BIOGRAPHICAL SKETCH 2018

NAME: MOFFITT, TERRIE EDITH

eRA COMMONS USER NAME (agency login): TMOFFITT

POSITION TITLE: Nannerl O. Keohane University Prof (Duke), Prof of Social Behaviour & Development (KCL)

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE (if applicable)	Completion Date MM/YYYY	FIELD OF STUDY
University of North Carolina, Chapel Hill	BA	05/1977	Psychology
University of Southern California, Los Angeles	MA	05/1981	Psychology
University of Southern California, Los Angeles	PHD	05/1984	Clinical Psychology
UCLA School Medicine, Neuropsychiatric Inst.	Other training	1983	Clinical Intern Neuropsych
UCLA School Medicine, Neuropsychiatric Inst.	Postdoctoral Fellow	1984	Beh. Neuroscience, Geron.

A. PERSONAL STATEMENT

I am Associate Director of the Dunedin Longitudinal Study, which follows a 1972 birth cohort in New Zealand. I also founded the Environmental Risk Longitudinal Twin Study (E-Risk), which follows a 1994 birth cohort in the UK. Longitudinal research is an inherently horizon-scanning enterprise, and I relish forward planning, anticipating new trends, and asking new questions. My team has a good track record as first adopters of new research technologies; for example, we were among the first cohorts to collect DNA, in 1996, and among the first cohorts to use retinal imaging, in 2009 (reference 3). My work is about as inter-disciplinary as it gets; my team often generates discoveries when we make dataset combinations across previously unconnected disciplines. I am a licensed clinical psychologist, with specialization in neuropsychological assessment. I have a published record of collaboration with criminologists, economists, geneticists, epidemiologists, sociologists, demographers, gerontologists, statisticians, neuroscientists, medical scientists, even opthalmologists and dentists. The resulting products make an impact **(ISI H-index of 122 and Google scholar index of 165, Nov. 2017).** I bring to projects expertise in longitudinal methods, developmental theory, clinical psychopathology, neuropsychological assessment, and genomics in behavioral science. I also draw on expertise from a broad network including the Pepper Center for the Study of Aging, Center for Computational and Genomic Biology, Social Science Research Institute, Population Research Institute (all at Duke Univ.), UNC Center for Developmental Science, and King's College London's Institute of Psychiatry Centre for Social, Genetic, and Developmental Psychiatry. My team emphasizes representing our science accurately to the media, and promotes public understanding of science (see references 4,5; and www.altmetric.com). I have a track record of bringing in large-scale research projects on-time and on-budget. Over the past decade, I have provided data to over 100 senior investigators, at 49 Universities, in 14 countries. I also have an appetite for mentoring young scientists. I provide them with high-quality data with which they can train and develop independent ideas. I meet for 2 hours weekly with each graduate student or postdoc involved in my projects. I have trained 27 young scientists; have placed them in top-flight and influential positions; they have won more than 45 prestigious early-career awards and fellowships. My Hispanic and African American trainees have held NIH minority supplement fellowships. My team also welcomes undergraduates; 8 of them have co-authored publications in the past 5 years, and all have gone on to medical school or a PhD program. Web page with full publication list: www.moffittcaspi.com. Publication list: http://www.ncbi.nlm.nih.gov/sites/myncbi/collections/mybibliography/; http://orcid.org/0000-0002-8589-6760;

1. Moffitt TE, Caspi A, Rutter M, Silva PA. Sex differences in antisocial behaviour: Conduct disorder, delinquency, and violence in the Dunedin Longitudinal Study. Cambridge: University Press; 2001.
2. Moffitt TE, Caspi A, Rutter M. Strategy for investigating interactions between measured genes and measured environments. Arch Gen Psychiatry. 2005 May;62(5):473-81. PubMed PMID: 15867100.
3. Shalev I, Moffitt TE, Wong TY, Meier MH, Houts RM, et al. Retinal vessel caliber and lifelong neuropsychological functioning: retinal imaging as an investigative tool for cognitive epidemiology. Psychol Sci. 2013 Jul 1;24(7):1198-207. PubMed PMID: 23678508; PubMed Central PMCID: PMC3713191.
4. Moffitt TE, Poulton R, Caspi A. Lifelong impact of early self-control. The American Scientist. 2013

5. Predict My Future, 4-episode documentary, http://www.moffittcaspi.com/content/science-us, https://app.curiositystream.com/video/1268

B. POSITIONS AND HONORS

Positions and Employment

1985 - 2007	Assistant-, Associate- (1989), Full-Professor (1993), University of Wisconsin, Madison
1991 -	Associate Director, Dunedin Multidisciplinary Research Unit, University of Otago, New Zealand
1997 -	Professor, Social, Genetic, & Developmental Psychiatry Centre, Institute of Psychiatry, London
2007 -	Nannerl O. Keohane University Professor, Duke University
2010 -	Board of Trustees, Nuffield Foundation, UK

Other Current Experience and Professional Memberships

- National Advisory Council on Aging, US NIH
- American Journal of Psychiatry Associate Editors Board
- The Health and Retirement Study Data Monitoring Board, National Institute on Aging
- Chair, Jacobs Foundation Prize Jury, Switzerland
- Natl. Register of Health Service Providers #50256; NC Licensed Psychologist #4428

Honors

1993	Award for Early Career Contribution to Psychology, American Psychological Association
1999	Elected Fellow, Academy of Medical Sciences
2002	Wolfson Merit Award, The Royal Society
2003	Elected Fellow, American Society of Criminology
2003	Eleanor Maccoby Book Award, American Psychological Association
2004	Elected Fellow, British Academy
2005	Elected Fellow, both Academia Europaea, and American Psychopathological Association
2006	Distinguished Research Award (Child-Adolescent Psychopathology), Am Psychol Assoc
2007	The Stockholm Prize in Criminology, Sweden
2008	Elected Thorsten Sellin Fellow, American Academy of Political & Social Sciences
2008	Rema Lapouse Award, American Public Health Association
2008	Distinguished Scientific Contribution Award, Internatl. Soc. Study of Behavioral Development
2009	Elected Fellow, Association for Psychological Science
2009	The Klaus-Grawe Prize for Research in Clinical Psychology, Klaus-Grawe Foundation
2010	Ruane Prize for Outstanding Child and Adolescent Psychiatric Research, NARSAD
2010	Jacobs Research Prize for Productive Youth Development, Klaus J. Jacobs Foundation, Switzerland
2012	Top 10 Criminologists worldwide, Cohn & Farrington, Scholarly Influence in Criminology, NY: Nova.
2013	In the world's top 400 biomedical scientists, Boyack et al. European J. of Clinical Investigation.
2014	Honorary Doctorate, Basel University, Switzerland
2016	Distinguished Career Research Award from American Psychological Assn.
2016	Luminary Prize from the Avielle Foundation, www.aviellefoundation.org
2017	Honorary Doctorate, Catholic University of Leuven, Belgium
2018	Matilda White Riley Award from NIH.

C. Contributions to Science (in order from earliest in my career to most recent)

1. **Life-course Persistent versus Adolescence-limited antisocial behavior.** In a 1993 theoretical paper, now cited over 10,000 times, I proposed that young people engaging in antisocial behaviors can be characterized in a taxonomy of two distinct types: One type of antisocial behavior is called "life-course persistent" (LCP). It is a neurodevelopmental disorder afflicting primarily males, with very low prevalence in the population, genetic predisposition, adverse family environment, early childhood onset, and persistence of violent offending into midlife. The other type is called "adolescence limited" (AL). It affects females as well as males, is common, limited mainly to the adolescent developmental stage, and emerges in the

context of peer social relationships. This developmental taxonomy has had wide ranging influence in psychology, criminology, psychiatry, neuroscience and the law. It has been codified in the DSM diagnosis of conduct disorder, cited in Supreme Court decisions, and it won the coveted 2007 Stockholm Prize.

a. Moffitt TE. "Life-course-persistent" and "adolescence-limited" antisocial behavior: A developmental taxonomy. Psychological review. 1993; 100:674-701.
b. Moffitt TE, Caspi A. Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways, among males and females. Development & Psychopathology. 2001; 13:355-375.
c. Moffitt TE, Caspi A, Harrington H, Milne BJ. Males on the life-course-persistent and adolescence-limited antisocial pathways: follow-up at age 26 years. Dev Psychopathol. 2002 Winter;14(1):179-207. PubMed PMID: 11893092.
d. Moffitt, T.E. (2018). Male antisocial behavior in adolescence and beyond. Nature Human Behaviour. DOI 10.1038/s41562-018-0309-4. PubMed PMID: in process.

2. **Discoveries about mental disorder.** We were the first to report, in 2003, that over half of adult patients with psychiatric disorder have their first diagnosable disorder before age 15 (and 75% before age 18), suggesting that most of the burden of adult mental disorder could be prevented by effective treatment of young people. We were also first to report, in 1998, that the underlying structure of adult DSM mental disorders comprises two factors, internalizing and internalizing. Continuing our work on the structure of psychopathology, we have confirmed that all adult psychiatric symptoms fit onto a single dimension of severity with symptoms of thought disorder at the extreme end,"p". We also initially reported that the lifetime prevalence of anxiety, depression, and substance dependence is at least double what the mental-health community has been led to believe by retrospective surveys. People markedly underreport the amount of mental illness they've suffered when they recall their history in interviews years after the fact. We demonstrated this by repeatedly assessing for mental disorders while following cohorts forward. These findings have all been replicated multiple times. At the least, our finding that most of us will experience an episode of mental disorder if we live long enough should reduce stigma against mental illness.
   a. Krueger RF, Caspi A, Moffitt TE, Silva PA. The structure and stability of common mental disorders (DSM-III-R): a longitudinal-epidemiological study. J Abnorm Psychol. 1998 May;107(2):216-27. PMID: 9604551.
   b. Kim-Cohen J, Caspi A, Moffitt TE, Harrington H, Milne BJ, et al. Prior juvenile diagnoses in adults with mental disorder: developmental follow-back of a prospective-longitudinal cohort. Arch Gen Psychiatry. 2003 Jul;60(7):709-17. PMID: 12860775.
   c. Moffitt TE, Caspi A, Taylor A, Kokaua J, Milne BJ, et al. How common are common mental disorders? Evidence that lifetime prevalence rates are doubled by prospective versus retrospective ascertainment. Psychol Med. 2010 Jun;40(6):899-909. PubMed PMID: 19719899; PMCID: PMC3572710.
   d. Caspi A, Houts RM, Belsky DW, Goldman-Mellor SJ, Harrington H, et al. The p Factor: One General Psychopathology Factor in the Structure of Psychiatric Disorders?. Clin Psychol Sci. 2014 Mar;2(2):119-137. PubMed PMID: 25360393; PMCID: PMC4209412.

3. **The importance of self-control for health, wealth, and life success**. One of our projects that has attracted the most attention from policy makers is about the importance of self-control skills mastered in childhood for success in all aspects of adult life. We reported that childhood self-control is more important than socioeconomic status (SES) or IQ for adults' physical health, addiction, crime, suicidality, wealth, life satisfaction, and parenting of the next generation. The findings have been viewed as lending support to the movement for quality early-childhood education. Our work on impulse-control goes back to a series of our highly–cited papers in the 1990's on the role of self-control in antisocial development. Recently we reported that people who don't take care of their money don't take care of their health. Using a unique data source (our subjects' credit ratings), we found a strong connection between financial health and cardiovascular health, unaccounted for by income. This connection was explained by self-control in early childhood. We further showed that a brief measure of conscientiousness can identify which now-healthy young-adult patients will develop health problems in the future. This line of translational research suggests that a 5-item questionnaire given in the waiting room at the GP's office can tell doctors which patients need more motivational counseling to promote healthy behaviors and prevent later onset of disease.
   a. White J, Moffitt TE, Caspi A, Jeglum-Bartusch D, Needles D, et al. Measuring impulsivity and examining its relation to delinquency. Journal of abnormal psychology. 1994; 103:192-205. PMID: 8040489

b. Moffitt TE, Arseneault L, Belsky D, Dickson N, Hancox RJ, et al. A gradient of childhood self-control predicts health, wealth, and public safety. Proc Natl Acad Sci U S A. 2011 Feb 15;108(7):2693-8. PubMed PMID: 21262822; PubMed Central PMCID: PMC3041102

c. Caspi A, R.M. Houts, D.W. Belsky, HL Harrington, S. Hogan, S. Ramrakha, R. Poulton, T.E. **Moffitt** (2017). Childhood forecasting of a small segment of the population with large economic burden. Nature Human Behaviour. 1, 0005. PubMed PMID: 28706997; PubMed Central PMCID: PMC5505663

d. Israel S, Caspi A, Belsky DW, Harrington H, Hogan S, et al. Credit scores, cardiovascular disease risk, and human capital. Proc Natl Acad Sci U S A. 2014 Dec 2;111(48):17087-92. PubMed PMID: 25404329; PubMed Central PMCID: PMC4260546.

4. **Gene-environment interaction (GxE).** Genetic endowment contributes to psychiatric disorders by shaping how people respond to environmental pathogens. Of course, this idea was around for many years, but it remained an abstract statistical concept in psychiatry until 2002/2003 when we provided the first empirical evidence of GxE interactions, in conduct disorder and depression. The work has not been uncontroversial (although oft-replicated), but lost in the debate over the use of candidate genes is the transformative nature of our discoveries. First, geneticists can leverage information about environmental exposures to identify genetic effects. Second, neuroscientists who are looking for gene-to-brain connections can uncover these connections by experimentally manipulating participants' exposure to relevant environmental stimuli. Our GxE interaction papers are also proving to be influential in the public's understanding of genetic science. These reports vividly contradict the public's pervasive belief in genetic determinism, by showing how genetic effects on health and behavior often depend on lifestyle factors under human control. This work launched a new era in the study of how gene-environment interplay causes human behavior, and was recognized with prestigious prizes from NARSAD, the Sackler Foundation, and the Jacobs Foundation.
   a. Caspi A, McClay J, Moffitt TE, Mill J, Martin J, et al. Role of genotype in the cycle of violence in maltreated children. Science. 2002 Aug 2;297(5582):851-4. PubMed PMID: 12161658.

   b. Caspi A, Sugden K, Moffitt TE, Taylor A, Craig IW, et al. Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. Science. 2003 Jul 18;301(5631):386-9. PubMed PMID: 12869766.

   c. Caspi A, Moffitt TE. Gene-environment interactions in psychiatry: joining forces with neuroscience. Nat Rev Neurosci. 2006 Jul;7(7):583-90. PubMed PMID: 16791147.

   d. Caspi A, Hariri AR, Holmes A, Uher R, Moffitt TE. Genetic sensitivity to the environment: the case of the serotonin transporter gene and its implications for studying complex diseases and traits. Am J Psychiatry. 2010 May;167(5):509-27. PubMed PMID: 20231323; PubMed Central PMCID: PMC2943341.

5. **Aging in young people, an opportunity for prevention.** I am now leading the Dunedin Study into the study of aging. To prevent onset of age-related diseases and physical and cognitive decline, interventions to slow human aging and extend health span must eventually be applied to people while they are still young and healthy, before organ damage sets in. Yet most human aging research examines older adults, many with chronic disease, and little is known about aging in healthy young humans. This knowledge gap is a barrier to extending health span. We have put forward the case that gero-science should invest in researching processes of aging in young adults. We developed a measurement model of the pace of aging in a young-adult birth cohort by using repeated waves of biomarkers collected across the third and fourth decades to quantify the pace of coordinated physiological deterioration across multiple organ systems (e.g., pulmonary, periodontal, cardiovascular, renal, hepatic, metabolic, and immune function). Our findings provided proof of principle that it is possible to quantify individual variation in the pace of aging in young adults who are still free of age-related diseases. Using our measurement of the pace of aging, we are pinpointing factors that slow or speed the pace of aging, and factors that characterize slow-aging young adults. We compared 11 purported measures of aging (pace of aging, bioage, genomic methylation clocks, telomeres), a comparison which revealed that these measures are virtually uncorrelated and therefore cannot be measuring the same thing. We are working toward applying the pace of aging as an outcome measure in preventive clinical trials of anti-aging therapies.

a. Moffitt TE, Belsky DW, Danese A, Poulton R, Caspi A. (2017). The longitudinal study of aging in human young adults: Knowledge gaps and research agenda. J of Gerontology: Biological Sciences and Medical Sciences. PubMed PMID:28087676; PubMed Central PMCID: PMC5233916

b. Belsky DW, Caspi A, et al. and Moffitt TE. (2015). Quantification of biological aging in young adults. Proceedings of the National Academy of Sciences. 77, 601-617. PubMed Central PMCID: PMC45522793

c. Belsky DW, Caspi A, et al. and Moffitt TE. (2017). Impact of early personal history characteristics on the Pace of Aging: Implications for clinical trials of therapies to slow aging and extend healthspan. Aging Cell. PubMed PMID: 28401731; PubMed Central PMCID: PMC5506399

d. Belsky DW, Moffitt TE, et al. (2017). Telomere, epigenetic clock, and biomarker-composite quantifications of biological aging: Do they measure the same thing? No. American J of Epidemiology. Pubmed PMID 29149257

D. RESEARCH SUPPORT

Ongoing Research Support

2017/01/01-2021/12/31 P005918, UK Medical Research Council
MOFFITT, TERRIE Edith (PI)
Midlife Pace of Aging in the Dunedin Study, 2017-2021
This project supports data collection in the Dunedin longitudinal study.

2009/03/01-2020/03/31 R01 AG032282, National Institute on Aging (NIA)
MOFFITT, TERRIE EDITH (PI)
Aging in 1000 healthy young adults: Phase 45 of the Dunedin Study 2016-2020
Aims to track biological aging in young to midlife adulthood, and study its social and genomic correlates.

2015/07/15-2020/02/28 R01AG049789 National Institute on Aging, (NIA)
MOFFITT, TERRIE EDITH and HARIRI, AHMAD (Co-PIs)
Neural signatures of healthy and unhealthy aging: The Dunedin Study.
Aims to collect and study brain imaging data in the Dunedin cohort.