

## Concept Paper–Dunedin Multidisciplinary Health and Development Study

**Provisional Paper Title:** Aging out: Criminal careers and biological aging

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**P.I. Sponsor:** Terrie Moffitt  
(if the proposing author is a student or colleague of an original PI)

**Today's Date:** 8/20/19

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Please describe your proposal in 2-3 pages with sufficient detail for helpful review.

### **Objective of the study:**

#### **Part I. Biological Age→Criminal Careers (“Aging Out”)**

According to criminological theory, the process of desisting from crime occurs for a variety of reasons (e.g., maturation, new social ties, and the exogenous effect of age). However, the role of physical health (as denoted by biological age) in the desistance process has not yet been studied. This study will explore how biological age affects the latter part of the criminal career.

We develop two competing hypotheses for how biological age might affect desistance:

**H1.** *Accelerated biological age will shorten the criminal career.*

Assuming that a criminal lifestyle requires physical strength to maintain, individuals who experience accelerated biological aging will be forced to abstain from physically demanding criminal acts.

**H2.** *Accelerated biological age will lengthen the criminal career.*

Assuming that the types of prosocial opportunities open to individuals with an antisocial/criminal background are more physical in nature (e.g., construction, road work), individuals who experience accelerated biological aging will be less able to take advantage of such opportunities and may continue in their antisocial/criminal lifestyles.

## Part II.

### Criminal Careers→Biological Age (“The Body Keeps Score”)

Criminal offending peaks in the early twenties and declines thereafter. This age-crime relationship has been observed across time, place, and crime type. The association is so pervasive that most criminological theories have proffered an explanation for it. For instance, the age-crime relationship has been said to exist because of 1) developmental processes that influence behavior (Moffitt, 1993), 2) socially-timed events that affect societal roles/relationships (Sampson & Laub, 1993), or 3) because of purely exogenous (i.e., unexplainable) effects of age on crime (Gottfredson & Hirschi, 1990). The latter explanation—that age is an exogenous factor—led Gottfredson and Hirschi, two of the field’s leading theorists, to claim that the impact of age on crime is “inexplicable.” This has become the dominant paradigm for many criminologists, such that age is viewed as a cause of the frequency and length of the criminal career (aging→crime).

In contrast to the criminological perspective, a voluminous literature conceptualizes age as the level of integrity of the organ systems within the body. This conception of age assumes that age is at least partially an *outcome* affected by one’s lifestyle and experiences. From this perspective, age is not exogenous but rather a part of the developmental/life-course process.

Juxtaposing these two viewpoints, we propose that one lifestyle component that is likely to affect aging is the criminal career. The criminal career is defined based on one’s *participation, frequency, seriousness, and length* of their involvement in criminal activity (Blumstein et al., 1986). We, thus, hypothesize that each of these components of the criminal career will influence aging. This perspective reverses the causal direction that is typically assumed in criminology (crime→aging).

Drawing from the criminal career paradigm in criminology, we develop four hypotheses:

- H3.** *Individuals high in antisocial behavior will experience accelerated biological aging (i.e., compared to those low in antisocial behavior), and individuals whose antisocial acts rise to the level of illegality will experience even more rapid biological aging.*

**H4.** *Individuals who report higher numbers of antisocial/criminal acts over a given time will experience accelerated biological aging compared to those who commit fewer such acts.*

**H5.** *Individuals who commit more serious antisocial/criminal acts (e.g., violence) will experience accelerated biological aging compared to those who commit less serious acts (e.g., property offenses).*

**H6.** *Individuals who begin committing antisocial/criminal acts early in life and persist until later life will experience accelerated biological aging compared to those individuals who begin offending later and cease offending earlier.*

**Data analysis methods:**

- Descriptive analyses (e.g., measures of central tendency)
- Bivariate analyses (e.g., correlation, *t*-tests)
- Multivariate analyses (e.g., multiple regression)
- Growth curve analyses

**Variables needed at which ages:**

Concept	Variable or Questionnaire Name	Phase
Offending	SRD questionnaire variety score matched for item content	P15 onward
	Official criminal record dated convictions	P15-38
	Conduct disorder measures scale	P7 to 26
Biological Age	Pace of aging	P26 to P45
	Methylation clocks <ul style="list-style-type: none"> <li>• Horvath</li> <li>• Hannum</li> <li>• Levine</li> </ul>	P26 to P45

\*Appropriate covariates to be decided upon later.

**Significance of the Study (for theory, research methods or clinical practice):**

The significance of this study will be to 1) inform criminological theory about the impact of offending on the body via biological aging, 2) demonstrate that biological age/aging can affect desistance process, and 3) introduce the criminal career/lifestyle as a salient predictor of biological age to the aging literature.

## **References cited:**

- Blumstein, A. (Ed.). (1986). *Criminal Careers and "Career Criminals"*, (Vol. 2). National Academies.
- Hirschi, T., & Gottfredson, M. R. (1983). Age and the explanation of crime. *American Journal of Sociology*, 89, 552–584.
- Moffitt, T. E. (1993). A developmental taxonomy. *Psychological Review*, 100(4), 674-701.
- Sampson, R. J., & Laub, J. H. (1993). *Crime in the making: Pathways and turning points through life*. Harvard University Press.
- Sugden, K., Hannon, E. J., Arseneault, L., Belsky, D. W., Broadbent, J. M., Corcoran, D. L., ... & Prinz, J. A. (2019). Establishing a generalized polyepigenetic biomarker for tobacco smoking. *Translational psychiatry*, 9(1), 92.

## Data Security Agreement

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Today's Date	8/21/2019

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Please initial your agreement

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PTT	My project is covered by Duke or Otago ethics committee OR I have /will obtain ethical approval from my home institution.
PTT	I will treat all data as "restricted" and store in a secure fashion. My computer or laptop is: a) encrypted (recommended programs are FileVault2 for Macs, and Bitlocker for Windows machines)
	b) password-protected c) configured to lock-out after 15 minutes of inactivity AND d) has an antivirus client installed as well as being patched regularly.
PTT	I will not "sync" the data to a mobile device.
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PTT	I will not share the data with anyone, including my students or other collaborators not specifically listed on this concept paper.
PTT	I will not post data online or submit the data file to a journal for them to post. <i>Some journals are now requesting the data file as part of the manuscript submission process. The Dunedin Study Members have not given informed consent for unrestricted open access, so we have a managed-access process. Speak to Terrie or Avshalom for strategies for achieving compliance with data-sharing policies of journals.</i>
PTT	I will delete all data files from my computer after the project is complete. Collaborators and trainees may not take a data file away from the office.  The data remains the property of the Study and cannot be used for further analyses without an approved concept paper for new analyses.

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