity. Recently, Rocque (2015) has revitalized the maturational reform approach to desistance. He asserted that the Gluecks’ notion of maturation was not as tautological as early critics assumed because the Gluecks had made efforts to define maturation independent of offending (p. 343). Rocque (2015, 2017) posited a five-part conceptualization of maturation, the components including: 1) psychosocial, 2) neurocognitive, 3) cognitive transformation/identity, 4) adult social role, and 5) civic/communal maturation. With such a guiding framework, it is hoped that the natural phenomena captured by the maturational process may be studied together as a revitalized approach to studying desistance.

Despite the incorporation of biological phenomena like “neurocognitive maturation” (i.e., brain development), the concept of maturational reform—even as restated by Rocque (2015,

2017)—falls short of what is considered full incorporation of the aging concept. Maturational reform suggests that some sort of idealized state has been achieved, one in which the offender has developed the requisite biopsychosocial capacity/drive to reform from crime. It is here that I wish to draw out the distinction between maturation and physical aging.

Let us consider the anatomical and physiological processes wrapped up in the developmental process of maturation. These processes are, in a real sense, “pre-programed” (i.e., ontogenetic; see generally Elder & Shanahan, 2007): following along a very specific progression of developmental milestones, the whole time with a predetermined end-state as the goal. This is not to say that maturational processes do not interact with environments; quite the reverse, as environmental stimuli (e.g., malnutrition) may delay or even halt certain processes (e.g., puberty; Soliman et al., 2014). But this process-driven conceptualization of maturation needs to be differentiated from aging. Aging is commonly defined by the geroscience field as “a progressive, generalized impairment of function, resulting in an increasing vulnerability to environmental challenge and a growing risk of disease and death” (Kirkwood, 2005, p. 438). Aging is then, in a sense, antagonistic towards maturation—the idealized state. Put simply: maturation builds, aging degrades. (Note: life-span developmental psychology would place both maturation and aging under the heading of “life-span development”, noting that the entire human life span is characterized by growth, maintenance, and decline in varying proportions [Baltes, 1987]. The dichotomy draw here between maturation and aging should only be taken to reflect general trends of growth and decline that predominate in the early and later parts of the life course, respectively.)

Maturation and aging are not mutually exclusive in that, aging does not begin as soon as “maturity” is achieved. This is mostly because maturation is not a singular process; rather,

maturation is better thought of as a collection of processes that vary in terms of onset, duration, and cessation. Thus, maturation is not defined by a discrete time period in an individual's lifetime; rather, it is a continuous process that is more or less begun or ended according to a distribution of underlying processes, each of which being at different stages of completion. Assuming that some processes are completed earlier than others, some organ systems may also begin to age before all of maturation is "completed." Thus, maturation and aging are two intimately related, overlapping, and somewhat antagonistic processes that each deserves attention. Given criminology's history of considering what might be considered maturational reform, I focus on physical aging and its role in the degradation of the criminal career.

The Reason for Aging

It may be curious to note, but geroscientists are not completely certain why we age. Despite the many theories of aging (i.e., as many as 300+; Medvedev, 1990), most geroscientists agree that aging, unlike maturation, is not a fully preprogrammed process (i.e., that aging is *supposed* to happen). The evolutionary logic is straightforward. Consider a heuristic example wherein a species possesses a gene "for" aging (i.e., a gene with the ultimate function of making its host more vulnerable to disease and death at an early age). This gene would be self-limiting in that, any individual organism that possessed a "functional" copy of it would likely have less fitness (i.e., in terms of survival and reproduction). Additionally, any individual with a "dysfunctional" version of the gene would have greater fitness (i.e., they would survive longer and likely produce more offspring). Thus, selective pressures against any such an "aging gene" would be strong, resulting in the speedy removal of said gene from the gene pool.

A key consideration needs to be aired at this point. Selection pressure is ultimately manifested in the form of reproductive success. If a gene resides in an organism that does not

reproduce, then the fitness of that gene is seriously compromised (cf. eusocial species; Dawkins, 2016). If, however, a gene has no impact on its host's ability to reproduce (e.g., it does not express itself until after the reproductive period is concluded), then it is effectively hidden from the pressures of natural selection. In this way, "genes for aging" may still persist within the human genome, insofar as they are expressed after reproduction is concluded. There is a family of aging theories based around this insight, including mutational load (Medawar, 1952), antagonistic pleiotropy (Williams, 1957), and disposable soma theory (Kirkwood, 1977). The upshot of this evolutionary detour is simply this: aging in humans, as in other species, is predominately a bug, not a feature. To the extent that "genes for aging" exist, they are evolutionary stowaways—they are not supposed to be there.

As aging represents a genetic pathology of sorts, I hypothesize that its effects on the criminal career are also pathological (i.e., at least from the offender's viewpoint). For many offenders, aging represents a hijacking of criminal capability, not a change in moral orientation (e.g., Piquero & Moffitt, 2005). The corollary of this possibility is that aging differentially affects the commission of some crime types (e.g., robbery, assault) more than others (e.g., white-collar crime). (Note: although the current study examines crime generally, a nature extension of the work herein would be to examine the effects of age on specific crime types, stratified by physical cost. For example, violent, property, and drug crimes). The aging process, being a "progressive, generalized impairment of function..." (Kirkwood, 2005, p. 438), may exert its effects on the persistent offender by reducing their ability to maintain their previous levels of offending. The result being that aging offenders are likely to commit crimes that are fewer in number, less frequent, and are lower in seriousness (e.g., they may commit property or public order crimes as opposed to violent crime).

Aging has been theorized to influence desistance in a physical way for decades; however, the aging effects that have typically been studied in the desistance process have been positive cognitive changes (e.g., identity transformations, agency development, growth in risk aversion) rather than antagonistic physical ones. One reason for the dearth of quantitative research on the physical effects of aging on desistance has been due to an inability to operationalize such a concept. Recently, however, the field of geroscience has made advances in the operationalization of a concept that offers insight into the physical toll of aging. This concept is referred to as “biological age.”

Biological Age

In order to set the stage for a discussion of biological age, it will be helpful to make a distinction between two easily conflated terms: age and aging. For the purposes of this discussion, age will refer to an individual’s chronological age (i.e., years lived since birth). Aging, as defined earlier, will describe the progressive, generalized impairment of function experienced by individuals with the passage of time. It should be noted that age is often used as a proxy for aging (i.e., the amount of aging experienced by an individual can be approximated if their chronological age is known). However, because age is not wholly consonant with aging, a more accurate proxy (i.e., one based on more than chronological age) is possible. This is the role of biological age—to assess the biological state of an individual in order to better approximate the progression of that individual in the aging process.

Biological age has intrigued aging researchers for decades because it represented a means of predicting why some individuals die sooner and some later than would be predicted by their chronological age. Though intuitive, this task has proven difficult as chronological age has for many years been the single most robust and consistent predictor of morbidity and mortality

(Costa & McCrae, 1980). Recently, however, methods for estimating biological age have gained ground and have begun to outperform chronological age in the prediction of morbidity and mortality. The significance of this is that geroscientists have finally managed to decouple chronological age from the individual level variation in aging. For the study of desistance, this opens up the possibility of directly modeling the physical changes associated with aging that for too long have been the province of theory alone.

Quantifying Biological Age

The advances in the measurement of biological age have come with the identification of new biomarkers of aging. Baker and Sprott (1988) set down one of the first definitions of biomarkers of aging: “a biological parameter of an organism that either alone or in some multivariate composite will, in the absence of disease, better predict functional capacity at some later age than will chronological age” (p. 223). Compared to approaches that rely on single biomarkers (e.g., telomere length), the composite approach has become favored by aging researchers due to its higher levels of consistency and predictive validity (Jylhävä et al., 2017).

Though details vary widely between approaches, these new composite methods for quantifying biological age can all be shown to follow a three-fold procedure. First, identify biomarkers that change reliably with chronological age. Second, train an algorithm on an aging phenotype (e.g., chronological age, mortality) using the identified biomarkers as inputs. Third, scale the algorithm to produce scores that are comparable to chronological years. This final point is important because chronological age is often used as a natural benchmark to which measurements of biological age are compared. For example, suppose an individual is chronologically 45 years of age but they received a score of 47 from a measure of biological age.

This would indicate that their aging has advanced two years beyond that which would be expected for someone their age.

Most assessments of biological age are based on measurements of biomarkers at a single point in time and, thus, produce cross-sectional snapshots of the biological process of aging that the individual is experiencing (Belsky et al., 2015a). The limitation in the cross-sectional approach is that it cannot give you a clear indication of how fast an individual's biological age is advancing relative to their chronological age nor when differences between biological and chronological age were acquired. In an attempt to overcome this shortcoming, Belsky and colleagues (2015) developed a measure of biological age that captures the rate of longitudinal change in an individual's biological state. Using data from the Dunedin Longitudinal Study (i.e., the same data used in the current study), they estimated the within-individual change from 18 biomarkers across three time points (i.e., when the sample was 26, 32, and 38 years of age). These slopes were then combined to form a score called "Pace of Aging".

Measures of biological aging like Pace of Aging provide an ideal means for assessing the role of biological aging in the desistance process. Desistance represents a process of decreasing involvement in and eventual termination of the criminal career (Laub & Sampson, 2001). But biological aging is not universally applicable as an explanation of the desistance processes. The desistance process is most consistent with an explanation based on physical limitations brought on by the biological aging process when considering those offenders who persist in offending past emerging adulthood (i.e., ages 18-25; Arnett, 2000) when most individuals desist (Piquero et al., 2002). Accordingly, persistent offenders who engage in offending behaviors into adulthood (i.e., after age 25) are likely to have a more advanced biological age compared to offenders who demonstrate more desistance by the time signs of biological aging begin to appear.

The Current Study

The current study seeks to explore the desistance process, and specifically the non-normative desistance process experienced by more high-rate offenders later in life. Given this focus, the current study hypothesizes a role of biological aging in the desistance process that has heretofore never been tested empirically. The guiding hypothesis for this study is:

H3—Non-normative desistance will be associated with more advanced aging throughout middle adulthood.

With the use of newly developed methods for operationalizing biological age (i.e., “Pace of Aging”; Belsky et al., 2015a), I again use data from a prospective birth cohort, the Dunedin Longitudinal Study, to assess the role of aging in the non-normative desistance process

Methods

Data

To analyze the relationship between crime and biological aging, I will use data from a prospective birth cohort study, the Dunedin Multidisciplinary Health and Development (Dunedin Longitudinal) Study. The Dunedin Longitudinal Study includes prospective data collection on $N = 1037$ individuals born in Dunedin, New Zealand, between 1972-73. The Dunedin study has conducted 13 phases of data collection at ages 3, 5, 7, 9, 11, 13, 15, 18, 21, 26, 32, 38, and 45. At the most recent phase (phase 45, collected in 2018-19), $N = 938$ individuals participated (i.e., 94% of the original sample still living).

The Dunedin Longitudinal Study is unique for its extensive collection of information on criminal (i.e., both self-reported and official) and deviant behavior (i.e., multisource, from self, parent, and teachers) throughout the life course. Additionally, the Dunedin Study has been a pioneering force in the incorporation of biological/physiological data into sociological study designs. They were among the first studies to collect candidate gene data in the early 2000s (e.g.,

see Caspi et al., 2002) and are still at the cutting edge of biomedical research with their inclusion of genome- and epigenome-wide information. The above factors make the Dunedin Study an ideal resource for analyzing the role of biological aging in the criminal career across the life course.

Measures

Self-Reported Offending Variety. A self-reported offending interview was administered at ages 26, 32, 38, and 45 years using a 1-year retrospective window at each wave. Four types of offenses were assessed. Property offenses included items such as vandalism, breaking and entering, motor vehicle theft, embezzlement, shoplifting, and fraud. Rule offenses included items such as reckless driving, public drunkenness, soliciting or selling sex, giving false information on a loan or job application, and disobeying court orders. Drug-related offenses included using and selling various types of illicit drugs. Violent offenses included items about simple and aggravated assault, gang fighting, robbery, arson, and forced sex.

Offense items were used to create a *Self-Reported Offending Variety* scores, calculated by assigning 1 point for a “yes” response to each different offense and then summing across all types (see Figure 4.1 for a visual presentation of Self-Reported Offending Variety across Phases 26-45). Variety scores typically correlate with frequency (Monahan & Piquero, 2009), but the variety score is preferred over frequency because variety is less skewed, does not overweight trivial offenses (Sweeten, 2011), and is less affected by recall errors (e.g., “Have you shoplifted?” is more accurately recalled than “How many times have you shoplifted?”) (Moffitt et al., 2001). The *Self-Reported Offending Variety* scores ranged from 0 to 30 offense types; higher numbers indicated greater crime involvement.

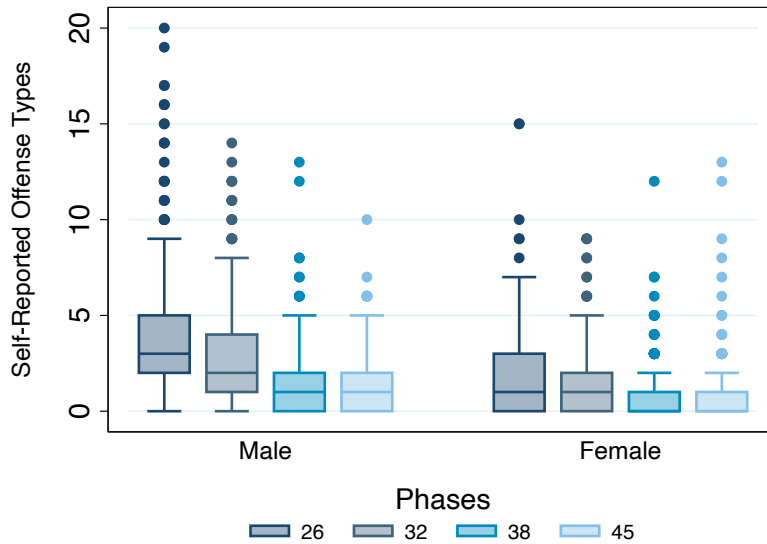


Figure 4.1. *Self-Reported Offending Variety across phases 26-45, stratified by sex.*

Pace of Aging. The *Pace of Aging* measure of biological age was created using data collected at phases 26, 32, 38, and 45 (see Belsky et al., 2015a). *Pace of Aging* was calculated in three steps. First, data on 19 anthropometric/blood-based biomarkers were collected at each phase and then standardized using z -scores ($M=0$, $SD=1$). The 19 biomarkers were (refer to Table 3.1 in chapter 3): body mass index, waist-hip ratio, glycated hemoglobin (HbA1C), leptin, blood pressure (mean arterial pressure), cardiorespiratory fitness (VO2Max), forced expiratory volume in one second (FEV1), forced vital capacity ratio (FEV1/FVC), total cholesterol, triglycerides, high-density lipoprotein (HDL) cholesterol, apolipoprotein B100/A1 ratio, lipoprotein(a), creatinine clearance, urea nitrogen, C-reactive protein, white blood cell count, gum health, and caries-affected tooth surfaces. Some variables were reverse coded so as to ensure that higher scores always indicated greater physiological deterioration (i.e., more advanced age). Next, longitudinal mixed effects growth models were used to estimate random slopes of all 18 biomarkers from phase 26 to 45. Finally, the individual slopes for each study member were summarized into a single *Pace of Aging* score.

The resulting *Pace of Aging* variable was normally distributed in the sample, suggesting that some individuals age faster and some slower than their chronological age would suggest. Figure 4.2 displays the distribution of Pace of Aging scores for male and female participants and provides a reference point (i.e., the red line) for the amount of deterioration expected for a single chronological year (i.e., $Pace\ of\ Aging=1$). To aid interpretation, *Pace of Aging* was scaled within sex so that the central tendency of the measure reflected the physiological change expected over the course of a single chronological year (i.e., $M=1$) for both males and females. On this scale, the sample ranged in the *Pace of Aging* from nearly 0 to around 2.5 years' worth of physiological change per chronological year. For example, a participant who scored a 1.2 is expected to have biologically aged the equivalent of 1.2 years for every 1 chronological year that passes.

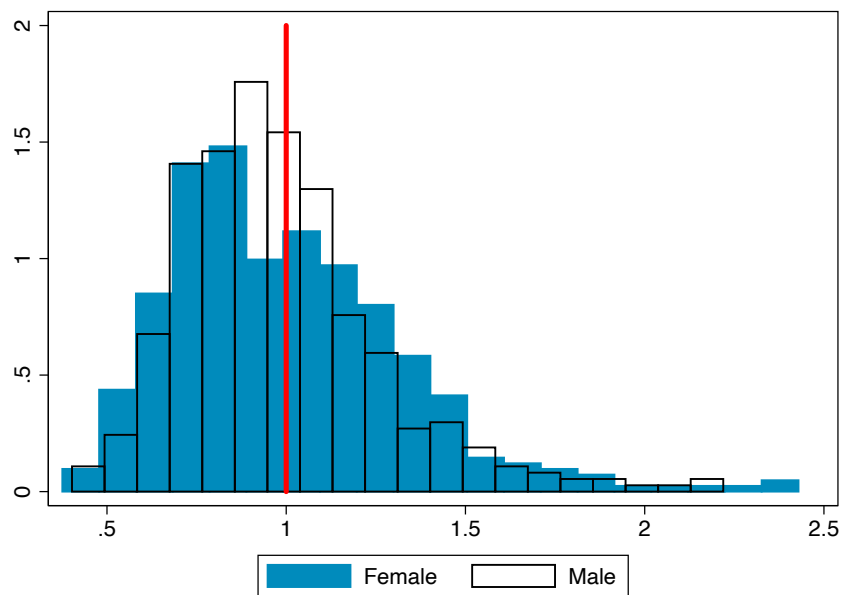


Figure 4.2. Histogram of Pace of Aging from phase 26-45, stratified by sex Covariates. This study uses *Self-Reported Offending Variety* to predict trajectories of desistance over the latter part of the life-course (i.e., age 26-45). It is important to condition these

trajectory models on variables relevant to selection into the various trajectories. With this in mind, I attempt to condition the trajectory model (described below) on variables from two broad domains: offending behaviors and childhood environment/characteristics.

Two types of offending covariates were used to adjust for offending behavior during and prior to the time period of focus in the current study (i.e., age 26-45). First, prior offending behavior was captured by using the average level of self-reported offending variety from age 15-21. This measure of offending differs from the measure used as the outcome of interest in the trajectory analysis (described above) in that fewer items comprise this earlier variety measure (i.e., 12 vs. 44 items). This shorter version was preferred over the longer version because it was consistently asked of Dunedin respondents from age 15 onward, whereas the longer version was not used until age 18.

Participants were asked how many times in the past year they had committed 12 different deviant/criminal behaviors, including: running away from home, carrying a hidden weapon, destroying property, setting fire to property, braking into a building to steal, stealing less than \$100, stealing more than \$100, stealing from a store, stealing a motor vehicle, using force to rob, using marijuana, and using a harder drug (than marijuana). In order to create a variety index of offending, all non-zero responses were given a value of 1 and the resulting items were summed together and then averaged across Phases 15-21. Because this variable represents an average across time, it was entered into the trajectory model as a time-stable covariate.

Criminal convictions during the trajectory period were also included in the trajectory model, but as a time-varying covariate. Official data was used to identify criminal convictions received between ages 26 and 45. Official conviction records were obtained through a search of the central computer system of the New Zealand police that provides details of all New Zealand

convictions and Australian convictions communicated to the New Zealand police. Searches for all convictions occurring from the age from which conviction was permissible (14 years) were conducted after each assessment at ages 26, 38, and 45. *Conviction Status* was coded so as to capture criminal conviction received prior to the start of the observation period in the current study (i.e., 26-45 years of age). For example, any respondent who received a criminal conviction between Phase 26 and 32 received a 1 on their Phase 32 *Conviction Status* variable and a 0 otherwise.

Three additional covariates were included in the trajectory analysis to adjust for confounding due to factors observed during childhood. These covariates were all time stable and included IQ, SES, and poor health. *Childhood IQ* was assessed with the Weschler Intelligence Scale for Children-Revised (WISC-R; Wechsler, 1974) and was administered to the study members at ages 7, 9, and 11 years. IQ scores for the three ages were averaged and standardized ($M=100$, $SD=15$). *Childhood SES* was measured by assessing the cohort members' families on a six-point scale that assessed parents' occupational statuses, defined based on average income and educational levels derived from the New Zealand Census (Poulton et al., 2002). Parents' occupational statuses were assessed when participants were born and again at subsequent assessments up to age 15 years. The highest occupational status of either parent was averaged across the childhood assessment.

Childhood Poor Health was measured from a panel of biomarkers and clinical ratings taken at assessments from birth to age 11 years (Belsky et al., 2015b), including motor development (at ages 3, 5, 7, and 9 years), overall health (at ages 3, 5, 7, 9, and 11 years; rated by two Unit staff members based on review of birth records and assessment dossiers including clinical assessments and reports of infections, diseases, injuries, hospitalizations, and other

health problems collected from children’s mothers during standardized interviews), body mass index (at ages 5, 7, 9, and 11 years), tricep and subscapular skinfold thicknesses (at ages 7 and 9 years), and, finally, forced expiratory volume in one second (FEV1) and the ratio of FEV1 to forced vital capacity (FEV1/FVC; at ages 9 and 11 years). To calculate the childhood health measure, assessments were standardized using z-scores ($M=0$; $SD=1$) within age- and sex-specific groups. Cross-age scores for each measure were then computed by averaging standardized scores across measurement ages. The final *Childhood Poor Health* score was calculated by taking the natural log of the average score across all measures, resulting in a normally distributed childhood health index. High scores indicate poorer average health in childhood.

Analytic Plan

The analysis for the current study will unfold in three steps. First, I describe the sample with regard to the distribution of key variables. Second, I explore the desistance patterns identified in the sample by estimating a group-based trajectory model (GBTM) (Nagin, 2005) with the *traj* suite (Jones & Nagin, 2013) in Stata version 14 (StataCorp, 2015). GBTMs are a specialized case of the finite mixture model used in longitudinal settings and the maximum likelihood parameter estimates have been shown to be asymptotically unbiased and asymptotically normally distributed in sample sizes as small as $n = 500$ (Loughran & Nagin, 2006). GBTMs work by situating cases into subgroups, each with a unique trajectory on an outcome that is described by a finite set of polynomial functions (i.e., of age, time, etc.). Model fit is monitored, and the best-fitting model is selected primarily on the basis of the Bayesian Information Criterion (BIC), which is calculated as (Nagin, 2005, p. 64):

$$BIC = \log(L) - 0.5k \log(N) \quad (5)$$

where L is the model-specific maximum likelihood, N is the sample size, and k is the number of parameters in the model. The BIC is a model fit index that penalizes model fit as a function of the number of model parameters, thus preferring parsimonious solutions. Subgroup assignment is achieved through the use of conditional probabilities derived from changes in individuals' levels on an outcome over time. For the current analysis, I will be predicting trajectories of *Self-Reported Offending Variety* during middle adulthood as a way to test for heterogeneity in patterns of desistance, conditional on time-stable and time-varying covariates.

Third, and finally, I will use *Pace of Aging* and the other covariates to predict trajectory group membership. Having identified cases with statistically similar trajectories across time, GBTM allows for the simultaneous prediction of group membership using logistic regression. Because the number of trajectory groups identified in the current analysis exceeded two (described below), a series of multinomial logistic regressions were used to estimate the impact of *Pace of Aging* and other lifestyle characteristics on trajectory group membership.

Results

I begin by describing the sample in terms of the key variables for the analysis (see Table 4.1). After listwise deletion on *Self-Reported Offending Variety*, *Pace of Aging*, *Conviction Status*, *Tobacco Pack-Years*, and childhood covariates, a sample of $N=845$ remained. Ten additional cases were removed due to their outlier status on offending variety (more details below), leaving a final analytic sample of $N=835$. The analytic sample was 50% male. A chi-square test for independence revealed that trajectory group membership did not significantly vary across sex ($\chi^2=3.67$; $P>0.05$). As expected, *Self-Reported Offending Variety* declined over time from Phase 26-45, from participants reporting an average of 2.83 ($SD=2.93$) crimes in the past year at Phase 26 and an average of 0.87 ($SD=1.45$) crimes in the past year at Phase 45. Also

expected were statistically significant sex differences in offending variety across all phases in favor of males. The patterns observed in self-reported offending variety were mimicked by the declining, male-biased rates of criminal convictions across phases.

Table 4.1. Univariate Statistics of Key Variables and T-Test/Chi-Square of Group Mean Differences Across Male (N=412) and Female (N=423) Respondents (Total=835).

	Mean (Proportion)	SD	Min	Max	t-stat (χ^2)
Self-Reported Offending Variety					
<i>P26</i>	2.83	2.93	0	20	-10.99***
<i>P32</i>	2.13	2.18	0	14	-9.89***
<i>P38</i>	1.04	1.51	0	13	-6.65***
<i>P45</i>	0.97	1.45	0	13	-4.70***
Pace of Aging	1	0.30	0.38	2.43	1.29**†
Conviction Status	(0.23)	-	0	1	(57.16***)
Childhood IQ	100.00	15.00	39.87	143.27	-2.19*
Childhood SES	3.81	1.11	1	6	-1.13
Childhood Poor Health (z-score)	0	1	-2.64	2.7	-0.44

*= $P < 0.05$, **= $P < 0.01$, ***= $P < 0.001$.

Note: *t*-statistics and chi-square values correspond to mean and proportion tests across male and female respondents. †*F*-statistic: *Pace of Aging* was scaled within sex so that both male and female participants would have a group mean of 1, thus a variance ratio test was employed to assess distributional instead of mean differences. With the exception of *Pace of Aging*, all significant differences were in favor of male respondents.

Pace of Aging was normally distributed in the sample ($M=1$; $SD=0.30$), suggesting that some members of the sample were aging slower ($Min=0.38$) and some faster ($Max =2.43$) than would be expected for a single year of chronological time. Sample means of *Pace of Aging* could not be compared across sex because it was scaled within sex so that both male and female respondents would have a mean of 1. Thus, a variance ratio test was employed, and statistically significant differences were observed ($F=1.29$; $P < 0.01$) with female respondents having a larger standard deviation in *Pace of Aging*. Among the covariates only *Childhood IQ* ($t=-2.19$; $p < 0.05$) varied significantly across sex with a small bias towards males in the sample.

Group-Based Trajectory Modeling Analysis

I now turn to the prediction of desistance trajectories in the Dunedin Longitudinal Study sample. Given the low rates of offending among females identified by the prior descriptive analysis, the Group-Based Trajectory Modeling (GBTM) analysis was conducted only on the male subsample. This approach was selected because it is consistent with the prior research on offending trajectories (Odgers et al., 2007) and will help focus the analysis on the portion of the sample that contains the majority of the variation in criminal behavior beyond age 26. After listwise deletion on Pace of Aging and the covariates, the analytic sample was $N=384$ cases.

Like most offending measures, self-reported offending was heavily skewed at each phase prompting the use of a zero-inflated Poisson (ZIP) distribution for the GBTM. ZIP models are appropriate when a measure contains more zeros than is assumed by the Poisson assumption (Lambert, 1992). ZIP models assume that there are two groups within the sample—a zero-only group and a group that demonstrates variation in the outcome with which distinct trajectories may be estimated. Having established the model distribution, the model-fitting process commenced, and a three-step procedure was used. First, different polynomials were repeatedly fit to a one-group model. These polynomials were used to describe the none-zero group assumed in the ZIP model, and they included a constant (i.e., intercept), linear, quadratic, and cubic terms (see the models listed for step 1 of Table 4.2).

The model fit of the resulting solutions were primarily compared based on the Bayesian Information Criterion (BIC; Schwarz, 1978), but also with a number of supporting metrics (described below). The BIC is a model fit statistic that is similar to the Akaike Information Criterion (AIC; Akaike, 1974), but the BIC is notable for exerting a harsher penalty on free parameters and thus preferring more parsimonious models. Thus, the BIC works against the

tendency of adding groups to a model in order to increase likelihood-fit statistics. As the specified models improve in their fit to the data, the BIC will decrease (i.e., approach zero). Two forms of the BIC were relied on: one that scales the penalty on free parameters based on the number of observations across phases (i.e., unadjusted BIC) and one that scales the penalty by also adjusting for the analytic sample (i.e., sample-adjusted BIC; Sclove, 1987). Looking at Table 4.3, we see the cubic form produced the best model fit according to BIC values (i.e., it produced the values closest to zero). Thus, a cubic term was adopted for the second step in the model selection process.

Table 4.2. Results from The Model Fitting Process of The GBTM Analysis.

Number of Groups	Poly-nomial	Unadjusted BIC	Adjusted BIC	APP	OCC
Step 1					
1	0	-4109.02	-4107.64	-	-
1	1	-4109.02	-3881.90	-	-
1	2	-3886.89	-3884.12	-	-
1	3	-3876.44	-3872.98	-	-
Step 2					
2	33	-2931.67	-2919.88	.99, .95	162.81, 211.06
3	333	-2931.67	-2842.78	.91, .91, .98	77.60, 39.23, 990.98
4	3333	-2847.36	-2820.33	.87, .92, .90, .99	67.59, 45.09, 144.29, 85894.23
5	33333	-2879.35	-2844.692	.86, .81, .93, .90, .99	56.45, 1455.04, 52.72, 147.23, 87490.42

Notes: BIC= Bayesian information criterion; APP=average posterior probability of assignment; OCC=odds of correct classification. Polynomials: 0=intercept only; 1=linear; 2=quadratic; and 3=cubic. **Bolded** models were selected based on model fit. All models adjusted for Pace of Aging, childhood IQ, SES, poor health, and the average offending involvement from age 15-21.

The second step of model fitting involved the identification of the correct number of trajectory groups in the data. In a similar fashion to step one, polynomials were repeatedly fit to the ZIP model and model fit was compared across models in terms of the BIC statistics. In this

case, however, each model added a cubic term until models with polynomial terms 2-6 were estimated. BIC values were compared across these models in the hopes of identifying the optimal number of trajectory groups supported by the data. In order to determine the appropriate number of groups 2- through 5-group models were fit, each group taking the cubic form. Model fit was evaluated with the use of a variety of fit statistics, including BIC and the sample-adjusted BIC, as well as the average posterior probability (APP) of assignment and the odds of correct classification (OCC). First, the models were evaluated based on BIC. As in step 1, the model with the largest BIC (i.e., closest to zero) is the preferred model. Both adjusted and unadjusted BIC statistics identified the 4-group model and the model with the best fit. In terms of APP (a statistic roughly similar to “entropy” used in growth mixture modeling; Sweeten, 2018), all four groups within the 4-group solution were beyond the 0.70 threshold recommended by Nagin (2005) with the lowest APP being 0.87.

Next, I examined the OCC for each group, which is calculated as (Nagin, 2005, p. 88):

$$OCC_j = \frac{APP_j/1 - APP_j}{\hat{\pi}_j/1 - \hat{\pi}_j} \quad (6)$$

where the numerator represents the odds of correct classification into group j based on the maximum posterior probability rule. The denominator represents the odds of correct classification into group j based on random assignment, with the probability of assignment to group j being $\hat{\pi}_j$, the estimated base rate in the population. As model accuracy improves beyond chance, the OCC will exceed 1, with a value of 5 being the minimum necessary to indicate high assignment accuracy. Moving to the OCC, all four groups had OCCs in excess of 5 (i.e., the minimum value suggested by Nagin, 2005) and they ranged from 45.09 to 85894.23. These high odds suggest that this group assignment was a significant improvement over chance.

Finally, the group proportions of the 4-group model were examined. Three groups contained acceptably large group sizes (i.e., $n=60$, 102 , and 213), while one group was substantively small, containing only 2.3% of the sample ($n=9$). Despite its size, the 4-group model was retained in the hopes of avoiding the small trajectory group from being imperfectly incorporated into the remaining trajectories. This would have been problematic, as the small group represented a cluster of individuals who were abnormally high in offending (i.e., compared to the rest of the sample) throughout the entire observation period.

A Four-Group Model of Desistance

Having identified four different trajectories with which to describe desistance, a brief description of these groups is in order (a visual depiction of the three trajectory groups is provided in Figure 4.3). The first trajectory group (i.e., 25% of the sample) demonstrated a substantively low level of offending over time, which led to members of this group being labeled “Abstainers”. This label is drawn from prior work on offending trajectories (e.g., Moffitt, 1993; Moffitt et al., 1996) and is used to signify the exceedingly low level of offending displayed by this group over time. The individuals of the second desistance trajectory were labeled “Normative Desisters”. The choice of the “Normative” label is appropriate for this group as the trajectory group comprised the majority of the sample (i.e., 56.5%) and, being a population representative birth cohort, the majority by definition is the norm.

The third group displayed much higher rates of self-reported offending during the observation period, gaining its members the label of “Non-Normative Desisters”. The Non-Normative group began with a high level of self-reported offending at Phase 26, experienced a sharp decline throughout Phases 32 and 38, before leveling off at Phase 45. This group was comprised of a small portion of the sample (16.4%) and, as expected with a smaller group, it also

demonstrated a large amount of variance. The final group, as was mentioned above, was very small, containing only eight individuals (2.1% of the sample). These individuals demonstrated a high level of offending during the observation period, with virtually none of its members reporting less than five crime types perpetrated in the last year. This trajectory group was labeled “Persisters” due to their consistent pattern of offending over the observation period.

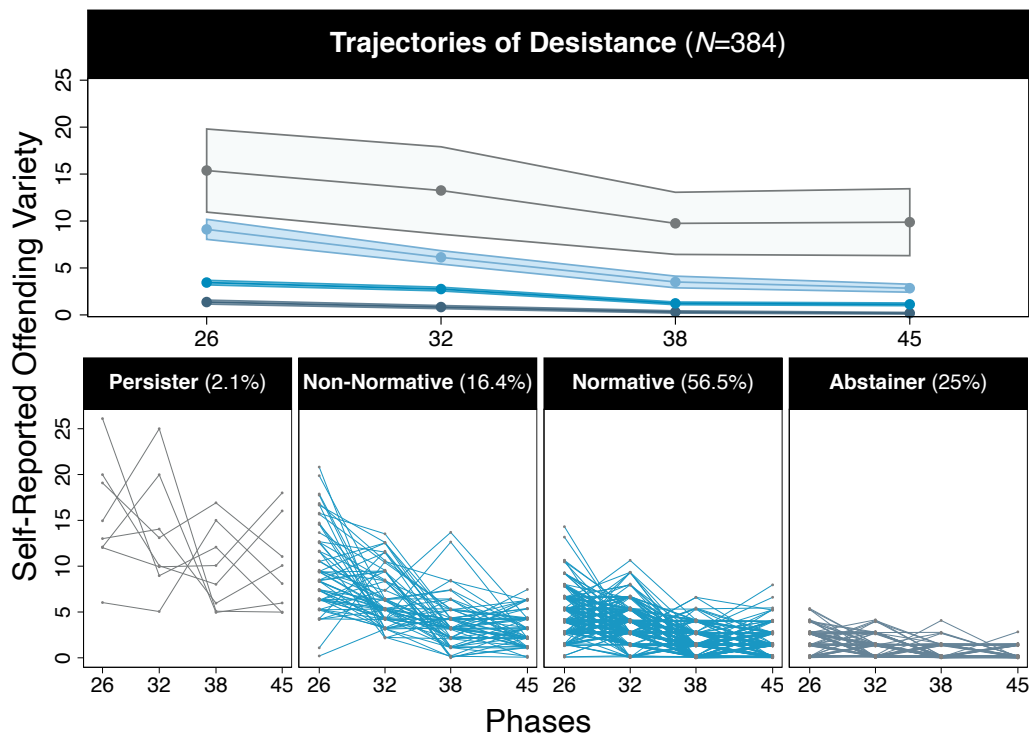


Figure 4.3. Four-group trajectory model of Self-Reported Offending Variety from age 26-45.

Predicting Trajectory Group Membership—Does Pace of Aging Differ Across Trajectory Groups?

Having identified the model producing the best fitting solution to the data, I now turn to the analysis predicting group membership using *Pace of Aging* and other covariates. This analysis took the form of a multinomial logistic regression estimated simultaneously with the 4-

group GBTM model, thus the estimates reported in Table 4.3 represent the prediction of group membership probabilities having adjusted for the key variables and covariates during their initial estimation. Multinomial logistic regression allows for group comparisons by selecting a reference group and then using a logistic regression framework to compare each group to that reference group. As the *Persister* group contained only eight individuals, I place little emphasis on the estimates derived from this group. Thus, I do not use this group as a reference group nor do I report their results.

Table 4.3. Prediction of Offending Trajectory Group Membership among Males ($N=384$) with Pace of Aging and Covariates Using Multinomial Logistic Regression.

	<i>b</i>	SE	OR	95% CI
Non-Normative vs Normative				
Pace of Aging	1.36†	0.78	3.90	[0.84, 18.02]
Average Early Offending	0.80***	0.15	2.22	[1.66, 2.96]
Childhood IQ	0.04*	0.02	1.04	[1.00, 1.08]
Childhood SES	-0.18	0.20	0.84	[0.57, 1.23]
Childhood Poor Health (z-score)	0.19	0.24	1.21	[0.75, 1.94]
Constant	-8.07**	2.55		
Non-Normative vs Abstainer				
Pace of Aging	0.26	1.03	1.29	[0.17, 9.63]
Average Early Offending	3.51***	0.60	33.39	[10.41, 107.08]
Childhood IQ	0.04	0.03	1.04	[0.99, 1.09]
Childhood SES	-0.13	0.26	0.88	[0.52, 1.47]
Childhood Poor Health (z-score)	0.07	0.31	1.08	[0.59, 1.97]
Constant	-7.82*	3.16		
Normative vs Abstainer				
Pace of Aging	-1.10	0.81	0.33	[0.069, 1.60]
Average Early Offending	2.71***	0.58	15.07	[4.86, 46.72]
Childhood IQ	0.00	0.02	1.00	[0.96, 1.03]
Childhood SES	0.05	0.20	1.05	[0.71, 1.56]
Childhood Poor Health (z-score)	-0.12	0.23	0.89	[0.57, 1.40]
Constant	0.25	2.14		

† $p < 0.1$; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Non-Normative vs. Normative Desisters. Turning to the first model of Table 4.3, we see that Pace of Aging did predict group membership in the expected direction (i.e., higher Pace of Aging predicted an increase in the probability of being placed in the Non-Normative group over the Normative group of desisters. However, this prediction was not statistically significant ($b=1.36$; $SE=0.78$). Despite the probability of membership in the Non-Normative group increasing almost four-fold for a one-unit increase in *Pace of Aging* ($OR=3.9$), the high degree of variability in the effect ($CI=0.84-18.02$) suggests that caution be used when interpreting this finding as even marginal support for this study's hypothesis.

Moving to the other covariates in the first model, only two predicted membership in the Non-Normative group over the Normative group of desisters to a statistically significant degree: *Average Early Offending* and *Childhood IQ*. Committing one additional type of crime during adolescence/early adulthood (i.e., age 15-21) results in a little more than a two-fold increase ($OR=2.22$; $CI=1.66-2.96$) in the odds of being classified as a Non-Normative desister over a Normative desister. This finding is expected, as the commission of more crime early in life has been repeatedly found to predict more offending later in life. Lastly, Childhood IQ showed a small influence on the probability of group membership, with a one-point increase in childhood IQ was associated with 4% increase ($OR=1.04$; $CI=1.00-1.08$) in the odds of being classified as a Non-Normative desister.

Non-Normative Desister vs. Abstainer. The second model compared the highest trajectory group highest in offending (i.e., excluding the Persister group) to trajectory group that was lowest in term of offending. Despite expectations, the effect of *Pace of Aging* on the probability of group membership did not attain statistical significance, although it was in the expected direction ($b=0.26$; $SE=1.03$). A one-unit increase in *Pace of Aging* did increase the

probability of being classified as a Non-Normative desister by almost 30% (OR=1.29), but the effect of *Pace of Aging* was again found to be very heterogenous (CI=0.17-9.63). With regard to the other covariates, only Average Early Offending predicted a statistically significant increase in the odds of being classified as Non-Normative compared to being classified as an Abstainer (OR=33.39; CI=10.41-107.08).

Normative Desisters vs. Abstainers. Moving to the final group comparison, *Pace of Aging* again did not predict group membership to a statistically significant degree. Interestingly, however, the effect of Pace of Aging on in this final model reversed its sign ($b=-1.10$; $SE=0.81$), suggesting that an increase in Pace of Aging predicted a lower probability of being classified as a Normative desister compared to an Abstainer. This would suggest that Abstainers, despite being lower in offending, may experience a more advanced *Pace of Aging* compared to the Normative desisters (i.e., counter to the expectation of this study). Considering the large standard error of the effect, however, this finding should not be interpreted as a substantive result of the analysis. Lastly, *Average Early Offending* predicted a significant increase in the odds of being classified among the Normative desisters compared to the Abstainers (OR=15.07; CI=4.86-46.72). As the Normative group still demonstrated some degree of offending while the Abstainer group demonstrated virtually none, this result was expected.

Looking at the results as a whole, *Pace of Aging* largely performed in the expected manner, if not to the hypothesized degree. After adjusting for range of covariates, an increased *Pace of Aging* did demonstrate a trend of also increasing the probability of being classified among the Non-Normative desister compared to the other groups. This trend was non-significant, however, a result that may be due to the high degree of variability in *Pace of Aging* within the groups. This variability is visible in group-specific means of *Pace of Aging* presented in Figure

4.4. Despite the higher average level of *Pace of Aging* among Non-Normative desisters, after adjustment for covariates it did not predict trajectory group membership.

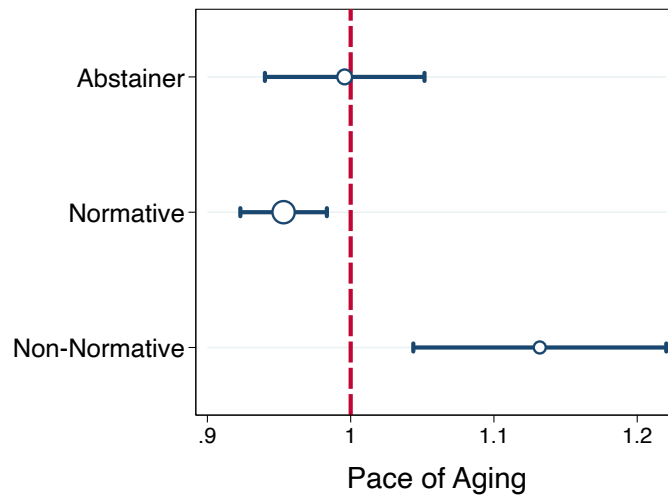


Figure 4.4. Group-specific Averages of Pace of Aging.

Discussion

Uncertainties remain about one of criminology’s “laws”: the age-crime curve. While theories explaining the early curvature of the age-crime phenomenon are plentiful, fewer have offered insight into its latter half—and fewer still have been tested (Rocque, 2017). This study focused on the non-normative desistance process and provided one of the first tests of the (biological) aging-out hypothesis. Leveraging newly developed methods for quantifying biological age, this study used data from the Dunedin Longitudinal Study to assess whether “Pace of Aging” was associated with variation in offending (desistance) trajectory in middle adulthood.

Using group-based trajectory modeling (GBTM; Nagin, 2005), a 4-group of desistance was identified and then trajectory group membership was predicted with a longitudinal measure of aging, referred to as “Pace of Aging” (Belsky et al., 2015a). Although there is a clear substantive *pattern* that emerged in the data (i.e., that Non-Normative desisters have the highest

average level of Pace of Aging), group comparisons conducted with multinomial logistic regression did not find a statistically significant association. The small group sizes in the current analysis contributed to a high degree of heterogeneity in the effect of Pace of Aging (and thus large confidence intervals). Future studies with increased statistical power will be needed to determine if the heterogeneity observed here is indeed hiding a truly distinctive level of advanced aging among high-rate offenders or is merely an artifact of the current analysis.

This study attempted to demonstrate an association between offending beyond the normal point of desistance and biological aging. The hypothesis being that biological aging (i.e., generalized physical deterioration) as a substantive influence on the criminal career should be concentrated among those individuals who persist in offending long enough to experience its negative effects. Yet the findings from the current study do not allow me to reject the null hypothesis.

Future studies that attempt to examine the perspective on biological aging examined here should consider a few points. First, the trajectories observed using the GBTM approach appeared to be largely similar with regard to shape, only differing in terms of level of offending over time. This result may point future researchers to different methods of examining longitudinal change that do not assume multiple/distinct trajectories of desistance (e.g., latent growth curve models). Such approaches may still allow researchers to capture of the variation in desistance while not sacrificing samples size through the partitioning of samples that is required by group-based approached.

Second, it is possible that the high degree of heterogeneity in observed effects across trajectory groups, if not solely due to group size, may have been the result of the specific choice of offending measure. Though commonly used among criminologists to estimate gross levels of

offending involvement, variety measures of offending run the risk of obscuring meaningful variation in outcomes that are more associated with some crime types than others. For instance, it may be that advanced biological aging is more associated with individuals who specialized in either violence or drug related offenses, while property offenses are not associated with aging outcomes. Hypotheses in this vein may be best explored within large population samples or else institutional samples so that adequate statistical power for crime type comparisons may be reliably achieved.

Limitations

This study used a prospective cohort study and a novel measure from the field of geroscience, Pace of Aging, to operationalize an aspect of the age-crime literature—biological aging—that has heretofore been outside the reach of criminology. Despite the strengths the study, it also contained several limitations. First, the results of this study can only be described as correlational, as the primary dependent and independent variables were measured over the same period (i.e., between ages 26 and 45). As direction of influence cannot be established, no causal interpretation would have been possible should the analysis have identified the hypothesized relationship.

Second, this study did not examine female offenders. The focus on male participants was largely due to the limited variation in offending among female participants, as the Dunedin Longitudinal Study is a population sample and the prevalence of female offending is generally quite low in the population (Steffensmeier & Allen, 1996). Future research efforts should focus on justice-involved samples in order to better understand how biological age influences one of the most impactful social behaviors—crime. The female incarceration rate has been on a steady climb for the past several decades (Carson, 2020; Zeng, 2020) And while still far below the

incarceration rate of males, this segment of the justice-involved population should receive more empirical study in the hopes that they too may benefit from the better-informed policy decisions that result from such efforts.

Third, the age of the study sample may have prevented the current study from an association between offending and biological aging. For instance, it may be that the effects of offending occur after the chronological age of 45. Although, Pace of Aging has been shown to predict variation in a number of outcomes include cognitive performance, physical tasks, subjective aging, and others (Belsky et al., 2015), it may be that lifestyle-factors of offending do not demonstrate an association with aging until much later in the life course. Fourth, and finally, the size of the current study sample raised concerns regarding the statistical power of the observed effect. It is my hope that, with improved statistical power, a future study may be able to observe some signal through the noisy results observed in the current study.

Conclusion

This study's primary hypothesis was that biological aging would be associated with the non-normative desistance process. It is among these more persistent offenders that biological aging may be expected to be substantively impactful in shaping the desistance process. Offenders who desist at the normative time (i.e., early adulthood) never attain a biological age advanced enough to impose the kinds of physical limitations that would drive such an "aging out" effect. The findings of the current study suggest that non-normative desisters do not necessarily have an advanced biological age; however, additional evidence is needed before a firm conclusion can be reached. In light of the current study's findings and its limitations, there is much work to be done in exploring this newly operationalized dimension of the aging process and its role in the criminal career.

Chapter 5 —From Age to Aging: Discussion and Conclusions

Discussion

The criminological literature assumes that age is an exogenous force in the life course of offenders. Modern geroscience has complicated the purely temporal view of age by directly attention away from *age* as a temporal state and towards *aging* as a biological process. The move from age to aging necessitates an appreciation for and quantification of the biological factors that undergird the aging process. Geroscience has begun to develop measures of biological age that capture the underlying system integrity of a body and outperform chronological age in the prediction of morbidity and mortality.

This dissertation integrated biological age with developmental/life-course criminology by leveraging these newly developed methods in three analyses that probed long-held criminological questions. These included: 1) the health consequences of contact with the criminal justice system, 2) the long-term effects of early offending on aging, and 3) aging out in the non-normative desistance process. These analyses involved the use of two cutting-edge methods for quantifying biological age (“Pace of Aging”; Belsky et al., 2015a; “PhenoAge Acceleration”; Levine et al., 2018) in two independent longitudinal datasets (i.e., the Dunedin Longitudinal Study, Health and Retirement Study) that were collected in two different countries (i.e., New Zealand, the United States). In each case, the results suggest age is more complicated than criminological theory has acknowledged. This is the first evidence that viewing age through a biological lens is not only possible but also useful for exploring the age-crime relationship in more depth.

Below I review the major findings reported in the previous chapters. While doing so, I will draw connections across analyses as well as outline policy implications. Finally, I will

discuss the utility of updating developmental/life-course theories in criminology to account for biological aging.

Area 1—The Health Consequences of Criminal Justice Contact

In chapter 2, I explored the association between incarceration and biological age in the representative sample of elderly Americans in the Health and Retirement Study (HRS). This analysis provided a useful first test case because if we assume that greater involvement in the criminal lifestyle will advance biological age, then it would follow that those who experience incarceration would be most impacted. After all, those who experience incarceration have not only exhibited criminal behavior (i.e., assuming no wrongful convictions) but they have also proceeded through every level of the criminal justice system (i.e., arrest, indictment, conviction, and incarceration). Given the representativeness of the HRS sample, I was also able to examine how the association between incarceration and biological age breaks across demographic lines. This last point was a crucial part of the analysis, especially given the marked disparities that exist in minority communities across both incarceration rates and health burden.

The findings from the chapter 2 generally supported the conclusion that incarceration is associated with having an accelerated biological age, usually in the 1-2 ½ year range. This means that former inmates will be older biologically than they are chronologically, increasing their likelihood of experiencing early onset of age-related morbidities (e.g., cardiovascular disorders, pulmonary disorders, cancers). When broken down by sex and race/ethnicity, three patterns emerged: 1) males demonstrated higher biological age than females, regardless of race or incarceration status; 2) Black respondents had the highest biological age, regardless of incarceration status; and 3), Black respondents were the only racial group that did not show signs of higher biological age as a function of incarceration status.

The findings from this first study must be considered in light of a number of limitations. First, the measure used to assess biological age (i.e., PhenoAge Acceleration) might not be well-calibrated for members of minority groups. This possibility would suggest that the pattern of findings for Black respondents in the HRS represent more of a statistical artifact than reality. This possibility may be unlikely, however, because the PhenoAge measure of biological age has been shown to perform similarly across the different major racial/ethnic subpopulations in the United States (e.g., Liu et al., 2018). Alternatively, it is possible that the Black HRS respondents experienced differential attrition, thereby inducing a group-specific survivor bias. For instance, if the unhealthiest Black respondents (and especially those who experienced incarceration) died prior to the estimation of PhenoAge this would leave only their healthier (i.e., biologically younger) counterparts in the sample. The result of such a survivor bias would be a deflated estimate of PhenoAge among Black respondents who experienced incarceration, thus making the incarceration experience appear to have no, or even opposite-signed, effects.

Second, the imprecise measurement of incarceration may have impacted the results in various ways. Though a weakness of the study, the measurement of incarceration in the HRS is typical for large-scale longitudinal studies. There currently exists a dearth of studies that include both in-depth information on criminal justice contact and high-quality biomedical data of the sort needed to estimate biological age. Using the HRS, afforded the ability to strike a balance between quality biomedical data, information on incarceration, and representative sample characteristics (i.e., the sample is old enough to avoid right-censoring on incarceration and to show substantive signs of aging).

Third, it is possible that incarceration is just a proxy for other lifestyle factors/circumstances that are actually causal in the relationship. If that is the case, it would

mean that the incarceration experience simply identifies at-risk individuals but does not itself contribute a causal effect to their rate of biological aging. The propensity score matching (PSM) analysis in chapter 2 attempted to rule out this possibility by adjusting for a range of childhood background factors before estimating the association between incarceration and aging. The results from this portion of the analysis revealed that the incarceration effect remained even after adjusting for a major source of confounding (i.e., childhood disadvantage). Because I was unable to adjust for contemporaneous aspects of the criminal lifestyle, the findings from this chapter should be considered suggestive, but not conclusive in terms of whether the pattern of results indicate a true “incarceration effect”.

Recognizing this limitation provided the motivation for the next analysis, which assessed criminal behaviors and events that precede incarceration as predictors of aging in mid-life. Exploring the events and behaviors that precede incarceration helped provide insight into whether the results uncovered in chapter 2 were the result of an impact of incarceration or whether incarceration is simply a proxy for other lifestyle factors/circumstances.

Area 2—The Long-Term Health Effects of Offending Behavior

In chapter 3, I used data from the prospective birth cohort of New Zealanders in the Dunedin Longitudinal Study to explore the association between offending behavior in the first quarter century of life and biological aging throughout middle adulthood. Building on the results from the previous chapter, I examined aspects of offending that precede incarceration—including 1) level of offending, 2) pattern of offending, and 3) criminal convictions—as predictors of aging. The analysis in chapter 3 improved on the chapter 2 analysis in that all of the data used were prospective instead of the retrospective self-reported measures available in the HRS. With

the Dunedin Longitudinal Study data, I was able to better account of the timing of events in order to sort out the relationships of interest.

The chapter 3 analysis found that level, but not pattern, of offending was associated with a more rapid pace of aging throughout middle adulthood after adjusting for childhood covariates. As with the HRS analysis, I attempted to adjust for selection into the criminal justice system when assessing the impact of criminal justice contact. Using an inverse probability of treatment weighting (IPTW) framework, I was able to estimate the average treatment effect of receiving a criminal conviction by age 26 on later pace of aging. The results suggested that, even after adjusting for selection, the receipt of a criminal conviction impacted one's pace of aging. Sensitivity analysis revealed simultaneity bias did not likely inflate the observed association.

The results of chapter 3 point to an association between early offending behavior and pace of aging throughout middle adulthood. At a higher, more conceptual, level, the key contribution of chapter 3 is that it demonstrated the age-crime relationship is not necessarily unidirectional. Thus, age might exert some influence on offending, but not the reverse. The results from the analysis of the Dunedin Longitudinal Study demonstrated that biological age is a process that is also sensitive to offending behavior. Thus, age, when viewed from a biologic perspective, might be influenced by crime in return. Having observed the interplay between biological age and the early part of the criminal career, the final step for this dissertation was to explore the latter part of the criminal career: is aging associated with desistance from crime?

Area 3—Biological Age and the (Non-Normative) Desistance Process

In chapter 4, I explored the association between biological aging and non-normative desistance in males using data from the prospective birth cohort of New Zealanders in the Dunedin Longitudinal Study. This analysis drew on the distinction between normative and non-

normative desisters and hypothesized that biological aging would play an outsized role in the latter group. Using group-based trajectory modeling (GBTM), I identified four patterns of desistance in the data. Preliminary results suggest that the Non-Normative desister group presented with the highest overall levels of biological aging (“Pace of Aging”; Belsky et al., 2015a). But these group differences were not statistically significant once subjected to a multivariate analysis. The null effect of this study suggests that, while Pace of Aging may be responsive to lifestyle factors and life events (e.g., Belsky et al., 2017), perhaps it does not vary to such a degree as to be able to distinguish between different trajectory groups. The small group sizes in this study make these findings tentative and in need of further testing with a larger sample.

Though not definitive, the analyses undertaken in chapters 2-4 have provided preliminary evidence for an association between the criminal lifestyle—be it offending behavior or contact with the criminal justice system—and changes in biological aging. It is important to emphasize that these results are preliminary. The limitations of each study—especially their inability to fully rule out all other factors of the criminal lifestyle that could explain the observed associations—must be kept in mind when interpreting these results. Each chapter explored a different aspect of the criminal career and, in each case, demonstrated the relevance of biological age to criminological processes. Having described the empirical results, I will now turn to a discussion of the theoretical implications of examining the age-crime relationship from a biological perspective.

Theoretical Considerations

For nearly two centuries, criminology has studied how offending patterns change with age. As described in chapter 3, criminological theories have generally fallen into one of two

camps when seeking to explain the role of age in the criminal career: 1) age exerts an exogenous influence on crime or 2) age is only important insofar as it indicates the onset of socially-timed events that exert a real causal influence on crime. These perspectives point the causal arrow in one direction only—from age to crime. By shifting the focus from temporal age to biological aging, however, the path is opened for the causal flow to run from (biological) age to crime and back.

Measures of biological age capture the idiosyncratic changes in bodily integrity and health to which a measure of chronological age would be insensitive. The major implication for criminology being that age becomes tied to the bodily health of the offender. Herein lies the opportunity to merge the age-crime and crime-health literatures: because biological age is tied to the bodily integrity of the offender, health will necessarily decline as a function of offenders' biological age. Just as examining minority status brings with it certain connotations relating to socioeconomic status, so too will biological age connote things about health.

Biological age as a proxy for health brings with it several benefits, including an ability to assess variation in health that is both subclinical (i.e., below clinical thresholds) and unknown to the offender themselves (i.e., an offender need not feel consciously ill for a measure of biological age to detect signs of declining health). Because measures of biological age is now considered the best predictors of morbidity and mortality (i.e., even outperforming chronological age; Levine, 2013; Levine et al., 2018), it thus provides a mechanistic link between lifestyles that wear on the body's integrity and later disease morbidities and mortality.

With this strong linkage to health comes a limitation on the utility of biological age: biological aging does not begin in earnest until sometime in early adulthood, when health begins to decline at the population level. All of the age-related variation in offending behavior that

precedes adulthood (i.e., the majority of the age-crime curve) is thus out of reach for measures of biological age. As shown in chapter 3, it is possible that early offending behavior exerts an influence on biological age later on in the life course, but concurrent measurement and analysis of biological age and early offending are not possible. In other words, the utility of biological age for criminology lies in its ability to combine age- and health-related processes with the analysis of the criminal career/lifestyle. If a phenomenon is particularly associated with chronological age and not associated with health, then the utility of employing a measure of biological age will be limited.

An Integrated Vision of Biological Age and Developmental/Life-Course Theory

This dissertation has uncovered some of the first evidence that offending, and its consequences like incarceration, impact pace of aging. Evidence also indicated that faster pace of aging may impact the desistance process. In short, this dissertation has shown that age and crime are tightly intertwined in more complicated ways than has heretofore been appreciated. No longer will it be sufficient to argue that age is an exogenous influence on the criminal career (Hirschi & Gottfredson, 1983). Instead, with the results from the current dissertation in hand, we now know that the reality is much more complicated: offending influences aging, which goes on to influence offending. The impact of these findings on developmental/life-course theory is, therefore, important to consider.

It may turn out that scholars need to re-visit assumptions about the influence of age in developmental/life-course theory. One way forward is to consider a “multi-system” approach, where chronological, social, and biological age as viewed complementary influences. For instance, take the age-crime curve—a sharp increase in offending during childhood/adolescence, a peak during late adolescence, and a steady decline throughout adulthood. During the initial rise

and peak, the effects of chronological age would predominate due to the use of chronological age by society to time certain events. For instance, 18 marks the age of majority and the end of compulsory education, 21 marks the full removal of prohibitions on the purchase of alcohol and tobacco, and myriad other events (e.g., marriage, joining the work force or military) are expected to fall in this age range though they are less formally prescribed. These societal milestones, the attainment of which is entirely a function of chronological age, exert a large influence on crime through their social implications. The means by which socially-timed events actually affects changes in offending behavior is through their impact on psychosocial maturity (Rocque, Beckley, & Piquero, 2019) and the adoption of adult roles (e.g., Sampson & Laub, 1993)—social age.

Biological age becomes more salient in early adulthood. As the consequences of life choices accumulate and health behaviors (or their absence) become more ingrained into a lifestyle, biological age begins to exert influence on the offending behavior through physical limitations and disease morbidity. This process may become expedited if one's home environment is particularly caustic or, as we saw in chapter 2, one experiences a massively stressful life event (e.g., incarceration). The case of criminal justice contact could be particularly pernicious for biological aging as both the experience itself and the stigma that follows after jail/prison sentence are detrimental to health (e.g., Pearlin, 1989).

Thus, it may be useful to conceptualize age in multiple ways, each having a different impact on the criminal career. Chronological and social age impacts offending in adolescence and throughout adulthood. Biological age is influenced by early offending and, in turn, begins to influence the criminal career by impacting the desistance process in adulthood. As biological aging advances and morbidities accrue, persistent offenders (i.e., non-normative desisters) will

slowly begin the desistance process. Though much work is still needed, it is possible that biological aging might confound prior theories of cognitive transformations for explaining the desistance process. For example, if the ravages of age prevent an offender from committing crimes, it is possible that, through post hoc rationalization, they begin to outwardly espouse a lifestyle and a mindset that does not include antisocial behavior. This could cause the desistance process to appear to be related to cognitive transformation, while the true causal agent is aging.

We should keep in mind, though, that physical limitations on a persistent offender's ability to offend may not impact their antisocial attitudes. They may not be able to break into homes any longer, but they will still kick the dog (Piquero & Moffitt, 2005). Future studies on persistent offending/non-normative desisters will be needed to adjudicate these conflicting hypotheses.

The Limitations of Biological Age

Biological age represents the confluence on chronological and biological age; however, some consideration of the limitations of the modern measures of biological age are in order. First, measures of biological age are often developed in one population and deployed in another. For instance, the PhenoAge measure of biological age (Levine et al., 2018) was developed using a specific data source, the NHANES cohort. The estimates derived from the training sample are only as valid in a new sample to the extent that the original training data are representative of the new sample. This was not an issue for the HRS analysis, as the biomarker levels were highly concordant with the original NHANES data (see VBS documentation, 2016). But this concern could impact other studies that attempt to apply a measure like PhenoAge to other data sources.

Second, biological age must not be confused with maturation because, as was addressed in chapter 4, maturation represents the gradual approach toward some idealized state whereas

aging represents a gradual decline in integrity (i.e., away from an idealized state). Assessing biological age too early in the life course runs the risk of conflating it with maturation. The real utility of biological age is typically located in the latter part of the life course. This is because biological age is highly concordant with chronological age during the early part of the life course, only diverging as the consequences of life events and health behaviors accrue.

Finally, a practical limitation to measures of biological age is they require high-quality biomedical data. Even cross-sectional measures like PhenoAge (Levine et al., 2018) require sensitive information on almost a dozen biomarkers that are only acquired through a blood draw. Longitudinal measures, like Pace of Aging (Belsky et al., 2015a), require at least three repeated measures. Acquiring the requisite data is the largest hurdle, however, as the individual weights and algorithms needed to construct a given measure of biological age are usually made available by the developers. Despite the difficulties associated with collecting the requisite data, several large-scale longitudinal studies have begun to include items that can be used (e.g., The Add Health, CARDIA, HRS, and MIDUS studies). As discussed in chapter 2, however, many of these studies fall short in their sparse collection of criminal justice-related information. Recognition of these points helps identify opportunities for future work to expand on the data collection efforts that are currently available in hopes that more detailed insight can be gained into the relationship between age, aging, and the unfolding of the criminal career.

Conclusion

It is my hope that the work undertaken in this dissertation will ultimately lead to the integration of biological age into developmental/life-course theories of crime. Biological age is not a complete theoretical explanation of crime; it is a complementary factor that can help

scholars improve their understanding of the criminal career by illuminating the variegated ways aging influences offending, and vice versa.

Table 5.1. Glossary

Term	Definition	Source
Age	See "chronological age".	
Aging	The time-dependent functional decline that affects most living organisms.	<u>Lopez-Otin et al., 2013</u>
All-cause mortality	Mortality rate according to any/all causes.	
Biological age	The organ system integrity of an organism at a single point in time.	
Biological aging	The declining integrity of multiple organ systems.	<u>Belsky et al., 2015</u>
Biomarkers of aging	The biological parameters of an organism that either alone or in some multivariate composite will, in the absence of disease, better predict functional capability at some late age, than will chronological age.	<u>Baker & Sprott, 1988</u>
Cause-specific mortality	Mortality rate according to a specific cause.	<u>Last et al., 2001</u>
Chronological age	The number of years lived since birth.	
Healthspan	The period of life spent in good health, free from the chronic diseases and disabilities of aging.	<u>Kaeberlein, 2018</u>
Life expectancy	The average lifespan of a given species.	
Lifespan	The number of years lived from birth to death.	
Morbidity	Any departure, subjective or objective, from a state of physiological well-being.	<u>Last et al., 2001</u>
Mortality	Death. See also "all-cause mortality".	
Pace of aging	The rate of physiological deterioration.	<u>Belsky et al., 2015</u>

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Appendix

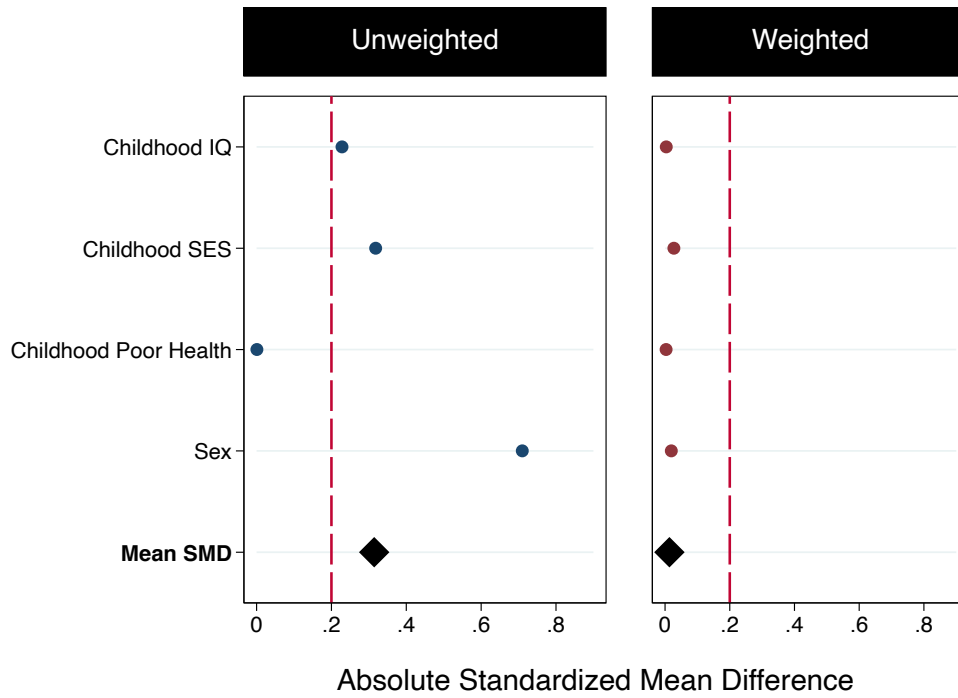


Figure A1. Absolute standardized mean differences across all covariates (CJ covariates removed), before and after weighting with inverse probability of treatment weights (see model 3, Table 3.6).

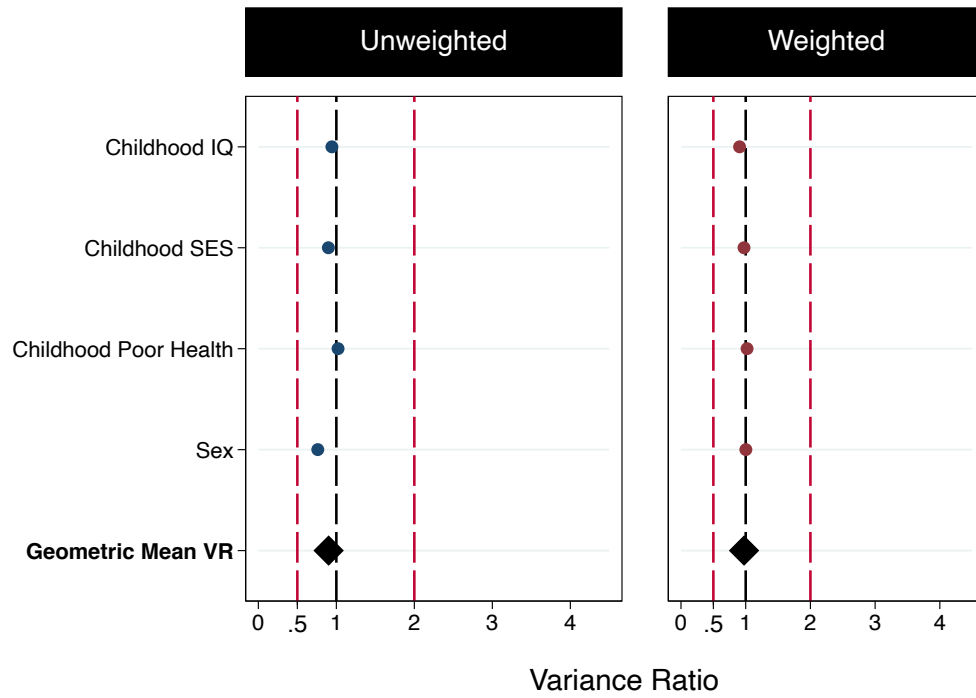


Figure A2. Variance ratios across all covariates (CJ covariates removed), before and after weighting with inverse probability of treatment weights (see model 3, Table 3.6).

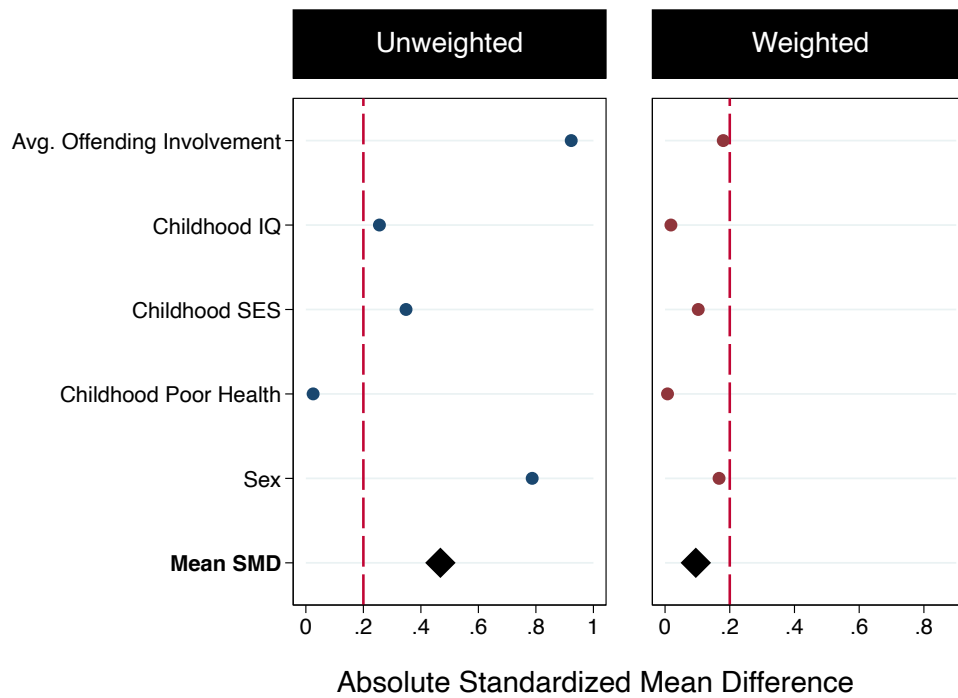


Figure A3. Absolute standardized mean differences across all covariates, before and after weighting with inverse probability of treatment weights. CJ variables were temporally corrected, with Average Offending Variety being restricted to between age 15 and 18 (Developmental Trajectories were dropped; see model 4, Table 3.6).

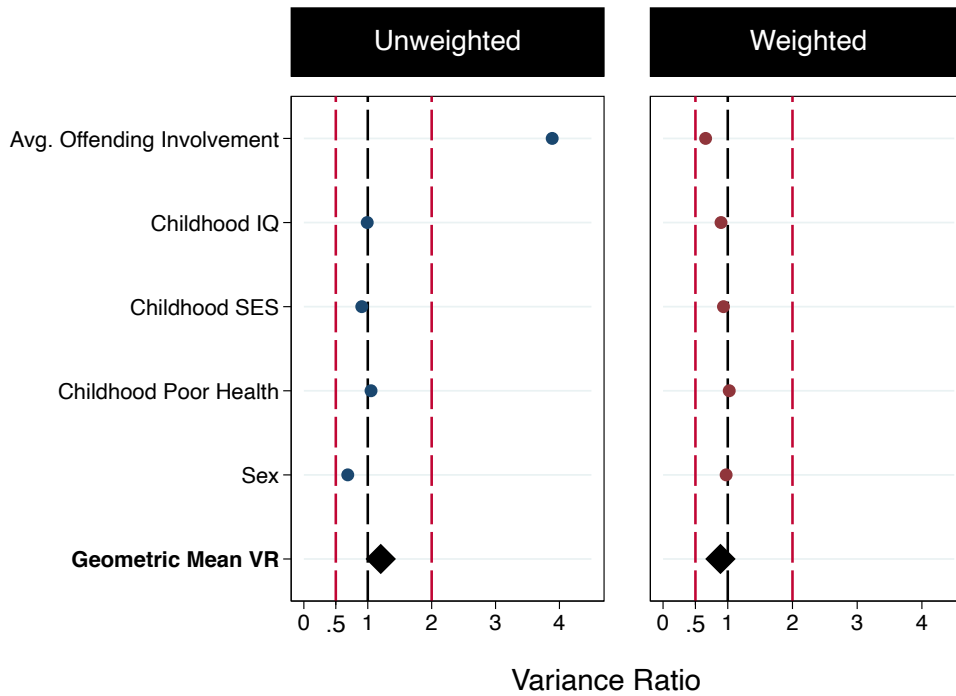


Figure A4. Variance ratios across all covariates, before and after weighting with inverse probability of treatment weights. CJ variables were temporally corrected, with Average Offending Variety being restricted to between age 15 and 18 (Developmental Trajectories were dropped; see model 4, Table 3.6).