The Reproductive Demography of Suicide

Maximilian Stiefel[a]

[a]University of California, Santa Barbara

Introduction

Suicide is one of the most traumatic and least scientifically understood mortality outcomes. It results from a combination of health and behavioral factors that compound and interact over the life course. Suicide rates in the U.S. are on a pernicious rise within some demographic groups, and the cost bore to families and communities is immeasurable. To aid in efforts towards suicide risk reduction, this project has three aims: 1) the role neighborhood quality plays in suicide risk, 2) the interacting biological and environmental influence on suicide risk, and 3) the reciprocal relationship of suicide risk and reproductive demography. Our presentation at EPC will concern the last aim: the reproductive demography of suicide.

The primary exposure-outcome relationship we study concerns how poor neighborhood quality exposes individuals to a variety of compounding adversities that increase stress load, reduce healthful behaviors, and limit access to health-promoting resources. Individuals who experience greater adversity from poor neighborhood quality may exhibit increased risk for mental health complications, compromised social networks, and constrained socioeconomic opportunities. These forces work together to reduce health status. Biological disposition moderates the relationship between neighborhood adversity and health outcome. Different health outcomes produce variation in reproductive histories, i.e., disparities by health status as well as systematic gaps in preferred and realized reproductive outcomes. We therefore hypothesize that individuals who experience greater neighborhood adversity are at a higher risk for being suicide afflicted, this relationship is moderated by the familial standardized incidence ratio (FSIR, a proxy for genotypic risk for a health outcome), and individuals at high-risk for suicide, as measured with their FSIR or by being suicide afflicted, exhibit distinct patterning of life history events, i.e., births, marriages, and divorce, across the life course.

To do this, we first compare fertility histories (quantity, life course timing, and spacing of offspring) for the suicide afflicted and high-risk populations against the general population. High-risk status is determined by a Familial Standardized Incidence Ratio (FSIR), a phenotypic proxy for genetic risk. High FSIR (high-risk) for suicide individuals have compromised development of the 'capital trinity', which comprises human, social, and psychological capital. This occurs through mental health disorder and future discounting, among other mechan-

CONTACT Email: maximilian.stiefel@gmail.com

ics, and is moderated by social, economic, and place-based context. These forms of capital represent the individual features of 'what I know', 'who I know', and 'who I am', respectively. Good mental health status may have advantages for developing each type of capital at different life course stages. Similarly, those who discount future scenarios may choose to limit investment in capital trinity development. A limited capital trinity reduces likelihood for mate matching and increases likelihood of unplanned pregnancies.

Second, we are reciprocally interested in how fertility histories influence suicide likelihoods. The population with children, holding FSIR constant, may exhibit increased time to and/or decreased occurrence of suicide. Furthermore, there may be life course and fertility history composition effects. Having children at later as opposed to earlier ages could reduce suicide risk, there may be an absolute or threshold effect of child quantity on suicide risk, and increased birth spacing may allow mothers to adapt to child rearing and recover more effectively from postpartum depression if they are in the high-risk group. Many of these are hypothetical relationships based on adjacent literatures that we plan to test in the reproductive demography of suicide context. We hypothesize that 1) suicide afflicted and high-risk individuals exhibit lower marriage rates, higher divorce rates, lower lifetime reproductive success, and lower age at first birth, 2) high-risk individuals who have children experience a protective effect and exhibit increased time to and decreased occurrence of suicide, 3) birth interval length negatively correlates with suicide risk, and 4) not all child quantities are the same–low and high quantities increase suicide risk.

Literature Review

Mental Health as a Demographic Determinant

Mental health status is an important determinant for population-level mortality, fertility, and migration rates. The diversity of conditions under the umbrella of mental health, or psychopathology, includes depression, anxiety, and schizophrenia. These disorders affect both behavior and physiology. Borrowing from the proximate determinants framework proposed by Bongaarts (1978), we understand that behavior and biology (physiology) affect fertility. A prominent paper recently demonstrated that substance use disorders (SUD) and suicide can be responsible for unexpected and negative shifts in mortality schedules (Case and Deaton 2015). Mental health outcomes are the result of a dynamic process involving many factors such as behavior (exercise, sleep, etc), biology (genetic risk, stress, etc.), social support (families, employment, etc.), and environmental context (various types of pollution, crime, etc) (Allen et al. 2014; Bovier, Chamot, and Perneger 2004; Lorenc et al. 2012; Consortium et al. 2009; Krabbendam and Van Os 2005; Lichtenstein et al. 2009).

A useful definition for mental health comes from the World Health Organization, "a state of well-being in which an individual realizes his or her own abilities, can cope with the normal stresses of life, can work productively, and is able to make a contribution to his or her community" (WHO 2010). Several theoretical frames help us understand the effects of mental health on demographic outcomes. The first is human capital theory, popularized by

Becker (1964), which provides an outline of the types of somatic assets humans necessarily develop over their lifespan for accomplishing life course transitions, among many other things. Research has shown that mental health problems limit human capital accumulation, particularly when problems begin in early life (Currie and Stabile 2006; Currie and Stabile 2007 Almedom 2005). Another frame is that of social capital theory, which has a long history stretching back to Durkheim (Portes 1998). Social capital theory posits that resources, such as social networks–the relationships and connections therein–and social cohesion, provide for greater individual and societal success. Social capital occurs through many mechanisms, one of which is social learning. This concept provides a framework for how and what people learn, which is constrained by content and transmission biases (Sear 2015). Coleman (2000) gives evidence to suggest that social capital influences human capital accumulation, which could function through mental health (McKenzie and Harpham 2006). There is also evidence that mental health problems affect social cohesion and the creation of effective social networks (De Silva et al. 2005). Furthermore, a newer concept has been developed–positive psychological capital–that comprises factors present in individuals with good mental health such as optimism and resiliency. Considerable agreement exists that these three types of capital–human, social, and psychological–all work in a dynamic way to influence individual and societal success (Luthans and Youssef 2004).

Reproductive Demography of Suicide

The following framework assumes that suicide results from a combination of mental health status and other non-observable factors. Few studies concern life event patterning, such as age at first birth (AFB) and lifetime reproductive success (LRS), for individuals with mental health disorders. However, demographic and health researchers are thoroughly interested in reproductive health, specifically how reproductive outcomes affect mental health (Brockington and Guedeney 1999; Fergusson, John Horwood, and Ridder 2006; Fisher, Mello, Izutsu, et al. 2009; Organization and Society 2009). McGrath et al. (1999) examined differences in fertility and fecundity for patients with psychosis. They found that individuals with mental health disorders exhibit lower LRS and men with non-affective disorders displayed the biggest differences. The authors neglect to mention any potential pathways for decreased physiological functioning through stress exposure that might affect fecundity–the biological ability to have children. There are many ways physiological stress could affect fecundity, such as certain stress-related hormones affecting reproductive organ functioning. This has been a popular area of inquiry for some time (Chrousos 2009), especially for non-human species (Greenberg and Wingfield 1987). The authors do highlight key ways mate selection capabilities might differ for the study population, such as schizophrenic males having impaired social skills. Another article reviewed the impacts of severe mental health disorder on reproductive health (Matevosyan 2009), however it only focused on 'adverse' outcomes such as sexually transmitted infections, female cancer, unwanted pregnancies, and sexual dysfunction. The authors found evidence that individuals with mental health disorder have more lifetime sex partners, low contraceptive usage, higher rates of unwanted pregnancies, and are at high risk

for sexually transmitted infections.

An evolutionary approach to understanding fertility outcomes presents the capital trinity as an important determinant. Phenotypic variation, the range of observable physiologic and behavioral characteristics present in a population, is the result of natural selection, an evolutionary process guided by fitness maximization, i.e., passing on as much genetic material as possible (by producing surviving offspring). These characteristics give relative advantage over peers by increasing energy allocation efficiency, e.g., through improved immune response, which allows for longer lives and more children. Living longer is not just ancillary to having more children; longevity bestows other advantages on fitness. The grandmother hypothesis and pooled energy budget theory propose two ways social capital increases fitness. Evolutionary science suggests individuals achieve higher fitness (increased fertility) through somatic and extra-somatic investments, i.e., personal and material, respectively (Sear 2015). Better mental health increases our ability to make these kinds of investments.

From a demographic and economic perspective, marriage behavior has changed over the past few centuries. Although the changes have not been asymptotic, they tend towards some general directions: lower rates of marriage, higher rates of divorce, higher ages at first marriage, cohabitation as an alternative to marriage, among others. Classic theories on marriage markets describe reasons for union formation as production/consumption complementarities and risk pooling. Changes in marriage behavior have occurred for several reasons, as described by Stevenson and Wolfers (2007). These mechanistic factors include birth control, household technology, wage and legal marriage structures, and a shift in the marriage market matching function (greater opportunities to meet mates, which creates a positive feedback for divorce rates). Mental health problems decrease competitiveness in the marriage market, both during the transition to adulthood, which comprises life course transitions (e.g., leaving the nest) and for developmental task completion (e.g., autonomy) (Brazil and Clark 2017). A "failure to launch" from mental health problems might lead to lower lifetime reproductive success.

From an empirical lens, we can see the capital trinity working to produce disparate life course trajectories across racial/ethnic groups (Mulia et al. 2017). Across variation in marriage behavior there appears to be a socioeconomic/educational gradient. Lundberg, Pollak, and Stearns (2016) demonstrate that risk for marriage positively correlates with human capital, which is a function of mental health. There is therefore much more inquiry to undertake surrounding mental health and fertility: what are the mediating factors that lead to differences in fertility patterning between non-diagnosed and diagnosed individuals, how do we attribute weight to each of those factors, does access to mental health services moderate the effect of diagnosis on fertility, and many others. Despite numerous studies looking at the adjacent and reciprocal relationships, there currently exists a large gap in the literature on fertility behavior and outcomes of individuals with mental health disorder.

Data

We plan to utilize the Utah Population Database (UPDB) for this research. UPDB has proven promising for studies concerned with neighborhood effects and health disparities (Smith et al. 2011; Zick et al. 2013; Fan et al. 2014). The UPDB is one of the world's richest sources of linked population-based information for demographic, genetic, and epidemiological studies. UPDB has supported numerous biomedical investigations in large part because of its size, inclusion of multigenerational pedigrees, and linkages to numerous data sources. The UPDB now contains data on over 9 million individuals from the late 18th century to the present.

UPDB represents Utah's population that appear in administrative records. The holdings of the data grow due to longstanding efforts to update records as they become available including statewide birth and death certificates, hospitalizations, ambulatory surgeries, and driver licenses. UPDB creates and maintains links between the database and the medical records held by the two largest healthcare providers in Utah as well as Medicare claims. The multigenerational pedigrees representing Utah's founders and their descendants were constructed based on data provided by the Genealogical Society of Utah (GSU). Pedigrees spanning the past 80 years have been expanded extensively based on vital records and, together with the GSU data, form the basis of the deep genealogical structure of the UPDB. Studies using UPDB data have been approved by the University of Utah's Resource for Genetic and Epidemiologic Research and its Institutional Review Board (*Utah Population Database* 2017).

According to the UPDB Limited Query Tool, there were 30,633 suicide since 1900 in Utah across 13 Residence Districts, an average of 2,357 suicides per geographic unit (SPGU), which comprise 28 of the 29 Utah Counties (1,049 avg. SPGU). Utah contains 588 Census Tracts (52 avg. SPGU, 78 max, 15 min) and 1,690 Census Block Groups (18 avg. SPGU, 27 max, 6 min). Descriptive statistics of total population counts for CT and CBG from the 2017 American Community Survey 5-Year Estimates are 27,035 max, 5,084 avg., 136 min and 18,752 max, 1772 avg., 110 min, respectively.

We include individual records from the UPDB for those over 18 and alive during a time window that varies by Census Tract of residence and observation, since individuals may move among Census Tracts. We base this 'inclusion' time window on a maximum allowable amount of neighborhood change. The purpose of this is to allow any current, static neighborhood quality measurement to accurately estimate exposure over retrospective time. Variables representing the built environment are the best example of these types of measurements. The first step in constructing the inclusion time window is to calculate the degree of neighborhood change (DNC) distribution, which is the average percentage change by Census Tract in median household income (chained to dollars for the year we gathered neighborhood quality measurements) since 1900. We constrain the inclusion time window for each Census Tract from present back to the year reaching the lower bound of the first standard deviation for the DNC distribution. The inclusion time window therefore contains Census Tracts for as long as they have not exceeded the DNC threshold since the year we gathered any non-longitudinal neighborhood quality measurements. We include all individuals who have lived their whole life continuously in Census Tracts not exceeding the DNC threshold. We exclude individuals

if they ever lived in a Census Tract that exceeded the DNC threshold. For example, included individual A lived in three Census Tracts, all of which fell under the DNC threshold. Excluded individual B lived in three Census Tracts, the least recent of which fell under the DNC threshold, however the second most recent exceeded the DNC threshold.

We use a case-control design for analysis. Cases comprise the suicide afflicted and controls will be matched to each case based on the following set of observable socio-demographic and risk factor characteristics: age, sex, race/ethnicity, socioeconomic status, personal experience with a suicide afflicted individual, socio-geographic social isolation as measured by population density, and religious affiliation. This approach will limit the confounding effect of unobservable risk factors: prior suicide attempts, drug and alcohol misuse, mental health disorder, access to lethal means, behavioral social isolation, chronic disease and disability, and lack of access to behavioral care. We construct pedigrees for each individual in the sample using the oldest records contained within UPDB. We necessitate data on residential mobility, life histories, and socioeconomic/demographic characteristics to include as controls and for making among-group comparisons in our statistical modeling. The following provides a summary of UPDB variables that we plan to use:

Residential mobility

– Driver license (height, weight, gender, address). Provides place of residence (1 million geocoded addresses with Eastings, Northings, and census tracts and blocks; connected to census designated neighborhood characteristics)

Life histories

– Vital records (birth, death, ICD cause of death, marriage, divorce)
– Employment (industry, occupation)

Methods

Familial Standardized Incidence Ratios

Construct familial standardized incidence ratios (FSIR) for suicide for specific aim #2. Kinship Analysis Tools (University of Utah, Salt Lake City, Utah) software was developed to estimate the magnitude of familial risk using the FSIR based on this database (Kerber 1995). Use of the FSIR requires a large genealogic database and a linked population-based disease registry. The FSIR is based on the ratio of the observed to the expected incidence of a disease occurring in a pedigree, multiplied by the kinship-weighted coefficient (Kerber 1995). This statistic measures the excess relative risk attributable to familial factors. The UPDB is population based, and the linkage to the UPDB meets the criteria for analysis using the FSIR. We assign a FSIR to each individual in our sample. This score typically only contains detail on suicide occurrence rates within a pedigree. However, since we are interested in life course timing of suicide events, we will also construct an age-at-death adjusted FSIR, standardized

for period and cohort life expectancy. This allows us to account for individuals who were unable to finish their reproductive careers.

Formal Demographic Analysis of Suicide

Compare fertility histories–1) quantity, 2) timing, and 3) spacing of offspring–for suicide afflicted and high-risk (measured by FSIR) groups against the general population for Specific Aim #3. We employ the following models: 1) Poisson to estimate child quantity as count data, 2) Cox proportional-hazard for ages at first and last births, and 3) GLMs with restricted cubic splines (or another non-parametric transformation) to analyze inter-pregnancy intervals (birth to start of next pregnancy) since the hypothetical relationship of mental health status and birth spacing is multi-directional. We also present descriptive statistics of marriage, divorce, and employment rates for FSIR quantiles and suicide affliction, further stratified by socioeconomic status. Then we model the impacts of fertility histories on suicide risk: time to and occurrence of suicide. Model selection for each of these is as follows: 1) Cox-proportional hazard for time to suicide, and 2) Logistic GLMs for occurrence of suicide (to produce risk ratios). We construct these models and the explanatory variables of interest based on literature suggesting that time to suicide and occurrence of suicide have positive and negative relationships, respectively, for the following fertility history characteristics: ever had a child (binary), developmental stage of first birth (ordinal categories), child quantity (continuous), child quantity (ordinal quantiles), and birth intervals (ordinal categories and continuous, the former used to align with prior studies). We include NQI as an interaction term in our models to capture any moderating effects.

References

Acevedo-Garcia, Dolores et al. (2003). "Future Directions in Residential Segregation and Health Research: A Multilevel Approach". In: *American Journal of Public Health* 93.2.

Allen, Jessica et al. (2014). "Social determinants of mental health". In: *International Review of Psychiatry* 26.4, pp. 392–407.

Almedom, Astier M (2005). "Social capital and mental health: An interdisciplinary review of primary evidence". In: *Social science & medicine* 61.5, pp. 943–964.

Becker, Gary S (1964). "Human capital theory". In: *Columbia, New York* 1964.

Beckie, Theresa M. (2012). "A Systematic Review of Allostatic Load, Health, and Health Disparities". In: *Biological Research for Nursing* 4.14, pp. 311–346.

Berger, Maximus and Zoltan Sarnyai (2014). ""More than skin deep": stress neurobiology and mental health consequences of racial discrimination". In: *Stress* 18.1, pp. 1–10.

Berger, Shelley L et al. (2009). "An operational definition of epigenetics". In: *Genes & development* 23.7, pp. 781–783.

Bongaarts, John (1978). "A Framework for Analyzing the Proximate Determinants of Fertility". In: *Population and Development Review* 4.1, pp. 105–132.

Bovier, Patrick A, Eric Chamot, and Thomas V Perneger (2004). “Perceived stress, internal resources, and social support as determinants of mental health among young adults”. In: *Quality of Life Research* 13.1, pp. 161–170.

Brazil, Noli and William AV Clark (2017). “Individual mental health, life course events and dynamic neighbourhood change during the transition to adulthood”. In: *Health & place* 45, pp. 99–109.

Brockington, Ian and Antoine Guedeney (1999). *Motherhood and mental health*.

Case, Anne and Angus Deaton (2015). “Rising morbidity and mortality in midlife among white non-Hispanic Americans in the 21st century”. In: *Proceedings of the National Academy of Sciences*.

Chrousos, George P (2009). “Stress and disorders of the stress system”. In: *Nature reviews endocrinology* 5.7, p. 374.

Clark, David (1989). *Urban Decline*. Routledge.

Coleman, James S (2000). “Social capital in the creation of human capital”. In: *Knowledge and social capital*. Elsevier, pp. 17–41.

Colen, Cynthia G. (2011). “Addressing Racial Disparities In Health Using Life Course Perspectives: Toward A Constructive Criticism”. In: *Du Bois Review* 8.1, pp. 79–94.

Consortium, International Schizophrenia et al. (2009). “Common polygenic variation contributes to risk of schizophrenia and bipolar disorder”. In: *Nature* 460.7256, p. 748.

Currie, Janet and Mark Stabile (2006). “Child mental health and human capital accumulation: the case of ADHD”. In: *Journal of health economics* 25.6, pp. 1094–1118.

– (2007). “Mental health in childhood and human capital”. In: *The problems of disadvantaged youth: An economic perspective*. University of Chicago Press, pp. 115–148.

De Silva, Mary J et al. (2005). “Social capital and mental illness: a systematic review”. In: *Journal of Epidemiology & Community Health* 59.8, pp. 619–627.

Diez Roux, Ana V. and Christina Mair (2010). “Neighborhoods and health”. In: *Annals of the New York Academy of Sciences* 1186.1, pp. 125–145.

Ellis, Bruce J and W Thomas Boyce (2011). “Differential susceptibility to the environment: Toward an understanding of sensitivity to developmental experiences and context”. In: *Development and psychopathology* 23.1, pp. 1–5.

Epel, Elissa S et al. (2004). “Accelerated telomere shortening in response to life stress”. In: *Proceedings of the National Academy of Sciences of the United States of America* 101.49, pp. 17312–17315.

Fan, JX et al. (2014). “Geographic scale matters in detecting the relationship between neighbourhood food environments and obesity risk: an analysis of driver license records in Salt Lake County, Utah”. In: *BMJ Open* 4.8.

Fergusson, David M, L John Horwood, and Elizabeth M Ridder (2006). “Abortion in young women and subsequent mental health”. In: *Journal of Child Psychology and Psychiatry* 47.1, pp. 16–24.

Fisher, Jane, M Cabral de Mello, T Izutsu, et al. (2009). “Mental health aspects of women’s reproductive health”. In: *Geneva: World Health Organization*.

Ganzel, Barbara L., Pamela A. Morris, and Elaine Wethington (2010). “Allostasis and the Human Brain: Integrating Models of Stress From the Social and Life Sciences”. In: *Psychological Review* 117.1, pp. 134–174.

Greenberg, Neil and John C Wingfield (1987). “Stress and reproduction: reciprocal relationships”. In: *Hormones and reproduction in fishes, amphibians, and reptiles*. Springer, pp. 461–503.

Karriker-Jaffe, Katherine J (2013). “Neighborhood socioeconomic status and substance use by U.S. adults”. In: *Drug Alcohol Depend* 133.1, pp. 212–21.

Karriker-Jaffe, Katherine J, Huiguo Liu, and Renee M Johnson (2016). “Racial/ethnic differences in associations between neighborhood socioeconomic status, distress, and smoking among U.S. adults”. In: *J Ethn Subst Abuse* 15.1, pp. 73–91.

Karriker-Jaffe, Katherine J, HuiGuo Liu, and Lauren M Kaplan (2016). “Understanding Associations Between Neighborhood Socioeconomic Status and Negative Consequences of Drinking: a Moderated Mediation Analysis”. In: *Prev Sci* 17.4, pp. 513–24.

Kerber, Richard A (1995). “Method for calculating risk associated with family history of a disease”. In: *Genetic epidemiology* 12.3, pp. 291–301.

Krabbendam, Lydia and Jim Van Os (2005). “Schizophrenia and urbanicity: a major environmental influence—conditional on genetic risk”. In: *Schizophrenia bulletin* 31.4, pp. 795–799.

Lichtenstein, Paul et al. (2009). “Common genetic determinants of schizophrenia and bipolar disorder in Swedish families: a population-based study”. In: *The Lancet* 373.9659, pp. 234–239.

Lorenc, Theo et al. (2012). “Crime, fear of crime, environment, and mental health and well-being: mapping review of theories and causal pathways”. In: *Health & place* 18.4, pp. 757–765.

Lundberg, Shelly, Robert A Pollak, and Jenna Stearns (2016). “Family inequality: Diverging patterns in marriage, cohabitation, and childbearing”. In: *Journal of Economic Perspectives* 30.2, pp. 79–102.

Luthans, Fred and Carolyn M Youssef (2004). “Human, social, and now positive psychological capital management: Investing in people for competitive advantage”. In:

Matevosyan, Naira R. (2009). “Reproductive Health in Women with Serious Mental Illnesses: A Review”. In: *Sexuality and Disability* 27.2, pp. 109–118.

McEwen, Bruce S. and Peter J. Gianaros (2010). “Central role of the brain in stress and adaptation: Links to socioeconomic status, health, and disease”. In: *Annals Of The New York Academy Of Sciences, The Biology Of Disadvantage* 1186, pp. 190–222.

McGrath, John J et al. (1999). “The fertility and fecundity of patients with psychoses”. In: *Acta Psychiatrica Scandinavica* 99.6, pp. 441–446.

McKenzie, Kwame and Trudy Harpham (2006). *Social capital and mental health*. Jessica Kingsley Publishers.

Mulia, Nina et al. (2008). “Social disadvantage, stress, and alcohol use among black, Hispanic, and white Americans: findings from the 2005 U.S. National Alcohol Survey”. In: *J Stud Alcohol Drugs* 69.6, pp. 824–33.

Mulia, Nina et al. (2017). “Racial/ethnic differences in life-course heavy drinking from adolescence to midlife”. In: *J Ethn Subst Abuse*, pp. 1–20.

O’Campo, Patricia et al. (2015). “The Neighbourhood Effects on Health and Well-being (NEHW) study”. In: *Health & Place* 31, pp. 65–74.

Okena, Barry S., Irina Chaminea, and Wayne Wakelandc (2015). “A systems approach to stress, stressors and resilience in humans”. In: *Behavioural Brain Research* 282.144-154.

Organization, World Health and Key Centre for Women’s Health in Society (2009). *Mental health aspects of women’s reproductive health: a global review of the literature*. World Health Organization.

Portes, Alejandro (1998). “Social capital: Its origins and applications in modern sociology”. In: *Annual review of sociology* 24.1, pp. 1–24.

Rollings, Kimberly A., Nancy M. Wells, and Gary W. Evans (2015). “Measuring Physical Neighborhood Quality Related to Health”. In: *Behavioral Sciences* 5, pp. 190–202.

Sear, Rebecca (2015). “Evolutionary contributions to the study of human fertility”. In: *Population Studies*.

Smith, Ken R. et al. (2011). “Effects of Neighborhood Walkability on Healthy Weight: Assessing Selection and Causal Influences”. In: *Social Science Research* 40.5, pp. 1445–1455.

Stevenson, Betsey and Justin Wolfers (2007). “Marriage and divorce: Changes and their driving forces”. In: *Journal of Economic Perspectives* 21.2, pp. 27–52.

Utah Population Database (2017).

WHO (2010). “Mental health: Strengthening our response (Fact sheet No. 220)”. In: *Geneva, Switzerland: Author*.

Williams, David R. and Chiquita Collins (2001). “Racial Residential Segregation: A Fundamental Cause of Racial Disparities in Health”. In: *Public Health Reports* 116.

Zick, CD et al. (2013). “Re-visiting the relationship between neighborhood environment and BMI: an instrumental variables approach to correcting for residential selection bias”. In: *Int J Behav Nutr Phys Act*.