I'D GIVE MY RIGHT KIDNEY TO BE ALTRUISTIC: THE SOCIAL BIOGEOGRAPHY OF ALTRUISM IN THE UNITED STATES OF AMERICA

by

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SIGNED: Rafael Antonio Garcia

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If I have seen further, it is by standing on the shoulders of Giants.

~ Sir Isaac Newton

My intellectual odyssey to this point would not have been possible without the support, kindness, and occasional kick in the pants from those that took interest in my growth as a potential scientist.

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Jake, we started this journey together; it seems appropriate that we see its end together. Had you not been such a strong advocate for me, I would almost certainly be doing something completely different with my life. We do not always see eye-to-eye, but I only have the utmost respect for you and see great value in your perspective. You have been essential to my development as a researcher – framing the way I think about reality, theory, and this little game called Science.

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Lastly (but certainly not least), I would like to thank my family for their support. Many of you somehow knew that that know-it-all kindergartener would be in school forever, and it looks like you were right.

RAG

DEDICATION

This dissertation is dedicated to my wonderful, patient, and understanding family.

LaShuna, you have been the best partner I could possibly ask for on this crazy journey together.

To my children, I hope that this might inspire you to reach far and with all you have.

I love you all more than I ever imagined possible.

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ABSTRACT

The purpose of this dissertation is to model biosocial determinants of group-directed altruistic behavior – exploring the nomological net around it. To do this a study will be presented to determine existing associations among various biological and social predictors and test a life-history-derived causal cascade using a partially exploratory and partially confirmatory statistical technique called Sequential Canonical Analysis to ultimately predict living-donor, non-directed kidney donations (NDKD). Toward that end, some important methodological considerations first need to be discussed. The first consideration revolves around the level of analysis and how this frames the cascade model and its interpretation. Following a general discussion, an exercise in some of the general principles is provided – investigating the higher-order factor structure of the Big-5 personality constructs across two levels of analysis. The second consideration is the use of unit-weighted factor scores and their appropriateness. Following the theoretical discussion, a demonstration is provided – deriving an estimate of genetic relatedness from a set of heterogeneous data sets. Once the methodological considerations have been discussed, the primary cascade model is presented in two parts: 1) the measurement model – operationalizing the measures incorporated into 2) the structural model – testing the proposed causal cascade using Sequential Canonical Analysis. A discussion follows in which the results are summarized, limitations are articulated, and further research directions are explored.

CHAPTER 1: GENERAL INTRODUCTION

The purpose of this dissertation is to model biosocial determinants of group-directed altruistic behavior – exploring the nomological net around it. To do this, a study will be presented to a) determine existing associations among various biological and social predictors, b) test a life-history-derived causal cascade using a partially exploratory and partially confirmatory statistical technique called Sequential Canonical Analysis, and c) ultimately predict living-donor, non-directed kidney donations (NDKD). Toward that end, some important methodological considerations first need to be discussed**. The first** consideration revolves around the level of analysis and how this frames the cascade model and its interpretation. Following a general discussion, an exercise in some of the general principles is provided – investigating the higher-order factor structure of the Big-5 personality constructs across two levels of analysis**. The second** consideration is the use of unit-weighted factor scores and their appropriateness. Following the theoretical discussion, a demonstration is provided – deriving an estimate of genetic relatedness from a set of heterogeneous data sets. Once the methodological considerations have been discussed, the primary cascade model is presented in two parts: 1) the measurement model – operationalizing the measures incorporated into 2) the structural model – testing the proposed causal cascade using Sequential Canonical Analysis. A discussion follows in which the results are summarized, limitations are articulated, and further research directions are explored.

Literature Review

Altruistic acts are behaviors performed by organisms that result in a benefit (increased fitness) for one or more other organisms – typically resulting in a perceived detriment to the self

(decreased fitness). These behaviors are notoriously difficult to account for using evolutionary-based models (Baston, 2010; Massey et al., 2010). Some extraordinary acts (like living-donor, non-directed kidney donations; NDKD) have such high potential fitness costs (death) that many genetics-based models cannot account for the risk level without very high coefficients of relatedness (comparable to those seen in eusocial insects; Campbell, 1975). Because of this, many researchers investigating altruistic acts reject predictive evolutionary based models (e.g., kin selection, inclusive fitness, reciprocal altruism, sociality) outright in favor of a mere descriptive approach. A review of the literature, however, fails to find *any* body of work attempting to explain these altruistic acts from a life history framework – though some have articulated ideas consistent with such a framework (Baston, 2010).

This dissertation investigates altruism by focusing primarily on living-donor, non-directed kidney donations. These are donations by individuals that elect to donate a kidney without specifying a recipient, knowing that they will live from that point forward with only one functioning kidney. To truly investigate altruism, it is essential that these donations be from living donors and not from the near-deceased (as the former has clear potential fitness costs, while the latter may not). Kidney donations designated "non-directed" ones by the United Network for Organ Sharing (UNOS) mean that they are not earmarked for specific individuals such as a sibling, parent, offspring, or any recipient known to the donor.

NDKD are infrequent (~200 NDKD in the USA in 2010; Organ Procurement and Transplantation Network, 2014) – making it difficult to do research on these extraordinary acts of altruism. Additionally, it is virtually impossible (at present) to know *a priori* who will ultimately elect to and complete a NDKD. Thus, it becomes immensely difficult to gather information on who these individuals are before the event of interest (donation).

One approach that gets around these limitations is to investigate NDKD rates across group-level data. By choosing a group-level of analysis (e.g., state or province) researchers can investigate the group-level cultural factors that might influence NDKD rates. The approach of using group-level data to examine psychological phenomena is a technique commonly used to investigate infrequent but theoretically-interesting events (e.g. homicide - Vandello & Cohen, 1999; infection rate - Fincher, Thornhill, Murray, & Schaller, 2008).

Consistent with this approach, living-donor, non-directed kidney donations exhibit limited levels of interstate traffic in organs (Organ Procurement and Transplantation Network, 2014) – favoring hypotheses that individual donors are directing their donations to any member of their specific local group and that the nature and composition of that group would, therefore, influence their decision to donate.

Brethel-Haurwitz & Marsh (2014)

One such study was published in *Psychological Science* by Brethel-Haurwitz and Marsh (2014). In that paper, the authors attempt to establish a relationship between a nationally-representative well-being index (Gallup-Healthways Well-Being Index; WBI) and acts of extraordinary altruism (per capita living-donor, non-directed kidney donation rates; NDKD). More specifically, they aimed to establish that well-being promoted extraordinary altruism.

The Gallup-Healthways Well-Being Index (WBI) is a state-level composite measure of self-reported well-being in the United States comprised of: *life satisfaction* (current and expected in five years), *emotional health* (experience of various emotional indicators; e.g., smiling, worry), *physical health* (e.g., reported sick days and feeling well-rested), *healthy behavior* (e.g., exercise and health diet habits), *work-environment* (job satisfaction and relationship with

supervisor), and *basic access* (e.g., feeling safe and having enough money for basic needs). Although Brethel-Haurwitz and March took WBI at face value, the composite is consistent with the Covitality factors used in life history research (Weiss, King, & Enns, 2002; Figueredo et al., 2005; Figueredo, Vásquez, Brumbach, & Schneider, 2007).

Brethel-Haurwitz and Marsh found a relationship between NDKD and WBI (Pearson r = .52), which held true even after controlling for covariates (β = .50). Included in their list of covariates were: Gini coefficient (a measure of income equality), median income, % non-Hispanic White, male-to-female ratio, median age, educational attainment, poor mental health, and poor physical health – of which only % of non-Hispanic White and median age achieved statistical significance. These covariates were included atheoretically because "both well-being and altruistic donations *may covary* with sociodemographic variables that are not predicted to drive the well-being/altruism relationship" [emphasis added]. No further explanation was given into why these effects may exist or why the two statistically significant effects were found. Although the establishment of a relationship among variables is a critical first step, it is curious when one does not attempt to go beyond mere description toward mapping out a 'nomological net' (Cronbach & Meehl, 1955).

Given the desire of the researchers to establish a *causal* relationship between WBI and NDKD, these analyses and handling of covariates are tragically inadequate. At a minimum, rationale should be provided on *why* the covariates were included and *why* the two significant covariates were found. What 'flavor' are these covariates? Are they mishandled mediators, moderators, or confounders? The term covariate itself is vacuous, demoting the variables to nuisance. "Confounds are alternative hypotheses towards which one has a negative attitude" (Figueredo, personal communication). Significant covariates imply a larger nomological network

(theoretical framework) that needs to be found and incorporated. What theoretical framework might help unpack some of these relationships?

Life History and Super-K

Life history (LH) theory is a mid-level theory derived from general evolutionary theory (MacArthur & Wilson, 1967; Wilson, 1975) that describes the strategic allocation of bioenergetic and material resources toward solving adaptive problems. While originally developed to explain between-species differences in reproductive strategies, LH theory has been extended to explain within-species differences such as between-group differences and between-individual differences (Rushton, 1985; Cabeza de Baca & Figueredo, 2014). Whatever level of analysis being investigated, instances are typically categorized as being primarily Slow LH or Fast LH – based on the pattern of resource allocation preference observed. With respect to individual difference, resources are typically classified into different components: *somatic effort* (growth and maintenance of the self) and *reproductive effort* (parental vs. mating effort; Cabeza de Baca, Figueredo, & Ellis, 2012).

Slow LH strategists are individuals that invest highly in somatic effort and parental effort over mating effort. The general strategy is to have a few children that can be invested in heavily (low quantity, high quality). Slow LH strategists are described as long-term oriented, possessing greater levels of executive functioning and engaging in far more mutualistic social behaviors (Figueredo & Jacobs, 2010; Figueredo, Cuthberson, Kauffman, Weil, & Gladden, 2012; Wenner, Bianchi, Figueredo, Rushton, & Jacobs, 2013) – engaging in more mutualistic behaviors such as general altruism (Figueredo et al. 2005) and presumably extraordinary altruism (NDKD).

Fast LH strategists, on the other hand, are individuals that invest highly in mating effort over somatic and parental effort. The general strategy is to have many children (ideally from several genetically diverse partners; Wolf & Figueredo, 2011) and invest very little in each (high quantity, variable quality). Fast LH strategists are described as short-term oriented, having little need for executive functioning and engaging in far more antagonistic social behaviors (Figueredo & Jacobs, 2010; Figueredo et al., 2012; Wenner et al., 2013).

Slow LH strategists reliably produce offspring of greater phenotypic fitness than fast LH strategists. These individuals have greater mental and physical health (covitality), social effectiveness (General Factor of Personality), and other personality traits indicative of greater somatic and parental effort (Rushton, 1985, 2000; Thornhill & Palmer, 2004). Collectively, this constellation of characteristics has been called the "Super-K Factor" (Figueredo, Vasquez, Brumbach & Schneider, 2004).

LH and Genetic Relatedness

To combat the adverse effects of random mating on reliably producing offspring of greater phenotypic fitness, slow LH strategists practice *assortative pairing* (Rushton, 1989; Figueredo & Wolf, 2009). Generally, assortative pairing refers to the selection of any partner (social or sexual) on the basis of phenotypic similarity. The consequence for assortative pairing is a group of individuals that is more genetically homogeneous than would be expected under conditions of random mating. In the specific case of *assortative mating*, individuals select mates on the basis of phenotypic similarity (which presumably corresponds to genotypic similarity; Miller, 2000). The result is offspring that are more related to each parent than would be predicted by *panmixia* (true random mating) – preserving slow LH characteristics in the subsequent

generation (Wolf & Figueredo, 2009). Naturally, the assortative pairing of social partners further facilitates the possibility of an individual's offspring having a pool of individuals that are more genetically homogenous from which to select their eventual sexual partner(s).

Further, according to Genetic Similarity Theory (Rushton, Russel, & Wells, 1984) individuals in more genetically homogeneous groups will be more likely to perform acts of altruism toward unknown in-group members (e.g. kidney donations). Because of the increased genetic relatedness among individuals, helping anyone within the in-group (even a stranger) will still result in a fitness payoff. Thus, assortative pairing promotes reliably phenotypically fit offspring, increased likelihood of further assortative mating of the next generation, and (potentially) incremental fitness advantage by the actions of altruistic conspecifics.

Shaping Life History

Ecological pressures contribute to the overall *harshness* and *predictability* of the environment (Ellis, Figueredo, Brumbach, & Schlomer, 2009). Harshness refers to the *mean level* extrinsic morbidity and mortality, while predictability refers to the *variance* or stability of extrinsic mortality in a given environment. Extrinsic sources of morbidity and mortality are sources beyond control of the individual; this is in contrast to sources of mortality is within control of the individual (intrinsic). The more agency afforded the individual (i.e. the more control individuals have in their continued existence), the more individuals will invest in somatic effort and parental effort (slow LH strategies), producing more physically robust individuals (competing in quality). Conversely, when afforded less agency, individuals will invest more in mating effort (fast LH strategies), producing a greater quantity of heterogeneous offspring (competing in quantity).

Several ecological parameters have been identified to impact the degree of *environmental agency* (the degree to which individuals can control sources of extrinsic morbidity and mortality). The earliest LH models focused on population density (Pianka, 1970). Environments with greater population densities (specifically those near the environmental carrying capacity) have increased resource competition – favoring slower LH strategies. Environments with lower population densities, on the other hand, do not have such increased competition – permitting niche generalization and favoring faster LH strategies (Figueredo, Woodley, Brown, & Ross, 2013)

Another well-researched ecological parameter is infectious disease. Historically, pathogen burden (total exposure to infectious diseases) has been a primary and direct driver of extrinsic mortality (McNeill 1998; Wolfe, Dunavan, & Diamond, 2007). Environmental pathogens often act as "random death machines" sometimes decimating entire continents until some immune response (physiological or behavioral) can be employed to combat it (e.g. The Black Death). Thus, any model attempting to model group-level LH effects should include an indicator of parasite burden when able. It is simply one of the best indicators we have of extrinsic morbidity and mortality (Fincher & Thornhill, 2008).

As slow LH strategists thrive best in environments with greater environmental agency, behavioral and social efforts are made to keep their social and sexual relationships stable. This has already been pointed out in the previous section when discussing genetic similarity; slow LH strategists assortatively pair to reduce the variability in local social groups, resulting in highly predictable social ecological conditions. Slow LH strategists can further reduce the social variability by treating conspecifics all on relatively equivalent footing (Black, Peñaherrera Aguirre, Chavarria Minera, & Figueredo, 2017). Interestingly, this social stability can give rise to

a certain amount of phenotypic differentiation – allowing each individual to pursue their own role within the available niche-space. This individual diversification can complement slow LH strategies to yield greater Human Capital (Fernandes & Woodley, 2017).

Kidney Donations

Thus, there are two possible evolutionary dynamics for the undirected altruistic behavior seen in NDKD. The first is, as stated previously, Genetic Similarity Theory (Rushton, Russel, & Wells, 1984), which predicts that individuals in more genetically homogeneous groups will be more likely to perform acts of altruism toward unknown in-group members (kidney donations, in this case). The second is multilevel selection theory (Wilson & Sober, 1994), which predicts that, partially contingent upon the genetic variability both within and between competing social groups, groups that cooperate more (living-donor, non-directed kidney donations, in this case) will tend to outcompete groups that do not. As these groups produce more 'cooperators,' the altruistic trait increases in frequency.

The second is multilevel selection theory, which predicts that group-directed altruism is in fact group-*selected* altruism, as a direct result of competition between groups, whereas the more conventional Genetic Similarity Theory views the aggregate behavior as merely the aggregate of genetic self-interest among the constituent individuals. The present study is not specifically designed to discriminate or decide between these two theoretical explanatory frameworks; however, there is a possibility that certain patterns of empirical can favor one view over the other. For example, a prevalence of living-donor, directed kidney donations might be construed to favor Genetic Similarity Theory interpretations of the data, whereas a prevalence of

living-donor, non-directed kidney donations might be construed to favor multilevel selection theory interpretations..

Dissertation Overview

The primary objective of this research is to model potential biosocial determinants of group-directed altruistic behavior (living-donor, non-directed kidney donations; NDKD) at the aggregate state level for each of the 50 United States of America. A sequential canonical cascade model will explore possible relations among various biological, psychological, and sociological constructs/variables. The structural model for the social biogeography of altruism incorporated: population density, parasite burden, proportion of Whites, genetic relatedness, biodemographically estimated LH strategy, social equality, strategic differentiation of biodemographically estimated LH strategy, median income, familism, *Alpha* and *Beta* Aggregate Regional Personalities (ARPs), empathy, living-donor directed kidney donations, and living-donor non-directed kidney donations. To accomplish this primary objective, this dissertation will begin with two methodological considerations relevant to the implementation and interpretation of this cascade model: level of analysis considerations and considerations pertaining to the use of unit-weight factor scores.

Predictions

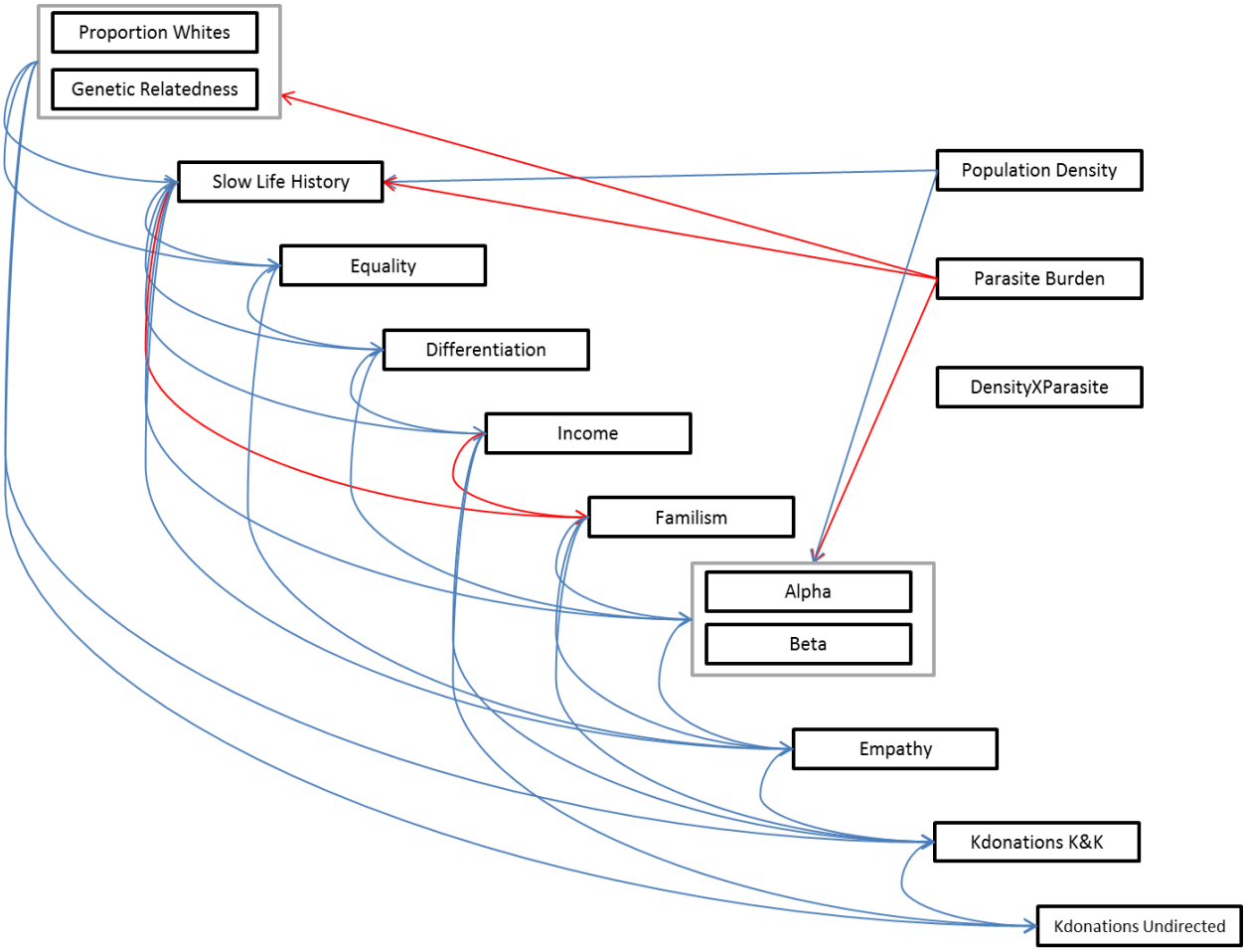


Figure 1. *A priori cascade model predictions*

Blue paths are predicted to have positive coefficients, while red paths are predicted to have negative coefficients. Any differential effect(s) of Proportion Whites versus Genetic Relatedness is (are) unknown as are the specific relations among the higher-order Big-5 variables (*Alpha* and *Beta*) and Altruism.

Predictions are depicted in Figure 1. The specific effects expected by theory were:

1. Proportion Whites will be inhibited by Parasite Burden, because persons of European extraction tend to avoid settling areas that are high in parasites, probably due to their

generally lower natural resistance to infections as a consequence of having evolved in more temperate climates (Cabeza de Baca & Figueredo, 2017).

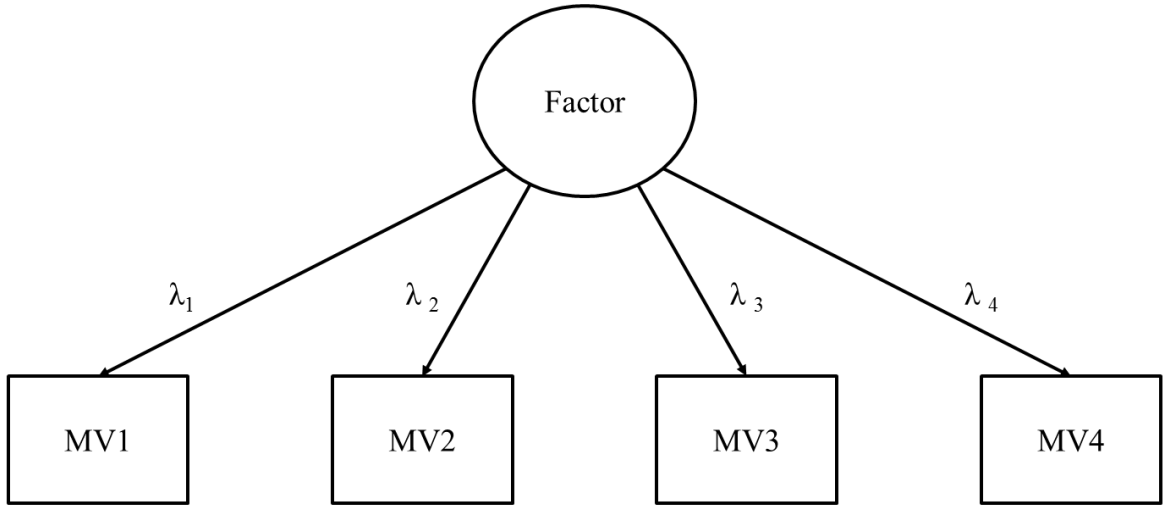
2. More Genetic Relatedness will be inhibited by increased Parasite Burden because higher extrinsic sources of mortality will favor genetic diversity (Fincher & Thornhill, 2012; Woodley of Menie, 2009; Figueredo & Wolf, 2009)
3. Super-K will be promoted by: (a) more Genetic Relatedness because it serves as a partial mediator between Super-K and Parasite Burden; (b) greater Population Density as increases in density promote higher levels of competition for resources leading to the production of more competitive offspring; and (c) lower levels of Parasite Burden as fewer sources of extrinsic mortality allow individuals to allocate effort toward circumventing sources of intrinsic mortality.
4. Increased Equality will be promoted by: (a) Super-K because slower life histories will promote more mutualistic social schemata (Figueredo & Jacobs 2010) and (b) more Genetic Relatedness because the more interrelated a state is the more prone it will be to help conspecifics because of inclusive fitness.
5. Increased Strategic Differentiation will be promoted by: (a) increased Equality and (b) increased Super-K because this kind of specialization (niche-splitting) has been shown to be present within and between populations when the social environment is highly predictable (Black, Peñaherrera Aguirre, Chavarria Minera, & Figueredo, 2017).
6. Increased median state Income will be promoted by: (a) increased levels of Strategic Differentiation because division of labor have been a proposed means of increasing productivity (Smith, 1776; Ricardo, 1871) and (b) Super-K because slower strategists are more future-oriented and thus more likely to have stayed longer in formal education.

7. Increased Familism will be promoted by: (a) lower Income because larger composite families are more likely to be from poorer backgrounds and (b) Fast Life History because life history theory predicts faster strategists will reproduce at higher rates with shorter intergenerational and interbirth intervals.
8. Increased *Alpha* will be promoted by: (a) increased Familism because both Agreeableness, Conscientiousness, and Emotional Stability are essential to hierarchical socialities (Triandis, 1995), (b) increased Equality because interpersonal Aggregate Regional Personalities (ARPs) will facilitate the "group hug" ideologies of egalitarian worldviews, and (c) Super-K because life history theory predicts that mutualistic orientation (interpersonal ARPs) are developed by slower life history individuals and groups.
9. Increased *Beta* will be promoted by: (a) higher levels of *Alpha* because* ARPs like Openness to New Experiences and Extraversion are potentially self-actualized adaptations to prosocial environments, (b) increased Familism because self-actualized ARPs are promoted by positive experiences with the closely attached, (c) decreased Equality because Openness to New Experiences and Extraversion promote self-uniqueness, and (d) increased population density because greater densities increase the likelihood that individuals will be in forced interactions with those of different life experiences.
10. Increased Empathy will be promoted by: (a) increased levels of *Alpha* because prosocial ARPs should facilitate the number of positive social interactions and facilitate "role-taking" behaviors and (b) Super-K because life history theory predicts that other-directed orientation (prosocial ARPs) are developed by slower life history individuals and groups.

11. Increased Kidney Donations (K&K) will be promoted by: (a) increased levels of Empathy because empathizing with the situations of friends and family should serve as a motivation to act altruistically, (b) increased Familism because regions that value families more heavily should be more willing to make personal sacrifices for the familial group, (c) increased Income because kidney donations are serious medical procedures needing recovery time and enough financial stability to compensate for any work time missed as well as pay for any unexpected medical complications, and (d) increased Genetic Relatedness as groups with higher mean coefficients of relatedness will have higher average family coefficients as well making them more willing to act altruistically toward kith and kin according to inclusive fitness.
12. Increased Kidney Donations (Undirected) will be promoted by: (a) increased Kidney Donations (K&K) because the special case of undirected donations may be an extreme case of an altruism mechanism evolved to help an individual's local community, (b) increased Empathy because empaths often exhibit prosocial behaviors like altruism, (c) increased Income because kidney donations are serious medical procedures needing recovery time and enough financial stability to compensate for any work time missed as well as pay for any unexpected medical complications, and (d) increased Genetic Relatedness because groups with higher mean coefficients of relatedness will be more willing to act altruistically toward an unknown stranger as according to inclusive fitness theory.

CHAPTER 2: METHODOLOGICAL CONSIDERATIONS I. ESTIMATING LATENT SCORES

Many procedures for generating latent factor score estimates can be found in the literature. One major division is the use of differential-weighting versus unit-weighting of indicators to generate estimates (Bentler & Woodworth, 1979). The two procedures are characterized in Figure 2.



Differential Weighting

$$\text{Factor} \approx w_1*zMV1 + w_2*zMV2 + w_3*zMV3 + w_4*zMV4$$

Unit Weighting

$$\text{Factor} \approx \frac{z(MV1) + z(MV2) + z(MV3) + z(MV4)}{4}$$

Figure 2. *Characterization of two weighting procedures for estimating factor scores*

Differential-weighting procedures use some criterion to give more weight (w; where $w \propto \lambda$) to manifest variables (MVs) that "better represent" the underlying factor. Unit-weighting procedures given equal weight to each MV (implied that w = 1.0).

Simulation studies show that the outcomes of the two methods highly correlate; however, unit-weighting typically yields results that generalize better across research studies and various study conditions (Schmidt, 1971). The literature does not appear to provide a theoretical rationale for why this discrepancy might be the case. I propose that just as the mean of a sample of participants' scores is often the best estimate of a population parameter in the absence of any additional information, the mean of several standardized, manifest indicators is often the best estimate of a latent factor.

Theoretical Justification for the Generalizability of Unit-Weight Factor Scores

According to critical multiplism (Cook, 1985; Shadish, 1986), participants (people) within a population is not the only dimension along which sampling occurs. Any dimension along which generalization *can* occur is sampled. That is to say, any time a researcher makes a decision to not exhaust all possible instances of a dimension and wishes to make inferences along that dimension, sampling has occurred. Using a scheme articulated by Cronbach (1982), a given study typically samples: *units* (persons or groups), *treatments* (predictors or potential causes), *observations* (outcome measures), and *settings* (social context).

When the observations chosen come from a theoretically-defined class of outcome measures caused by the same underlying process, the measures represent manifest indicators of a latent construct. The selection of these indicators is a sampling of all possible manifestations of the latent construct. As a sample, it follows that approximations of the "true" latent score (population parameters) can be estimated by aggregating scores from a sufficient number of manifest indicators that have been appropriately (*representatively*) sampled. What the

correspondence is between each indicator and the underlying latent construct is initially unknown and to be determined.

When considering a sample of individuals, the arithmetic mean of a sample's scores is typically used as the best estimate of any individual's score within that given sample (blue box in Figure 3). The underlying logic is that each individual score deviates randomly from the sample statistic and these deviations will approximate a unit-normal distribution according to the Central Limit Theorem (Laplace, 1812; Galton, 1889).

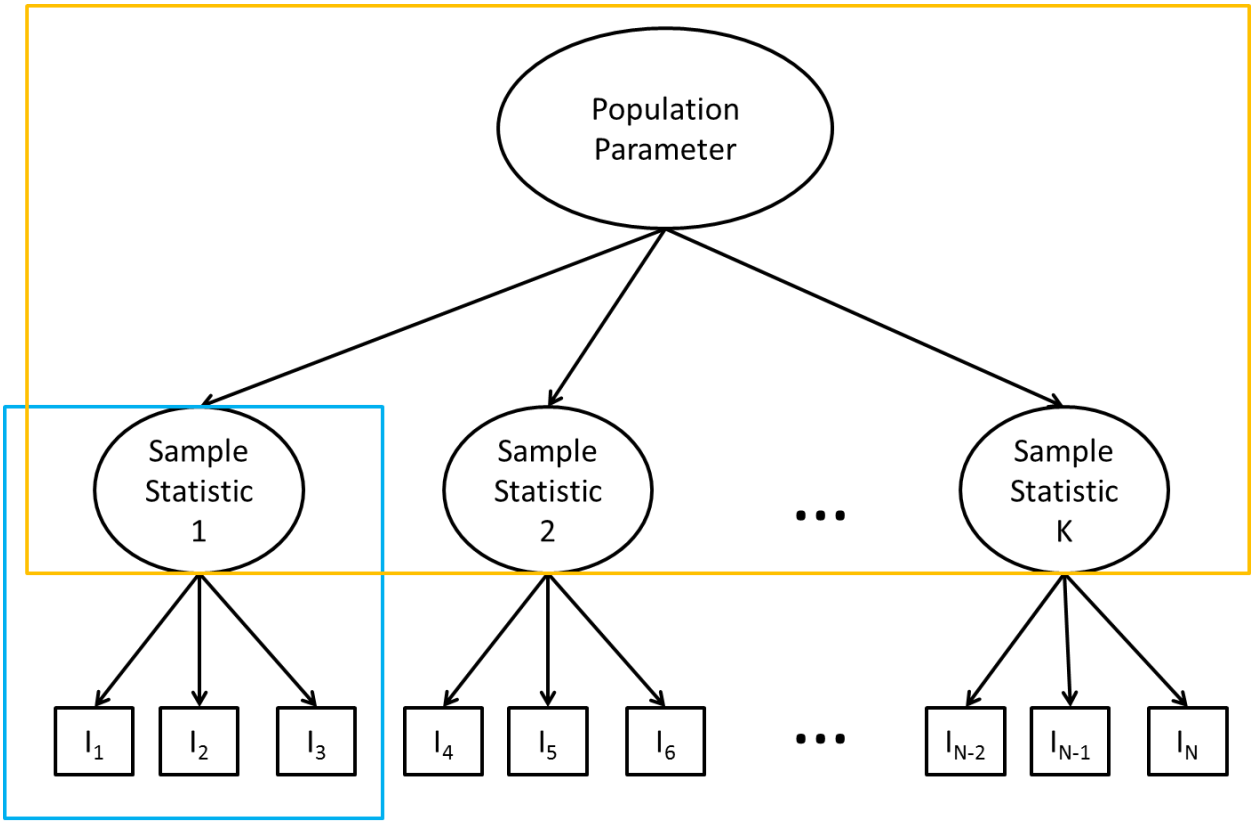


Figure 3. *A representation of estimating effects from samples of individual cases (Ii)* The blue box depicts that sample statistics are derived from a sample of individual cases, while the orange box depicts that population parameters are derived from a sample of sample statistics.

Sample statistics, likewise, deviate randomly from the population parameter. To mitigate these random deviations, researchers attempt to collect large, representative samples or aggregate several heterogeneous samples to estimate population parameters (orange box in Figure 3). The effect of either attempt is to sample across the range of a dimension in such a way that single parameters can be estimated confidently based on the sampling distribution. In both of these cases, individuals (units) are all "created equal" (unit-weighted). This is done, in part, because extraneous variables are unidentified or irrelevant. Individuals *can* be weighted differentially, but are not typically unless extraneous variables are identified and their prevalence in the population is known (e.g. weighting cases based on race, socioeconomic status, group membership).

Thus, by aggregating a sufficiently large and representative sample of individuals and/or aggregating several heterogeneous samples of individuals, reliable and externally valid estimates of population parameters can be calculated from the mean of the sample(s). In other words, through these aggregation procedures, researchers can reduce the effect of *error variance* (measurement and sampling error) on distorting population estimates.

As manifest indicators are sampled from the latent construct (population), it follows that individual indicators will, likewise, deviate randomly from the latent construct. Thus, the mean of the manifest indicators will be a reasonable estimate of the underlying latent construct (just as the mean of several sample means is a reasonable approximation of the population parameter; orange box in Figure 4).

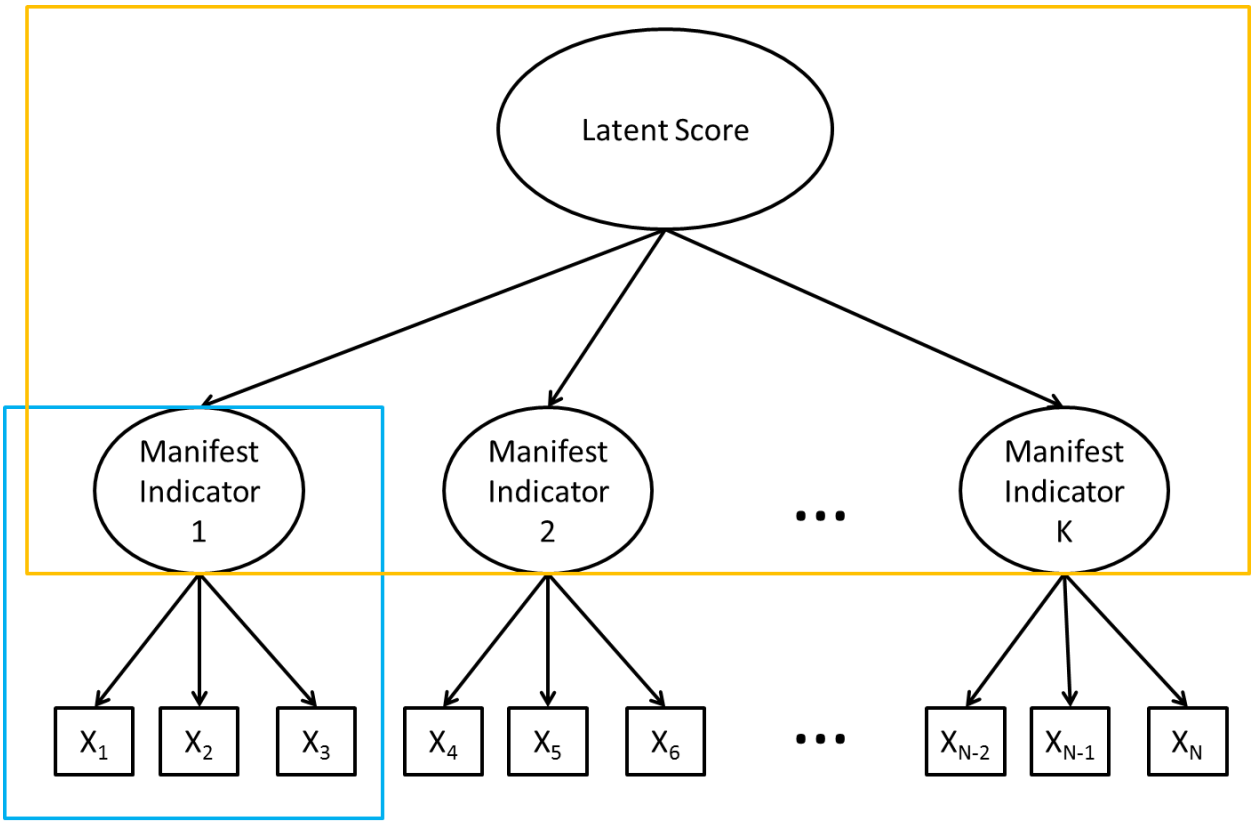


Figure 4. *A representation of estimating scores from samples of items (Xi)*

The blue box depicts that manifest indicators are derived from a sample of items, while the orange box depicts that latent scores are derived from a sample of manifest indicators.

Now why choose the unit-weighting procedure over the differential-weighting procedure? The latter will better fit the sample data, but the former will generalize better. Unit-weighting generalizes better as it more closely parallels the sampling procedures used in the sampling of individuals (units), which is designed to estimate the population parameters. Whether discussing individuals from a population of potential participants or indicators from a population of potential measures, in both cases some instances will better represent the relevant underlying population (some individuals or indicators will deviate less than others from the true population value). Instances are thus given equal weight in an attempt to correct for systematic

differences that cancel out on the aggregate (individual differences in the case of individuals and specific variance components in the case of indicators). What is left is a single point estimate that approximates the true population value.

Unit-weighting confers benefits beyond just increasing the generalizability of discovered effects. Because the created composites are simply aggregated standardized scales, simple part-whole correlations can be used to determine the internal factor structure and coherence. These are elementary procedures that virtually anyone having taken a basic-level statistic class can understand. Unlike differential-weighting, which is typically done using path analytic and structural equations models (PA and SEM, respectively), unit-weighting typically has greater statistical power, and thus permits the use of smaller sample sizes to achieve the same results (Gorsuch, 1983). The sample size need only be large enough to produce stable correlation coefficients as opposed to PA and SEM which require a sample size of at least 5-10 per parameter being estimated (Bentler 1995), which amounts to 10-20 per manifest indicator (one parameter for the factor loading and one for the error component). This can become quite cumbersome as the number of manifest indicators increases.

Further, the interpretation of the unit-weighted model is more elegant. It relates to variance decomposition, but can simplified as: the unit-weighted factor score is simply the average z-score of all the manifest indicators.

Until "true" manifest-latent relationships can be derived (e.g. meta-analytic factor loading estimates), unit-weighting is a reasonable and powerful option based in sampling theory. While it is true that unit-weighting results in a loss of sample-level information, the decrease in resolution actually *facilitates* the discovery of more robust effects.

When considering the role of latent factors within a given research study, data analysts ought to consider using a unit-weighting scheme to derive factor scores – particularly when interested in identifying effects that will generalize to similar research context or perhaps even the real world (different settings). Unit-weighting can drastically reduce necessary sample sizes, which is of particular importance when dealing with expensive interventions of rare events.

Is 'White' Right?: Genetic Homogeneity, Race, and Kidney Donations

Racial classification is frequently used in research as a covariate, often with no theoretical rationale (Walsh, Katz, & Sechrest, 2002). Sometimes racial differences are merely proxies for behavioral differences in culture, while others may be innate physiological characteristics that are more/less concentrated in certain races. Research on altruism often finds racial differences, yet gives little to no discussion as to an underlying reason (e.g. Brethel-Haurwitz & Marsh, 2014). Further, many of those same studies claim that kin-selection models of altruism are insufficient to account for regional differences observed. These studies often take an overly simplified view of kinship – modeling only those connected via bloodlines within the last three or four generations. It is entirely possible for a group of individuals to be distantly related according to pedigree, but share a high genetic relatedness according to genome (Genetic Similarity Theory; Rushton, Russell, & Wells, 1984). The purpose of this analysis is to determine whether or not race proportions at the state level are simply a proxy for genetic relatedness when it comes to predicting living-donor, non-directed kidney donations (as in Brethel-Haurwitz & Marsh, 2014).

The question as to what influences altruism has probably plagued mankind since its first recording, particularly what is considered as extraordinary or true acts of altruism – acts in which

the actor seemingly receives no direct or indirect benefit (or seemingly incurs a detriment) from a social exchange. While the ecological validity of those claims is questionable (in many circumstances givers/actors receive things like social praise or increased status), some researchers have tried to determine what influences these extraordinary behaviors. Brethel-Haurwitz and Marsh (2014) looked at living-donor, non-directed kidney donations (NDKD) at the state level. The primary focus of the investigation was to examine the relationship between well-being (as measured by a Gallup poll) and NDKD. Interestingly, one of the effects found was glossed over in the results and discussion – a relationship between the proportion of self-identified racially-White individuals within each state (pWhites) and NDKD rates per capita. This effect was present even after controlling for median income, social equality, well-being, sex ratio, median state age, educational attainment, and measures of mental and physical health. The authors did not explain why that relationship was found, simply that it is exists.

Some argue that of the traditionally identified racial groups, those of ancestral European descent (self-identified Whites) are more genetically related (i.e. possess less genetic diversity) than those ancestrally from other geographic regions, most notably Africa (self-identified Blacks or African Americans; Lohmueller et al., 2008). These hypotheses derive from the recent African origin model. The logic being that as a small subpopulation quickly expanded out of Sub-Saharan Africa, founder effects restricted the available genetic pool and made them more susceptible to genetic drift. Further, geographic isolation restricted gene flow, genetically isolating the subpopulation from the original population. Less genetic diversity means more genetic homogeneity (a greater degree of relatedness).

There are reasons to be skeptical of these hypotheses, however. Historically, approximately 75% of the ancestors of modern African Americans originated from the Bight of

Biafra and West Central Africa, relocated during early colonial years of the United States rather than being a random sample of individuals from Africa (Walsh, 2001). Additionally, it is naïve to assume that being subjected to intercontinental travel and relocation (particularly under the conditions of *forced* relocation) to the Americas would not subject any group of people(s) to unique selective pressures – further restricting the group's genome. In fact, it is estimated approximately 16% of those shipped to the Americas died of sickness, starvation, or revolt (Curtin, 1972). While these reasons are all face valid, evidence still supports that Black or African Americans have a greater degree of genetic diversity than Whites in HLA genes (Cao et al. 2001), SNPs, and haplotypes (Stephens et al. 2001), in part, attributable to highly variable admixture (both among individuals of different African ancestries and with individuals of non-African ancestries; Bryc et al. 2010).

According to Genetic Similarity Theory (an extension of Hamilton's kin selection theory; Hamilton, 1963), individuals within social groups will tend to engage in more altruistic behaviors (engage in more asymmetric social exchanges of which they are on the losing end) with conspecifics (fellow in-group members) the more genetically-related the conspecifics (hereafter *kith and kin*). In other words: individuals are more likely to do favors for someone more genetically related to them than someone who is not.

Since those of White European descent are more genetically homogeneous than those of other racial categories (which is the US is predominantly of African descent), then it stands to reason that pWhites within state may *merely* serve as a proxy for genetic relatedness. If true, when it is added into a linear regression with a more sophisticated estimate of genetic relatedness, there should be no incremental explanatory power of pWhites.

Methods

This study is a state-level analysis of the 50 states within the USA (N=50). The proportion of Whites for each state was taken based on 2010 Census data, additionally, the per capita living-donor, non-directed kidney donation rates were acquired from *United Network for Organ Sharing* (UNOS), and an estimate of genetic relatedness was constructed from four data sets: two data sets comprised of ancestral indicators of genetic relatedness that served as the primary data (Dataset) and two data sets comprised of a proportional breakdown of ancestral compositions by state (Procedure)– crossed to result in four unique composites. These composites were created using a unit-weighted factor score estimation procedure from all indicators within each 'Dataset X Procedure' combination. Each state, thus had four composite score estimates of genetic relatedness. A unit-weighted procedure was used for factor score creations as the sample size was low (N=50) and the unit-weighted procedure is less likely to fit sample specific sources of variability and effects are more likely to generalize from one set of study conditions to the next.

Measures.

Living-donor, non-directed kidney donations. Kidney donation rates were obtained from the Organ Procurement and Transplant Network overseen by United Network for Organ Sharing (UNOS; Based on Organ Procurement and Transplantation Network data as of March 4, 2016). These data included a state-by-state breakdown for all living-donor, non-directed kidney donors from 1999 to 2010. Per capita donation rates were calculated by dividing the total number of kidney donations within the time period by the 2010 U.S. Census population estimates (U.S. Census Bureau, 2011). This was done as the research question was most relevant to the donation base rate and not the absolute number (which will be confounded with population size).

Proportion of Whites (pWhites). The proportion of self-identified White, non-Hispanic residents within each state was taken from 2010 US Census population data (U.S. Census Bureau, 2011).

Genetic relatedness. To determine genetic-relatedness, two weighting schemes were implemented on two datasets. The weighting schemes were derived from two different commercial genetics companies, each using different *ancestral categorization* criteria. The datasets were taken from two different studies, each with unique indicators tapping into different parts of the genome stratified by *continent of origin*. The result was four unique composites that were created using heterogeneous weighting procedures and heterogeneous genetic indicators. Thus, each composite should possess unique biases and error variances – reducing the likelihood of inflating the trait scores with common method variances. The resulting four variables were aggregated using a unit-weighting procedure (Gorsuch, 1983) to estimate a single common trait score for each of the fifty US states.

Scheme 1 - 23andMe. The first weighting scheme utilized summary statistics provided by a sample of 162721 23andMe customers (Bryc, Durand, Macpherson, Reich, & Mountain, 2015). 23andMe is a private genetics company that provides a genotyping service to its customers for a fee. Customers of the service provide basic demographic information including self-reported racial identification as well as salivary samples for genotyping. The racial identifications were condensed into three broad but colloquially accepted categories: African American, European American, and Latinos. Within each of the racial identification, the percent of African, Native American, and European ancestry was determined for each US state.

This procedure took the percentages within each state's ancestral categorization, grouped them according to the limitations of the specific dataset, and generated a weighted average

genetic relatedness estimate. Once that was done, a second weighted average was generated based on the proportions of each racial classification according to the 2010 US Census. The result was a single weighted estimate of genetic relatedness for each dataset used.

Scheme 2 - Ancestry. The second weighting procedure utilized proportions of ancestry derived from a sample of over 250000 AncestryDNA customers (Granka, 2014). Like 23andMe, AncestryDNA is a private genetics company that provides a genotyping service to its customers for a fee. The proportional ancestry of each customer was determined for 26 world regions. The results were aggregated by state (interactive graph available at http://blogs.ancestry.com/ancestry/2014/04/04/a-genetic-census-of-america/).

This procedure took the proportions within each state's ancestral classification, grouped them according to the limitations of the specific dataset, and generated a weighted average genetic relatedness estimate. Then, just as in Procedure 1, a second weighted average was generated based on the proportions of each racial classification according to the 2010 US Census. The result was a single weighted estimate of genetic relatedness for each dataset used.

Dataset 1 - Li. The first dataset contained summary statistics from fifty-two genetic samples taken from various locations worldwide (Li et al., 2008). Three indicators of genetic diversity/similarity were available including: geographic distances from the origin of *Homo sapiens*, haplotype heterozygosities, and an admixture index. The bivariate correlations among these indicators ranged from moderate to strong ($range_{|r|}$ = [.32, .91]). These indicators were aggregated using a unit-weighting procedure to produce an estimate of the common trait score for each location. These trait scores were weighted using Schemes 1 and 2 to yield two estimates of genetic homogeneity for each of the fifty US states. For Scheme 1 aggregation of several sample sites was necessary to yield single African, European, and Native American estimates of

genetic diversity/similarity for each state. For Scheme 2 each of the sample sites was plotted onto a map of the 26 ancestral regions provided by AncestryDNA. Each location was assigned the proportions of its nearest region. In the event of overlapping regions, proportions of all overlapping regions was summed and considered the appropriate weight. In the event of multiple sample sites within the same region, the average trait score of the cohabiting locations was multiplied by the same appropriate weight.

Dataset 2 - Jorde. The second dataset contained summary statistics from a sample of 255 individuals assumed to be representative of the genetic diversity within three continental populations: Africa, Asia, and Europe (Jorde et al. 2000). Five indicators of genetic diversity/similarity were available including: autosomal restriction-site polymorphisms (RSPs), autosomal *Alu* polymorphisms, mtDNA control-region sequences (hypervariable sequences 1 and 2), and Y-chromosome polymorphisms. The best available estimate of the bivariate correlations among these indicators was strong ($range_{|r|}$ = [.74, .99]). These indicators were aggregated using a unit-weighting procedure to produce an estimate of the common trait score for each continental population. These trait scores were weighted using Procedures 1 and 2 to yield two estimates of genetic homogeneity for each of the fifty US states. For Procedure 1 continental population trait scores were only provided for African and European estimates of genetic diversity/similarity as no proportion of ancestry was available for self-identified Asians. For Procedure 2 each of the 26 ancestral regions provided by AncestryDNA were aggregated into their appropriate continent.

Analysis. All univariate and multivariate analyses were performed using SAS 9.4. A simple simultaneous regression was performed in which the per capita living-donor, non-directed

kidney donation rates (NDKD) were predicted by genetic relatedness and proportion of self-identified racially-White individuals within each state (pWhites) within each state.

Results

Measurement models. The results of the part-whole correlations analysis can be seen in Tables 1-3.

Table 1 shows the part-whole correlations of the various composite measures of genetic variance. All correlations were very strong (magnitudes ranging from .69 to 1.00) and negatively correlated with the reversed unit-weighted average (as all indicators are measures of genetic diversity).

Table 1. *Part-whole correlation structure of genetic variance composites*

Indicator	**23andMe X Li**	**Ancestry X Li**
Geo Distance	-.85	-.91
Haplo Het	-1.00	-.93
Admixture	-.79	-.69

Indicator	**23andMe X Jorde***	**Ancestry X Jorde**
RSP	-1.00	-.97
Alu	-1.00	-.97
HVS1	-1.00	-.97

HVS2	-1.00	-1.00
Y	-1.00	-.97

Note: It is unusual to find part-whole correlations with a magnitude of 1.00. These unexpected correlations are inflated due to there being so little variance in the measures.

The intercorrelations among estimates ranged from moderate to very strong (range .45-.93) and, and all had excellent Cronbach's *alphas* (ranging from .80-1.00), consistent with a single unitary trait, as expected (Table 2).

Table 2. C*orrelations among genetic variance composites*

	23andMe X Li	**Ancestry X Li**	**23andMe X Jorde**	**Ancestry X Jorde**
23andMe X Li	(.85)			
Ancestry X Li	.76	(.80)		
23andMe X Jorde	.87	.51	(1.00)	
Ancestry X Jorde	.82	.45	.93	(.99)

Note: Cronbach's *alphas* with each composite are in parentheses.

These four estimates were thus combined using a unit-weighting factor score procedure to yield a single estimate of Genetic Relatedness. Table 3 shows the part-whole correlations of Genetic Relatedness. All correlations were very strong (ranging from .76 to .97) and positively correlated with Genetic Relatedness (after reverse coding).

Table 3. *Part-whole correlation structure of Relatedness*

Composite	**Relatedness**
23andMe X Li	.97
Ancestry X Li	.76
23andMe X Jorde	.93

Ancestry X Jorde	.90

Note: * denoted indicator is reversed;
Cronbach's *alpha* = .97

Test of incremental validity. Genetic Relatedness and pWhites fit the data reasonable well ($F(2,47) = 7.56$, $p = .0014$, $R^2 = .24$). As shown in Table 4, both Genetic Relatedness and pWhites demonstrated incremental validity over the other ($pR^2 = .14$, $p = .0052$ and $pR^2 = .07$, $p = .0427$, respectively) and shared very little variance with each other ($pR^2 = .04$, $p = .6901$).

Table 4. *Incremental validities: Donations per capita*

Total R^2	**Shared R^2**	**pR^2**	
		Relatedness	**pWhites**
.24*	.04	.14*	.07*

Note: * denotes $p < .05$

Discussion

Demographic variables are often included with very little thought into the potential causal mechanisms that may underlie differences between individual demographic groups. In fact, they often serve as crude proxies for some causal mechanism. The purpose of this study was to test a hypothesis regarding the role proportion of self-identified racially-White individuals within each state (pWhites) played in predicting living-donor, non-directed kidney donations (NDKD).

The results of this study demonstrated that pWhites did not merely serve as a proxy for Genetic Relatedness (as estimated through a composite measure from heterogeneous indicators from heterogeneous sources). In fact, as far as proxies go, pWhites would be a poor proxy as it

only shared 4% of its variance with Genetic Relatedness. While pWhites demonstrated incremental validity in predicting NDKD, it should be noted that the unique variance accounted for by pWhites was approximately half of the unique variance accounted for by Genetic Relatedness (~7% and ~14%, respectively).

Ultimately, the ability of pWhites to predict NDKD is still an unknown. This study demonstrated that genetic similarity is not likely driving this association, though it does, in its own right, appear to be predicting altruistic actions at the state level. There are many ecocultural factors that could account for the propensity of high pWhites states to participate in more NDKD. Walsh, Katz, and Sechrest (2002) identified nine such ecocultural factors that might impact disease management.

Going back to the fundamentals and asking "what might my variable represent, causally?" are important steps not taken enough in the literature, particularly with many of the auto-include demographic variables. The primary hypothesis of this study (that pWhites was an imperfect proxy of Genetic Relatedness) does not seem to be supported; however, new insight has been gained from this research in terms of the nomological net (Cronbach & Meehl, 1955) surrounding NDKD. Further research is needed to expand this net, incorporating more nodes and painting a clearer story as to what factors influence NDKD.

CHAPTER 3: METHODOLOGICAL CONSIDERATIONS II. LEVEL OF ANALYSIS

According to differential-K theory (Rushton, 1985), group (population) differences in life history strategies arise as a consequence of ecological pressures on individuals within each group. Thus, one set of ecological pressures will "push" the average "fit" phenotype of one group in one or more directions, while a different set of pressures will "push" the average "fit" phenotype of another group in another one or more directions; phenotypic shifts of individuals result in comparable net shifts in the aggregate (group level). This type of thinking is an example of an assumed generalization. An assumption is being made that causal forces at one level that yield differences (ecological pressures acting on individuals) will result in corresponding differences at higher levels of aggregation (state or regional level).

The Simpson's Paradox

Whenever drawing inferences across levels of analysis, it is essential to first test for *ergodicity* (high correspondence between levels of analysis). Typically, researchers do this by looking at mean differences and variance magnitudes between different levels of analysis – checking for statistically similar means and variances. If consistency across levels is found, results found at one level are assumed to translate to others. If consistency is not found, either a relevant predictor (typically considered a confounder) is unaccounted for and/or there is some emergent characteristic inherent to the one level of organization that yields phenotypically distinct relationships not found at other levels or organization.

The Simpson's paradox (also called the amalgamation paradox; Simpson, 1951) is a logical fallacy in which an effect found at one level is incorrectly generalized to another level. This problem in inference arises from poor aggregation or division of data. That is, the categories

or groups that lower-level data is being organized into are confounded by some other number of variables (Kievit, Frankenhuis, Waldorp, & Borsboom, 2013).

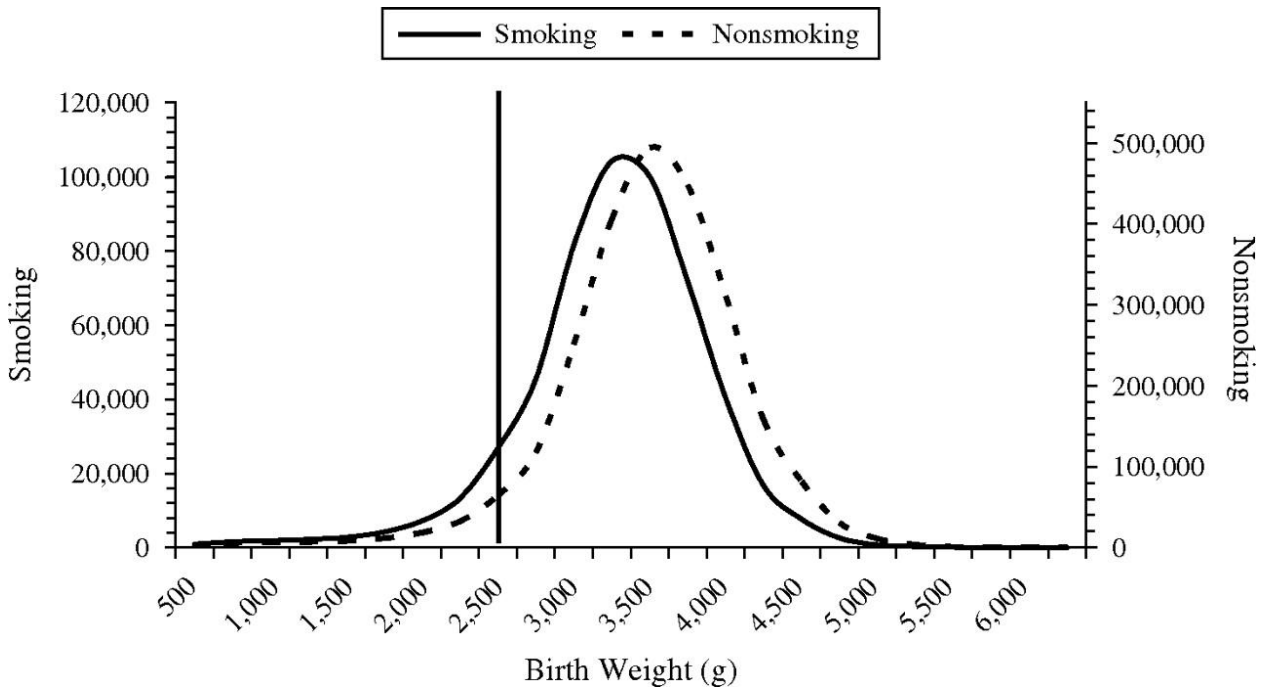


Figure 5. *Simpson's paradox involving smoking status and infant birth weight*
The vertical line indicates the low-birth weight cutoff (from Hernandez-Diaz, Schisterman, & Hernan, 2006).

A clear example of this paradox can be seen in the relation between low-birth rate and infant mortality rates of children born to smokers and non-smokers (Hernandez-Diaz, Schisterman, & Hernan, 2006). Babies classified as 'low-birth weight' typically have a significantly greater infant mortality rate than babies classified as 'normal-birth weight' or above. Additionally, babies born to smoking mothers are more likely to be classified as 'low-birth weight' than babies born to non-smokers. This would lead one to conclude that infant mortality should be greater among babies born to smoking mothers; however, 'low-birth weight'

babies born to smoking mothers have *lower* infant mortality rates than 'low-birth weight' babies born to non-smoking mothers. The reason for this paradox is that the variable used to partition the data (smoking status) differentially affects the other key variables: birth-weight and infant mortality rate. The distribution for birth weight for babies born to smoker mothers is shifted toward lower weights (see Figure 5). Functionally, this means that more babies that are otherwise healthy cross into the 'low-birth weight' category. This will pull the average infant mortality rate for the 'low-birth weight' babies of smoking mothers closer toward that of 'normal-weight' babies.

Testing Ergodicity of Higher-Order Personality Structures

We often see personality descriptions being used to describe more than just differences between individuals. Some researchers will use estimates of common personality inventories to describe larger geographic regions as having particular personality characteristics or suites of characteristics (Rentfrow, Gosling, & Potter, 2008; Rentfrow, Gosling, Jokela, Stillwell, Kosinski, & Potter, 2013). Making such inferences across levels of analysis without empirically testing for ergodicity (high correspondence between levels of analysis) can lead to faulty inferences and (depending on the nature of the research) poor policy recommendations. While previous personality research of this type takes precautions to test for consistence between levels, it is typically done so with respect to each personality factor individually (i.e. means and variance) and not the relations among them (i.e. factor structures). This analysis used data from each of the fifty United States of America to investigate the higher-order factor structures of the Five-Factor Model of Personality (also known as the Big-5) at the level of individual respondents and state-level Aggregate Regional Personalities (ARPs).

Probably the single most utilized personality scheme, the Five-Factor Model of Personality (Costa & McCrae, 1985; Digman & Takemoto-Chock, 1981; hereafter Big-5) is often used as a predictor of various social behaviors. These five domains (Openness to New Experiences, Conscientiousness, Extraversion, Agreeableness, and Emotional Stability) are used separately or in combination to yield pop research predicting everything from taste preferences (Meier, Moeller, Riemer-Peltz, & Robinson, 2012) to social and cognitive outcomes (Hart & Hare, 1994; Wolfe & Johnson, 1995).

Research into the Big-5 has been done at many levels of organization from specific person by situation cases to global international comparisons. With the advent of web-based data collection and the relative ease of setting up these protocols, studies investigating higher-levels of organization (i.e. state-level and above) are becoming more commonplace (e.g. Rentfrow et al., 2013). The nature of these studies typically falls into simple description of Big-5 differences among colloquially discussed regions (e.g. using regions like “The Midwest” or “The South” when investigating the United States of America), ranking regions (national regions, states, providences, etc.) along one or all of the domains, or creating personality profiles that group regions into stereotypically-common clusters (using procedures like latent class analysis). In each of these cases researchers will most commonly pool individual-level scores within a single domain and create a single region-level estimate for the regions of interest. While this is a convenient way of grouping individual cases into regions, great care must be taken in the interpretation of these results. Incorrectly interpreting the results of such a study can lead to incorrectly generalizing effects across levels of aggregation – Simpson’s paradox (Simpson, 1951). Tests of ergodicity are needed to correctly interpret the generalizability of these effects.

These tests for ergodicity, however, rarely test for relations beyond mean and variability consistency. What are often ignored are the structural (measurement) relations among variables. In other words, correlations *can* be observed among variables that exist at multiple levels, but they are either not compared at all or are compared in only a superficial sense. One of the primary purposes of this first analysis is to look at the interrelations among the Big-5 personality domains and to determine whether or not a higher-order factor structure exists in the data, and if it does, whether or not that measurement relationship applies once the data has been aggregated.

Why look at high-order factors of the Big-5? The Big-5 was originally developed to determine *the basic* personality factors. Since the initial development of these factors, examination of the Big-5 intercorrelations has revealed reliable higher-order factor structures. The two most common higher-order factor schemes are the Big-2 (*Alpha* and *Beta*; Digman, 1997) and the Big-1 (General Factor of Personality; GFP; Musek, 2007).

Alpha and *Beta* are two higher-order personality factors that have been characterized as *stability* and *plasticity* (DeYoung, Peterson, & Higgins, 2002). *Alpha* represents stability in emotional, social, and motivational domains, and has been hypothesized to be associated with individual differences in serotonergic functioning. Thus, higher levels of *Alpha* are believed to result in greater degrees of socialization and have been negatively linked to the expression of negative emotions in situations of social stress (DeYoung, Peterson, & Higgins, 2002). Put simply, individuals with higher levels of *Alpha* are more likely to respond to socially stressful situations with less distress. *Beta*, on the other hand, represents flexibility in behavior and cognition, and has been hypothesized to be associated with individual differences in dopaminergic functioning. Thus, higher levels of *Beta* are believed to result in greater degrees of personal growth and self-actualization and have been positively linked to the expression of

positive emotions in situations of social stress (Depue & Collins, 1999; Gray et al., 1997). Put simply, individuals with higher levels of *Beta* are more likely to respond to socially stressful situations with eustress. In individual-level analyses, these two factors are often shown to have a weak to strong bivariate correlation, leading many researchers to suggest a second-level higher-order factor (GFP).

The General Factor of Personality (GFP) is a higher-order personality factor that has been characterized as *social effectiveness* (Dunkel & Van der Linden, 2014). GFP represents higher levels of both *Alpha* and *Beta*, meaning more emotional, social, and motivational stability (social maintenance) in conjunction with greater behavioral and cognitive flexibility (social cultivation) – a personality profile that is typically considered socially desirable. As stated above, individual-level analyses often support the existence of GFP modeled directly or by first modeling *Alpha* and *Beta* (Rushton & Irwing, 2009). What remains to be established, however, is whether or not state-level Aggregate Regional Personalities (ARPs) support either higher-order factor scheme. This study will test the ergodicity of the higher-order factor structure between the individual and State levels.

As has been demonstrated time and again (for a recent review, see Figueredo, Woodley of Menie, & Jacobs, 2015), the Individual-level analyses of the Big-5 domains of human personality should support a single General Factor of Personality (GFP) model. It is hypothesized, however, that the results of the State-level analysis do not support a single GFP model. At this higher level of organization, *Alpha* and *Beta* will act more independently and, thus, not form a single common factor

Methods

To determine the factor structure of the Big-5 at the Individual and State levels, a part-whole correlation analysis was used to describe relations among three unit-weighted composites (*Alpha*, *Beta*, and GFP) and each of the Big-5 domains.

Sample. Data used in this study were downloaded from the myPersonality Project (Kosinski, Matz, Gosling, Popov, & Stillwell, 2015). This project was set up in June 2007 and ended date collection in 2012 with the purpose of collecting data from Facebook on common psychosocial measures, social network activity, and demographic information. From this extensive database (N = 3.1 million), only cases that had all of the relevant variables were included in these analyses. That is, only individuals with Big-5 domain scores (Openness to New Experiences, Conscientiousness, Extraversion, Agreeableness, and Emotional Stability) and a reported current location within one of the United States of America (N_S = 50) were included in these analyses (N_I = 402495). The number of cases within each state was proportional to the 2010 U.S. Census population estimates (U.S. Census Bureau, 2011; Pearson's *r* = .99) and over six hundred cases were present for each state (range: 623-44229, median: 5461) with forty-eight states possessing at least one thousand cases.

Measures. The myPersonality Project collected its Big-5 data using various lengths of the International Personality Item Pool (IPIP) Big-5. This measure has been shown to correlate strongly with other established measures of the Big -5 (most notably, the NEO PI-R; average domain Pearson's *r* = .90 after correcting for unreliability; Goldberg, et al. 2006). Item-level data was not available in this dataset; however, the five aggregate domains (Openness to New Experiences, Conscientiousness, Extraversion, Agreeableness, and Emotional Stability) were available and utilized in this study.

Analysis. All univariate and multivariate analyses were performed using SAS 9.4. At each level of analysis (Individual and State) an analysis of the part-whole correlations between the higher-order factors and each of the Big-5 domains was performed. This was done by creating unit-weighted common factor scores (Gorsuch, 1983) for Stability (*Alpha*), Plasticity (*Beta*), and the General Factor of Personality (GFP) and taking bivariate correlation s between those scores and the scores for each Big-5 domain using SAS PROC CORR.

A part-whole correlation analysis was done to look at the relations among three unit-weighted composites (*Alpha*, *Beta*, and GFP) and each of the domains. This procedure was used for two primary reasons. Firstly, because the procedure is based entirely on correlations, the minimum sample size required is determined only by the sample size required for effect size stability. This is essential for the State-level analyses as the total sample size is N=50. Secondly, unlike the results of Confirmatory or Exploratory Factor Analysis, part-whole correlation-based procedures will tend to generalize better across samples as the latent factor estimation does not fit to sample-specific conditions to the same degree. This procedure is similar to procedures used in classical test theory and scale construction for item selection. In this case, however, the analysis is not being used to determine which items to drop, but the structural relations among the indicators (parts) and composite scores (whole) to test higher-order factor structure.

Unit-weighted factor scores (i.e. wholes) were constructed using SAS PROC STANDARD and the DATA step. To create these higher-order Aggregate Regional Personalities (ARPs), the arithmetic average was taken of the standardized (mean = 0, standard deviation = 1) state-level estimates of the Big-5 domains. This was done for GFP (all five domains), *Alpha* (Agreeableness, Conscientiousness, and Emotional Stability) and *Beta* (Openness to New

Experiences and Extraversion). Bivariate correlations between ARPs and each Big-5 domain were then computed and assessed.

For the Individual-level analysis, State membership was factored out of each domain before creating ARPs using SAS PROC REG in order to account for any systematic differences between individuals due to geographic location and not due purely to individual differences. Due to the large sample size, the Individual-level intercorrelations should be stable and closely approximate the population estimates (N = 402495). Because of this assumption an absolute cutoff of Pearson's r = .40 was used as that is typically considered an acceptable part-whole correlation (factor loading).

For the State-level analysis, a cutoff of Pearson's r = .60 was used. This cutoff was chosen as that is the point at which the correlation stabilize within a corridor window of generally acceptable part-whole correlations (between [.40, .80]) with a high degree of confidence (95% confidence in correlation stability; Schönbrodt & Perugini, 2013).

If a domain "belongs" to a particular ARP, it will be above the pre-determined cutoff; otherwise, the domain is determined to not belong to that ARP. If all five domains load highly and positively onto the GFP ARP, then a single GFP model is assumed to be the best model as it is the most parsimonious model meeting the criteria. If instead a two-factor model is a better fit then Agreeableness, Conscientiousness, and Emotional Stability will be above threshold for *Alpha* but not for *Beta*, and Openness and Extraversion will be above threshold for *Beta* but not for *Alpha*.

Results

The results of the part-whole correlations analysis can be seen in Tables 5 and 6.

Individual-level. The Individual-level data are consistent with a GFP model, as is typically found in the literature. The part-whole correlations for each Big-5 domain were above the Pearson's $r = .40$ cutoff (Table 5 – GFP column). Additionally, the data was consistent with an *Alpha* and *Beta* model (Table 5– *Alpha* and *Beta* columns). In this case, *Alpha* and *Beta* significantly and positively correlate ($r = .25$, $p < .0001$), further supporting a GFP model.

Table 5. *Part-whole correlation structure of Individual-level personality analysis*

Indicator	**Part-Whole Correlation**		
	GFP	*Alpha*	*Beta*
Conscientiousness	**.57***	**.69***	.12*
Agreeableness	**.60***	**.71***	.15*
Emotional Stability	**.71***	**.77***	.27*
Openness to New Experiences	**.43***	.06*	**.76***
Extraversion	**.62***	.32*	**.76***

Note: N = 402495; Bolded correlations are above the $|r| = .40$ cutoff; * denotes $p < .0001$; Correlation between *Alpha* and *Beta* is $r = .25$, $p < .0001$

State-level. The State-level data are not consistent with a GFP model. Only two part-whole correlations were above the Pearson's $r = .60$ cutoff: Conscientiousness and Emotional Stability. Agreeableness, Openness to New Experiences, and Extraversion failed to meet the cutoff with an average correlation of .46 (Table 6 – GFP column). The data were, however, consistent with an *Alpha* and *Beta* model (Table 6 – *Alpha* and *Beta* columns). In this case, *Alpha* and *Beta* did positively correlate but that correlation is not statistically significant (r = .11,

p = .4118), indicating that *Alpha* and *Beta* aggregated at the State level operate more or less orthogonally. In other words, they behave as if they are independent.

Table 6. *Part-whole correlation structure of State-level personality analysis*

Indicator	**Part-Whole Correlation**		
	GFP	*Alpha*	*Beta*
Conscientiousness	**.75***	**.78***	.24
Agreeableness	.45	**.79***	-.35
Emotional Stability	**.87***	**.84***	.39
Openness to New Experiences	.48	.11	**.77***
Extraversion	.45	.07	**.77***

Note: N = 50; Bolded correlations are above the $|r|$ = .60 cutoff; * denotes $p < .0001$; Correlation between *Alpha* and *Beta* is r = .12, p = .4118.

Discussion

The goal of this analysis was to determine whether or not ergodicity in the higher-order factor structure of the Big-5 existed between the individual level and the state level. This is one of the few (if not the first) case of a test of ergodicity being applied to factor structures across levels of aggregation.

The results of this study failed to demonstrate a high correspondence between the individual level and state level higher-order factor structures. The individual-level data supported a General Factor of Personality (GFP), as is typically found in individual difference research of higher-order Big-5 factors (van der Linden, te Nijenhuis, & Bakker, 2010; Rushton & Irwing, 2009; Figueredo, Woodley of Menie, & Jacobs, 2015). The state-level data, however, supported

a Big-2 model in which *Alpha* and *Beta* had only a weak, positive correlation (not statistically significant).

It should be noted that with more a more lax state-level criterion (r = .40 cutoff instead of r = .60), the GFP model would be supported. Due to the small overall sample size, however, the stricter criterion was needed to ensure stable correlation estimates with a high degree of confidence (Schönbrodt & Perugini, 2013). Further, the GFP model would yield a rather asymmetric GFP with the average *Alpha* part-whole correlation being approximately .70, while the average *Beta* part-whole correlation would be closer to .45.

Cross-validation on independent samples will need to be performed in the future to verify this discrepancy between the individual- and state-level factor structures, but for the time being is seems that *Alpha* and *Beta* function independently at the state-level. This is likely due to the fact that group selective pressures are not always the same as individual selective pressures. For example GFP will serve individuals well in any complex social system, but individual groups may differentially use *Alpha* or *Beta* to solve local group adaptive problems. A region in which several genetically-restricted groups co-exist among other heterogeneous groups would not be conducive to high *Beta* conditional upon certain ecological conditions, such as high parasite burden. In contrast, several genetically-diverse groups co-existing among other heterogeneous groups would not likely be subject to the same pressure against interaction among groups and may, in fact, receive pressures that facilitate such interactions (e.g. intergroup commerce).

CHAPTER 4: THE MEASUREMENT MODEL

The purpose of this chapter is to provide the anatomy (parts) of the causal cascade model. Operationalizations of the relevant constructs being investigated are presented. While many constructs have a single operationalization, some constructs are modeled as composites representing common factors. The internal structure and performance of these composites are discussed where relevant.

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The Anatomy of the Model

Methods

Sample. The present study models the social determinants of group-directed altruistic behavior, operationalized as the rate of kidney donations recorded in each state for the period spanning 1988-2015, using the United States of America (N = 50) as the units of analysis at the aggregate state level.

Measures. All of the data used in this study is publically-available. Most of the sources are entirely open source, while others are free but require permissions for use.

Community ecology

Parasite burden. A parasite burden index was taken from a previous study investigating parasite-stress in the US (Fincher & Thornhill, 2012). This index was created using the *Morbidity and Mortality Weekly Report*'s 'Summary of Notifiable Diseases, United States' produced by the Center for Disease Control. The incidence counts provided by the CDC of various infectious diseases were converted into incidence rates by dividing by the CDC-reported state population size. Data from 1993 to 2007 was collapsed for each state in order to create a stable estimate of parasite burden over a 15-year period. These incidence rates were combined

using a unit-weighting procedure (Figueredo, McKnight, McKnight, & Sidani, 2000; Gorsuch, 1983) to yield a single parasite burden score. A list of the infectious diseases included in the index can be found in Electronic Supplement 5 of Fincher and Thornhill (2012).

Social ecology

Population density. Estimates of population density were derived for each US state by taking its 2010 US Census population estimates (U.S. Census Bureau, 2011) and dividing by the square land area of each state (U.S. Census Bureau, 2010a). The final result was the population per square mile.

Proportion of Whites. The proportion of self-identified White, non-Hispanic residents within each state was taken from 2010 US Census data (U.S. Census Bureau, 2011)

Genetic relatedness. Estimates of genetic relatedness were calculated using data from four studies investigating ancestral histories (Bryc, Durand, Macpherson, Reich, & Mountain, 2015; Granka, 2014) and genetic markers (Li et al., 2008; Jorde et al., 2000). These data were combined using the unit-weighting procedure described in Chapter 2. More positive scores indicate more genetic relatedness (genetic homogeneity).

LH strategy. State-level socio-demographic data were collected from various 2010 US Census data. Biometric indicators of LH strategy include: teen birth rate (per 1000 women ages 15-19), infant mortality rate (deaths per 1000 live births), life expectancy at birth, and rate of child deaths (per 100000 children ages 1-14). These indicators were combined using a unit-weighting procedure to yield a single biometric LH strategy score (K-factor).

State-level estimate of covitality were taken from the Gallup-Healthways Well-Being Index (Gallup, 2011). This composite score is comprised of six subscales: general life evaluation, emotional health, physical health, healthy behaviors, access to basic care, and perception of work

environment. As the bivariate correlation between K-factor and covitality was $r = .67$ ($t(48) = 6.253$, $p < .0001$), the two indicators were combined to yield a unit-weighted, higher-order Super-K Factor (Super-K). More positive Super-K scores indicate slower LH strategies

Equality. The Gini index (U.S. Census Bureau, 2010b) was used as a proxy for social equality. Previous work has used the index as a measure of social equality that correlated with general regional variations in egalitarianism (Black, Peñaherrera-Auirre, Minera, & Figueredo, 2017). The Gini index was reversed so that more positive scores indicate more Equality.

SD-IE of biodemographic-estimated LH strategy. The average part-whole correlation for the biodemographically-estimated LH strategy factor and its indicators were calculated for each US state. This was calculated by taking the arithmetic average of the cross-product of the standardized biodemographic-estimated LH strategy factor with each of the standardized indicators. Effects positively associated with this SD-IE variable indicate strategic differentiation (as the part-whole correlations increase as a function of the predictor), while negative associations will indicate integration (as the part-whole correlations decrease as a function of the predictor).

Cultural ecology

Income (Human Capital). Median household income was obtained from 2010 US Census data (U.S. Census Bureau, 2012a).

Familism. A measure of state-level familism was adapted from a measure of collectivism created by Vandello and Cohen (1999), restricting the breadth of the construct to characteristics relating to either the nuclear or extended family and not to any higher levels of social organization. This restriction was imposed to test more focused hypotheses derived from inclusive fitness and multilevel selection theories than is usually done in conventional studies of

collectivism. This measure of familism was derived by creating a unit-weighted factor from four state-level indicators found within the 2010 US Census data (U.S. Census Bureau, 2012b): number of family members in the household, percentage of people living alone (reversed), percentage of elderly people (65 +) living alone (reversed), and percentage of households with grandchildren in them. More positive numbers indicate a higher degree of familism.

Higher-order factor structure of Big 5 (group). Group-level personality factors were estimated using data from the myPersonality Project (Kosinski, Matz, Gosling, Popov, & Stillwell, 2015). The exact factor pattern was determined in the procedure described in Chapter 3. *Alpha* was a unit-weighted average of state-level averages of Agreeableness, Conscientiousness, and Emotional Stability, while Beta was a unit-weighted average of state-level averages of Openness to New Experiences and Extraversion. These "aggregate regional personalities" (ARPs) are thus unit-weighted composite group-level personality aggregates that reflect the underlying higher-order factor structure of the Big-5.

Cooperation

Empathy. In an attempt to get at some sort of proximate measure in predicting NDKD, state-level Empathy scores were included. The Total Empathy Scores of the Interpersonal Reactivity Index (Davis, 1983) were taken from Bach, Defever, Chopik, and Konrath (2017). This was chosen as it has been shown to associate with indicators of Super-K such as life satisfaction, emotional intelligence, and self-esteem (Eisenberg et al., 2006; Mayer et al., 2000; Richardson et al., 1994) as well as higher rates of prosocial behaviors like volunteering, donating money to charity, and helping others in need (Davis, 1983; Grühn et al., 2008; Konrath, 2014; Wilhelm & Bekkers, 2010)

Altruism. Altruism was measured by the per capita living-donor, kidney donations for each of the fifty US states. Two forms of altruism were considered in this study: altruism toward kith and kin (per capita living-donor, directed kidney donation rates; Kidney Donations (K&K)) and altruism toward strangers (per capita living-donor, non-directed kidney donation rates; Kidney Donations (Undirected)). Rates will be obtained via the method described in Chapter 3. The total number of kidney donations in each category was divided by 2010 US Census adult population estimates.

Results

Statistical Analyses. All univariate analyses were performed using SAS 9.4. Part-whole correlations representing factor structure coefficients were estimates using SAS PROC CORR. See Chapter 2 for the rationales underlying this procedure.

Measurement models. The results of the part-whole correlations analysis can be seen in Tables 7-12.

Table 7 shows the part-whole correlations of the various Slow LH indicators. All correlations were very strong (magnitudes ranging from .82 to .93).

Table 7. *Part-whole correlation structure s of slow life history (LH)*

Slow LH Indicator	Part-Whole Correlation
Life Expectancy	.93
Teen Birth Rate*	.89
Infant Mortality*	.82
Child Death*	.84

Note: * denoted indicator is reversed; N = 50; $p < .0001$

Table 8 shows the part-whole correlations of the two Super-K indicators. Both correlations were very strong ($r = .90$).

Table 8. *Part-whole correlation structure of Super-K*

Super-K Indicator	**Part-Whole Correlation**
Slow LH	.90
Covitality	.90

Note: * denoted indicator is reversed; N = 50; $p < .0001$

Table 9 shows the part-whole correlations of the various Strategic Differentiation indicators. All correlations were very strong (magnitudes ranging from .92 to .97).

Table 9. *Part-whole correlation structure of Strategic Differentiation*

Strategic Differentiation Indicator	**Part-Whole Correlation**
Life Expectancy x Slow LH	-.97
Teen Birth Rate* x Slow LH	-.95
Infant Mortality* x Slow LH	-.95
Child Death* x Slow LH	-.92

Note: * denoted indicator is reversed; N = 50; $p < .0001$

Table 10 shows the part-whole correlations of the various Familism indicators. All correlations were very strong (magnitudes ranging from .77 to .92).

Table 10. *Part-whole correlation structure of Familism*

Familism Indicator	**Part-Whole Correlation**
# of family members in household	.90
% of people living alone*	.92
% of elderly (65+) living alone*	.84
% of households with grandchildren	.77

Note: * denoted indicator is reversed; N = 50; $p < .0001$

Table 11 (modified from Chapter 3) shows the part-whole correlations of the various Big-5 indicators. All correlations were very strong (magnitudes ranging from .77 to .84).

Table 11. *Part-whole correlation structure of Alpha and Beta*

Indicator	**Part-Whole Correlation**	
	Alpha	*Beta*
Conscientiousness	.78	
Agreeableness	.79	
Emotional Stability	.84	
Openness to New Experiences		.77
Extraversion		.77

Note: N = 50; $p < .0001$; Correlation between *Alpha* and *Beta* is $r = .12$, $p = .4118$.

Table 12 (from Chapter 2) shows the part-whole correlations of the various genetic relatedness composites. All correlations were very strong (magnitudes ranging from .63 to .97).

Table 12. *Part-whole correlation structure of Genetic Relatedness*

Composite	**Genetic Relatedness**
23andMe X Li	.97
Ancestry X Li	.76
23andMe X Jorde	.93
Ancestry X Jorde	.90

Note: N = 50; $p < .0001$

Discussion

This chapter outlined the key constructs to be integrated into the causal cascade model presented in the next chapter (Chapter 5). The composites representing Slow LH, Super-K, Strategic Differentiation, and Familism all performed exceptionally well – indicating single common factors. Composites derived in Chapters 2 and 3 were also reiterated here. Specifically, higher-order personality structure is being represented by the Big-2 (*Alpha* and *Beta*) and not a General Factor of Personality as it fails to replicate at the state-level (Chapter3). Additionally, Genetic Relatedness is being represented by a single higher-order composite created from four

lower-order composites (as described in Chapter 2) – comprised of molecularly distinct indicators and two unique ancestral estimation procedures.

CHAPTER 5: THE STRUCTURAL MODEL

The purpose of this chapter is to provide the physiology (performance) of the causal cascade model. The constructs discussed in Chapter 4 are integrated into a causal cascade represented by a system of linear regression equations. The results of the statistical analyses are presented and a discussion of the immediate nomological net surrounding living-donor, non-directed kidney donations follows. Additional interesting effects are also discussed.

The Physiology of the Model

Methods

Statistical Analyses. All multivariate analyses were performed using UniMult 2.

Sequential Canonical Analysis (SEQCA) was utilized in this study in order to accomplish two necessary functions: 1) to model a theoretically-specified *cascade* of hypothesized effects and 2) to permit exploratory analyses for unspecified residual relations (Figueredo, Garcia, Cabeza de Baca, Gable, & Weiss, 2013; Figueredo & Gorsuch, 2007).

While most statistical analyses are classified as either confirmatory or exploratory, SEQCA is unique in that it has both confirmatory and exploratory features. On the confirmatory side, a series of hierarchical regressions (sequential or Type I SS) is constructed such that the outcome of the previous step serves as the first predictor of the next step (Step 1: Y1 = X; Step 2: Y2 = Y1 + X). This ordering gives partitioning priority to variables entered earlier in the model specification, assuming a direct effect of the outcome of the previous step on the current (i.e. in Step 2 Y1 is assumed to have a direct effect on Y2).

The exploratory side of SEQCA comes from the testing of the direct distal effects after controlling more causally proximate variables (the mediators of any indirect effects). All of these

residual effects are tested and cannot be pre-specified as can be done in methods such as Structural Equations Modeling. Any effect automatically estimated by the model, following the procedure described, that is not specified as a priori hypotheses should therefore be considered an exploratory result, which must eventually be subjected to cross-validation on an independent sample to be fully supported by the empirical evidence. As there are not likely to be more than 50 states in the USA any time in the near future, conceptual cross-validation will have to be carried out on the sub-national regions of other polities (Black, Peñaherrera Aguirre, Chavarria Minera, & Figueredo, 2017; Cabeza de Baca & Figueredo, 2017; Fernandes, Figueredo, Garcia, & Wolf , 2017; Fernandes, & Woodley of Menie, 2017; Figueredo, Cabeza de Baca, & Peñaherrera Aguirre, 2017; Figueredo, Fernandes, & Woodley of Menie, 2017) or replication within the 50 states in the USA across time**.**

Model. This cascade can be described by the following system of linear equations:

1. Proportion Whites = $\beta_{4,1}$* Population Density + $\beta_{4,2}$* Parasite Burden + $\beta_{4,3}$* DensityXParasites
2. Genetic Relatedness = $\beta_{5,4}$* Proportion Whites + $\beta_{5,1}$* Population Density + $\beta_{5,2}$* Parasite Burden + $\beta_{5,3}$* DensityXParasites
3. Super-K = $\beta_{6,5}$* Genetic Relatedness + $\beta_{6,4}$* Proportion Whites + $\beta_{6,1}$* Population Density + $\beta_{6,2}$* Parasite Burden + $\beta_{6,3}$* DensityXParasites
4. Equality = $\beta_{7,6}$* Super-K + $\beta_{7,5}$* Genetic Relatedness + $\beta_{7,4}$* Proportion Whites + $\beta_{7,1}$* Population Density + $\beta_{7,2}$* Parasite Burden + $\beta_{7,3}$* DensityXParasites
5. Strategic Differentiation = $\beta_{8,7}$* Equality + $\beta_{8,6}$* Super-K + $\beta_{8,5}$* Genetic Relatedness + $\beta_{8,4}$* Proportion Whites + $\beta_{8,1}$* Population Density + $\beta_{8,2}$* Parasite Burden + $\beta_{8,3}$* DensityXParasites

6. Income = $\beta_{9,8}$* Strategic Differentiation + $\beta_{9,7}$* Equality + $\beta_{9,6}$* Super-K + $\beta_{9,5}$* Genetic Relatedness + $\beta_{9,4}$* Proportion Whites + $\beta_{9,1}$* Population Density + $\beta_{9,2}$* Parasite Burden + $\beta_{9,3}$* DensityXParasites

7. Familism = $\beta_{10,9}$* Income + $\beta_{10,8}$* Strategic Differentiation + $\beta_{10,7}$* Equality + $\beta_{10,6}$* Super-K + $\beta_{10,5}$* Genetic Relatedness + $\beta_{10,4}$* Proportion Whites + $\beta_{10,1}$* Population Density + $\beta_{10,2}$* Parasite Burden + $\beta_{10,3}$* DensityXParasites

8. *Alpha* (A + C + ES) = $\beta_{11,10}$* Familism + $\beta_{11,9}$* Income + $\beta_{11,8}$* Strategic Differentiation + $\beta_{11,7}$* Equality + $\beta_{11,6}$* Super-K + $\beta_{11,5}$* Genetic Relatedness + $\beta_{11,4}$* Proportion Whites + $\beta_{11,1}$* Population Density + $\beta_{11,2}$* Parasite Burden + $\beta_{11,3}$* DensityXParasites

9. *Beta* (O + E) = $\beta_{12,11}$* *Alpha* + $\beta_{12,10}$* Familism + $\beta_{12,9}$* Income + $\beta_{12,8}$* Strategic Differentiation + $\beta_{12,7}$* Equality + $\beta_{12,6}$* Super-K + $\beta_{12,5}$* Genetic Relatedness + $\beta_{12,4}$* Proportion Whites + $\beta_{12,1}$* Population Density + $\beta_{12,2}$* Parasite Burden + $\beta_{12,3}$* DensityXParasites

10. Empathy = $\beta_{13,12}$* *Beta* + $\beta_{13,11}$* *Alpha* + $\beta_{13,10}$* Familism + $\beta_{13,9}$* Income + $\beta_{13,8}$* Strategic Differentiation + $\beta_{13,7}$* Equality + $\beta_{13,6}$* Super-K + $\beta_{13,5}$* Genetic Relatedness + $\beta_{13,4}$* Proportion Whites + $\beta_{13,1}$* Population Density + $\beta_{13,2}$* Parasite Burden + $\beta_{13,3}$* DensityXParasites

11. Kidney Donations (K&K) = $\beta_{14,13}$* Empathy + $\beta_{14,12}$* *Beta* + $\beta_{14,11}$* *Alpha* + $\beta_{14,10}$* Familism + $\beta_{14,9}$* Income + $\beta_{14,8}$* Strategic Differentiation + $\beta_{14,7}$* Equality + $\beta_{14,6}$* Super-K + $\beta_{14,5}$* Genetic Relatedness + $\beta_{14,4}$* Proportion Whites + $\beta_{14,1}$* Population Density + $\beta_{14,2}$* Parasite Burden + $\beta_{14,3}$* DensityXParasites

12. Kidney Donations (Undirected) = $\beta_{15,14}$* Kidney Donations (K&K) + $\beta_{15,13}$* Empathy + $\beta_{15,12}$* *Beta* + $\beta_{15,11}$* *Alpha* + $\beta_{15,10}$* Familism + $\beta_{15,9}$* Income + $\beta_{15,8}$* Strategic Differentiation + $\beta_{15,7}$* Equality + $\beta_{15,6}$* Super-K + $\beta_{15,5}$* Genetic Relatedness + $\beta_{15,4}$* Proportion Whites + $\beta_{15,1}$* Population Density + $\beta_{15,2}$* Parasite Burden + $\beta_{15,3}$* DensityXParasites

Results

Cascade model. Table 13 displays the results of the Sequential Canonical Analysis Cascade Model. Parameter estimates are semipartial correlation coefficients (*sR*; see Cohen and Cohen, 1983), statistically controlling for all hierarchically prior predictors (as described previously).

Table 13. *Sequential Canonical Analysis predicting non-directed kidney donations*

Criterion Variables	Prior Criterion Variables	Predictor Variables	*df*	Semipartial Correlation	*p*(H0)
Proportion White		Population Density	1/46	-.19	.12
		Parasite Burden	**1/46**	**-.57**	**<.0001**
		Density X Parasites	1/46	.02	.86
Genetic Relatedness	Proportion White		1/45	.15	.16
		Population Density	1/45	-.18	.10
		Parasite Burden	**1/45**	**-.65**	**<.0001**
		Density X Parasites	1/45	-.08	.49
Super K	**Genetic Relatedness**		**1/44**	**.55**	**<.0001**
	Proportion White		1/44	-.09	.39
		Population Density	**1/44**	**.41**	**.0002**
		Parasite Burden	**1/44**	**-.23**	**.02**
		Density X Parasites	**1/44**	**.22**	**.03**
Equality (-GINI)	**Super K**		**1/43**	**.31**	**.003**
	Genetic Relatedness		**1/43**	**.39**	**.0003**
	Proportion White		**1/43**	**.35**	**.0008**
		Population Density	**1/43**	**-.43**	**<.0001**
		Parasite Burden	1/43	.07	.50
		Density X Parasites	1/43	.18	.07
Strategic Differentiation	**Equality (-GINI)**		**1/42**	**.38**	**.001**
	Super K		**1/42**	**.29**	**.01**
	Genetic Relatedness		1/42	.19	.10
	Proportion White		1/42	-.10	.39
		Population Density	1/42	-.04	.70
		Parasite Burden	1/42	.00	.90

		Density X Parasites	**1/42**	**.46**	**.0002**
Income	**Strategic Differentiation**		**1/41**	**.17**	**.01**
	Equality (-GINI)		1/41	.09	.18
	Super K		**1/41**	**.74**	**<.0001**
	Genetic Relatedness		1/41	-.12	.07
	Proportion White		**1/41**	**-.18**	**.009**
		Population Density	**1/41**	**.43**	**<.0001**
		Parasite Burden	1/41	-.03	.66
		Density X Parasites	1/41	.11	.10
Familism	**Income**		**1/40**	**.29**	**.006**
	Strategic Differentiation		1/40	-.04	.71
	Equality (-GINI)		1/40	-.03	.78
	Super K		**1/40**	**-.20**	**.05**
	Genetic Relatedness		1/40	-.01	.91
	Proportion White		**1/40**	**-.68**	**<.0001**
		Population Density	1/40	-.06	.54
		Parasite Burden	1/40	-.05	.59
		Density X Parasites	1/40	-.09	.38
Alpha (State level)	**Familism**		**1/39**	**.46**	**.0001**
	Income		**1/39**	**-.36**	**.002**
	Strategic Differentiation		**1/39**	**.26**	**.02**
	Equality (-GINI)		**1/39**	**.26**	**.02**
	Super K		1/39	.11	.32
	Genetic Relatedness		1/39	.00	.90
	Proportion White		1/39	-.02	.84
		Population Density	1/39	-.21	.06
		Parasite Burden	1/39	.13	.24
		Density X Parasites	1/39	.02	.88
Beta (State level)	***Alpha* (State level)**		**1/38**	**.12**	**.03**
	Familism		**1/38**	**.52**	**<.0001**
	Income		**1/38**	**-.12**	**.04**
	Strategic Differentiation		**1/38**	**-.13**	**.02**
	Equality (-GINI)		**1/38**	**-.66**	**<.0001**
	Super K		**1/38**	**-.14**	**.02**
	Genetic Relatedness		**1/38**	**-.16**	**.005**
	Proportion White		**1/38**	**-.11**	**.04**
		Population Density	**1/38**	**.21**	**.0004**
		Parasite Burden	**1/38**	**-.12**	**.04**
		Density X Parasites	**1/38**	**-.12**	**.03**
Empathy (State level)	*Beta* (State level)		1/37	-.10	.48
	Alpha (State level)		1/37	-.05	.70
	Familism		1/37	-.11	.44
	Income		1/37	.09	.54
	Strategic Differentiation		1/37	-.18	.21
	Equality (-GINI)		1/37	-.19	.17
	Super K		**1/37**	**.40**	**.007**
	Genetic Relatedness		1/37	.05	.75
	Proportion White		1/37	.10	.46
		Population Density	1/37	.02	.87
		Parasite Burden	1/37	.00	.90
		Density X Parasites	1/37	-.03	.85
Kidney Donations (K&K)	Empathy (State level)		1/36	.21	.08
	***Beta* (State level)**		**1/36**	**-.36**	**.004**
	***Alpha* (State level)**		**1/36**	**-.26**	**.03**
	Familism		1/36	-.01	.90
	Income		**1/36**	**.46**	**.0004**

	Strategic Differentiation		1/36	.04	.76
	Equality (-GINI)		1/36	.10	.40
	Super K		1/36	-.10	.41
	Genetic Relatedness		1/36	-.13	.29
	Proportion White		1/36	.11	.36
		Population Density	1/36	-.01	.92
		Parasite Burden	1/36	.09	.44
		Density X Parasites	1/36	.03	.81
Kidney Donations (Undirected)	**Kidney Donations (K&K)**		**1/35**	**.55**	**<.0001**
	Empathy (State level)		**1/35**	**.27**	**.002**
	Beta (State level)		1/35	-.15	.07
	Alpha (State level)		1/35	.13	.11
	Familism		**1/35**	**.37**	**<.0001**
	Income		1/35	.12	.14
	Strategic Differentiation		1/35	.04	.61
	Equality (-GINI)		1/35	-.13	.10
	Super K		**1/35**	**.31**	**.0004**
	Genetic Relatedness		1/35	.06	.42
	Proportion White		**1/35**	**.30**	**.0007**
		Population Density	1/35	.03	.73
		Parasite Burden	1/35	-.06	.47
		Density X Parasites	1/35	-.05	.55

Notes: Statistically significant effects are indicated in bold ($p < .05$)

The overall pooled multivariate effect size for the model was moderate (Pillai-Bartlett $V = 1.870$, E = .39, $p < .0001$). The results and significant associations of the models are presented below:

1. Proportion Whites was negatively predicted by greater Parasite Burden.
2. Genetic Relatedness was negatively predicted by greater Parasite Burden.
3. Super-K was predicted by greater Genetic Relatedness, greater Population Densities, negatively predicted by Parasite Burden, and a significant positive Population Density X Parasite Burden interaction.
4. Social Equality was predicted by Slow Life History, greater Genetic Relatedness, greater Proportion Whites, and greater Population Density.
5. Strategic Differentiation was predicted by greater Social Equality, Slow Life History, and a significant positive Population Density X Parasite Burden interaction.

6. Median state Income was predicted by greater Strategic Differentiation, Slow Life History, negatively predicted by Proportion of Whites, and greater Population Density.
7. Familism was predicted by greater median state Income, Fast Life History, and negatively predicted by Proportion Whites.
8. *Alpha* was predicted by greater Familism, lower median state Income, greater Strategic Differentiation, and greater Social Equality.
9. *Beta* was predicted by greater *Alpha*, greater Familism, lower median state Income, less Strategic Differentiation, less Social Equality, Fast Life History, less Genetic Relatedness, negatively predicted by Proportion Whites, greater Population Density, negatively predicted by Parasite Burden, and a significant negative Population Density X Parasite Burden interaction.
10. Empathy was predicted by Slow Life History.
11. Kidney Donations (K&K) was predicted by lower levels of *Beta*, lower levels of *Alpha*, greater median state Income.
12. Kidney Donations (Undirected) was predicted by greater Kidney Donations (K&K), greater Empathy, greater Familism, Slow Life History, and greater Proportion Whites.

A summary of the results and their relationships to their predictions is depicted in Table 14.

Table 14. *Results and their relations to predictions*

Outcome variable	Replications from previous work	Untested theoretical predictions	Exploratory relationships
Proportion Whites		Parasite Burden	
Genetic Relatedness		Parasite Burden	
Super-K	Population Density; Parasite Burden	Genetic Relatedness	
Social Equality	Super-K	Genetic Relatedness	Population Density; Proportion Whites
Strategic Differentiation	Social Equality; Super-K		
Median State Income	Strategic Differentiation; Super-K		Population Density; Proportion Whites
Familism	Super-K	Median State Income	Proportion Whites
Alpha	~~Super-K~~	Familism; Social Equality	Median State Income; Strategic Differentiation;
Beta		*Alpha*; Familism; Social Equality; Population Density	Parasite Burden; Median State Income; Strategic Differentiation; Super-K; Genetic Relatedness; Proportion Whites
Empathy		~~*Alpha*~~; Super-K	
Kidney Donations (Kith & Kin)		~~Empathy~~; ~~Familism~~; Income; ~~Genetic Relatedness~~	*Alpha*; *Beta*
Kidney Donations (Undirected)	~~Income~~	Kidney Donations (Kith & Kin); Empathy; ~~Genetic Relatedness~~	Proportion Whites; Super-K; Familism

Note: Relationships in normal case were detected as predicted except: (a) relationships that were hypothesized, but not detected are ~~struck out~~; (b) relationships that were detected but in the opposite direction to the hypotheses are in red; and (c) relationships in the 'Exploratory' column were not hypothesized.

Discussion

The purpose of this study was to partially map the nomological net around group-directed altruistic behavior. The model tested in this study demonstrated several biological, social, and cultural variables contribute to living-donor, non-directed kidney donations (NDKD) at the state level. Specifically, NDKD were predicted by 1) greater kidney donations to kith and kin (indicating that NDKD may be an extraordinary, almost pathological form of the general altruism trait), 2) greater levels of empathy (indicating that the ability to empathize and perspective-taking may serve as a prosocial motivator), 3) greater degrees of familism (indicating that strong family values may promote a "giving" cultural norm), 4) slower life history strategies (indicating that mutualistic social schema), and 5) greater proportion of self-reported Whites.

It should be noted that of the final SEQCA step, while familism, slow life history, and proportion of self-reported Whites were not initially predicted, the only truly surprising effect is the proportion of self-reported Whites (pWhites). In fact, the behavior of pWhites was rather unusual. pWhites predicted: social equality (positively), median state income (negatively), familism (negatively), *beta* (negatively), and NDKD (positively). Based on these findings, what is the proportion of self-reported Whites measuring? One possible explanation is that pWhites may be a proxy for *postmaterialistic values* (Woodley of Menie, Sarraf, Pestow, & Fernandes, 2017). Postmaterialism is characterized by the spread of individualistic (non-familistic), secular, and anti-hierarchical (social equality) values (Welzel, 2013). Postmaterialism has also been linked to pathological altruism, which some could argue NDKD could be classified as (Oakley, 2013; Oakley, Knafo, Madhavan, & Wilson, 2012). Postmaterialism by its design forgoes

economic security (median state income) in favor of autonomy and self-expression. The only predictor that does not fit this pattern is the negative relationship between pWhites and *Beta*; however, the effect is very small (*sR* = -.11) and may simply be an artifact of restriction of range for *Beta*. pWhites being a proxy for postmaterialistic values would also be consistent with the finding that parasite burden predicts pWhites, as increased environmental pressures (of which greater parasite burden would be indicative) would restrict the ability of postmaterialism to spread (Woodley of Menie, Sarraf, Pestow, & Fernandes, 2017).

Another unusual finding was the strong positive relationship between Familism and *Beta* (sR = .52). As stated for pWhites, part of this large effect may be an artifact of restricted range for *Beta*; however, that cannot account for the entirety of the correlation's magnitude. One possibility is that the *% of households with grandchildren* item of *Beta* is driving this effect. A household with grandchildren will also have grandparents and presumably the intermediate generation. Three generations will typically span 50 -75 years of experience among all members of the household. Living in close quarters with such varied historical upbringings can force a certain degree of openness, particularly for older generations adapting to the new, more liberal computer age. If this item is removed from Familism and rerun in the SEQCA, the pattern of effects does not change; however, the effect size does drop in magnitude noticeably (sR = .38).

CHAPTER 6: GENERAL DISCUSSION

The primary objective of this dissertation was to explore the nomological net surrounding group-directed altruistic behavior (operationalized as living-donor, non-directed kidney donations; NDKD) using a causal cascade model derived from life history theory. To accomplish this Sequential Canonical Analysis (SEQCA) was implemented incorporating various biological and social predictors to ultimately predict NDKD.

Had the goal of this dissertation been simply to *predict* NDKD, a SEQCA would not have been necessary. In fact a simple hierarchical regression would yield virtually identical results to the last step of the SEQCA ($R^2 = .72$, including statistically significant predictors only within this restricted model). This project, however, was about more than mere prediction. Its goal was to attempt to *explain* NDKD from a LH framework. Explanation in this sense is to anchor the results into theory either existing or developed in light of the empirical findings (Shmueli, 2010).

The entire set of equations in SEQCA permits one to map a series of mediating processes – gaining insight into not just what predicts the ultimate outcome but also what may give rise to those predictors. This is necessary as a single multiple regression/correlation model can only detect *direct* effects and is by itself incapable of detecting *indirect* effects. Such an in-depth exploration articulates a specific exploratory model that can be empirically supported (or rejected) in subsequent studies. The purpose of exploratory research must be understood to function more in theory *construction* than in theory *testing*. As such, the next section intentionally uses causal language to articulate a *tentative* causal theory of NDKD – being quite aware of the fact that these causal statements are drawn from correlational data. What follows is therefore the theory that was constructed using this procedure.

A Causal Life History Theory of Living-Donor, Non-Directed Kidney Donations

A tentative theoretical life history theory of NDKD is depicted schematically in Figure 6. For the verbal description that follows, the following assumptions will be made: 1) parasite burden can be used as a proxy for overall *environmental agency*; 2) proportion of Whites can be used as a proxy for *postmaterialistic values*; and 3) median state income can be used as a proxy for overall *human capital*.

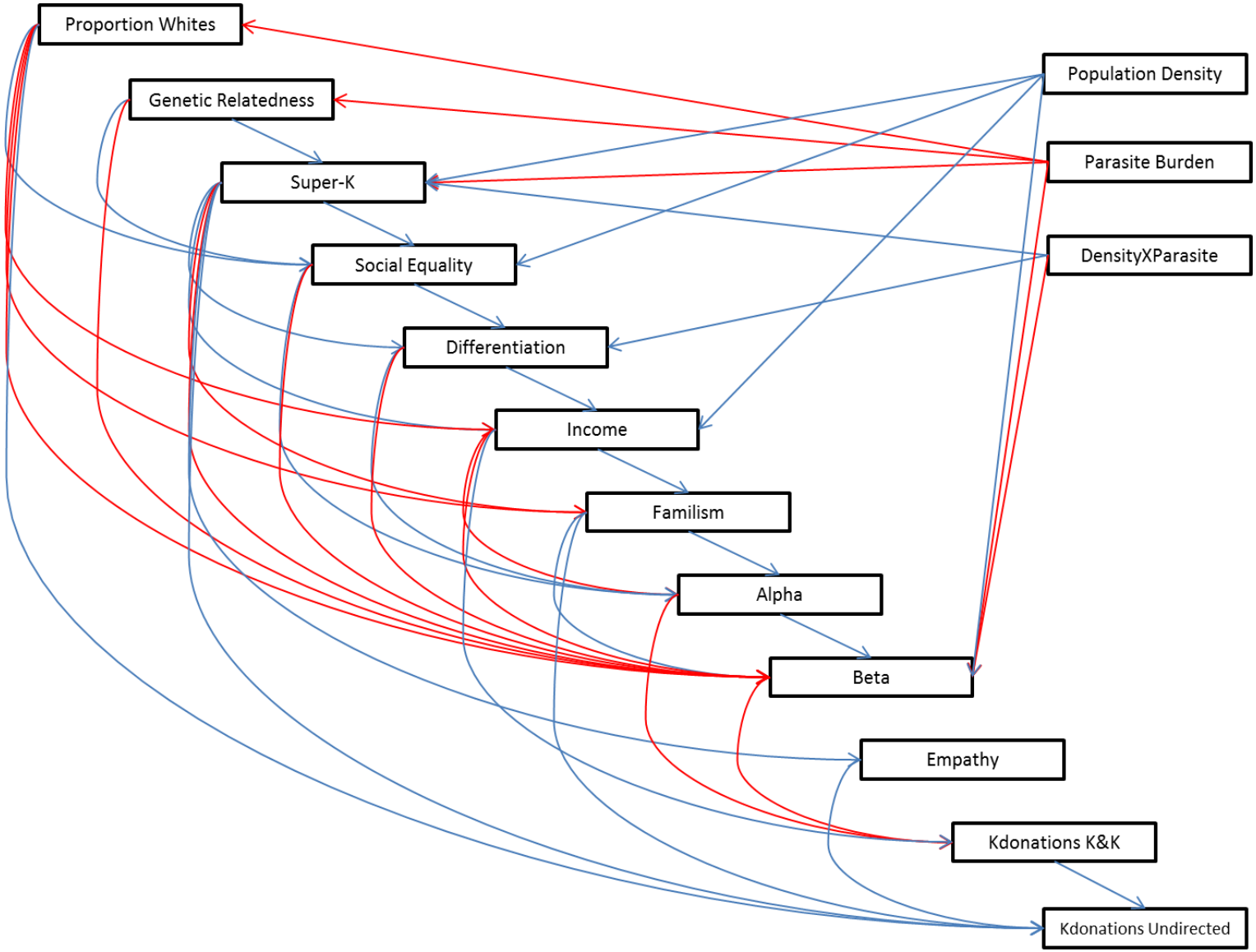

Figure 6. *Causal life history theory of living-donor, non-directed kidney donations*

Environmental agency inhibits the effects of any postmaterialistic cultural values and restricts regional degrees of genetic relatedness. Regions with greater degrees of genetic relatedness, greater population densities, and more environmental agency facilitate the development of slower life history strategies within human populations. Mutualistic social schemata possessed by slow life history strategists promote the development of higher degrees of social equality. This greater social equality is further promoted within more genetically homogeneous regions, regions of greater population density, and regions inhabited by a majority of persons holding more postmaterialistic values. Social equality and slower life history strategies allow for regional strategic differentiation of life history traits – permitting more specific adaptation to local conditions. Strategic differentiation, slower life history, and greater population density yield increased human capital, while higher levels of postmaterialistic values reduce it. Greater family cohesion (familism) arises when there is more human capital, faster regional life history strategies, and generally lower postmaterialistic values. Higher aggregate levels of the Stability personality factor (*Alpha*) is promoted by familism, strategic differentiation, and social equality; however more available human capital results in a reduction of dispositional Stability. Higher levels of dispositional Stability, familism, and population density result in higher aggregate levels of the Plasticity personality factor (*Beta*), while human capital, strategic differentiation, slow life history strategies, genetic relatedness, postmaterialistic values, and parasite burden result in lower levels of dispositional Plasticity. Slower life histories foster greater empathy in regional populations. Directed kidney donations are facilitated in regions with higher levels of human capital and hindered when the levels of both higher-order personality factors (Stability and Plasticity) are elevated. Greater empathy, stronger familism, slower life history strategies, higher or more prevalent postmaterialistic values, and a greater

propensity for directed kidney donations create circumstances that foster greater non-directed kidney donation rates.

As a whole, the theory derived from these results was not consistent with Genetic Similarity Theory (GST). GST views the aggregate altruistic behaviors as an aggregate of individuals acting in their self-interest – attempting to maximize their inclusive fitness. Rather than genetic relatedness directly promoting more altruistic behaviors it fosters a more general prosocial/mutualistic *ethos* (positively predicting Super-K and Social Equality) that, in turn, encourages more altruistic behaviors (directly and indirectly). Further, if GST had been correct, the bivariate correlation between Genetic Relatedness and Kidney Donations (Kith & Kin) should have been *greater* in magnitude than with it and NDKD as the theory would predict altruism to be stronger among kith and kin than unknown conspecifics. This was not the case (Pearson's r = .19 and .44, respectively).

This prosocial/mutualistic *ethos* characterization is, however, consistent with Multilevel Selection Theory (MST). Altruistic behaviors (like NDKD) are, by definition, an incurred fitness cost to the giver, while conferring a fitness benefit to the recipient. These altruistic and prosocial behaviors *should* result in greater group-level fitness. Supplementing MST, Realistic Conflict Theory (RCT; Campbell, 1965) characterizes a differential propensity towards altruism based on group membership. In circumstances of greater intergroup conflict, *intra*group relationships are strengthened, while *inter*group relationships are simultaneous weakened – resulting in what is known in social psychology as in-group bias (Jackson, 1993). Without having a clear set of fitness outcomes it is difficult, however, how to determine what the groups are competing for. At this time, no clear empirically-based decision can be made with reasonable certainty.

Testing and Disconfirmation of Relationships

As an underlying assumption of the theory articulated here is that the processes described here are universal and not unique to the 50 United States, cross-national cross-validation is a logical next step. It should be noted, however, that any handful of effects failing to replicate cross-nationally (or even cross-temporally) would not be a strong disconfirmation of this tentative causal model. As these social biogeographic studies are investigating complex interrelated relationships, it is unreasonable to expect a perfect 1:1 replication across any two areas with unique ecological and historical backgrounds. Any failure to verify a relationship could be a disconfirmation, but under the right circumstances, it may just as well be an indication of a Type II error in the model.

Furthermore, taking many of these concepts and testing them at lower levels of aggregation (e.g. class of college freshmen) would first require finding alternative indicators. For example, Super-K was measured by the Gallup Well-Being Index, life expectancy, infant mortality, child mortality, and teen birth rates. While the Gallup Well-Being Index has an individual-level assessment that could be administered, the other variables are (by definition) group-level characteristics. Theoretically-derived and empirically-verified, ergodic facsimiles would need to be utilized in a *conceptual replication* as opposed to a *direct replication.*

All that being said, disconfirmation is possible. Considering just the last step of the sequential canonical analysis: NDKD were predicted by 1) greater kidney donations to kith and kin, 2) greater levels of empathy, 3) greater degrees of familism, 4) slower life history strategies, and 5) greater proportion of self-reported Whites. If these are, in fact, causal forces driving NDKD, then individuals that give kidneys should exhibit most/many of these characteristics. As such, a study could be executed assessing the levels of these characteristics of those that have

given a kidney. In other words, follow-up self-report assessment and interviews should demonstrate that NDKD individuals have greater levels of empathy, greater degrees of familism, slower life history strategies, and greater postmaterialistic values (assuming that proportion Whites is simply a proxy for them).

Of the exploratory relationships found, the behavior of Proportion Whites is most curious. As states previously, the variable may simply be a proxy for postmaterialistic values. If that is the case, then an incremental validity test similar to that done in Chapter 2 would be able to disconfirm that tentative hypothesis. To accomplish this, an alternate measure of postmaterialistic values would need to be acquired with state-level estimates and tested against Proportion Whites.

Limitations and Further Directions

This study presents a causal model of social biogeographic predictors of NDKD. Since the United States of America is currently limited to 50 states (plus maybe a few territories) and is unlikely to gain any new states in the future, no independent direct replication of this model is likely; however, systematic, functional replications are possible with alternative measures, other polities, and for data covering other time periods. For example, similar social biogeographic work has already used Spanish, Italian, and Mexican subnational regions to analyze various LH-related effects (Figueredo, Cabeza de Baca, & Peñaherrera Aguirre, 2017) – incorporating NDKD would simply be a matter of acquiring the additional data (assuming that equivalent NDKD data exist at a similar level of precision as the UNOS data). If these effects are, in fact, more generally due to the ecological conditions specified by the tested model, meaning outside the original 50 United States, then the exact geographic location of the subnational regions

modeled should be incidental as long as polities with a similar range of characteristics can be found.

Further elaboration is needed to unpack the precise causal mechanisms that underlie these biological, psychological, and socio-cultural effects. A mediational cascade model is designed in such a way that *ideally* there would be as few residual direct effects as possible, providing greater model parsimony, after the hypothesized indirect effects have been accounted for, and supporting any claims of complete ("full") rather than partial mediation. The entire causal chain could thus be accounted for using fewer model parameters, and greatly simplifying any excess complexity within the theory. For example, one of the major criticisms against a psychometric approach to investigating life history theory is a lack of association typically found with more traditional biometric approaches. This study, however, found that biometric life history (Super-K) and psychometric life history (Empathy) contributed to NDKD. Further, they were strongly related to each other such that biometric life history gave rise to psychometric life history. With a stronger, more comprehensive estimate of psychometric life history, it is entirely possible that the relationship between biometric life history and NDKD will be fully-mediated by psychometric life history – depicting a causal chain from the biodemographic indicators through the psychological mediators to the actualized behavior.

Implicit within these social biogeographic models is the expectation that certain state-level mechanisms may apply to influence individual-level behavior. As elaborated on in Chapter 3, these are empirical questions that need to be tested explicitly. Within the scope of this research, however, many of the group-level effects tested and verified have been conceptually confirmed at the individual-level (e.g. the role Super-K in prosocial behaviors and attitudes). In

fact, there is only one clear instance of a Simpson's paradox and that is the higher-order factor structure of the Big-5 (Chapter 3).

This would seem to indicate that the Differential-K description of group-level differences is correct; they are simply the sum of the individual-level differences. While possible, this explanation seems insufficient. Individuals do not exist in a void, but instead nested within ecologies that include the groups within which they belong. It is rather naïve to assume that individuals fail to extract any information regarding group cultural norms while interacting with in-group members. Many of these regional aggregates appear to reflect cultural norms (or at least function as proxies for such norms). For example, a state with a high mean level of empathy will have a greater empathy norm than a state with a very low mean level of empathy. These norms will function to differentially reinforce certain social behaviors among individuals. So, while the Differential-K description would state that individual differences create group differences, it also seems plausible that group differences create individual difference.

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