THE EVOLUTION OF LIFE HISTORY TRAITS IN ICELAND: 1650-1950

By

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ABSTRACT OF THE DISSERTATION

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I analyzed heritability of lifespan and fertility over 300 years of Icelandic history, using computer simulations, a genealogical database called Íslendingabók (the book of Icelanders), and genetic data –single nucleotide polymorphisms (SNP's)-from modern populations. There was no evidence that either lifespan or reproduction is heritable. There was, however, substantial evidence that parental investment (PI) has enhanced both the survival and reproduction of the people of Iceland. There was also strong evidence of a quantity-quality tradeoff for reproduction and lifespan; each additional child conceived by parents substantially reduced the lifespan and reproduction of all current and future offspring.

I also searched for evidence of sexual antagonism (SA) in the Icelandic database. SA occurs when the reproductive interests of males and females conflict. Whenever variance in reproductive success is different between the sexes, each sex will have different and often conflicting strategies. Genes that benefit one sex are transmitted to opposite sex offspring (e.g. mothers to sons) which then have deleterious effects (Andersson, 1994). There was some suggestive, although not statistically significant, evidence of SA effects in Iceland (see chapter 3). Post-hoc testcrosses between high lifetime reproductive

success (LRS) males and low lifetime reproductive success (LRS) females produced more grandchildren through their sons than through their daughters. Post-hoc testcrosses between high LRS females and low LRS males in contrast produced more grandchildren through their daughters than through their sons. Although statistically insignificant, these data did produce effects hypothesized by sexual conflict theory in both directions.

Finally, I searched for evidence of reproductive advantages to sex biased parental investment (Trivers-Willard) in two separate databases. In one publicly available, on-line genealogy from the United States, there was significant evidence suggesting that sons from large families have more children than daughters from large families. Also, daughters from small families are more likely to have more children than sons from small families. In Íslendingabók, however, there was no evidence that biasing the sex ratio based on family size would confer any advantage to its practitioners. The discrepancy in these results suggests that Trivers-Willard effects may depend on yet to be determined environmental, social, or cultural variables.

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Chapter 1: A Brief History of Iceland

Life for the settlers of Iceland over the past millennium could be described in Hobbesian terms - solitary, poor, nasty, brutish and short. As the poorest country in Europe prior to the middle of the 19th century, Icelanders commonly experienced mass starvations and periods of extreme deprivation. Several times since the founding of the island, the Icelandic population teetered on the verge of extinction (Karlsson, 2000).

In this chapter, I will provide a brief outline of Icelandic culture and history. Understanding the historical context is critical to understanding any analysis of the genealogy of Iceland, also known as Íslendingabók (meaning "book of the Icelanders"). Historic, legal and cultural factors and practices have changed over time and, accordingly, affect the life history traits of both the individuals and the populations living on the island. All issues of concern in this paper (sex ratios, sexual conflict, lifespan, reproductive success, birth order, etc…) result from environmental and genetic factors.

Geology and founding date of the island

Iceland is a geologically young land; it began to rise above the Atlantic Ocean approximately 40 million years ago (Walker, 1964). The island lies along a major tectonic plate, known as the mid-Atlantic ridge that splits the Eurasian plate from the north and South American ones. A rift crosses most of the island, separating at a rate of about two centimeters per year (Brantley, 1994). The entire island is lined with major volcanic hotspots, and every few years major volcanic eruptions occur. Because Iceland is geologically new and violent volcanic activity is common, the ecosystem is extremely fragile (Redfern, 2001; Edgran and Penver, 1977).

Once domesticated animals were introduced to the island the ecology began to deteriorate rapidly. This process was accelerated into and throughout the middle ages (13th and 14th centuries). Therefore Iceland could have sustained much larger populations earlier, rather than later, in its history. Sheep grazing and other human activities severely degraded the fragile environment in the first few centuries after the island was colonized through erosion and soil depletion (Sveinbjarnardottir, 1992). To the first settlers of Iceland who came from Norway and Denmark, the Icelandic landscape looked remarkably similar to their home countries. The similarities, however, were largely a mirage (Karlsson, 2000).

Iceland has an extremely short growing season due to its northern location and lack of sunlight in the winter - a small portion of the northern part of Iceland enters the Arctic Circle (The Island of Grimsey). Also the islands' crust is constantly being recycled so that soon after grasses and thin layers of topsoil are created, they are destroyed. Originally, volcanoes were the primary culprit for this constant soil transformation, but soon after colonization, the livestock (primarily sheep imported from Scandinavia and horses imported from Ireland) caused the erosion and damage to the fragile and thin topsoil to accelerate (Diamond, 2005).

The settlement date for Iceland is generally agreed upon by most scholars (see Karlsson, 2000 for review). The earliest histories and the best archaeological evidence put this date at 871 A.D. The archaeological record that supports this settlement date is found just above a layer of volcanic ash (known as the settlement layer) that has been dated to this period (Vésteinsson and McGovern, 2012). Comparisons of this layer with the ice cap in Greenland can be dated to precisely 871 A.D. (Sveinbjarnardóttir, 2012).

There is no other country from this time period whose settlement date can be determined with this degree of certainty.

By the end of the 10th century the population of Iceland was probably around 10,000 people. These estimates come from a variety of sources, but rely heavily on Íslendingabók (Thorgilsson, 1930). Many scholars agree that a century later, by 1100, the population of the island had exploded to between 70,000 and 100,000 individuals (Karlsson, 2000). This number has been disputed, however, and some historians have claimed that the island could never (prior to the 20th century) have supported more than 50,000 individuals (Thorarinsson, 1961; Karlsson, 2000).

Some general cultural factors before the 17th century

Icelandic society during the commonwealth period (before the 13th century) was, by European standards, relatively egalitarian but there were still wide gaps in wealth and income (Karlsson, 2000). For example, slavery existed until the 13th century and although women could inherit property, farms were usually passed through first born sons. Substantial dowries were also paid by the families of the bride (Gudmundsson, 1997). Although Iceland was more economically homogeneous than the rest of Europe, wealth was still unequally stratified between land owners, farmers, and tenant laborers (Karlsson, 2000).

Most of the Icelandic population throughout the settlement period (prior to the17th century) consisted of farmers and their land. The primary class distinction was between farmers and their servants (Karlsson, 2000). Childhood was short, particularly for males, and after the age of twelve boys could sit on courts and act as judges. At age sixteen, young men were entitled to receive their inheritance. Women did not receive

inheritances until the age of twenty although they were often married much earlier (Siggurdsson, 1998). As we will see, particularly in Chapters 4A and 4B, this will have an important effect on the degree to which offspring were a drain on parental resources versus the extent to which they were a benefit to parents.

Sex differences in social status were quite strong until recently. Males typically occupied higher positions and legal rights of women were restricted (Karlsson, 2000). For instance, women were forbidden from carrying weapons or entering politics and generally had little choice in whom they married. They could inherit property, but were always second to any male heirs and could only receive property in the form of a dowry paid to their husbands. Indeed, female social status was often both constrained and maintained by the social status of their fathers and husbands (Hastrup, 1984).

The relationship between wealth and reproduction

Until the 20th century Iceland was primarily an agrarian society, with relative socioeconomic homogeneity (Helgason et. al., 2008). Iceland underwent industrialization extremely late, by European standards, and was still a predominantly agricultural society well into the 20th century (Karlsson, 2000**)**. Up until the middle of the 20th century, there had been two primary ways to make a living in Iceland: farming (primarily sheep grazing), and fishing (Gudmundsson, 1997). Therefore, the island did not undergo the *demographic transition* (typically defined by industrialization and a decline in mortality followed by a decline in fertility) until after World War II (Helgason et. al., 2005; Kaa, 2002). These characteristics will prove to be important assets when I later analyze the selection pressures over the course of centuries. One important advantage is that in Iceland, the association between wealth and reproductive success

remained positive longer than anywhere else in Europe (Lawson and Mace, 2011; Voland, 1998). This positive association between wealth and reproduction is a pattern that human behavioral ecologists and evolutionary psychologists believe was typical through most of our species' history (Coal, 1973; Caldwell, 1976; Caldwell et al., 2006**)**. In fact, it is a basic assumption among evolutionary psychologists that status (defined by access to resources; particularly amongst males) has been an important driver of sexual selection in humans (Caldwell, 1976; Buss, 1988). Such a late demographic transition means that the genealogical and genetic data I use can be reliably analyzed without having to be concerned with dramatic shifts in population structure or changes in fundamental assumptions about the relationship between status, wealth, mortality and fertility.

The demographic transition has been a puzzle and major concern in many academic disciplines ranging from economists and sociologists to demographers and anthropologists. During the transition, wealth and reproductive success are decoupled, so that poorer families have more offspring (Thompson, 1919; Caldwell, 1976; Caldwell et al., 2006) and wealthy families have fewer. This has posed a theoretical quandary for evolutionary theorists in many disciplines because resources are often considered to be currency that can be converted into offspring (Mace, 2000; Caldwell, 1976; Galor & Weil, 2000; Van de Ka & Dirk, 1987). The first populations that experienced the demographic transition lived in Northern Europe around 1800 (Demeny, 1968) and the process is predicted to end globally in sub-Saharan Africa by 2100 (Liods, 2003). Major demographic changes can cause problems when analyzing data that bridge the time

periods where these changes occur and researchers often need to make some serious adjustments to compensate for substantial changes in population growth and structure.

Evolutionary theorists typically argue that resource acquisition functions to maximize an organism's reproductive success (RS). So the only reason that humans accumulate wealth and property is to eventually convert it into offspring. Therefore the declining fertility rates amongst the wealthy which characterize the demographic transition seem to violate this assumption. Evolutionary biologist Monique Mulder offers a possible solution to this puzzle. She argues that reproductive decision making in humans is driven by a brain designed by natural selection to maximize material wealth rather than offspring (Mulder, 1998). Accumulating property is an end in itself rather than a means to an end. Humans acquire resources solely to have more resources and not as a way to increase RS. Prior to the demographic transition more resources would naturally have resulted in more surviving offspring through both increased access to the opposite sex and lowered mortality of offspring. So this strategy would have succeeded; wealthy individuals would have produced more children. Whether more resources results in more offspring since the demographic transition, however, is questionable at best; and most evidence points in the opposite direction (poorer families within the same population are more fecund)(Mace, 1998). The extent to which widespread access to birth control has played a role in these changes is still unclear. It is likely, however, that access to birth control only further severed an already strained relationship between wealth and reproduction that had already begun with the demographic transition.

Evolution also provides some tentative support for Mulders hypothesis. Natural selection, for instance, does not need to provide an incentive or reward for sexually

reproducing organisms, such as humans, to produce offspring. A strong sex drive is sufficient. It has even been suggested by some anthropologists that individuals in primitive cultures may not make the connection between sex and pregnancy. Even if this is dubious speculation, it is easy to imagine that our ancestors may not have fully understood the timing of important physiological processes such as menstruation, ovulation or gestation (Malinowski, 1927).

In addition, monogamy was increasing alongside the rising tide of democratization at precisely the same time that the demographic transition was occurring (Henrich, Boyd and Richerson, 2012). The trend towards monogamy will tend to reduce intrasexual (male-male) conflict, and may also have helped to further weaken the relationship between wealth and reproduction. Some researchers have suggested that monogamy is the result of gene culture co-evolution. It's increase over the past few centuries has been interpreted as a way to reduce intergroup violence. This can benefit elite males who are also the primary losers from monogamous social systems (Henrich, Boyd and Richerson, 2012). The relationship between democratization and the demographic transition may be coincidental and the causes of both are complex and not well understood. It is clear, however, that increased monogamy will tend to reduce the relationship between wealth and reproduction simply because the variance of male reproduction will tend to be reduced. Fewer males will be completely isolated from the pool of breeding males while on the upper end fewer will be able to have unrestrained reproduction. In theory the RS of high status males is now limited to the RS of their wives. Of course the replacement of polygyny by serial monogamy (multiple wives separated in time by divorce) complicates this issue as well.

The increasing cost of children was perhaps the most important factor disrupting the relationship between wealth and reproduction, and will be discussed at length in a future section subtitled '*Public education and the Demographic Transition: The Cost of Children*' later in this chapter. The perceived increasing costs of children is probably one of the most important factors that caused the sharp fertility decline seen in the late 18th and early 19th century across much of Europe. Some evidence also suggests that a quantity-quality tradeoff for number of children produced is increasing in importance around this time, and that widespread access to public education is an important cause of the rising costs of children (Becker, S. O., Cinnirella, F., & Woessmann, L. 2010). The tradeoff between offspring number and their condition (quantity vs. quality) are important results from our analysis of Íslendingabók. This tradeoff will be discussed in much greater detail in chapter two.

Malthus and the demographic transition

The discussion over what population size the island could sustain has been controversial. The writing of Thomas Malthus contributes to this debate. Malthus (1798) argued that because human populations increase at a rate greater than food supplies, the only controls on infinite population growth were famine, disease and violent competition such as war. Malthus' ideas played a key role in shaping Charles Darwin's theory of evolution by natural selection. His influence can be seen in a passage Darwin wrote in October of 1838 (Vorzimmer, 1969):

> "I happened to read for amusement Malthus on Population... it at once struck me that under these circumstances favorable variations would tend to be preserved, and unfavorable ones to be destroyed. The result of this would be the formation of new species." (pp. 527).

This passage was written twenty-one years before the publication of Origin of Species (1859). Malthus classified the types of forces, or "checks" against population growth into two categories: misery (positive) and vice (preventative). One category was "positive checks", or those that increased the mortality rate such as disease, famine or war. The second type was "preventative checks" and these would lower the birth rate. The checks could be induced by a faltering economy and depressed wages but they also included the postponement of marriage, contraception and increased rates of prostitution.

Prior to the industrialization of Europe, Malthus' predictions appeared to be correct. Whenever warfare, disease or catastrophic events (e.g. volcanoes) occurred, compensatory economic factors, such as wages, reacted strongly. Both positive and preventative checks therefore brought the population back into balance (Lee, 1987; Lee, 1997; Lee and Anderson, 2002). But Malthus' most alarming prediction - that a dramatic increase in mortality would result from massive disease and starvation as population outstripped food supplies failed to occur. The reason that Malthus' more dire predictions failed to come to fruition is generally attributed to the demographic transition which began in Northwestern Europe around 1800.

The classic demographic transition is experienced in three phases. The initial phase is typically associated with a drop in mortality. This is then followed by a decline in fertility, and finally a severely aging population. This is currently happening in countries like Japan and parts of Europe, where the elderly are rapidly outnumbering the young (Cohen, 2003). The classic declines in mortality seen in the demographic transition are, for the most part, well understood. They are the result of modern medicine, improved hygiene, a monopoly on violence held by the state and vast improvements in nutrition

(Lee, 2003). These declines in mortality also affect the costs of children throughout the period of PI. The commensurate declines in fertility, associated with the demographic transition, however, are not as well understood (Galor and Weil, 2000). These concepts have important implications in Chapter 2 when I analyze the quantity-quality tradeoffs for family size.

The demographic transition and public education: the cost of children

Although there were schools appearing in Reykjavik and some villages in Iceland by the 19th century, all of them were privately funded and concentrated mostly on itinerant and short term so that school did not to interfere with work at home, such as farming, herding livestock, or fishing. Schools typically formed on local farms for the winter and would then close in the spring (Karlsson, 2000). The educational system in Iceland was not rigorous or widespread until well into the 20th century.

The rise of public education has often been cited as a primary cause of the demographic transition (Kirk, 1996). As more of an importance is placed on education, children become increasingly costly to parents and begin to provide less of an immediate economic benefit. In 1907 public education became mandatory in Iceland for all children aged 10-14 and soon after the first University was founded. But for most ages groups secondary education was sporadic well into the 20th century, (Karlsson, 2000; Helgason, pers. comm.).

The cost of children

As previously discussed, RS and socioeconomic status (SES) are often negatively correlated in modern societies (Vining, 1986) and this is probably a new trend that

occurred after the demographic transition. It is also violates a fundamental assumption of sociobiology. Rather than helping parents with chores or helping to raise their siblings, researchers have found that children in middle to upper class American families provide little or no help to parents (Konner, M., 2010) . The costs of parental investment (PI) and the benefits of children to parents are fundamental questions in evolutionary biology. Measuring the costs and benefits of children to parents will be discussed in greater detail in the next chapter. In the literature to date, however, the best demonstration of a significant quantity-quality tradeoff for family size comes from a study in Ghana of 2,461 offspring groups within a polygynous population confronted by adverse conditions (Meij, 2009). The authors showed that the risk of mortality increased by 2.8% for each additional child produced.

Costs and benefits of children to parents is a central concern as I analyze the role of PI on several life history traits that are closely tied to fitness. Some important questions are as follows. Prior to the demographic transition, when did children become independent of their parents? At what age do children provide a net benefit to parents through provisioning or by helping to raise siblings? It may be hard to imagine how, in our current society, four year olds could contribute to family resources or assist with rearing younger siblings, but developmental psychologists have suggested that humans can be self-sufficient by the age of seven or even earlier (Katz et. al., 1973) (e.g. youth street gangs in Rio de Janeiro, Brazil or Bogota, Colombia). In addition, when farm land is transferred via marriage and inheritance, as was often the case in Iceland (Karlsson, 2002) the benefits of children may be even higher because younger siblings, who are less likely to inherit land, may be better off helping their older siblings or parents, rather than

pursuing mates themselves. Under these conditions, the search for a mate may be delayed and the degree to which children help their parents increased.

The ivory billed woodpecker provides useful insight into behaviors that arise when resources are scarce. These birds are limited to a restricted number of nesting sites so individuals will contribute heavily to provisioning their nieces and nephews until new sites become available for them to occupy with a mate (Wilson and Nowak, 2012). A similar process may have occurred in Iceland, where a farm or piece of land became available following an individuals' death, or perhaps on larger scales following major disasters.

For the purposes of this analysis, I will concentrate on Icelandic history from the 17th century through the present. Although the genealogical record (Íslendingabók) in Iceland goes back to the 9th century, the first centuries were not recorded in a systematic and rigorous manner. The first national census in Iceland, and likely the first ever in the world, was in 1703 (Karlsson, 2000). It is therefore typical to regard the genealogical data from 1650 and after as the most accurate; and to treat the time before with greater skepticism (Gunnar Gundarson: *pers. comm.*). For this dissertation all of the analyses and reported results are conducted on the population after 1650 to ensure the greatest reliability.

CHAPTER 2

Parental Investment Increases Both Offspring Reproduction and Lifespan in Iceland

Abstract

What effect do parents have on the reproduction and lifespan of their offspring? This question is difficult to answer in part because humans live long lives which make it difficult to obtain accurate genealogies and other records of life history traits. In Iceland, however, there is an electronic database called Íslendingabók (the book of Icelanders) which has recorded all life history traits of the Icelanders since the country's founding in 871 A.D. This database helps to answer questions on the evolution of life history traits, such as lifespan and fertility. Parents affect the evolutionary success of their offspring in two ways: by direct parental investment (PI) and by transferring genetic material. I distinguish between the effects of genetics and PI on offspring reproduction and lifespan by comparing the fertility and lifespan of full siblings with that of parent-offspring pairs. Using both genealogical *and* genetic data I am able to show that an individual's reproduction and lifespan are better predicted by the reproduction and lifespan of their full siblings than that of their parents. Because full siblings share genetic material and parental resources while parents and offspring only share genetic material, the difference between the full sibling correlation in reproduction and lifespan and the parent offspring correlation in these traits is unlikely to be caused by genetics and is therefore most likely the result of shared parental resources and or shared parental investment(PI). Using a variety of statistical methods and simulations, I was also able to show that parental

influence (PI and resources), but not genes, has had a major impact on the reproduction and lifespan of children in Iceland. In addition, I demonstrate a consistent tradeoff between the quantity and quality of one's offspring. The correlation between parents and offspring in reproduction and longevity is negative and this is indicative of a quantity-quality tradeoff. As parental reproduction and lifespan increases, the reproduction and lifespan of all of their children consistently and systematically declines.

Introduction:

Traits that are closely tied to fitness, such as longevity and reproduction, generally have lower heritability than traits less related to fitness, such as hair or eye color (Price and Schluter, 1991; Visscher et. al., 2008). This observation has often been interpreted as support for Fishers' (1930) fundamental theorem of natural selection which states that, for a population in equilibrium, there is no additive genetic variance in total fitness (Sheldon, 1994; Glazier, 2002). That is to say that for populations which are not invadable by mutants, or are evolutionarily stable (Smith and Price, 1973), there are no changes in the population's average fitness that can be attributed to changes in allele frequencies.

The reason that life history traits which are closely tied to fitness tend to have low heritability is simple. Traits that are highly correlated with genetic fitness will rapidly go to fixation, so variance and therefore heritability decreases. This is because traits that are closely tied to survival and reproduction become maximized and ultimately stabilize within a population (Hill et al., 2008). A trait that is critical to survival, such as a heart with four chambers, becomes fixed in a population. Once this occurs, heritability falls rapidly (Visscher et. al., 2008). Following this logic, most people with faulty hearts

should have problems caused by environmental factors (e.g., they had poor diets) rather than for genetic reasons (e.g. three chambered hearts).

It is important to note, however, that heritability is heavily dependent on the environment. For instance, height used to have a much lower heritability in Iceland than it does today. This trait is roughly 70% heritable today (Helgasson, personal communication; see table 2.6). This change is attributable to environmental change. Height depends on nutrition, which constitutes most of the environmental variance in this trait (Lai, 2011). The more that nutrition becomes stratified and unequally distributed amongst a population, the more that variance in height within that population is due to the environment, and less is therefore due to genetic variance. Because there is less variance in nutrition today than there was one hundred years ago, less of height depends on the environment, and more of it depends on the effect of genes. As the environmental variability that affects a given trait increases, heritability necessarily decreases.

Despite the expected low heritability for traits closely related to fitness, there are no expectations that such traits are not influenced by ones familial environment. Differences between families are the result of both environmental differences (PI, familial environments, investment, resources etc.) and genetic differences (Fisher, 1930; Kosova et. al., 2010). One question that has not received much attention, in part due to the difficulty of teasing apart genetic and environmental effects, is the heritability of family size. In this chapter I aim to answer this question. In addition to analyzing the relative impact of the environment and genetics on family size (e.g. LRS); I will also attempt to determine which environmental factors have the greatest effect on reproduction.

The life history trade-off between the number of offspring produced and their quality is a fundamental precept and assumption in evolutionary biology (Lack, 1947; Smith and Fretwell, 1974; Stearns, 1992). This idea relies on the fact that resources are limited and is based on the following principles: 1) as investment in offspring number is increased, investment per offspring is decreased, 2) increasing parental investment in offspring increases their reproductive success, and 3) maternal reproductive success is strongly dependent on the number of offspring that she can raise, successfully bring to sexual maturity, and ultimately on how many can produce grandchildren. Such success is often measured by lifetime reproductive success, and, ultimately by lifetime grand offspring success (Roff, 2002; West, 2010).

The evidence for a quantity-quality tradeoff in humans is limited. Other than the aforementioned study in Ghana that demonstrated a tradeoff between offspring number and mortality (the more children the greater their risk of death) (Meji, 2009), such studies usually analyze small samples. In one study by Hagen et al. (2006) there was no significant evidence of a quantity-quality tradeoff in a small Ecuadorian Shuar community. In a larger three generation study in preindustrial Finland, a tradeoff between offspring quantity-quality tradeoff was found for reproduction amongst those of low socioeconomic status (Gillespie et al., 2008). Other studies have used school performance as a proxy for the condition of offspring and as a reflection of PI. Of the few studies that focus on educational achievement in the United States researchers have reported a clear dilution effect (Blake, 1991, Downey, 1995). In these studies, the more siblings an individual had, the worse he or she performed in school. In larger families, later born children performed better than earlier births; but when teacher performance

was included as a variable, any effect of family size on school performance was eliminated (Hanushek, 1992). In addition, public education is a relatively new factor in human evolution so educational success may not be an appropriate measure for individual fitness in the past or even today (Smith et al., 2001). Beyond these studies, there have been relatively few reports which test for a potential quality-quantity tradeoff in family size. Perhaps more importantly, there is a lack of multi-generational studies with large sample sizes, both of which are critical if we want test the evolutionary significance of this tradeoff (Gillespie et al., 2008).

Despite the lack of good quantity-quality tradeoff data in humans, such work has been recorded in many other species, beginning with Lack's classic study on optimal clutch size in herring gulls (*Larus argentatus;* Lack, 1947). Lack demonstrated that female gulls produce the "optimal" number of eggs that she may care for (not too many so she is incapable of properly caring for all, but not too few so that she cannot maximize her reproductive success). Indeed, some of the best evidence for a tradeoff between offspring quantity and quality has come from birds (Gustafsson & Sutherland, 1988; Smith et al., 1989; Merilä and Wiggins, 1995; Gillepsie et al., 2008).

Because of the depth and accuracy of Íslendingabók, Iceland is an excellent place to search for the existence of a quantity-quality tradeoff among offspring in humans. First, I examined whether more offspring always lead to more grand-offspring. But more specifically, I sought to determine the extent to which the number of siblings one has affects ones reproduction and longevity. Quantity-quality tradeoffs in humans have been analyzed before using genealogical data, but never on a database with all of the unique and beneficial aspects of Íslendingabók. In addition, I developed a new method for

assessing the effects of parental and sibling investment in offspring (see methods) in order to determine the effect that helpers at the nest may have on an individual's reproduction and lifespan. Using this information, I examined what impact an individual's own reproduction and lifespan has on the reproduction and lifespan of their children.

Methods:

The database, Íslendingabók

Íslendingabók has several critical advantages over other genealogies. First, it is a population-based database that contains information about more than half of the one million individuals estimated to have ever inhabited the island (Gudmundsson et. al., 2000). A population-based genealogy is an advantage because it substantially decreases the possibility of sampling bias. In other words, when using a national census, one is less likely to over or under report any particular group (e.g., individuals who did not have children are likely to be underreported) (Helgason, pers. comm.).

Second, the database is extremely accurate. It includes all living Icelanders and most of their ancestors. An examination of mitochondrial DNA shows a maternal accuracy rate of 99.3% (Sigurardottir et al., 2000) while the error rate for non-paternity and lab error combined is less than 1.5% (Gudmundsson et al., 2000). This low of an error rate naturally invites skepticism. But I found a similar level of error (2%) estimating from the approximately 30,000 individuals who had been genotyped. For my own analysis this means that in approximately 2% of the cases, the category the genealogy assigned was not concordant with the percentage of SNP's they shared. For example, although two individuals were categorized by Íslendingabók as a father and son,

they had a coefficient of realtedness of .012, when they should have had a coefficient of relatedness of .5. Therefore, around 2% of males who are represented in the genelaogy as full siblings or offspring are not within the range of possible kinship coefficents for that particular group. This type of miscategorization is either due to extra-pair paternity or to bookeeping errors.

Third, the population has been isolated from the rest of Europe with little immigration to or emigration from the island. As a result multiple generations can be accurately traced back for centuries which also helps to reduce recording errors. Although Iceland was for the most part a closed population during the years analyzed (1650-1950), there is one important exception. At the end of the 19th century (1870-1910), there was a major emigration from Iceland during which approximately 20% of the population moved to Canada (see figure 2.1).

Fourth, birth control which creates potential problems with interpreting results was not widely available in Iceland until 1956 (Swanson, 1988). When women can more easily control their own fertility and reproduction, the analysis of reproductive correalates becomes more complicated and difficult to predict. At the very least, changes in reproductive patterns after the introduction of the widespread use of birth control may change reproductive patterns in unknown ways. And more importantly these new ways may not be typical of ancestral populations. In Iceland, however, these isssues and similar problems of interpretation caused by the demographic transition can be diminished while still preserving most of the data because Iceland did not undergo the demographic transition until after World War II. This is very unusual because in some

parts of Northern Europe the demographic transition was well under way by the early 19th century (Lesthaeghe, 1995).

The first national census in Iceland took place in 1703 (Karlsson, 2000). It is therefore typical to regard the genealogical data from 1650 and onward as the most accurate, and treat the time before this date with greater skepticism (Gunnar Gundarson, *pers. comm.;* Helgssson, *pers. comm*). So, for this dissertation, all of the analyses and reported results are conducted on the population from 1650 to 1960 to ensure the greatest reliability. Alas, this database does not contain any information on the socioeconomic status of the individuals contained in the databases, so this variable was not available to measure the "condition" of individual Icelanders.

Coefficient's of relatedness and estimating heritability

I used two main programs to extract the information that I needed from Íslendingabók to conduct my analyses. The first was Microsoft Access, a program developed by Microsoft Office. Microsoft Access is useful for organizing and connecting information from large relational databases. I used this program to extract all kinship relationships within the database all the way through and including 1st cousins. More precisely, I established and analyzed all relationships with a kinship coefficient (θ) [the probability that 2 alleles at a given locus – one chosen randomly from each individual – are identical by descent (IBD)] that was greater than or equal to 1/16 or similarly had a coefficient of relatedness (r) (the expected fraction of alleles that are identical by descent (IBD) that was greater than or equal to 1/8. The differences between a kinship coefficient (θ) and a coefficient of relatedness (r) are not particularly important to this paper. They do, however, have some effect on the values I generated for relationships between half

siblings and full siblings, which vary around ¼ and ½ respectively. They vary around these mean values because of meiosis and the independent assortment of chromosomes. Simply by chance some sibling or half sibling pairs may share more chromosomes than other pairs. This is not true, though, of parent- offspring pairs, where offspring each receive exactly half of their chromosomes from each parent. There is no variance around the mean as there is for full and half sibs or even cousins. Kinship coefficients are also of some interest when calculating relationship values between individuals in the database who are 'not related', by which we mean that they have very low (<.01) kinship coefficients.

Coefficients of relatedness, commonly symbolized with the letter (r) are often used in relation to kinship theory when describing the effect of natural selection on kinship altruism or traits that benefit kin (Hamilton, 1964). When describing the effect of selection on altruistic traits, the values between full siblings that vary around the mean of .5 do not have an effect. One reason that this is the case is because there would need to be a gene that would be capable of calculating these different measurements around the mean and this in unlikely (Trivers, 2012). It is also a mistake to think that a coefficient of relatedness applies to all genes. It doesn't because humans already share 99% of their genes, even those individuals that are considered unrelated. The 50% that full siblings share are those genes that are 'identical by descent from a common ancestor' and this is the important qualification made by Hamilton (Hamilton, 1964). Siblings may share 99% of genes altogether but only share 50% by descent from a common ancestor. The frequency that genes for altruism achieve in the population has no effect on selection for altruism (Trivers, 1985). Dawkins explains why genes for universal altruism are not

evolutionarily stable. Universal altruism genes which direct altruism towards all members of the species are invadable by mutant kinship altruist genes that only direct altruism towards kin. This is because universal altruists will favor all individuals including kin and kinship genes which are shared by full siblings will only favor kin and will spread at the expense of universal altruists (Dawkins, 1979). In other words universal altruist genes are not evolutionarily stable.

In these analyses, however, we are not concerned about the effect that the variance around the mean of .5 for the degree of relatedness between full siblings has on selection for altruistic traits. The genetic relatedness values generated for full siblings here are only used for calculating heritability (Visscher, et. al., 2006). Genetic relatedness may not affect the direction of selection for altruistic traits but it can be used to estimate the heritability of a given trait. For the entire genealogy (all of Íslendingabók), I obtained all relationship data on individuals that were first cousins or closer. For instance, I know how many first cousins everyone in the genealogy from 1650 onwards had, and I know who they were. I also obtained the same data for relationships such as great grandparents (r=1/8), maternal ½ aunts (r=1/8), and some particularly interesting relationships such as double cousins (r= ¼). Double cousins are rare, but they are interesting and helpful for the purposes of estimating family effects or heritability. They share all four of their grandparents but do not share parents. Although unusual, in isolated rural populations, such as Iceland, these relationships are more common. These relationships present opportunities to distinguish between environmental and genetic effects. Similar to the manner in which heritability estimates are ascertained by calculating the variation of phenotypes between identical and fraternal twins, double cousins and other relationships

between individuals who share some aspects of their environment and some proportion of genetic material (e.g. full siblings) can also be analyzed to estimate heritability.

Genealogical data

I used Microsoft Access to determine relationships between individuals in the genealogy. When the relationships became too complex, and the data too intensive for Microsoft Access, (relationship data between any two individuals in a genealogy with over 600,000 individuals), I wrote code in Visual Basic that was compatible with 'Macros' in Microsoft Excel (see appendix A). These programs extracted extended relationship data such as the number of maternal great grandchildren an individual produced. In the end, over 200 characteristics were obtained for each individual ("proband") in Íslendingabók. There are more than 600,000 individuals in the database and the traits of current interest (birth ratios, marriages, siblings, average lifespan, etc.) were extracted from the basic relationship data. Finally, I used a Perl script written for me by a colleague, Rolando DeAguiar, to once again confirm the consistency of the relationships and traits that I extracted via Microsoft Access, SPSS and Visual Basic (see Appendix C – Iceland Fast). This was done as a second check to make certain that the data were being extracted properly and without error. They were. The data was confirmed yet again when I compared it to previous analyses conducted by Agnar Helgason, the resident anthropologist at deCode genetics. As should be clear, even with the expansive computing power available at deCode genetics (over eighty 100 megabyte hard drives were devoted to calculating relationship data) the resources required were high and cluster and hard drive crashes were common, as were multiple overnight and weekend runs (e.g. 600,000 X 600,000 for each and every individual in the database

repeated for each and every possible relationship from full sibling to first cousin and beyond – 'the unrelated' individuals).

For the genealogy, I limited the database to all individuals in Íslendingabók that were born after 1650 and before 1910. I chose 1650 as an early cutoff because it is considered to be the first decade in which data becomes 'sufficiently' reliable (Helgason, *pers. comm.*). Most individuals who survived childhood are likely to be accurately recorded if they were born after 1650. Prior to this date, the genealogy is susceptible to a wide array of record keeping and reporting biases. Limiting it to those born prior to 1910 was done for several, many of them previously mentioned, reasons. First I wanted data prior to both urbanization and industrialization, specifically because the demographic transition may have changed fundamental population growth and dispersal patterns from those more typical of our ancestors evolutionary history (Barthold and Jones, 2012; McNicoll, 1992). Also, resources and reproduction are decoupled during the demographic transition. Although this did not occur in Iceland until after World War II but because some changes associated with the demographic transition, such as mandatory public education, occurred earlier in the 20th century I used a more conservative estimate of 1910 . I also wanted to make certain that all the subjects had completed their lifespans (102 years old was sufficient).

Genetic database

For the genetic analyses, I used all individuals born between 1897 and 1960 for which Single Nucleotide Polymorphism (SNP's) data was available. SNP's are single base pair differences between humans. They constitute approximately .05% of the human genome and must occur in at least 1% of the world's population to be classified as a SNP (Human

Genome Project Information, 2012). This method presents an easy and practical way to identify variation between individuals. Rather than search the entire genome for variants that may cause disease, researchers can concentrate on the small percentage of base pairs that differ amongst both humans and human populations, and can also calculate and compare extremely accurate kinship coefficients between all individuals. That is to say we can determine precise relationship values between any two individuals, whether they are closely related or not.

For the genetic analyses the dates I included in my analysis were based on maximizing sample size, and therefore used all individuals likely to have finished reproduction. For women this was likely to be true by age 52, and for men it was probably true by the same age, although there are certainly some exceptions. I therefore used all individuals born before1960 for which genetic data were available. For all individuals born after 1890 and before 1960, the database included genetic relatedness between 8,456 full sibling pairs and 3,386 parent-offspring pairs (confirmed by single nucleotide polymorphisms-SNP's).

Quantity vs. quality of offspring

All full siblings belong to discrete groups comprised of how many full siblings they have. This is synonymous with their parents combined reproduction (includes half siblings). Parent-offspring and full sibling relationships are therefore not entirely independent. An individual's net reproduction is the same as the number of siblings each of their children have plus one. The importance of this non-independence will become clear when I explain how the data was analyzed (see results).

To further tease apart the relative impact that genes and the environment have on offspring reproduction and lifespan, I analyzed the relationship between an individual's number of full siblings (e.g. parental RS-1) and their lifespan and reproductive success. Another simpler way of distinguishing between the quantity and the quality of offspring was to simply compare an individual's RS with the number of grandchildren they produced, and more specifically the number of grandchildren they produced per child.

Simulations

I wrote a code in Visual Basic that simulated a three generation genealogy. Three models were designed. The first, which henceforth will be called the 'Resources Model', was designed so that the number of children and grandchildren an individual produced depended solely on the number of resources the grandparental generation owned and then transmitted to their offspring. The second, which I called the 'Genetic Model', was designed so that the number of children and grandchildren an individual produced was determined solely by the alleles inherited from ones grandparents. The third model was the null model in which neither genes nor resources played a role. Reproduction was essentially randomly and was based solely on certain fixed parameters such as the mean number of offspring per generation and the standard deviation of each generation's size. In both the resource model and the genetic model, resources and alleles, respectively, are inherited by offspring. The key, and only, difference between the two simulations is that *resources* (and their effect on reproduction) are diluted across generations; alleles are not. I will now explain the details of each model.

The Resources Model

In the Resources Model, the first generation of N parents (the F1 generation) are assigned a 'resources' variable which is a random number drawn from a normal distribution with a mean of X and a standard deviation of Y. The parents are then additionally assigned a random number of children from the Poisson distribution with a mean of Z (the mean number of children produced in each successive generation) and this number is multiplied by the resources of parent (i). In the next generation (F2) the children inherit the resources from their parents which are distributed equally amongst parents (i) children. The children are then assigned a number of offspring (grandchildren of parent generation or F3) which is also taken from a Poisson distribution with a mean of Z and is modified (multiplied) by the resources they inherited from their parents. (See appendix A for the code for the Visual Basic simulation).

The Genetic Model

In the genetic model the first generation of N parents (F1) are randomly assigned two alleles with a population frequency of F. They are then assigned a genetic fitness effect of .5 (homozygous recessive), 1 (heterozygous) or 1.5 (homozygous dominant). So the fitness effect of the allele is additive; the more alleles one has the greater their fitness. The parent generation is then assigned a random number of children taken from a Poisson distribution and modified (multiplied) by the parents (i) genetic fitness. Children are randomly assigned alleles from their parents in a Mendelian way so that they have a 50% chance of receiving either allele from each parent. The grandchildren (F3) are assigned to each child (j) and are taken randomly from a Poisson distribution with a mean of (mean children produced each generation) modified by the genetic fitness effect they inherited from their parents. (See appendix A for Visual Basic simulation code for the

genetic model and see appendix B to run the macro in excel and alter any key data such as generation size, standard deviation for wealth, fitness, mean family size, number of simulations, etc.). I have run hundreds of simulations, altering numerous parameters to determine which effects were statistical aberrations, and which were real, replicable and reliable. Because genealogies are interdependent and complicated datasets, distinguishing between statistical anomalies and real effects is often difficult. Running the simulations repeatedly, and systematically changing parameters allowed me to determine which parameters were meaningful and which had the greatest effect on the relationships between full siblings and or parents on their relative reproduction.

Using kinship coefficients to estimate additive genetic variance

When I did my research at deCode in 2011, there were approximately 30,000 individuals whose genomes had been *fully* sequenced. This means that approximately 230,000 SNP's have been sequenced from each of these individuals. The goal is to ultimately sequence the entire Icelandic population of approximately 320,000 people (Statistics Iceland, 2008). The primary reason deCode genetics is sequencing these individuals is to search for the genetic causes of diseases. Because so many individuals have been sequenced, however, deCode has relatedness data (coefficients of relatedness or IBD values) between all of the individuals in this database (30,000 X 30,000 matrix- - 900 million precise relationship values between individuals). Not only are these data useful for distinguishing relatedness values between full siblings or first cousins, but they are also useful for matching phenotypes of so called 'unrelated' individuals. For example, if individuals have kinship coefficients of less than .01, these individuals are considered to be unrelated. Unrelated individuals can be used to determine if certain

traits are heritable. For example, imagine two unrelated individuals share a trait, such as a vulnerability to diabetes 1 *and* also share similar environments. We can then assume that the cause of the diabetes is not genetically inherited and is instead caused by a shared environment.

Using deCode's kinship values, heritability estimates can be obtained for any given trait by matching these low IBD values (unrelated individuals) with the percentage of a given phenotype that is shared. In samples or populations that have been thoroughly sequenced, or for which sufficient SNP data is available, heritability estimates are typically obtained in two different ways. The first way is to compare the kinship coefficients of full siblings which have a mean value of .25, but which may be considerably lower or higher due to meiosis and independent assortment, and then match these values against a given phenotype. These methods provide a way to achieve less biased heritability estimates that do not depend on what may be special or unique characteristics of twins (these studies are outlined in a paper by Visscher et al. in 2006). Twins raised together, or twins raised apart, are a rare and unique group of individuals, so it can be problematic to make assumptions about the population at large from these rare and unrepresentative samples. It is hard to imagine such biases when comparing the IBD values of full siblings, which presumably are unknown to the individuals themselves, (e.g. how am I genetically related to my full sibling- by .43 or .58?). We may not be able to detect our phenotypic relatedness but marker data can.

The second method involves comparing the IBD values between so called 'unrelated' individuals and comparing these individuals on a given phenotype. These methods are outlined in a paper by Yang et. al. (2010), and demonstrate that accurate heritability

estimates of height can be obtained from large samples of 'unrelated' individuals. The presumption is as follows. Because all humans are related, and some are more related than others; given a large enough sample size and variance, the diversity between matches on any given trait can be attributed to either additive genetic variance or environmental factors. If 'unrelated' individuals (individuals who have kinship values that are less than .01) share similar phenotypes then we can assume that this is due to shared environmental effects. For example if unrelated individuals in Iceland reach similar heights or achieve similar lifespan's and this is unrelated to their shared degree of relatedness, we can assume that these traits have low heritability's in the environments in which they were measured (e.g. 19th century Iceland).

Both methods (using IBD values between siblings and or unrelated individuals) require large samples, and thousands of markers, but are becoming increasingly feasible in an era of inexpensive and efficient DNA sequencing. In addition, both methods require intensive and vast amounts of computing space and processing speed. The comparison of large samples of individuals on both IBD values and phenotypes requires the use of Generalized Linear Models and other complex statistical software. Because the samples are not independent, however, mixed linear models are generally used to obtain heritability estimates for any given trait. This is always true when the variables are inter-related, such as they are in genealogies. I used software developed by Peter Visscher, which was freely downloadable off his website called Genome Wide Complex Trait Analysis, GCTA (Yang, et. al., 2011). This software requires data to be entered in matrices that are as wide and long as individuals you use (e.g. 5,000 individuals requires a matrix with 25,000,000 values or data points – in my case it was the relatedness

between every individual in the database). So it is easy to see how quickly computing power is absorbed by these statistical techniques, especially when multiple iterations are run. GCTA, the heritability estimation software, developed by Peter Visscher, uses a restricted maximum likelihood (REML) method to obtain heritability estimates. This method is typically used when samples are 'non independent' (for full methods and use of GCTA software for restricted maximum likelihood-GLMM-models see Visscher et. al., 2006).

There are two important methodological issues to note. The first is that when we average the RS or lifespan of all the grandchildren, the accuracy increases (Börger, et. al., 2006). One problematic and undesirable result of this is that the more grandchildren one uses the more inflated the correlation becomes. This is a basic statistical principle. The more measures we use, the closer we get to the 'real' measure. When sample sizes increase, the correlation also increases. This is why large sample size is so important for achieving significant results in statistical analyses. One undesirable result of this is that correlations between the reproductions of siblings from bigger families (e.g. more grandchildren) will necessarily be higher than those from smaller families. I controlled for this problem by programming into the selection process a random factor. The simulations choose two children in random order to eliminate this effect (e.g. the 1st is just as likely to be chosen as the 4^{th}). After running a series of tests I successfully controlled for this inflation effect. To briefly summarize: for the parent-offspring associations, I used the correlation between the average number of grandchildren per child to the average number of children for each set of parents (number of siblings plus the proband itself). The correlation between the children of full siblings (i.e. nieces and

nephews) were taken from the correlation between the number of full siblings an individual had plus one (again, to include the proband) and the average number his or her siblings children had by taking two at random and averaging them, to avoid the previously mentioned inflation effects. Therefore larger families no longer yielded larger, spurious correlations. This successfully eliminated the inflation of correlations and heritability estimates from the simulations so larger families no longer yielded larger correlations simply due to their size.

GCTA successfully eliminates interdependence and inflation effects by running multiple iterations through the restricted maximum likelihood (REML) statistical technique. The program itself assumes non independence of data (e.g. full siblings are not independent of offspring, nor are offspring independent of parents and full siblings).

Genome wide complex trait analysis (GCTA)

As previously explained, a generalized linear mixed model (GLMM) is often used when data sets are not normally distributed (non-Gaussian), exceptionally small, or when they are interdependent (Bolker et. al., 2009). In heritability studies, where family effects are high, heritability (additive genetic variance) is low and sample size is low (relative to the standard error) and the standard errors are high, a particular statistical technique known as a Restricted Maximum Likelihood Model (REML) is frequently used (Corbeil and Searle, 1976). In this study, REML was used to estimate the heritability for three traits: height, fertility and longevity. In the end, the standard errors for lifespan were too high and I was left to analyze height and reproduction alone. GCTA software, or genome wide complex trait association, developed by Yang et.al. (2006), is used to estimate additive genetic heritability. The sample I chose (all women under the age of 54

and all men under the age of 60) was exclusively based on maximizing sample size while eliminating most non-reproducing members of the population. For height, we retained 6,159 full siblings with genomic data (230,000 SNP's) available, 2,337 parent and offspring pairs, and for reproduction we retained 8,456 full siblings and 3,489 parent and offspring pairs.

A method for measuring the impact of parental care

All of the previously mentioned methods may be used to assess the impact of the environment or genes on any given trait. But the difference between full sibling correlations on a trait and parent-offspring correlations may be a useful method for determining a more specific environmental effect, that of parental investment (PI) on offspring. This may seem to be a misrepresentation of the evidence. We really just have correlations between full siblings and parents and offspring on a couple of traits. How is this a proxy for PI per offspring? In this paper I hope to persuade you that the difference between these two correlations is a measure of parental care or PI and will argue that the larger the gap between these correlations, (see figure 2.1 and table 2.1) the greater the effect of PI.

Quantity-quality tradeoff

The first discovery that attracted my attention was that the correlation between the RS of parents and offspring was consistently either negative or non-existent (see figure 2.1 and table 2.1). To put it another way, large families do not produce children who go on to produce large families. In fact the opposite is more often the case; children from large families tend to produce small families and children from small families tend to produce large families. This relationship alone is indicative of a quantity-quality tradeoff. It also

suggests that parents have an important *environmental* influence on their children. Individual who produce large families also produce more grandchildren. So there is no 'optimal' clutch size as in birds; the more children you have the, more grandchildren you will have. Even though parents and offspring steadily and consistently maintained a negative relationship over the centuries considered, parental production of children is strongly and always positively associated with grandchild reproduction ($r=.67$, $p<10^6$); see figure 1.1). But it is still important to analyze the *quality* of offspring in relation to family size. Specifically, does the expected number of grandchildren per child decrease as family size increases? Focusing solely on the number of grandchildren produced (the combined lifetime reproductive success of one's children) is not enough to answer this question because it fails to account for the 'quality' of the offspring produced, thereby discounting the effect of PI entirely. Even if the children produced by large families are of poorer evolutionary quality than those produced by smaller families, this result will usually be hidden (e.g. the correlation between an individual's RS and his or her grandparental RS will always be positive unless there is an extreme, almost impossibly high quantity-quality tradeoff) . The more offspring one produces, the more grandchildren they are likely to have, unless the quantity-quality tradeoff is exceptionally strong and the children from large families severely underperform the children from small families. For example, parents who have 5 children would need to produce 20 grandchildren (RS per child = 4) while parents who have 10 children would also need to produce 20 grandchildren (RS per child = 2) in order to eliminate the positive correlation between parental RS and grandparental RS. In other words there would need to be an impossibly sharp decrease in the RS of children as family size increased in order to

eliminate the positive correlation between the size of the F1 and F2 generations. So concentrating on the relationship between offspring produced and that of grandchildren produced is therefore a measure with limited used in determining if there is a quantity-quality tradeoff.

The reasons for a positive correlation between parental RS and grandparental RS are clear. But the reason that the correlation between children and grandchildren was so high ($r=.67$, $p<10^6$, see figure 1.1), despite the fact that the parent-offspring correlation was negative in most decades (see figure 2.1), was initially a puzzle. A negative parent-offspring correlation suggests a quantity-quality tradeoff and will naturally tend to reduce the strength of this relationship. But it seemed logical that the reason for such a high correlation between parental and grandparental RS was connected to PI and the 'quality' of the offspring produced. In evolutionary terms the production of grand-offspring is more important than RS and it soon became clear that the reason that the parent-offspring correlation was generally negative but the correlation between parental RS and grandchildren produced was so high (see figure 1.1) had something to do with *siblings*. I checked. It did. The number of siblings one has is the best predictor of both the lifespan and reproduction of an individual in Iceland in every decade analyzed (see results for details).

Siblings are the key

The key to measuring the impact of PI on offspring is to compare the difference between the correlation of parents and offspring on a trait and the correlation of siblings on that same trait. The difference or gap between the two indicates whether the effect of PI is more or less important; the greater the difference, the greater the importance of PI.

This method could be used, as was done here, within a single species (e.g. humans, see table 2.1 and figure 2.1) or even to assess the relative importance of PI between species. This technique and others including computer simulations (see tables 2.3 and 2.5), Restricted Maximum Likelihood methods estimating heritability (see table 2.6), and measuring the effect that the number of full siblings an individual has on life history traits (quantity-quality tradeoff, see table 2.2) are all ways to support the hypothesis that parents have had an important environmental, and non-genetic effect on their children's reproduction and lifespan.

Results:

An analysis of the data revealed that children do not reproduce like their parents. Their reproduction does however, correlate with their *grandparents* RS. The reproductive association between parents and offspring was negative in every decade between 1710 and 1890 (see Figure 2.1 – bottom line) but parental RS was still strongly correlated with grandparental RS ($r=.67$, $p<10^6$). The reason for these seemingly contradictory relationships soon became clear: an individual's reproduction is highly correlated with the reproduction of their full siblings. This was a consistent and clear relationship. It was true and consistently strong in every decade over the 300 years measured (see Figure 2.1 – top line). So the mitigating factor which was causing parental reproduction and grandparental reproduction to be so high was the positive reproductive correlation amongst full siblings. The reason that the correlation between an individual's RS and the number of grandchildren he or she produces is so high is because siblings reproduce alike (see figure 2.1). When siblings reproduce alike, they each produce a similar number of

grandchildren, and this magnifies the already naturally strong relationship between parental RS and grandparental RS.

The correlation between the RS of all full sibling pairs in Iceland across all generations between 1650 and 1960 was significant and high r=.130 (see Table 2.1). There were also some noticeable sex differences. The relationship between full brothers (r=.133 (p<.001)) was slightly lower than between full sisters (r=.145 (p<.001)) and there was an even lower association between opposite sex pairs, (brothers with sisters) (r=.117 (p<.001)). I did not conduct the statistical analyses required to determine whether any of these differences (e.g. the difference between the full brother's correlation and full sisters) were statistically significant. I also summarize parent-offspring correlations in reproduction (see Table 2.1). There are some "slight" differences in the regressions between mothers and fathers (e.g. fathers with sons; r=.001(p=0.766) vs. mothers with sons; r=-.014(p=0.003)) but it is not nearly as pronounced as the sex differences between same and opposite sexed siblings (see Table 2.1) and the data is of considerably less interest.

The association between the fertility rates of full siblings is relatively stable across decades, is always significant, and never falls below 0.1. The parent-offspring correlation, however, fluctuates considerably over time. In general, the trend is negative prior to 1890 with few exceptions. The full sibling correlations also change over time but they are far more stable (see Figure 2.1). Furthermore, the parent-offspring and full sibling curves are not correlated (r=.040, p=.838). The parent-offspring curve, however, is positively related to the population estimate at the end of each decade analyzed (r=0.545, p=0.005) while the full sibling curve is slightly but non-significantly and

negatively related to population (r=-0.298, p=0.148). Finally, the gap between the two curves is significantly and negatively related to population estimates at the end of each decades (r=-0.634, p=0.001).

The aforementioned negative associations are largely a function of the parent-offspring curve which exhibits greater volatility and contributes more to both gaps (full sibling correlation, and population which is not shown in fig 2.1). There is also a general trend for both curves to converge over time. The parent-offspring curve steadily rises while the full sibling curve steadily falls. It is also important that all of the data in all of the tables and figures in chapter 2 are standardized by decade. This was done to make the data comparable over time. Mortality and fertility rates are constantly changing and standardizing the data is the only way to analyze and effectively compare the data by decade so that the decades were comparable. All values for the reproduction and lifespan of all individuals were $\log_{10}$ transformed and then standardized by the decade in which the individual was born. Transformed values were standardized by decade in the following way [(log transformed value – mean of decade)/ standard deviation of decade)]. This provided variables that were all relative within the decade in which they were born. Decades can now be compared to one another, independent of rising or falling birth, mortality or population rates across decades and even centuries. Another basic reason for standardizing the data by decade is so that we can isolate variables that are not tainted by falling or rising population rates.

A quantity-quality tradeoff for reproduction and lifespan

There was a significant and consistent decline in both expected reproduction and longevity per additional child (see table 2.2). Children without siblings live longest and

reproduce most. Each subsequent child reduces the average longevity and RS of that sibling group (e.g. three full sibs live shorter lives and have fewer children than two full siblings, while they have more children and live longer than groups of four siblings). This pattern continues predictably and without exception for each subsequent child through the group 10+ and is true of both raw (computed from standardized values x standard deviation for each sibling category mean for all sibling categories across all decades) and standardized values. The p-value = 1.3×10^{-7}. This indicates a clear and unambiguous tradeoff between offspring quantity and quality. The more children parents have the worse each child in that family performs on measures of both lifespan and fertility. This is true even without any data on the resources or socioeconomic status of the families. Although it has never been confirmed, it is assumed that in Iceland and most other European pre demographic transition countries, wealth was positively associated with family size (Kirk, 1996). To some this contradicts the idea that the superior wealth of larger families itself is what leads to larger families. Currency is traded for offspring. Larger families, however, also run against a strong dilution effect: the more children parents have, the more these resources become divided. Wealth and PI are both subject to dilution and their effects will decrease in proportion to the production of each successive child. Genes also affect the lifespan and RS of children but unlike PI and resources, they are not affected by family size. While the transmission of genes to offspring is not impacted by number of children, PI and resources are. Lifespan and fertility are both highest for only children. Both traits successively and proportionately decrease with the production of each successive child, reflecting a strong and consistent quantity-quality tradeoff.

Simulation results

I ran multiple simulations of a three generation genealogy to distinguish between the simulated effects of parental resources and genes on offspring reproduction. I found that the Icelandic data most resemble a resource model, where offspring reproduction is primarily determined by parental resources. In this model, resources are divided equally amongst all full siblings. This was done to simplify the model although this was not strictly true in reality (e.g. first born sons usually inherited the farm) (Karlsson, 2000; see tables 2.3 and 2.5). Results reveal a negative association between parent and offspring reproduction and a positive one amongst full siblings. Unsurprisingly, a genetic model in which offspring inherit their parent's RS with some probability, elicits nearly identical parent-offspring and full sibling's correlations. Of course this is true because one's genetic fitness is not diluted by the number of siblings one has. So the simulations support the intuitively appealing argument that when full siblings behave alike while parents and their children do not, PI is more influential than genetics. The analysis of the real Íslendingabók data lends further support to this interpretation.

One interesting result that may not be as intuitively obvious is that as the resource standard deviation increases in the resources simulation, the parent-offspring and full sibling correlations converge. The parent-offspring correlation becomes less negative and approaches zero while the full sibling's correlation becomes less positive and also approaches zero. One interpretation of this result is that as income is distributed more unequally, PI becomes more important and genes less important.

When there are wide differences between families in access to resources, the previously mentioned 'dilution effect' may not be very important for rich families and the

same may be true for extremely poor families. The Rockefellers have enough no matter how many children they have and on the opposite end zero divided by three is the same as zero divided by twenty. Luck and genetic fitness may therefore play more important roles for those on both ends of the access to resources spectrum when resources are more variant. On the other hand, the opposite pattern is seen for heritability estimates with many traits. For instance as variance for nutrition increases, the heritability of height decreases. So what is happening here? Does increasing environmental or resource variance increase or decrease the importance of PI. One critical piece of information that would have helped to answer this question, income, was missing in Iceland. Additional reasons for these contradictory results are suggested in the discussion section.

Multiple iterations of the resources model revealed that when resources were distributed unevenly at the outset (the F1 generation), the subsequent economic inequality in the following generations diminished the otherwise strong relationship between the reproductions of full siblings and it increased the reproductive relationship between parents and offspring. Put another way, the full sibling correlation decreased and the parent-offspring correlation increased; they converged. This result also lends support to this interpretation that PI is less important under conditions of extreme economic inequality.

The genetic model in table 2.5 was based on 200 simulations. There were 1,000 parents (F1 generation) with an average reproduction per generation of two and a genetic fitness equal to one. The full sibling correlation was the relationship between the average number of grandchildren per child and the number of siblings one had. The full sibling correlation (Table 2.5) was strongly affected by the number of children that parents

produced. We standardized the effect of each successive child produced by selecting two randomly computer chosen children. Again, the reason the simulation picks the first two random siblings is to avoid inflating correlations which is the inevitable result of increasing comparisons and therefore sample size.

The most plausible model is one where parental resources are assigned randomly, and reproduction depends on the resources parents were allotted, as does the reproduction of their children, where parental resources are divided equally amongst them. It is important to note, however, that as the resources standard deviation (variability) increases, the parent-offspring and full sibling correlations converge (e.g., parent - offspring correlation becomes less negative and approaches 0 and the correlation between full siblings becomes less positive and approaches 0). The parent-offspring correlation rarely goes above 0, however. Why? As resources become more variable in the F1 generation, parent and offspring reproductions become more associated with one another, and full sibling's reproduction becomes less associated with one another. The reasons for these effects are the result of less noise being introduced into the correlations. As randomness increases, the correlations will always flat line. This is one of the fundamental functions of statistics and science – distinguishing patterns from randomness.

When parental reproductive success (RS) has a high standard deviation (10) so that parental reproduction does not depend on resources – full siblings are correlated at .89. If parental RS is dependent on resources, then we get a full sibling correlation of .23. This is true because full siblings are more similar when you take out the intervening factor

(noise). On the other hand, when parental RS is random and not tied to resources, the signal is louder when variance increases (see Table 2.3).

When both resources and genetics are run (Table 2.5), and resources in the F1 generation have a mean of 2 with a standard deviation of 2 and mean children per generation is equal to 2, the parent offspring correlation is .06 and the full sibling correlation is .21. When resources are distributed randomly in the parent generation and offspring resources are then divided equally by children, the simulation looks most similar to the actual data found in Islendingabok. In this case, the average parent offspring correlation is -.36 with a standard error of .06 and the average full sibling correlation is +.28 with a standard error of .027. But when resources are dependent on offspring number (children provide a net benefit to parents rather than a cost) so that parental resources are assigned based on the number of offspring that parents have (e.g. more offspring=more resources), both the parent offspring and full sibling correlations are predictably and strongly positive.

The simulation values provide a valuable source of information. I ran hundreds of simulations and changed all of the variables multiple times in an effort to measure the different effects of changing the mean reproduction of parents, the variance or distribution of resources, and altering the relative impact of alleles (see appendix A to run code and results will appear in excel, also see appendix B to run these results directly from excel). All one is required to do is enter the number of iterations, the original population (N), the mean of the Poisson distribution for children per generation, a standard deviation around this mean and the associations you would like as output (Appendix A can be copied into excel as a macro and run). Results of providing equal

weight to both genes and resources and is shown in table 2.5 under the genes and resources categories. Entering the same parameters for resources but not including a genetic effect produces effects in table 2.5 under resources and most resembles the real data shown in figure 2.1 or table 2.1.

The simulations suggest that parents do not have any genetic impact on their children's reproduction or lifespan. Instead they reveal that PI (measured as the correlation between the reproductions of full siblings) has an important impact on the reproduction of children.

Coincident with these results, I also demonstrate clear and unambiguous evidence of a quantity-quality tradeoff: as parental reproduction increases, the lifespan and reproduction of their offspring decreases. Furthermore, the genealogical and SNP data are consistent with simulations run in a resource model (where parental resources are the sole determinant of offspring reproduction and longevity) but not a genetic model (in which genes are the sole determinant of offspring reproduction and lifespan). Although I did not have any direct measure of PI, these findings support the hypothesis that PI in offspring, and not genes, has played an important role in determining both an individual's reproductive success (RS) and longevity in Iceland over the past three centuries.

The simulations have been run repeatedly and all variables have been changed independently to isolate their effects (e.g. fitness, resources, mean number of children per individual etc.). See Appendix B for simulation results in excel. This macro (in Excel and Visual Basic Macro) allows you to change any of the parameters and run any of the simulations discussed.

General parent to offspring correlation notes

When the population increases, the correlation between parent and offspring reproduction decreases, whereas when the population decreases, the parent to offspring correlation increases. One interpretation of this result is that as competition over resources increases as the population rises, the quantity–quality tradeoff is heightened so we see a stronger negative impact of parental reproduction on offspring (e.g. large families produce poor children and small families produce high quality children) (see discussion for a more detailed discussion of this effect). Finally, average reproduction per decade in Iceland is negatively associated with both the full sibling correlation (r= -.43) and the parent-offspring correlation (r= -0.31).

Table 2.2 provides a window on the impact of resources on reproduction. Mean reproduction is negatively associated with number of siblings (e.g. the more siblings one has the worse one reproduces; r= -.742 (p=0.006)). An ANOVA test for linearity showed that sibling number was a significant and negative predictor of reproduction (r= -0.052, $p<1.2\ X^{10-4}$) and lifespan (r= -0.082, p<0.001) lifespan.

The effect of birth intervals on similarity in reproduction and lifespan

To further test the effect that full siblings have on an individual's reproduction and lifespan, I looked at the effect of birth intervals between sibling pairs on the differences in lifespan and reproduction. There was a positive and significant association for both of these traits (see Table 2.4). The closer full siblings are in age the more similar are their lifespan and LRS.

Genome wide complex trait analysis (GCTA)

GCTA software was used to estimate heritability from the genetic data that was available in Iceland (see table 2.6). GCTA uses a restricted maximum likelihood model

(REML) to obtain estimates of additive genetic variance for any trait (Yang and Visscher, 2010). I used the software to estimate heritability for height, reproduction and lifespan. The results for height were shown for comparative purposes, as it is often used as a control to ensure the reliability of these methods (Visscher, 2006). Height has been repeated in multiple populations and generally varies between .7 and .8. This was my first time using GCTA and running a REML so I wanted a control to make certain I was running it correctly. For height, we estimated an additive genetic variance of around .70 when using both parents and offspring, and full siblings (see Table 2.6). This was the same estimate that other researchers at deCode genetics have found for the heritability of height using all 'unrelated' individuals in the genetic database at deCode Genetics (Helgason, pers. communication). All of these estimates are at the lower end of the range of heritability estimates for height from studies of various populations around the world which are usually around .8 (Visscher, 2010).

Using only full sibling pairs for which SNP data was available, I estimated a family effect (the effect of familial environment less genetic effects) of .14. For reproduction, the parent-offspring estimation was 0. Estimates of the family effect of full siblings were 0.09 familial and 0.00 additive genetic. The standard errors were quite high when I ran both matrices together and this was especially true for reproduction, so interpreting some of these results was problematic. They are, however, consistent with the estimates from the genealogy. I tried estimating heritability for longevity but the standard errors were too high so the results were not interpretable. I also ran the matrices with cousins and half sibs for a number of other traits controlling for various factors such as sex (in particular for height) or geographic region with varying degrees of success. Each run

was extremely costly in terms of computing power, and each analysis was run on hundreds of computers at deCode, known as the cluster. Time use on the cluster was also in high demand and I did not have constant access. One interesting thing to note here are that the straight correlations between IBD values (degrees of relatedness between full siblings or cousins or parent-offspring pairs are listed) are approximately half the value of the additive genetic variance estimates resulting from running a REML model in GCTA. In theory the correlations should be roughly half the h^2 values, and they are (see Table 2.6).

GCTA uses matrices of pairs of individuals as input. For example 4,000 full sibling pairs requires precise relationship values (based on the SNP data from deCode) for 16,000,000 pairs. In most cases the degree of relatedness is 0, but for the 4,000 full sibling pairs the IBD value (not the degree of relatedness) is entered. For full sibling pairs, this value is usually between .18 and .32 and on average is .25. In addition, another 2 column X 4,000 row matrix providing the phenotypic values for any given trait are entered (e.g. height for the 4,000 full siblings). When the two matrices are entered and a REML model is run, GCTA outputs both a heritability estimate and a standard error. For family effects (e.g. the environmental effect of being a full sibling, parent or child), the precise IBD values are replaced by one's for all relatives and all non-relatives are again entered as zeroes.

When the effect of being a relative (family effect) is strong, heritability (additive genetic variance) is low, and sample size is low, standard errors will be high. These factors will always increase the standard error of heritability estimates. Also, if you increase the IBD value (the degree of relatedness) the heritability proportionally

decreases (e.g. if you change the IBD value from ½ - as in the parent-offspring matrix - to 1 as in the family matrix – the heritability estimate is halved (.7 to .35). GCTA results show that there is no relationship between parent and offspring reproduction outside of reproduction. This is the only effect on children who have had at least one child as compared with those who have had zero. (See table 2.6 for all REML results for all heritability (additive genetic) and familial (environmental effects from being part of the same family).

Discussion:

An in-depth analysis of a multigenerational genealogy, spanning three centuries, known as Íslendingabók suggests that parental investment has had a positive effect on both offspring reproduction and lifespan. I demonstrate this effect in four ways. First, the lifespan and reproductive success of an individual's full siblings is the best predictor I found of an individual's longevity and reproduction (see figure 2.1 – top line and table 1). Second, computer simulations of a three generation genealogy most closely resemble a model in which resources, but not genes, affect a child's lifespan and reproduction (see table 2.3; table 2.5). It is possible; however, that some of this effect is the result of full siblings helping one another. In support of this interpretation, I found evidence that the closer siblings are in age, the more they resemble each other in both lifespan and fertility (see table 2.4). This suggests that peers (siblings close in age are often part of the same peer group and share friends) can have powerful effects on some life history traits. A third line of support comes from genetic data from the genotyped individuals in Iceland. The genetic relatedness between these full sibling and parent offspring pairs reveals low to no heritability for reproduction but suggests that there is a strong family effect on

reproduction (see table 2.6). Finally, there was a strong quantity-quality tradeoff for both reproduction and longevity. Parental reproduction affects the quality of children; the more children that parents have, the shorter the collective lives and reproduction of these children (see table 2.2). This is a linear relationship. It is also one of the primary causes of the negative relationship between the fertility of parents and offspring shown in figure 2.1. One important weakness of concluding from these results that PI has had an important impact on lifespan and reproductive success is that I do not have any direct measure of parental investment.

The tradeoff between offspring quantity and quality is a fundamental principle in evolutionary biology (Lack, 1974). Each additional child parents produce necessarily dilutes the total amount of investment (resources, time etc.) parents can invest in each individual child. This is necessarily a linear relationship, so that as an individual's number of sibling's increases, the amount that a given child in that family receives from the total parental pool of resources decreases. To put it simply, the more children parents have, the fewer grandchildren they have per child, and the shorter the lifespans of their children. It has been well established that as family size increases, academic performance declines, and this is generally seen as a function of a dilution effect (less resources per child) (Downey, 1995; Hanushek, 1992; Blake, 1981). A negative relationship between family size has also been found for IQ (Black, et. al., 2007) standardized test achievement scores (Grawe, 2008) and PI (Rosenzweig and Zhang, 2006), and a trade-off for both longevity and reproduction has been reported for a population in Ghana under difficult economic conditions (Meij, et. al., 2009).

Full sibling correlation vs. correlation between parent's and offspring

An analysis of the correlations between the reproductions of full sibling pairs and parent-offspring pairs reveals a strong correlation between the reproduction of full siblings, but not between parents and offspring (see Table 2.1 and Figure 2.1). There are also notable sex differences for the full sibling pairs so that same sex pairs reproduce more similarly than opposite sex pairs. There may also be some slight differences in the regressions between mothers and fathers but it is not as pronounced as the differences between same and opposite sex siblings. The ability to detect differences between opposite and same sex parent-offspring pairs, however, is likely diminished by the widespread practice of monogamy because a mother's and a father's RS are almost always identical. So these results may be quite different for polygynous populations.

Full siblings share parents; parents and offspring do not. But the parents of parents are the grandparents of these parent's children, so these relationships are all interrelated. I have suggested that the full sibling correlations reported here are evidence that PI has played an important role in the fitness of individuals in Iceland. Another way to think of the full sibling correlation is that it is the effect of genes plus the effect of familial environment and parental influence. Full siblings share the same parents, an identical number of siblings and genes. Parents and offspring share only genes. This is one important distinction between the two relationships and may be the ultimate cause of the differences we see between the two groups. Of course parents also often share a common household with their offspring but they do not share a common household during the key period of parental investment - PI prior to reproduction. Siblings do. The difference this may makes on the life history traits of an individual is not known but it could have an important effect.

The parent-offspring correlation appears to be driven almost entirely by parental reproduction. If it is positive, then the number of siblings one has (parental reproduction) is positively associated with offspring fitness (e.g. both longevity and reproduction). If it is negative, then the number of siblings is negatively associated with offspring fitness. In other words there is not a quantity-quality tradeoff for parents in periods when the parent-offspring correlation is positive (see figure 2.1). In addition, the effect of parental reproduction on the longevity of their children is significant and negative.

The risk of extinction

It is interesting to note that the three peaks of the parent-offspring correlation curve occur at the same time as the three greatest population declines during the period I analyzed (1650-1950). These events were the smallpox epidemic, the eruption of the Laki volcano and a massive migration to Canada. After the smallpox epidemic in which 26.4% of the population is estimated to have died (Adalsteinsson, 2007), the survivors may have had increasing opportunities to reproduce as farmland opened up and offspring from large families may not have been as constrained by resources as they had been previously. Parental resources may be less important at low densities while at high densities they may become more important. In other words as competition increases so too does the importance of PI. This interpretation is also supported by the previously mentioned (see results section) negative relationship between population and the parent-offspring relationship in reproduction. As population rises, the quantity-quality tradeoff increases in importance (parent-offspring correlation decreases). In a lizard population two morphs (K strategists and R strategists) were studied and the morphs that produced few larger eggs were favored at high densities while R strategists who produced more but

smaller eggs were favored at low population densities (Sinervo et al., 2000). Perhaps what we are seeing in Iceland during periods where the population may have been capped at 50,000 is opportunities opening up as farms became available during periods of severe population decline (e.g. following plagues, volcanic eruptions, widespread famine or large scale emigration) so that offspring quality became less important. During these periods selection favors R strategists. The generally negative parent-offspring correlation may become decoupled during these periods and can actually become positive (see Figure 2.1).

Conditions in Iceland begin to improve in the late 19th century and continue to do so throughout the 20th century. The consistently declining gap between the two trend lines (correlation lines) and the general upward trend (both lines) across the entire chart could be the function of two important historical trends. First, an improving environment and relative quality of life is expected to have this effect (see results section) and second, a decreasing importance of resources on reproduction should also have these effects. Height, for instance, becomes more heritable when the effects of environmental variance dissipate. In the case of Iceland it was easy and uniform access to nutrition that lowered environmental variance in height and likewise increased heritability estimates over time. Perhaps as resources become more accessible, the effect of PI on reproduction and longevity is lessened and the two curves converge (as seen in figure 2.1).

There may also be an explanation for the aberrations in Figure 2.1. The peaks in the parent-offspring correlation curve, and more subtly, the valleys in the full sibling correlation curve, are associated with birth decades which have had population crashes. Another way to put this is that the two lines converge (fueled primarily by a spike in the

parent–offspring correlation line) at three key events: the smallpox epidemic of 1707-1709 (26% estimated mortality rate; Adalsteinsson, 2007)(measurement based on all births 1707-1709), the Laki volcanic eruption of 1783 (1/4 of the population predicted to have died and 1/2 the livestock died) (Jackson, 1982;Thoranissin, 1961) (measurement based on all births in 1783) and during the massive migration to Canada between 1870 and 1910 in which more than 20% of Icelanders for the first time in their history voluntarily left the island in large numbers (Green and Green, 1993; see figure 2.1 for points on graph) (measurement based on births and mortality between 1870-1910). So are the offspring born in years of massive population decline benefiting from these crashes 30 years later when they begin to reproduce? Does this pattern follow a more familiar one that typically follows major extinctions? Massive mortality opens up new niches and opportunities for those that survive and their offspring. Certain species of fig wasps lay their eggs in specific species of fig trees (Machado et. al., 2005). The population of fig wasps is therefore limited to the number of fig trees that it can find. In Iceland, the limiting factor is likely farmland. And this was almost certainly true prior to industrialization (Karlsson, 2000). As mortality increased or populations left the island new niches may have opened up and the birth cohort for those years was therefore more successful.

These three population crashes were also characterized by extremely poor and difficult conditions on the island. So why then is the parent to offspring correlation in reproduction positively associated with population density while the full sibling correlation in reproductions seems to be negatively, although non-significantly, associated with it? Once again, the parent offspring correlation is in large part driven by

the quantity-quality tradeoff so that when the correlation is negative the tradeoff is high. So there is a higher tradeoff for parents when the population is low. There is also a bigger tradeoff when the gap between the two curves is high (see figure 2.1 and results section) suggesting that when PI is more important the population is relatively low. In other words, PI becomes less important as population increases. This may be because children are being influenced more by peer groups or siblings than by parents during periods of high population growth when there are more children (friends and siblings) around. It may also be because there is less competition for farmland when population is low so that PI becomes more important while random luck, other environmental factors and genes become less important.

The most plausible model, however, is that when parental resources are assigned to individuals randomly, reproduction depends on the resources an individual was allotted. So does the reproduction of one's offspring. This process occurs on a resource or PI level, but not a genetic one. The division of resources or PI amongst offspring is the defining characteristic of the quantity-quality tradeoff (Gillespie et. al., 2008) and is a general problem faced by all species. The converging of the curves at the end of figure 2.1 may be the result of the demographic transition and the respective increasing wealth of Icelanders. The wealthier a population becomes the less of an impact resources or PI will have on both longevity and reproduction. How much does increasing ones wealth from one hundred thousand to one million dollars increase one's ability to produce offspring? At the very least the relationship between wealth and reproduction is unlikely to be strictly linear. One aforementioned result of the simulations (see methods and results) demonstrates that as wealth is increasingly stratified – the standard deviation for

resources in the resource simulation increases – PI becomes less important. This often occurs after catastrophic events such as Laki or smallpox epidemics which are likely to strike all segments of the population equally. When this occurs (random factors or noise increases), it is not surprising that PI declines in relative importance. Geographic location (near volcanic eruptions) or the susceptibility to the contraction of contagious diseases becomes more important while PI becomes less important.

Average reproduction per decade in Iceland is negatively associated with both the full sibling correlation (r= -.43) and the parent-offspring correlation (r= -0.31). One possible interpretation of this fact is that in bad decades (defined by low average reproduction of that cohort) resources are more important. All groups (parents, offspring and full siblings) reproduce independently of one another when resources are scarce. Another way to say this is that siblings behave unlike, or even in opposition to, one another. Parents and their children are also independent of one another when resources are scarce. I predict that in the next 50 years both curves will continue to converge. As a society becomes wealthier, resources should have less of an effect on both reproduction and lifespan. The effect of cultural and environmental factors such as how one was raised and the quantity-quality tradeoff seen in earlier or poorer generations will become less determinate while randomness will begin to play a greater role in determining ones RS or lifespan. I should note, however, that at this point all of this is highly speculative.

Population crashes may reflect two contrasting possibilities. First, conditions could be poor and the environment harsh. The second possibility is that competition is low during these crashes and farmland plentiful. So far, the data suggests the latter. Parents are most important and the quantity-quality tradeoff is the highest when population and

competition for land is lowest. There are reasons to be skeptical, however. We should be cautious when inferring too much from these relationships because population changes over time are a complex and dynamic interaction of multiple factors. Using data on population changes alone as a proxy for conditions in Iceland is dubious at best. Reproductive rates per decade, as reported above, should also be treated with some skepticism. Here, I suggest that higher rates indicate better conditions. This may be true but, once again, there are a great number of factors that affect fertility. The mystery about the root causes of the demographic transition should be enough of a warning for us to be too confident in our assertions about the causes of fluctuating fertility rates over time.

Extinction risk and catastrophic events

A founder population is defined as a group formed by following an evolutionary bottleneck and is particularly susceptible to extinction due to reduced genetic diversity among the inhabitants. This has happened several times throughout Icelandic history and the island was nearly abandoned at least three times (Karlsson, 2000). Votes were actually taken on one occasion to decide upon the feasibility of surviving in a hostile and eroding landscape and abandoning the island permanently was a real consideration (Karlsson, 2002). When a population crash occurs at the same time that resources are becoming more unequally distributed (resulting in a declining quantity-quality tradeoff) the risk of extinction should increase. This is often referred to as the gamblers ruin (Harik, et. al., 1999). Island populations with small founding populations or bottlenecks also exacerbate both inbreeding depression (Frankham, 1995b) and the risk of extinction (Frankham, 1998).

The simulations add weight to these speculations. The resources data in Table 2.3 most closely match the real data from Islendingabok in Table 2.1. This further indicates that PI and resources are driving the full sibling association. The reason for this is likely due to the fact that, as resources are distributed more unequally (higher variation) amongst the population, the wealthiest parents have the most children (Kirk, 1996); and the effect of distributing resources amongst the offspring of wealthy families is lessened. So when the two curves converge (see figure 2.1) the population is either in serious decline or resources are being distributed more unequally. Another often underestimated factor in evolution is randomness and luck. Natural disasters can have beneficial effects on equality. Volcanoes may not distinguish between the wealthy and the poor (Adalsteinsson, 2007). Such random events can diminish the effect of wealth on both RS and lifespan. Natural selection also has few tools for dealing with or preparing for massive volcanoes. Still, the main tradeoff is between maximizing the number of children one has and the partitioning of parental investment (Trivers, 1972) and resources amongst them. A primary assumption underlying this principle is that as offspring number increases, the amount of PI and resources given to each child decreases. Following major natural disasters, some children prosper while others die, creating new niches while economic homogeneity is restored. The wealth gap seems to dissipate after these types of cataclysms (see Figure 2.1).

Wealth and family size

If an association between wealth and greater family size was true through most of Icelandic history, then the full sibling correlations in reproduction we observe are actually being diluted. They would be stronger if Iceland was truly an economically

homogeneous society with no social stratification or differences in wealth. If richer families are having more children, then the impact of dividing parental property is lower for these wealthier and larger families. In other words, parents who have big families have more resources from the outset, so that the effect of distributing them amongst their children is less than if these resources were distributed equally amongst the population, or if the population was sufficiently wealthy (see post 1950 Iceland on figure 2.1). The same logic applies to the full sibling correlation. In comparison to the rest of Europe, resources in Iceland were more equitably distributed. Despite this fact, there were substantial differences in wealth inherited across generations throughout Icelandic history (e.g., first born son received the farm) (Karlsson, 2000). So what we are seeing in table 2.2 and figure 2.1 are actually diluted by social stratification and inequality. Because conditions in Iceland began to improve in the late 19th century and continue throughout the 20th century (Karlsson, 2002) the effect of PI on basic life history traits may be reduced. The convergence of the parent-offspring and full sibling's curves suggest that this is the case. Once a certain threshold of economic prosperity has been met by a population, the diminishing pool of parental resources per child produced becomes less important, and the value of PI decreases. Reproduction is no longer as dependent on PI and is affected by other, unknown factors.

A central tenet of sociobiology is that wealthier parents should achieve higher RS (Nettle and Pollet, 2008). In contemporary societies this assumption is often violated, however, and an inverse relationship is usually seen between family size and wealth (see discussion on "the demographic transition", chapter 1; Vining, 1986). In Iceland, however, there is evidence that prior to 1950 wealthier families did have more children

(Kirk, 1996). The reproductive similarity that full siblings share as members of a group of a certain number of siblings (see table 2.2) diminishes as the parent-offspring correlation declines. So the intergenerational inheritance of wealth does not seem to be mitigated by the disbursement of wealth amongst large sibships from wealthy families. In other words, if wealthy families are larger, the inheritance of resources per individual child should be lower. So, although a quantity-quality tradeoff in number of children produced (e.g. wealthy families are bigger) should serve as a counterweight to an increasingly inequitable distribution of wealth, it is does not seem to have much of an effect on reproduction. After the demographic transition, whereby wealthy families tend to have smaller families, this effect should be much stronger, and both income and wealth inequality should continue to grow (Mace, 1998). The prospects are alarming. In the not too distant future it is possible that there will just be two classes: the super-rich and the impoverished as wealth is accumulated by a small and ever decreasing portion of the population.

When the two curves converge (see Figure 2.1) the population is often in serious decline. Furthermore, the simulation suggests that at these times resources are being distributed more unequally. If true, I suggest the following. After major disasters such as the Laki eruption of 1783 in which 1/3 to 1/4 of the population perished (Jackson, 1982; Thoranissin, 1961), new niches open up. Some organisms prosper and others perish (Walker and Valentine, 1984; Thuilerr, et. al., 1984). This process follows a simple stochastic model of extinction followed by speciation whenever the population falls below a critical level (Gomulkiewicz & Holt, 1995). Just as the mammals diversify and

thrive following the Cretaceous extinction 65 million years ago (Raup, 1986), the lucky farmers may thrive after major disasters such as the smallpox epidemic in Iceland.

Helpers at the nest

Some sociologists and demographers have argued that increasing PI is the major cause of lowered fertility (Mulder, 1988). Some models optimizing human fertility have even indicated that this was a major cause of the demographic transition (Mace, 1998). Another factor that may impact a tradeoff between the quantity and quality of offspring, however, is the extent to which children help their parents and siblings. Although we are used to thinking about parents investing in children, a behavior that is discussed less often is a form of investment that flows in the other direction: juveniles aiding parents and helping to raise their siblings. Although common in birds and other species (Rubenstein, 2011; Lessells and Avery, 1987; Emlen et. al, 1986), there is scant evidence to support this hypothesis in humans (Crognier, et. al., 2001).

Bigger families may have been richer in Iceland but, despite this countervailing pressure, there remains a significant quantity-quality tradeoff in Icelandic families. This tradeoff is a consistent and constant factor across several centuries of data. In addition to the likelihood that wealthy families were larger in Iceland, the extent to which offspring (especially later births) help their parents and their siblings will also tend to reduce the effect of a quantity-quality tradeoff. When children help their parents, their cost is obviously lessened.

Thus, children may not solely benefit parents through their ability to produce grandchildren. They may also benefit parents by working around the farm, helping to raise siblings or by aiding parents in the production of more offspring in other ways. By

the age of eight in some cultures, parental investment (PI) may shift to offspring investment (OI) in parents (Konner, 2010). It is hard to imagine this in modern western societies where children contribute little to family resources.

Ivory billed woodpeckers are one example of helpers at the nest. Nesting sites are both rare and competitive; so many offspring cannot raise their own broods until a breeding site becomes available (Chazaretta, 2011). Until then, young woodpeckers will provide substantial help for their parents, feeding and caring for siblings. The age at which PI in offspring switches to offspring investment in parents is species dependent, and probably depends on the individuals. The effect of helpers at the nest in our own evolutionary past may be underestimated (Konner, 2010). Farms in Iceland might be a limiting factor in the same way that nesting sites are for the Ivory billed woodpecker. Young adults may have had no choice but to help parents and siblings until they could either inherit or purchase farmland. These sites may take some time to open except in the case of major population declines, like Laki (see figure 2.1). Despite the countervailing pressures of helpers at the nest and wealthy families being larger, both of which should mitigate our ability to detect a quantity-quality tradeoff, we still see a strong familial effect of resource and PI dilution on life history traits of Icelanders.

Computer simulations

The simulations, replicable using code in appendix A and appendix B (in excel), all point in the same unambiguous direction. Full siblings behave similarly and parents and offspring do not when resources are permitted to affect reproductive success. This is true regardless of what parameters (e.g. standard deviations of family size or average fertility rates) are changed. The result is robust, replicable and important. The data from

Íslendingabók support a model in which resources, or likewise, PI influences life history outcomes of offspring and no other. A genetic model does not fit the data from Íslendingabók.

Simulations are valuable ways to understand basic underlying principles and distinguish statistical aberrations from real effects by isolating and simplifying a few variables. All simulations run in Visual Basic support the resource model. It is not a statistical artifact, and the results are that: 1) parental resources matter, and 2) there is a tradeoff between offspring number and reproduction; as sibling number increases, the ability to reproduce declines. The only inflation effect occurs in relation to family size. As family size increases the correlation amongst full siblings also increases. By taking two random full siblings, we eliminate this inflation effect and obtain results that are most similar to those from the real data in Íslendingabók. Statistically 'weighting' full siblings (e.g., full siblings from families of 10 counts as .1 while those from families of 2 count as .5) achieves the same effect.

The influence of peer groups

Correlations between the traits of full siblings (see table 2.1; figure 2.1; table 2.2) may also be described as correlations between parents and offspring, but within and across categories of offspring (e.g. family size or number of full siblings). This is because an individual's number of siblings is the same as his or her parents RS. The only difference is that the correlation between siblings as shown in figure 2.1 uses actual full siblings while just the categories of full sibling number, for instance all those individuals with three full siblings, are used in table 2.2 regardless of whether they are related or not. An analysis of variance provides a nearly identical result (r^2=.014 or r=.11) in table 2.2 as the

correlation among full siblings shown in figure 2.1 (r=.130) does. These results are consistent with the hypothesis that a shared familial environment has had an important effect on the reproduction and lifespan of the people of Iceland. These results may, however, be caused by a shared culture or perhaps a shared peer group (Harris, 1995). Judith Harris in a landmark study in 1995 reviewed the literature and evidence for the effect of parents on the personalities of their children. Once genetics was eliminated from the analysis, she concluded that parents did not affect the fundamental personality traits of their children in any detectable way. The key finding was that peer groups mattered far more than parents. If Harris' interpretation of the data is true, then the impact of peer groups on life history traits shared between siblings (or even friends) may also be important.

The correlations in longevity and reproduction amongst siblings may also extend to peer groups. Table 2.4 supports this conclusion. The closer the siblings are in age the more similar are their reproduction and lifespan (see table 2.4). This does not rule out the effect of parents, but rather suggests another possibility. Peer groups affect one another more than parents do. It would be interesting to calculate the correlation between friends on these traits (longevity and reproduction) and compare it with the one we found between siblings. Are siblings closer in age affecting one another more closely because of a shared household or simply because of a shared peer group? Some data supports the view that shared peer group is the more important factor. This can be seen in table 2.1 where brothers and sisters reproduce more alike than opposite sex offspring. This is particularly true of sisters where r=0.144, while for brothers and sisters the correlation is r=0.117.

GCTA and kinship coefficients

A restricted maximum likelihood (REML) model was used to estimate the genetic impact (heritability) of reproduction, longevity and height. Kinship coefficient values for all genotyped full sibling and parent and offspring pairs were used to generate these estimates. The standard errors for lifespan were too high to obtain an interpretable result. But the analysis of height and reproduction both achieved estimates that in the case of height were consistent with previous research (Yang, et. a. 2010), and in the case of reproduction with evidence previously presented in this paper. Using kinship coefficients for full siblings revealed no detectable heritability for reproduction but did show a significant familial effect (the environmental effect of being members of the same family). Essentially the kinship coefficient values between full siblings (which vary between .35 and .65) had no effect on fertility but did have a strong effect (.7) on height. The effect of being a member of the same family, however, did have an important impact on reproduction. This was just further confirmation of the evidence previously presented that the best predictor of an individual's reproduction is the reproduction of their siblings.

Quantity-quality tradeoff

In summary of some of the key results, 1) I found a negative association between the number of siblings one has and their reproduction and longevity (see table 2.2). 2) On average, the more siblings (or more parents children) one has, the less children one has. The latter finding may account for an important part of the negative correlation we often see between the reproduction and lifespan of parents and their children (see figure 2.1). On a population level this association is even stronger. Individuals who come from large sibships do far worse than those who come from small ones. From these analyses and

findings, I make the following prediction. In harsh decades, characterized by low average reproduction, the association between an individual's number of siblings (parental RS) and their reproduction will be stronger. In other words, children will behave more like their parents as the offspring quantity-quality tradeoff lessens in importance.

To understand the reasons for the generally negative correlation between parents and offspring and the positive correlation between full siblings (table 2.1 and figure 2.1) I examined the effect of sibling number on reproduction and longevity for all individuals born between 1650 and 1910. Table 2.2 reveals a primary cause of the negative parent-offspring correlation over time. Both reproduction and longevity decline as the number of siblings increases. The raw values were converted from the values standardized by decade by multiplying the standardized values by the standard deviation for each sibling category and adding it to the mean value for all categories. This was done to try to give a raw estimate of the differences that sibling category makes on lifespan and reproduction. Lifespan was added here also because it is less obviously correlated with reproduction and less confounded by parental reproduction. This table provides evidence of a quantity-quality tradeoff for parents. The more offspring parents produce, the shorter these children live and the fewer grandchildren the parents have per individual child. This does not mean that it is better to have fewer children of higher quality. The best predictor of grandchildren is still number of children ($r=.67$, $p<.01X10^{6}$). Of course without wealth data, we cannot be certain that this effect would hold true.

An ANOVA test for linearity was used to the association between sibling number and offspring reproduction and lifespan (see table 2.2). Sibling number was a significant and

negative predictor (r=-.052, p=.000) of both reproduction and (r=-.082, p=.000) lifespan. Parental reproduction also has an important effect on the correlation between full siblings on the traits of reproduction and longevity. The similarity amongst full siblings decreases as an individual's number of siblings increases. So the reproduction of those with only one sibling are more similar (r=.183, p=.000) than those with 10 or more (r=.077, p=.000) (see table 2.3, second column).

Predicting population growth rates

Total fertility rates (TFR) (broadly defined as the average number of children that would be born to a healthy woman over her reproductive lifetime, or estimated births per woman) inform demographers when predicting population growth rates. Using evolutionary theory and life history traits, these estimates illuminate our understanding of how major demographic changes affect both culture and society, and vice versa. This information may, in turn, provide evidence which will be useful to both governmental and non-governmental organizations (NGO's) with respect to the formulation and implementation of public policy.

In addition, this research can help predict demographic and population changes worldwide, as a function of PI and sibling behavior. It may also reveal the consequences of population change, which is currently one of the greatest threats to the planet. Estimates for the carrying capacity of the planet vary, but many demographers assert the limit has been passed. The world population now exceeds 7 billion and researchers have suggested that populations over a billion are unsustainable.

Even a mild improvement in population forecasting would be extremely helpful. It

is not always in the best interests of nations to reduce population growth, however. Some countries need to encourage growth for the sake of their economy. In the United States, growth problems are a vital concern. For example, social security is currently hemorrhaging funds due to a dysfunctional government and may leave current tax payers without retirement funds. Improved forecasting could avoid such disastrous outcomes and lead to better government estimates and, in turn, programs. China serves as another useful example. After four decades, this country is on the verge of ending their one child policy. Perhaps there are some, less draconian, measures that may be used to influence families to have more or less children.

Evolutionary biologist, E.O. Wilson has called our population growth more analogous to bacteria than primates. For environmentalists, reducing the population is always considered more beneficial for the planet. Some groups and governments, on the other hand, argue that growth is essential to economic development and sustainability. Evolving life history traits have important effects on political, economic and social structures. Fertility rates and patterns, for instance, will have significant impacts on the global distribution of population and wealth, while sex ratios and dispersal patterns have important effects on both culture and health. In sum, the biological and behavioral data presented here, combined with evolutionary theories, will improve our understanding of how major demographic changes affect both culture and society. This knowledge may, in turn, assist governmental and non-governmental organizations (NGO's) with the formulation of public policy.

Conclusion and future directions

The story for the settlers of Iceland is not unlike the story of billions of immigrants and their lives across the span of primate evolution. Although Iceland is one of the last places on the planet to be colonized by humans, it is just the last leg of a continuing saga of human migration (Smith, 1995). *Homo sapiens* have been deeply influenced by their siblings, their parents, and their families in countless ways. Parents influence their children through the combined effects of parental influence, siblings, environment, and their genes. The strong correlation between full sibling's reproduction and longevity supports the conclusion that parents exert a strong influence on their children through shared resources and household. Exactly how this happens is an important topic for future original research. Are full siblings affecting each other as peers do, or do they influence one another because they share a similar upbringing through shared parents? Of course both are possibilities as well. The quantity-quality tradeoff in reproduction and lifespan as family size increases is simply one aspect of this relationship. Parents affect their children via their siblings, but they face a serious tradeoff; the more offspring they have, the shorter their lives and the lower their RS. Only future research can parse more precisely the details of this influence.

But it is also possible that the two curves in figure 2.1 represent a much larger and far more general rule that is true in all sexually reproducing species. Perhaps by comparing the full sibling's reproduction correlation and the parent-offspring reproduction correlation investigators could develop a 'proxy' measure for the importance of PI in a species. This would not yield a 'raw' number for PI but might rather be used to compare the importance of parental investment between species. In any case, parental investment has had an important effect on human children in Iceland. Although this effect may be

declining, there is a cost to increasing reproduction. Increased reproduction results in a quantity vs. quality tradeoff for family size. The details of this relationship are not entirely clear. To what extent and at what age do children begin to benefit parents, and to what extent and during which periods of development do they cost parents? The costs and benefits of children to parents is a complicated question. It is highly variable and also depends on both cultural and sociological factors. Even within the same society and during the same time period, the costs and benefits of children are likely to fluctuate and depend heavily on socioeconomic and cultural factors. This study is an early attempt to try to tease apart some of these factors. But the evidence from the genealogy (in every decade over 350 years), the quantity-quality tradeoff, the genetic data, computer simulations, age effects (peer effects) and evolutionary theory all support the contention that parents have had an important, non-genetic effect on their children's reproduction and lifespan. The evidence presented here applies to Iceland between 1650 and 1950. Whether this is a general rule that applies to other parts of the world or at different times is not known. But due to the consistency (e.g. it is true in every decade over 350 years) of the data, it is likely that this phenomenon is true in other parts of the world, in other societies and at other times in human history. The extent to which it may be true or even whether it is true of other species is another question, and one which future research may investigate.

Chapter 3

Sexually Antagonistic Effects in Iceland

Abstract

Evolutionary theories of sexual selection and mate choice often assume that 'good genes' can be acquired from opposite sex partners (Pischedda and Chippindale, 2006; Foerster, et. al., 2006; Chapman, et. al., 2003; Parker, 1979; Trivers, 1972). This belief assumes such genes are transmissible from one generation to the next. In contrast, evidence has shown that males and females often have different optimal fitness outcomes. This conflict can result in sexually antagonistic (SA) effects where genes can have negative fitness consequences when inherited by the opposite sex. For example, genes which promote aggression may increase the fitness of males while reducing the fitness of females. Nevertheless, these genes can be transmitted from fathers to daughters just as they are to sons. While SA effects have been found in a variety of species, to my knowledge, few studies have explored this hypothesis in humans. This is primarily because the widespread practice of monogamy leads both parents to achieve the same reproductive success (RS). Accordingly, it is difficult to determine the traits that may produce differential RS among the sexes. For this study, I bypassed this issue by using the accurate and detailed database, Íslendingabók. I was able to examine cases of serial monogamy, where offspring were consistently produced. Specifically, I utilized a subset of the Icelandic population who had *second* families (e.g., widows, divorcees) where paired adult males and females may have differential RS. In doing this, I am able to demonstrate that there is a suggestive, but non-significant, SA effect in reproductive

success. Through sons, the mean number of grandchildren produced by relatively high fit males (fitness defined as lifetime reproductive success) and low fit females was higher than the mean grandchildren produced through the same parents' daughters. In addition, the mean number of grandchildren produced by relatively high fit females and low fit males through daughters was higher than the mean number produced through the same females' sons. Neither group, however, reached significance for either a one or two tailed test. This is likely one of the first analyses suggesting evidence of sexual conflict over reproduction in humans.

Introduction:

Mating strategies and physiology can generate traits that do not provide equal benefit to the two sexes (Pischedda and Chippindale, 2006; Foerster et. al., 2006; Chapman, et. al., 2000; Lande, 1980, 1987). For example, in fruit flies (*Drosophila melanogaster),* sexual selection favors increased wing length in males while natural selection favors reduced wing length (Wilkinson, 1987). A more famous example of this phenomenon is the length of a peacock's tail: sexual selection favors longer tails in males while natural selection favors shorter tails (Smith, 1991). This conflict between selective forces is known as runaway selection (Fisher, 1915). Accordingly, mothers and fathers may differentially affect the fitness (survival and reproductive success) of opposite sex offspring (Pischedda and Chippindale, 2006; Foerster, et al., 2006).

Because the reproductive interests of men and women do not always coincide, sexual conflict is expected in humans too. Such conflicts are played out over evolutionary time and can be resolved either by genomic imprinting (Day and Bonduriansky, 2004), sex limited gene expression (Rice and Chippindale, 2001) or reduced opposite sex heritability

(Bonduriansky and Rowe, 2005); all of which can limit gene expression to the sex which it benefits. Genetic conflict can remain unresolved, however, and may lead to a reduction in the average fitness of each sex (Fedorka and Mousseaum, 2004; Chippindale et al., 2001). There is, for instance, evidence that genes connected with homosexuality in human males may augment female fecundity when inherited by females (Camperio-Ciani, 2004). Discovering SA effects in humans may also encourage researchers to reevaluate theories of sexual selection based on the selection of partners with "good genes" (Kokko, 2001). These mates may, after all, produce opposite sex offspring with lower fitness. It may also affect sex biased investment (see Chapter 4A and 4B) patterns if fitness is not be reliably inherited by opposite sex offspring. This can occur when the given trait(s) is not equally beneficial to both sexes.

Hypotheses concerned with predicting when biasing investment towards one sex would be adaptive (also known as conditional sex allocation) often assume that genetic quality or condition can be *reliably* transmitted from one generation to the next. Different optimal fitness outcomes for males and females can result in genes which have negative fitness consequences when inherited by the opposite sex (Foerster, et al., 2007; Pischedda and Chippindale 2006; Chippindale et al., 2001). It is therefore necessary to explore how sexual conflict might produce a negative covariance in fitness between the sexes.

As previously stated, SA effects have been discovered in several species including fruit flies (*Dosphila melongaster:* Pischeddda and Chippindale, 2006), red deer (*Cervus elaphus*: Foerster, 2006), dungflies (*Sepsis cynipsea:* Blanckenhorn et al., 2002), and water striders (*Aquarius remigis*: Rowe, 1994; Watson et al., 1998). Here, the fitness of

sons and daughters can be negatively affected the opposite sexed parent. In humans, one study used the RS of opposite sex sibling pairs to explore the possibility of SA effects (Stulp et al., 2012) and found that for siblings of low height, sisters achieved higher RS than their brothers. For opposite sex pairs of average height, the brothers achieved higher RS than their sisters. This study suggests that height may be subject to sexual conflict in humans.

One difficult roadblock researcher's encounter when analyzing SA effects in humans stems from the widespread practice of monogamy, as described earlier. It is therefore impossible to untangle the effects of the parental sex on offspring. For this study, I developed a method to circumvent this issue. To isolate the sex specific fitness effects of mothers and fathers on sons and daughters, I used a subset of the population where adult males and females had children with more than one partner. Thus, even within monogamous pairs, each individual is more likely to have a different RS compared to their partner. This method was possible due to the large size of the Íslendingabók database.

Using this subset of couples, I predicted that when fathers have high lifetime RS and mothers have low lifetime RS, sons will have more offspring on average than daughters (see *Methods* for definition of these categories). I made the opposite prediction for the sons and daughters of high RS mothers and low RS fathers. In short, I predicted that the children of high RS mothers and low RS fathers would have more grandchildren through their daughters than through their sons. I also predicted that, in the opposite case, the children of high RS fathers and low RS mothers would have more grandchildren through their sons than through their daughters.

Methods:

The database

Long term and accurate life history data on humans is necessary to successfully answer my questions. In the fall of 2008, for a few weeks, I went to the offices of Decode genetics in Reykjavik, Iceland to work with Íslendingabók - a genealogical database encompassing the reproductive records of over 600,000 individuals and extending back to the founding of Iceland in the 9th century. Each record consists of a subject ID, a mother ID, a father ID, gender, date of birth and date of death. A Perl software program I constructed extrapolates all family relations from these basic data and generates 49 variables for each individual. The relationships between the subjects, family size, offspring sex ratios, birth order, birth intervals, lifespan, fertility and mortality rates are but a few of the analyses that can be run using these extrapolated variables. Due to privacy and proprietary concerns, however(deCode genetics controls access to Íslendingabók), the data may not be accessed off site. Following the trip in 2008, in 2010 I returned to deCode genetics and worked exclsively on Íslendingabók for 10 months.

This database has critical advantages over other genealogies. First, it is a population-based database that contains information for more than half of the one million individuals estimated to have inhabited the island (Gudmundsson et al., 2000). A population-based genealogy is useful for statical analyses as it substantially decreases the possibility of sampling bias. In other words, it increases the possibility that individuals are selected at random. Second, Íslendingabókis extremely accurate and it includes all living Icelanders and the country's inhabitants across centuries. An examination of

mitochondrial DNA show a maternal accuracy rate of 99.3% (Sigurardottir et al., 2000), while the error rate for non-paternity and lab error combined is less than 1.5% (Gudmundsson et al., 2000). This may seem low, but even if non-paternity rates are double or triple this number it improves the accuracy of the analysis. The problems associated with paternity uncertainty, which can present substantial difficulties when analyzing genealogical data, are thus minimized. Third, the Icelandic population is isolated from the rest of Europe with little immigration to or emigration, so multiple generations can be accurately traced back for centuries. Fourth, until the past few decades, Iceland has been a poor, agrarian society with socioeconomic homogeneity (Helgasson et. al., 2008). This is beneficial because extreme differences in resources can complicate interpretations of the data especially when these differences are unknown. Finally, Iceland underwent relatively late industrialization by European standards (Lesthaeghe, 1995), and was still a predoiminantly agricultural society until early in the 20th century. Therefore, Iceland did not undergo the demographic transition, which was marked by declining mortalityand fertility, until the late 19th century (Helgason et. al., 2005; Kaa, 2002). This is advantageous because potential problems with interpreting trends where 'voluntary' limitations may have been placed on fertility can be diminished while still preserving most of the data. We can also avoid having to understand the reasons for the demographic transition and can use data where fertility rates seem to have been maximized.

The first national census in Iceland, and likely the first ever in the world, was in 1703 (Karlsson, 2000). It is therefore typical to regard the genealogical data from 1650 and onward as the most accurate and to treat the time before with greater skepticism (Gunnar

Gundarson: *pers. comm*.). For this dissertation, all of the analyses and reported results use data after 1650 and prior to 1950 to ensure greater reliability. It should be noted there is no information on the socioeconomic status of the individuals in Islendingabok, so this variable was never considered to measure "condition". Future studies would benefit greatly by including this variable.

Creating the dataset

Although my analyses are not guided by the assumption that any of the traits under consideration are maladaptive (for a review of some of the methodological approaches of evolutionary psychologists and behavioral ecologists see Chapter 1; Irons, 1998; Smith et al., 2001; and Symons, 1987), I do acknowledge the changing socio-cultural circumstances of the Icelandic population over time. All analyses of reproductive success were tracked over time (typically by century) and changing patterns that are sensitive to varying environmental conditions, such as the demographic transition or industrialization, are standardized by decade.

Although cultural factors may play a role in the interpretation or in the analyses of these data, such variables are not the primary concern of this project. It is also possible that PI decisions may be influenced by shifting cultural practices over time, but these choices are not expected to be maladaptive or to systematically bias any findings. Furthermore, conscious family planning decisions (for example, the 'want' for children) are not immune to genetic influences or behavioral predispositions which override these 'deliberate' choices. For this reason, I am unconcerned with attempting to discern or speculate upon the reasons that couples may 'choose' to have more or less offspring and

will concentrate solely on reproductive outcomes (which is all that matters with regard to evolution).

As previously mentioned, for the purposes of this study, I sorted the dataset to only include parents who had children with multiple partners. The cutoffs for high or low LRS were somewhat arbitrary but served two purposes. First, the parents' LRS had to be 'sufficiently' different from each other, while maintaining a reasonable, although severely depleted, sample size. Second, couples were chosen if they were at least one standard deviation (SD) below or above the mean reproductive success of the average Icelander. In other words, also included in the study were individuals with the following family composition: one SD above the mean included males with 7 or more children (with one partner) and one SD below the mean included females with 2 or fewer children (also only with one partner). In addition, I included the children of mothers who had 5 or more children (one standard deviation above the mean) and whose father's had 2 or fewer children (one standard deviation below the mean).

These groups formed post hoc 'test crosses' and I was able to run an evolutionary experiment in reverse. The major constraint was sample size. I was ultimately left with the offspring of 1,289 pairs of high RS mothers paired with low RS fathers and 1026 offspring of low RS mothers paired with high RS fathers. There were two control groups: one was children of crosses between high RS mothers and high RS fathers and the other was children of crosses between low RS mothers and low RS fathers. I predicted high RS fathers crossed with low RS mothers would have more offspring through their sons, while high RS mothers crossed with low RS fathers would have more offspring through their daughters (figure 3.1).

Results:

The ex-post facto design described above (see methods) can be problematic. Determining causation, for instance, can be difficult because individuals have not been randomly assigned to treatment and control groups: assignments to groups were based on the possession of a particular trait. There are, however, no obvious reasons to expect biases in the data between males and females beyond the predicted SA effects.

In post-hoc test crosses (previously described in detail, see methods), the sons of mothers with low RS (low fit mothers) and fathers with high RS (high fit fathers) have higher RS than daughters ($p=0.103$). The average number of grandchidren produced through the daughters of these parents was 4.94 and the average number of grandchildren produced through their the sons was 5.19. In contrast, and as predicted, the daughters of low RS fathers and high RS mothers outperformed their sons ($p=0.107$). The daughters of these parents produced an average of 4.94 grandchildren while the sons produced an average of 4.7 grandchildren.

Discussion:

This study revealed evidence suggestive of sexual conflict in humans. When fathers with high lifetime reproductive success (LRS) mate with mothers with low LRS, more grandchildren are produced through their sons than through their daughters. Similarly, more grandchildren are produced through the daughters of high LRS mothers paired with low LRS fathers (see figure 3.1). Neither of these results reach significance but both are close when using one tailed tests ($p=.103$ and $p=.107$ respectively). As discussed, this study was conducted on a monogamous population, limiting the sample size to a subgroup of individuals who were involved with more than one partner over time.

Sexual conflict is generated when the genetic interests of male and females cannot be obtained simultaneously (Chapman et al., 2003; Parker, 1979;Trivers, 1972). Trivers (1972) argued that sexual conflict plays an essential role in the evolution of parental care when both males and females invest in offspring. On the behavioral level, each sex prefers to exert their own energy on future reproduction and mating, while desiring their mate to invest more heavily in shared offspring. In other words, each parent prefers the other to do more work in caring for offspring. The topic of sexual conflict has generated intense interest recently and the number of publications investigating it in the last decade now exceed those concerned with conventional sexual selection (Pizarri and Snook, 2003).

SA effects have now been discovered in several species. In an experimental population of fruit flies, the fitness of sons was negatively affected by mothers while the fitness of daughters was negatively affected by fathers. Testcrosses between high RS males and high RS females produce offspring with lower RS than do crosses between low RS males and low RS females (Rice, 1992; Pischedda and Chippindale, 2006). In a wild population of red deer, males with high lifetime RS produce daughters with relatively low lifetime RS (Foerster et al., 2007). Interestingly, no significant effects were found in any of the other predicted directions: high fit mothers had no detectable effect on the lifetime RS of sons, low fit fathers did not affect the RS of daughters, nor did high fit mothers significantly affect the RS of sons.

To my knowledge, this is the first study to date which suggests evidence of sexual conflict over reproduction in humans. Future studies can contribute to this area of inquiry through two lines of questioning. First, because this study was conducted on a

monogamous population (limiting the sample size), future studies should examine polygynous populations. There are ample data on Mormon populations who practiced polygyny in the 19th century and therefore facilitate the study of differential RS among males and females. Second, the genetic factors driving sexual conflict are not well understood. A better understanding of gene expression in males and females as well as inheritance patterns will allow researchers to identify the evolutionary consequences of these traits.

Chapter 4A: Reproductive Success is Differentially Inherited by Sex: Data Support a Reproductive Benefit to Biasing the Sex Ratio in the Manner Predicted by the Trivers-Willard Hypothesis

Abstract

The hypothesis that biasing both the sex ratio and subsequent investment towards males when mothers are in good condition and towards females when mothers are in poor condition is adaptive is known as the Trivers-Willard Hypothesis (TWH) (Trivers and Willard, 1973). There are two major issues I explore with respect to this hypothesis. First, do some human populations either adjust sex ratios or bias investment in favor of sons when either or both parents are in good condition and in favor of daughters when either or both are both in poor condition? Second, is there a reproductive benefit (measured in grandchildren) to biasing the sex ratio that depends on parental condition? In other words, do parents in good condition have more grandchildren through their daughters than through their sons? The first question asks whether or not individuals do in fact bias the sex ratio in ways consistent with the TWH while the second asks if they should. Would doing so provide a reproductive benefit? The first question has been tested thousands of times (see Google scholar, 2013; see West, 2010 for major review) and the second less frequently. In this study I used family size as a proxy for parental condition (the greater the parents reproductive success (RS), the better their condition) and found that there would be a benefit to having more sons when parental RS was high and a benefit to producing daughters when parental RS was low (see Figure 4A.1 and Figure 4A.2 and Tables 4A.1, 4A.2 and 4A.3). Sons from large family's produce more

children than daughters from large families while only daughters produce more children than only sons (see figure 4A.1 and 4A.2). The second question addressed in this study was whether big families either bias investment towards, or produce, more sons? I did not have the data to assess pre or post-natal investment but I did have secondary sex ratio (at birth) data. In the population from ancestory.com that I analyzed here, there was no relationship between parental reproduction and the sex of offspring. So there was no evidence that sex ratios were adaptively biased, although doing so would have provided a large benefit. In short, research here suggests that high RS parents should produce more sons and fewer daughters, but they don't. A major shortcoming of this study is a lack of data on the socioeconomic status of the individuals which has been suggested by Trivers and Willard to be a good measure of maternal condition in humans. Finally, of all the traits to which I had access, the only one which predicted sex of offspring was the age of the father (see table 4A.4). Older fathers produced significantly more daughters. If the age of the father is a negative predictor of parental condition (older fathers are in worse condition or have less resources remaining for parental investment) then there is also evidence for the TWH.

Introduction:

Fisher (1930) was the first to show that at a population level, if males and females cost the same, both sexes should be produced and invested in equally. Trivers and Willard (1973, "TW" hereafter) suggested that under certain conditions, however, deviations from an equal sex ratio should be favored in local breeding populations. It is important to note that these local deviations would be overridden in the overall population, though.

Three conditions are explicitly stated by TW: 1) the condition of young will be correlated with the mother's condition during period of parental investment (PI), 2) the condition of offspring at the end of the PI period will be correlated with their condition in adulthood, and 3) the sex with higher variance in RS will gain a greater reproductive advantage by being in better condition. Over two thousand papers have found support for this hypothesis (Google scholar, 2013). One of the earliest confirmations came from a study by Clutton-Brock of red deer (Clutton-Brock, et. al., 1984). And today, the TWH has become one of the most cited in history (Jones, et. al., 2009; see Cameron, 2004 for major review; Kanazawa, 2005, 2006, 2007; Grant, 2007; see Sheldon and West, 2004 for major review; West, 2010).

Underlying the logic of the TW hypothesis is that there are notable sex differences in the variance of reproductive success (RS) (Trivers, 1972). This variance may be so influential that offspring sex ratios are determined by the environmental conditions in which parents find themselves. Mothers in good condition may be preferentially selected to produce sons while those in poor condition, daughters. Mothers may also be able to preferentially produce sons or daughters in response to the quality of their mate. For instance, Long and Pischedda (2005) have shown that female fruit flies mated to younger males were more likely to produce sons, while those mated to older males were more likely to produce daughters. Importantly, the effect was only seen in the first 24 hours of mating and the offspring sex ratios were indistinguishable between females mated to old and young males after this period. So the timing of fertilization may also be important and therefore a number of variables must be investigated to analyze the TW hypothesis.

In another more recent study, Cameron (2004) conducted a meta-analysis across mammals to test for the TWH. When using measures of maternal condition near and around the time of conception, she found support for the theory 74% of the time. It is an interesting result, not just because it shows strong, unambiguous support, but also because it suggests a logical and highly plausible mechanism. It makes sense to measure condition just prior to major investments which will tend to diminish maternal condition, such as gestation, giving birth and raising offspring. This is especially true if mothers or parents more generally, are investing as predicted by TW (e.g. more investment in high quality males). Evolution is unlikely to have rebuilt a species specific mechanism for humans. So additional strengths of Cameron's work are not only that it is generalizable to all mammals, but also that it both uses a large sample and provides a logical, *a priori* evidence of a mechanism.

Still researchers should remain careful when using proxies to assess fitness. Some life history traits that are commonly used to indicate fitness may even be inversely related. For example some long-lived individuals may have lower reproductive success. One study found that post-menopausal women die earlier if they have had more offspring, and that the age at first childbirth was lower for women with shorter lifespans (Westendorp and Kirkwood, 1998). When genetic or developmental constraints prevent a trait from being maximized in both males and females at the same time, females should evolve preferences for traits that provide low fitness benefits to males but high fitness benefits to females (Trivers, 1985). Over evolutionary time these preferences should select for traits that lower average male fitness and raise average female fitness (Seger and Trivers, 1986).

These issues aside, the best and most obvious fitness proxy is lifetime reproductive success, or even better, number of grand-offspring produced as this includes the quality of the offspring produced (West, 2010). Conducting life history experiments with humans can be difficult because we are such a long-lived species (Olshansky, 1990; Guarente and Kenyon, 2000; Lindström, 1999; Strassman and Gillespie, 2002). Because generation time in humans is so long, researchers often seek variables that might serve as a proxy for lifetime reproductive success (LRS) (McGraw and Casswell, 1996).

For this study, I use LRS to evaluate the potential reproductive benefits of biasing the sex ratio. Before we attempt to determine if individuals do, in fact, bias either production or investment towards one sex or the other, it is necessary to determine whether or not this would be useful in the first place. In other words would favoring one sex over the other provide a reproductive or adaptive benefit? The present study was designed to answer the following question: does the reproductive success of sons and daughters differentially depend on the reproductive success of their parents? More specifically, do individuals with high LRS produce sons with high LRS and daughters with low LRS and do individuals with low LRS produce daughters with high LRS and sons with low LRS?

Methods:

In a pilot study, I analyzed a (genealogy) gedcom file with 3,000 entries from the family of another graduate student, Rolando DeAguiar. The effects were interesting but the sample size was too low, so I requested another gedcom file from the Church of Latter day Saints, family history library in Morristown, New Jersey. The only criterion was that the file should be as large and as well researched as they could find. I received a

file from them, with approximately 10,000 names, based on the descendants of William Brewster. Brewster was a well-researched individual who traveled to America on the Mayflower. Early arrivals to America are of great interest to many professional and amateur genealogists so these ancestries are considered to be more accurate and well researched than others and are constantly subject to update and verification. It should be noted, however, that the worst of these gedcom files achieved an accuracy rate as low as 58% as confirmed by DNA testing (Hahn, R. A., Truman, B. I., & Barker, N. D., 1996). I hope these files are more accurate than those, but I can't be certain. At the very least, however, these files are unlikely to be biased in any way that might perjure these results.

Next I looked for the largest genealogy file I could find at ancestry.com. To duplicate, go to awt.ancestry.com and type in Eeastus Sheffield. The file has 213,523 entries. This is the largest single gedcom file I could find and is the file I used in this study. Analyzing a single gedcom file has the advantage that it is impossible to repeat any individual because all individuals in a file have separate identification numbers. For ease of analysis, I converted the Gedcom file to excel using a program called Gedcom to excel converter, which was created by Rolando DeAguiar. It can be found at http://eden.rutgers.edu/~deaguiar/GedCom/.

The original Gedcom file contained a total of 212,480 individuals. Of these, 145,796 had both parents listed and all of these individuals were used in the first sample (see figure 4A.1). The rest were excluded because it was impossible to determine the parents RS, when the identity of either one of the parent's was unknown. On the other hand, if both parents were known then it was likely that the family size of the second generation was relatively accurate. The reproductive success of all of these individuals is

not certain, however, and when they were not listed as being a parent, it was not clear whether they did not have any children, or if this was just not known. The second group was likely the most accurate. In this sample, all individuals whose parents were either unknown or were not listed as parents themselves were excluded. This second sample included 56,043 individuals (see figure 4A.2).

I also examined Center for Disease Control data for all births in the United States in 2008, and analyzed the relationship between the sex of the children, the marital status of the mother, the age of both mother and father and a number of health characteristics of the mother such as smoking or history of heart disease.

Results:

Lifetime reproductive success is transmitted differently to sons and daughters. In both samples, only daughters have more offspring than only sons (Figures 4A.1 and 4A.2). In the first sample (figure 4A.1) only daughters have a mean LRS of 1.89 and only sons have a mean LRS of 1.51. In the second sample of 56,043 individuals (figure 4A.2) only daughters had an average LRS of 1.73 and only sons and average LRS of 1.58. In both samples, the mean LRS of sons was higher than daughters from parents who had a mean of two children or more. As parental RS rises above two, sons tend to increasingly outperform daughters in both samples. The regression for both samples reflects this trend. In the first sample (figure 4A.1) (only individuals without both parents listed excluded) the regression of daughters LRS on parents LRS is significant and negative ($r = -0.035$, $p<0.001$), while the regression of sons LRS on parents LRS is significant and positive ($r=.05$, $p<0.001$) (see figure 4A.2).

Importantly, it is not the magnitude of the correlation coefficient that is striking, but rather the difference in the correlation coefficient between sons and daughters. In the second sample (individuals without parents or offspring listed excluded) (table 4A.1) the regression of daughters LRS on parental LRS is positive and significant ($r=0.166$, $p<0.0013$) and the regression of sons LRS on parents LRS is also both positive and significant ($r=.294$, $p<.0014$) (see table 4A.2). The reasons for the slope differences between the samples are primarily the result of an overall increase in LRS for offspring of both sexes. This is due to the exclusion of all individuals not listed as parents in sample two. The pattern in the data is not reflected well by a regression, however, and as the data for parental LRS are categorical, the bar graphs **(**figures 4A.1 and 4A.2) display the pattern better than a regression line. For sample sizes and total sex ratios see table 4A.2. For unknown reasons mean male LRS (lifetime reproductive success=1.25) was higher than mean females LRS (lifetime reproductive success=1.05) in the first sample and was slightly higher in the second sample (males mean LRS=3.61 and female mean LRS=3.57) (see table 4A.3). In addition the mean reproductive success of the parents differed by sex and by sample. In the first sample (only individuals without parents excluded) the mean LRS of the parents of sons (5.47) was lower than that of females (5.60). In the second sample (all individuals without parents or offspring excluded) the LRS of the parents of sons (4.9) was slightly higher than the parents of daughters (4.8).

Female LRS is shown by red bars and male RS is shown by green bars in both samples. Mean number of offspring is on the Y axis and mean reproductive success of parents is on the X axis (figures 4A.1 and 4A.2). Sample sizes for *parents* (not offspring) mean LRS are as follows: males and females with 1 child respectively: 10918 and 8819;

with 2 children: 9563, 8114; with 3 children: 9131, 7897; with 4 children: 7339, 6550; with 5 children: 6606, 5709; with 6 children: 5971, 5063; with 7 children: 5669, 5208; with 8 children: 5266, 4848; with 9 children: 4791, 4414; with 10 children: 4154, 3558; with 11 children: 3165, 2989; with 12 children: 2337, 2165; with 13 children, 1271, 1126; with more than 13 children: 1617, 1519.

ADDITIONAL ANALYSES

Statistics from CDC National Vital Statistics System

Based on CDC statistics from 2008, unmarried mothers lost more male fetuses than married mothers. There was a 0.541816 percent male fetal death rate for all unborn children of unmarried mothers vs. a death rate of 0.520670 percent male fetal death rate for unborn children of married mothers. There are a number of explanations for these statistics but they are also consistent with a Trivers Willard effect. On average, a single parent should not be able to invest as much in children as two parents. The risk of females dying in utero (0.854) is lower for unmarried mothers than the risk of males dying in utero is for married mothers (0.930). There are no p-values here because these are not statistics; they are derived from the entire population (e.g. all births in the United States in 2008). The risk to daughters is greater when males are not present. Does this mean that mothers are cutting their losses because males are more expensive to birth and require more investment (see: CDC National Vital Statistics System marriages, divorces and mortality)? If so, Fisher's theorem explains why.

Does Trivers-Willard explain this? When the mother is married (good condition), males have a slightly higher risk of dying in utero. But when the mother is unmarried

(poor condition) male babies have an even higher risk than females of dying. In other words, daughters survive the transition from married (good condition) to unmarried (poor condition) better than male babies. This is consistent with the TWH. The effect sizes are small, however. For instance the switch from the unmarried to the married category results in a .00127% decrease in males born.

Abortions slightly increase the sex ratio at birth

The total abortions reported in the United States for the year 2000 was 857,749 (CDC vital statistics, Reproductive health, data and statistics, 2004). The abortion rate in 2000 was 245 per 1,000 live births and 87% percent are within the first 13 weeks of gestation. Importantly, abortion rates differ between married and unmarried women; 78% of abortions are to unmarried women. This is 8.8 times the rate for married women. The rate of abortions for unmarried women is 570 per 1,000 live births and for married women it is 65 abortions per 1,000 live births (CDC vital statistics, Reproductive health, data and statistics, 2004). Unmarried women are somewhat more likely than married women to give birth to females and unmarried women have the most abortions. So the sex ratio at birth would be lower had these births come to term. More women would be born if abortions were illegal.

Older fathers produce female biased sex ratios

The strongest and most significant effect I found in support of TWH was the effect of father's age on the sex of the child. Older fathers are more likely to produce girls and this is a nearly linear relationship (see table 4A.4); ($r=-.95125$, $p< 10^6$).

Discussion:

This is to my knowledge one of very few studies, in humans, to show that the reproductive success of sons and daughters differentially depends on the reproductive success of their parents. This study also confirms the fundamental logic behind the TWH. The heritability of LRS in this study follows the pattern predicted by Trivers and Willard. The sons of parents with low LRS have lower LRS than the daughters of these parents. At the same time, the sons of high LRS parents have higher LRS than the daughters of these parents. This indicates that when there is differential LRS, there would be a benefit to biasing the sex ratio as a function of the lifetime reproductive success of the parents.

Although we see no evidence for parental manipulation of the sex ratio, there seems to be a large potential benefit in doing so. The effect is significant. From these data, it is clear that the individual's in this sample would benefit by altering the sex ratio towards males when they have large families and towards females when they have small families. The mechanisms behind this phenomenon are not yet clear but many researchers have and are currently addressing this question (Chacon-Puignau and Jaffe, 1996; Cameron, 2004; Cameron and Dalerum; 2009). Future work should continue to replicate this hypothesis to further understand the specific requirements under which we should expect TWH to not only function but to also provide a reproductive benefit. One important discovery from CDC data is that spontaneous fetal abortion rates are higher for unmarried mothers carrying males than for those carrying females. Other CDC data also clearly and unambiguously demonstrate that the age of the father is important. One major drawback of this study is that I was not able to determine from the CDC data whether older fathers produced sons with higher LRS or daughters with higher LRS. This is a critical oversight because I am unable to determine if the female biased sex ratio produced by older fathers

is adaptive. Nevertheless, both marital status and age of the father should be included in future analyses as an a priori prediction of parental condition. They both seem to affect the sex ratio.

Chapter 4B: No Advantage Found for Biasing the Sex Ratio as a Function of Family Size Found in Iceland

Abstract

Using an Icelandic genealogy[i] (Íslendingabók), I searched for evidence that sons from large families are more fertile than daughters from large families. The Trivers Willard hypothesis (henceforth TWH) purports that the sex ratio of offspring and subsequent sex-biased investment can be affected by the "condition" of parents. The hypothesis relies on the fact that males have higher variance in lifetime reproductive success (LRS) than females, and that sons should benefit more from parents (in the original formulation of the hypothesis, maternal rather than parental condition was emphasized) that are in good condition than daughters . Although my analysis was not focused on testing the TWH, but rather on whether or not there would be a reproductive advantage for large families to produce more sons and for smaller families to produce more daughters, I did analyze sex ratios as a function of family size. My only assumption was that parental reproduction (family size) was a good proxy for parental condition. I did not discover any reproductive benefit (measured in grandchildren) for large families to produce more sons or for small families to produce more daughters in Iceland. If family size is a reasonable proxy for fitness (e.g., parents that produce large families are more fit than parents that produce small families) then there was no detectable reproductive benefit for large families to produce more sons, as they did not bear any more children than their daughters (see figure 4B.6). Also, daughters from small families did not out-reproduce sons from small families or daughters from large families. Neither

of these effects is consistent with a reproductive benefit to biasing the sex ratio. In addition, neither lifespan of parents (see figure 4B.4) (another proxy for condition), nor birth order (see table 4B.1) predicted the sex of children. The only factor to which I had access that influenced sex ratios was the age of the father. Older fathers gave birth to more daughters (see figure 4B.5). The age of the mother at birth did not have any detectable effect (see figure 4B.3). Importantly, some information both affecting maternal condition and predicted by Trivers and Willard themselves to have an impact is missing from the Icelandic dataset. There was no data, for example, on the wealth, occupation or status of any of the individuals in the genealogy. Therefore, any effect of socioeconomic status could be concealing a TW effect.

Introduction:

The Íslendingabók database is the most extensive and accurate genealogy in the world, recording life history data since the settlement of the island in 871 A.D. The TWH predicts that parents in "good condition" (e.g., fertile, long-lived, high socioeconomic status –SES -, intelligent) are more likely to produce sons, while those in poor condition (short lifespan, poor, low SES, low reproductive success) will be more likely to produce daughters (Trivers and Willard, 1973). Over two thousand studies have sought to find evidence for or against this hypothesis (Google scholar, 2013) and this 1973 paper has been one of the most cited in history (Jones, et. al., 2009; see Cameron, 2004 for major review; Kanazawa, 2005, 2006, 2007; Grant, 2007; see Sheldon and West, 2004 for major review; West, 2010).

Íslendingabók is a highly accurate genealogy of the Icelandic people that stretches back over 1,000 years and includes over 600,000 individuals. This database records all life history traits since first settlement of the island (Karlsson, 2002). As mentioned in an earlier chapter, the settlement date for Iceland is generally agreed upon by most scholars and the earliest histories and the best archaeological evidence put this date at 871 A.D. (Vésteinsson and McGovern, 2012). Also previously mentioned is that the genealogical record (Íslendingabók) in Iceland goes back to the 9th century, the first centuries were not recorded in a systematic and rigorous manner. It is therefore typical to regard the genealogical data from 1650 and onward as the most accurate.

Indeed, this extensive database allows for rigorous testing of the TWH across centuries. Here, I seek to demonstrate two things: 1) to determine whether parents adjust the sex ratio of their children dependent on their own condition, and 2) to determine whether or not there is a reproductive advantage for parents to adjust the sex ratio of their offspring dependent on their own conditions. Specifically, I ask the following questions. Do the sons of parents in good condition produce more children than do the daughters of parents in poor condition? Likewise, do the daughters of parents in poor condition produce more children than do the sons of parents in poor condition? Evolutionary success should be measured in grand-offspring and, because of its depth; Íslendingabók is a good opportunity to test this theory.

***Methods*:**

The database

Long term and accurate life history data on humans is necessary to successfully answer my research questions. In the fall of 2008, I went to the offices of Decode

genetics in Reykjavik, Iceland to work with Íslendingabók - a genealogical database encompassing the reproductive records of over 600,000 individuals and extending back to the founding of Iceland in the 9th century. Each record consists of a subject ID, a mother ID, a father ID, gender, date of birth and date of death. A Perl software program I constructed extrapolates all family relations from these basic data and generates 49 variables for each individual. The relationships between the subjects, family size, offspring sex ratios, birth order, birth intervals, lifespan, fertility and mortality rates are but a few of the analyses that can be run using these extrapolated variables. Due to privacy and proprietary concerns, however, Decode genetics controls access to Íslendingabók, the data may not be accessed off site. In 2010, I returned to deCode genetics and worked exclsively on Íslendingabók for a 10 month period.

The database has several critical advantages over other genealogies. First, it is a population-based database that contains information about more than half of the one million individuals estimated to have ever inhabited the island (Gudmundsson et. al. 2000). A population-based genealogy is a tremendous advantage as it substantially decreases the possibility of sampling bias. Second, the database is extremely accurate. It includes all living Icelanders and most of their ancestors. An examination of mitochondrial DNA shows a maternal accuracy rate of 99.3% (Sigurardottir et al. 2000) while the error rate for non-paternity and lab error combined is less than 1.5% (Gudmundsson et al. 2000). So even the problems of paternity uncertainty which can be major problmes in analyzing genealogical data are minimal. Third, the population has been isolated from the rest of Europe with little immigration to or emigration from the island so multiple generations can be accurately traced back for centuries. Fourth, until

the past few decades, Iceland has been a poor, agrarian society with relative socioeconomic homogeneity (Helgasson et al., 2008). Finally, Iceland underwent relatively late industrialization by European standards (Lesthaeghe, 1995), and was still a predoiminantly agricultural society until early in the 20th century. Therefore, Iceland did not undergo the demographic transition, which was marked by declining mortalityand fertility, until the late 19th century (Helgason et al., 2005; Kaa, 2002) so potential problems with interpreting trends where 'voluntary' limitations may have been placed on fertility can be diminished while still preserving much of the data and tracking trends in fecundity over time.

Under what conditions do parents produce more grandchildren through their sons than through their daughters? The answer to this question will provide insight into any potential mechanisms that natural selection may have devised to manipulate the primary sex ratio or subsequent sex-biased PI. Specifically, I predicted 1) that parents in poor condition would have more grandchildren through their daughters than through their sons, and that parents in good condition would have more grandchildren through their sons than through their daughters. If not, there is no reason to believe that a mechanism to bias relative investment in sons vs. daughters that depends on parental condition should have evolved.

Results:

There was no detectable association between family size and sex ratio ($r = .000$, $p = .772$) (see table 4B.1). In figure 4B.1 the numbers of siblings are represented on the X-axis and parental reproduction or family size on the Y-axis (less one for the individual proband). It was not necessary to precisely define large or small family to perform an

ANOVA because there was no relationship between family size and sex ratio, and creating arbitrary cutoff points would have unnecessarily created artificial categories. Neither was there any detectable relationship between lifespan of either parents and the sex ratio of their offspring r=.-04, p=0.210 (see figure 4B.4). Again the regression is the key statistic and creating arbitrary cutoffs between age groups would only have served to dilute the value of the data.

Maternal age at birth did not impact sex ratios (r=.04, p=.210). Neither did reproductive duration (the time between the first and last births in an individual's entire life was used as a proxy for reproductive effort; see figure 4B.2) see to affect sex ratios. Lifespan was also used (see figure 4B.4) as a proxy for an individual's condition, and it had no effect on the sex of children either.

The only trait that had any detectable influence on the sex of the child was the same one that I found in an on-line publicly available genealogy from ancestry.com - the father's age at the child's birth (see figure 4B.5). Older fathers are more likely to produce daughters(r= -.004, p=.029).

Discussion:

Measuring condition by a number of variables including lifespan, reproductive duration and reproductive rate (see table 4B.1), I found no relationship between parental condition and offspring sex ratio. The TWH relies on the critical assumption that sons of good-condition parents have a reproductive advantage over daughters and that daughters of poor condition parents have a reproductive advantage over sons. The generational depth of Íslendingabók allows us to determine if this population meets this assumption, which has never been tested on an Icelandic population before. I demonstrate that sons

from large families do not out-reproduce daughters from large families. If lifetime reproductive success is a good proxy for parental condition then there is no reproductive advantage to be gained by varying the sex ratio in the way predicted by TWH. Of course, this is not what T and W suggested. They suggested that we use SES as a proxy for condition. I did not have access to this data in Iceland. So one important drawback to studying the TWH in Iceland is the lack of data on either wealth or socioeconomic status for any of the individuals in the database. Such data was available in rudimentary form, in parish records, but these records had not been organized electronically into Íslendingabók and needed to be entered. The unorganized and un-entered (into records in Íslendingabók) include basic occupation such as priest, farmer, fisherman, landowner etc. but do not provide the detail one would want to really analyze the wealth or resources of the inhabitants of Iceland.

I used data I had available and, as long as family size (see figure 4B.1) can be used as a reasonable proxy for parental condition prior to the demographic transition, there was no evidence for a reproductive benefit for biasing the sex ratio (West, 2010). Although, lifetime reproductive success is often synonymous with an individual's fitness, I also used longevity and parental age (see figures 4B.2 and 4B.3) as proxies for 'condition'.

Future studies of the TWH in humans should address this critical assumption: Is there a reproductive advantage (measured in grandchildren) to be gained by good condition parents biasing investment towards sons and poor condition parents biasing investment towards daughters? Perhaps researchers with access to SES data should ask whether high SES parents produce sons with higher LRS than their daughters. Likewise do low SES parents produce daughters with higher LRS than sons? This is one way to determine if

there is an adaptive benefit to biasing the sex ratio or investment as suggested by Trivers and Willard.

Chapter 5:

Conclusion and future directions

In the past 40,000 years positive selection has been occurring at an accelerated rate. In comparison to the average rate of change since *Homo sapiens* diverged from our most recent common ancestors with other hominid species, the average rate of adaptive change has been at least 10 fold higher than the background rate (Hawks, 2007; Cochran and Harpending, 2010). Much of this genetic change has occurred in concert with major cultural and ecological changes, including the domestication of plants and animals (Laland et al., 2010). Not only is evolution by natural selection still occurring, it is happening at a pace far greater than is generally recognized. Analyzing a modern population, that is undergoing strong selection pressures across centuries, can add important insights into our understanding of general evolutionary principles in addition to deepening our knowledge of evolutionary theory.

Iceland was settled in 871 A.D. +/- 2 years (Vésteinsson, 1998) and has kept exceptionally accurate genealogical records from the middle of the 17th century to the present. These records are now stored in a database called Íslendingabók (the book of Icelanders). I used this database, in addition to some genetic data sequenced by deCode genetics (see Chapter 2), to analyze the evolution of some basic life history traits in Iceland over the past 3 centuries.

Genealogies are messy and complicated datasets. Most variables within them are interrelated. For example, the number of siblings an individual has is the same as the LRS of his or her parents. There are unavoidable inaccuracies and effects can change dramatically across decades (e.g. demographic transition). Important information can be

ascertained from good genealogies but care must be taken to standardize key variables such as longevity and reproduction over time (see figure 2.1).

The Trivers-Willard (1973) hypothesis has had varying degrees of confirmation and falsification over the years (see Freese, J., & Powell, B., 1999; Hewison, A. J., & Gaillard, J. M., 1999; Gaulin, S. J., & Robbins, C. J., 1991; Hrdy, S. B. 1987; Brown, G. R., 2001; Keller, M. C., Nesse, R. M., & Hofferth, S. ,2001;. Almond, D., & Edlund, L. 2007). Some have even suggested that it may not be testable (Brown, 2001). What do we mean by parental investment (PI) and maternal condition? How can we measure them? One reason that the theory is attractive to researchers is because it seems so testable and even null results are publishable.

Although, the TWH is an elegant and simple theory, testing it is not. In Chapter four, I found some evidence for TWH in a publicly available data set but less evidence for it in Iceland. Of course, in neither case did I have all the variables I would have liked (e.g. socio-economic status, birth weight, familial wealth and property). I don't know how much I have added to the debate over the TWH. In this dissertation, however, results are mixed and unclear (see Chapters 4A and 4B). Perhaps the most important addition I contribute to this ongoing debate is in recognizing the importance of determining if sex biased investment in offspring would provide a benefit to parents who did favor one sex or the other. In the publicly available genealogy there was a clear benefit for high RS parents to favor sons and for low RS parents to favor daughters. In Iceland this was not the case. The trait that predicted secondary sex ratios in all databases analyzed was the age of fathers. It predicted the sex of offspring in both genealogies and in data from the Center for Disease Control (CDC) for all births in the United States in 2008.

Another potential issue with detecting a TW effect is that the effects are expected to be subtle because Fisher's law of equal investment in the sexes is constantly at work driving sexes that are of equal cost to 50:50 ratios. So we often need huge sample sizes to determine if there is an effect. In additional, the possibility of sexual conflict violates one of the assumptions of the TW hypotheses – offspring condition at the end of PI will be correlated with mother's condition at conception. Sexual conflict (see chapter 3) puts this assumption into question for opposite sex offspring (sons). If good condition females are birthing poor condition sons due to SA effects, one of the fundamental premises of TW is violated. Parental ability to predict the future fitness of their offspring is also critical and is not discussed by Fisher. In addition, the more a mother is able to predict her child's future reproductive success, the more female biased the sex ratio will become. This logically follows from the fundamental principle that males are redundant. The only reasons Fishers equal investment in the sexes functions is because parents must not be able to predict with any degree of accuracy the future fitness of offspring. Life is too random and luck probably plays a much larger role than typically expected.

Measuring reproduction in contemporary environments is difficult. Demographers are not even in full agreement on the causes of the demographic transition, let alone population changes across centuries (see figures 1.1 for reproduction over Icelandic history). Many of these issues can be resolved by log transforming and standardizing the data but there may still be problems of which we are or may be unaware. As former secretary of defense Donald Rumsfeld has famously said, "There are known unknowns; that is to say, there are things that we now know we don't know. But there are also

unknown unknowns – there are things we do not know we don't know." (Rumsfeld, 2002).

Finally, I demonstrated convincing evidence (see chapter 2) that parental investment has played an important role in reproduction of both the lifespan and Icelanders over 3 centuries. I showed evidence from the genealogy, computer simulations and genetic data that the heritability of reproduction is undetectable but that parents likely have had a large impact on their children through a shared environment, common household, the transfer of resources and parental influence.

If I do use genealogical data again, I will only use data to which I have full access. Íslendingabók has many advantages including depth and accuracy, but the major problem is that I had to leave the data in Iceland. In any future investigations I will need to control the data myself. I have considered other ways to measure parental investment too. I have accessed the record of New Brunswick family court and have begun to analyze the age of divorce, whether or not custody was contested and the sex of the children. So far the results look interesting (see Figure 5.1) – boys increase risk of divorce early, girls late - but it is far too early to run meaningful statistics on this dataset. But these early results are consistent with a TW effect. The logic of the TWH would predict that parent's should divorce when their sons are either young (0-5) or old (14-18) but when their daughters are of intermediate age (6-12). The CDC also has excellent data and surveys on the health and demographics of the US population and I have begun to explore it as well.

Íslendingabók is a national treasure and a tremendous resource and working with it day after day for almost a year taught me many valuable lessons. In a database as large

as Íslendingabók, it is exciting to discover an original and novel result that also withstands multiple tests. I tested many hypotheses and was continuously disappointed by failures or tentative results. I spent the majority of my time in Iceland attempting to falsify a single result – parental investment has had a large and important impact on some key life history traits over three hundred years (see Chapter 2). The fact that this result is repeated decade after decade, and is sustained over centuries is convincing evidence that I have found something that is both important and interesting about what affects longevity and reproduction.

The best predictor of how many children an individual will have is how many children his or her full sibling has. The negative relationship between the fertility of parents and offspring is just one aspect of this main effect; siblings' reproductions are significantly and positively associated. Whether I can call this effect parental investment remains a question. Nevertheless PI has undoubtedly had a strong influence on humans in our recent history. The computer simulations, the Generalized Linear Mixed Model (GLMM) using the genetic data, and the tradeoff between the quantity and quality of offspring are all evidence supporting the hypothesis that PI has had a strong influence on reproduction (fitness) (see Chapter 2). This is the most important and original result of my work in Iceland. The results in chapter 3 which suggest sexual conflict are both original and intriguing. Unfortunately they do not reach statistical significance and require further research. Finally, the data and results concerning the TWH (see Chapters 4A and 4B) are interesting, but neither Íslendingabók nor publicly available genealogies contain information on either the occupation or SES of the individuals in the database. Both of these would help provide information on the condition of these individuals and

are important to support these findings and results. Without this information these results remain tentative.

Most of the results presented in this dissertation depend on lifetime reproductive success. Because LRS is often considered to be synonymous with fitness, these results are important and contribute to our understanding of some of the most significant selective pressures that have had and continue to have an impact on human evolution. Parents affect the reproductive success of their children primarily through the environmental effects of a shared household, family size and composition (siblings), PI and resources. Because traits that are strongly associated with fitness (LRS) are usually driven to fixation (Fisher, 1930), both the heritability and hence genetic variability of these traits tend to approach zero. The remaining environmental effects are then partitioned amongst factors that individuals have in common due to the effects of a shared household during the period of PI.

One of the most important of these environmental effects is that of family size or how many siblings an individual has. I also demonstrate an unambiguous quantity-quality tradeoff for family size. Each additional sibling reduces an individual's expected lifespan and LRS. This effect is significant, strong, and consistent across all decades analyzed. Although these results and analyses are meant to discover novel and generalizable evolutionary principles that are true for all human populations, none of these tests have previously been conducted on an Icelandic population.

This work will contribute to our understanding of basic evolutionary forces which may ultimately serve the public through an increased understanding of the effects that socioeconomic, technological and cultural change have on fertility, lifespan and sex

ratios. This mathematical research and modeling is unique in that it contributes to our understanding of population growth and its effects on resource availability, government policy and public health.

Chapter 1: Introduction: a brief history of Iceland

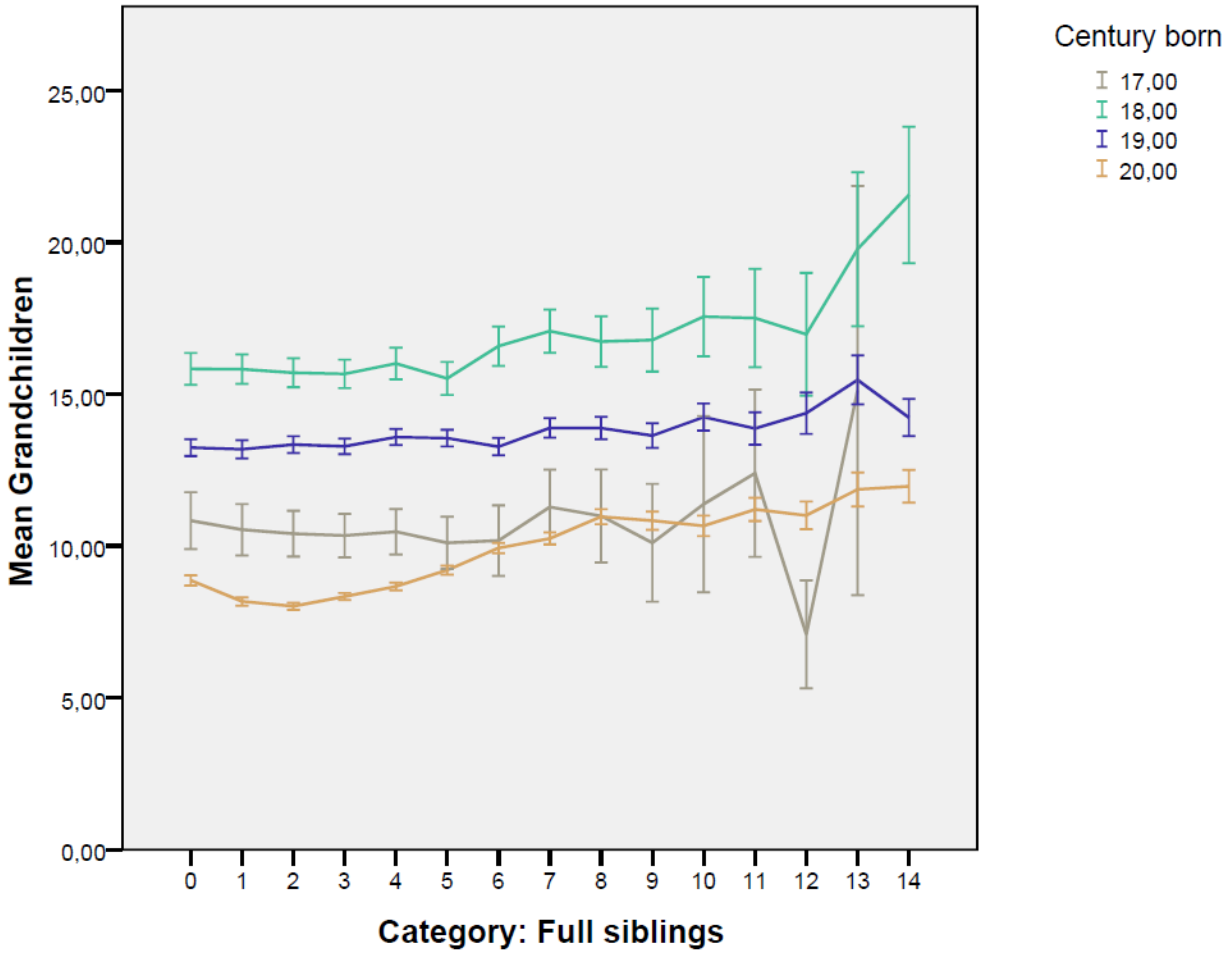

Figure 1.1: Descriptive statistics. Average grandchildren per individual by century

Chapter 2: Parental Investment Increases Reproductive Success and Lifespan in Iceland

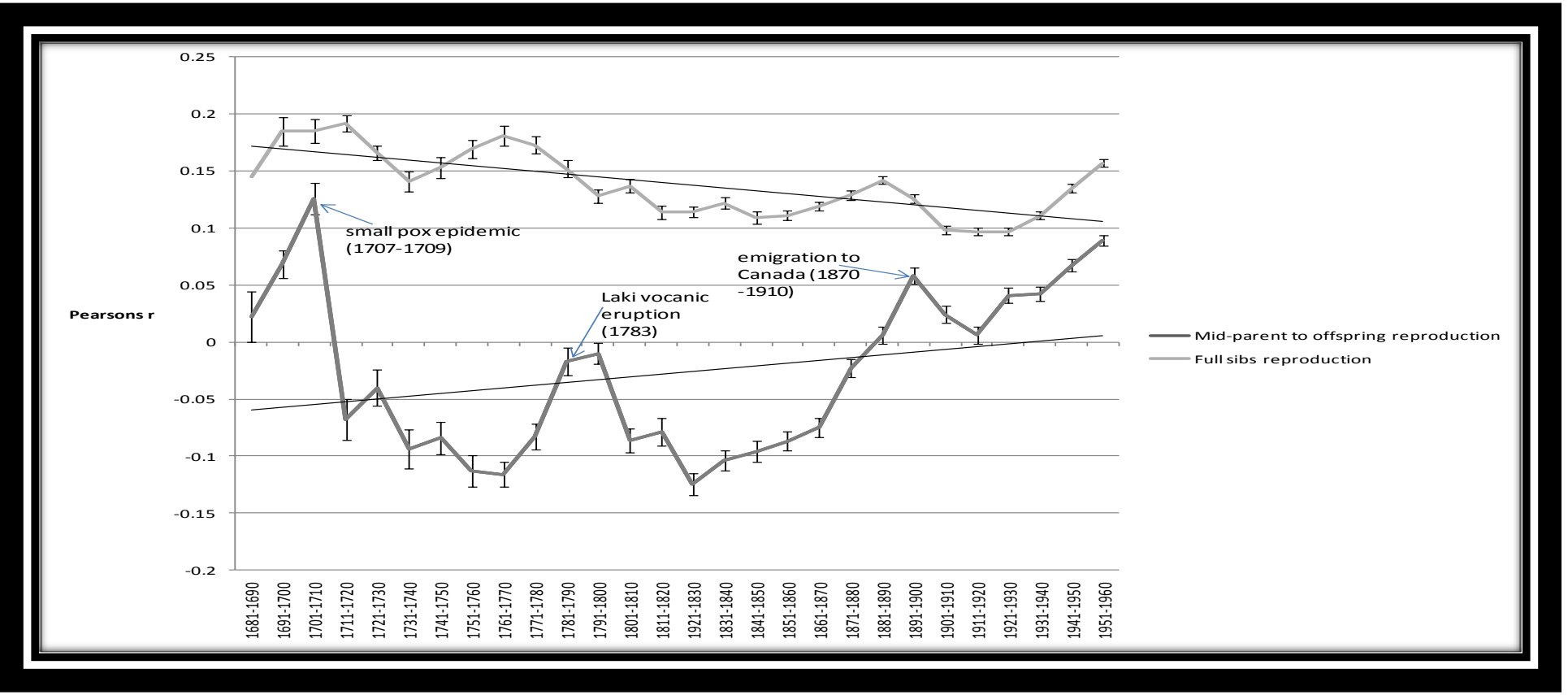

Figure 2.1: All values log transformed and standardized [(log transformed value – mean of decade)/ standard deviation of decade)] by individual's decade (z-score of mean of Lifetime reproductive success in which individual was born). Correlations are between parent and offspring reproduction and full sibling reproduction by decade. X axis-Pearson correlation standardized by decade. Y-axis by decade (1680-1960). Lower line is correlation between the combined reproduction (by decade) of parents and the average of their children's. Upper curve is the correlation (by decade) of full siblings correlation with average of all other full siblings correlations is weighted so that both correlations (offspring and average of full siblings is weighted by number (e.g. if an individual full sibling has 10 full siblings they each count as .1; similarly the mid-parent is simply (mothers + fathers lifetime reproductive success/2) offspring while offspring is weighted by number (e.g., 10 offspring count as .1 each).

Trait						
	Relationship	**Full siblings (all)**	**brothers**	**sisters**	**Opposite sex pairs**	****partial controlling for birth interval(all)**
RS		.130(.000)	.133(.000)	.145(.000)	.117(.000)	.130(.000)
		375249	169419	161509	187079	375246
	Relationship	**Mid-parent: offspring(all)**	**Father-son**	**Mother-daughter**	**Father-daughter**	**Mother-son**
RS		-.006 (.084)	.001(.766)	-.013(.004)	-.014(.003)	-.014(.003)
		93081	47407	49906	46918	46916

Table 2.1: Reproductive success for parent-offspring and full sibling pairs between 1650 and 1960. All comparisons weighted by number of siblings, sisters, brothers or offspring (e.g. a full sib pair in which the individuals have 4 full sibling is weighted ¼ as much as a single full sib pair). Reproductive success values are $\log_{10}$ transformed and standardized by decade [(log transformed value – mean of decade)/ standard deviation of decade)].

No. full sibs	Std reproduction	Avg. reproduction	N	Std Longevity	Avg. lifespan	N
0	.1292(.006)	2.71(.021)	24032	.0865(.007)	52.4(.238)	14736
1	.0793(.006)	2.55(.020)	24699	.0828(.007)	52.2(.241)	14049
2	.0666(.006)	2.51(.018)	28724	.0798(.007)	52.1(.223)	16448
3	.0641(.005)	2.50(.018)	31109	.0745(.006)	52.00(.213)	18560
4	.0525(.005)	2.46(.018)	30750	.0241(.007)	50.5(.217)	18535
5	.0259(.006)	2.38(.019)	27596	.007(.007)	49.97(.230)	17004
6	.0223(.006)	2.37(.020)	24284	-.0165(.008)	49.21(.246)	15305
7	.011(.007)	2.33(.022)	20477	-.0455(.009)	48.20(.266)	13317
8	-.0208(.008)	2.23(.025)	16134	-.0758(.010)	47.2(.303)	10472
9	-.0585(.008)	2.11(.028)	13010	-.1228(.012)	45.6(.341)	8341
10	-.0357(.010)	2.18(.032)	10454	-.1400(.013)	45.0(.375)	6844
>10	-.0768(.007)	2.04(.023)	21842	-.1731(.009)	43.7(.261)	14209

Table 2.3: Average reproduction and average lifespan from 1650-1910 are calculated in the following way: (standardized values [(log transformed value – mean of decade)/ standard deviation of decade] x standard deviation for each sib category) + mean for all sibling categories across all decades.

Table 2.2: The effect of parental reproduction on offspring longevity and reproduction.

Number of sibs	**FS correlation (Pearson‘s r)**	**N**	**Mean RS**	**N**	**Resources effect simulation average r (s.e.)**	**Resources effect F2 generation scaled RS to keep f3 generation at a mean of 2**	**Genetic effect simulation average r (s.e.)**
1	.183	39462	2.40(.014)	40470	.3325(.02)	.330(.02)	.1017(.03)
2	.170	49959	2.39	52626	.3204(.02)	.404(.02)	.1362(.02)
3	.145	53471	2.43	56842	.3086(.02)	.442(.018)	.1601(.02)
4	.129	49420	2.42	52006	.2939(.019)	.467(.014)	.1785(.01)
5	.120	41606	2.41	43240	.2841(.01)	.4845(.016)	.1922(.01)
6	.116	34526	2.43	35472	.2740(.015)	.495(.014)	.204(.016)
7	.100	28113	2.40	28656	.2675(.014)	.505(.014)	.212(.015)
8	.099	21642	2.28	21896	.2587(.014)	.513(.012)	.2186(.013)
9	.099	16788	2.37	16943	.2529(.013)	.519(.012)	.22619.013)
10	.078	13022	2.28	13120	.2443(.013)	.524(.012)	.233(.012)
>10	.075	27240	2.33	27331			

	Longevity difference	**RS difference**
Birth interval	.021(.000) 683962	.034(.000) 1195330

Table 2.4: The effect of the birth interval on lifespan and reproduction of full siblings (data from 1650-1910).

	Resources	Genes	Genes and resources
Parent-offspring correlation	**-.35 (.016)**	**.185 (.03)**	**.06 (.03)**
Full sibling correlation	**.28 (.027)**	**.201 (.03)**	**.21 (.02)**

Table 2.5: Simulations: **Resources model (resource mean = 2, resource standard deviation=2 and mean children per generation =2) Resources random – parental resources are randomly assigned and offspring resources are divided equally by children; Resources (dependent on offspring number) – parental resources are assigned based on the number of offspring parents have (e.g., more offspring=more resources).

	Relationship	Model	N	Correlation (p-value)	f^2(S.E.) (1)	f^2(S.E.) (2)	h^2(S.E.)(3)	Log Lkhd	LRT
RS	Full sibs	Family (1) IBD(3)	8456	.076(.000)	.093(.139)		0.00(.272)	-4726.747	.026
		Family(1)		.076(.000)	.071(.011)			4726.76	
		IBD(3)	8456	.076(.000)			0.137(.022)	4726.975	
	Parent-offspring	Family (1) IBD(3)	3489	.071(.000)	.0210(1.06)		.00001(2.1)	610.028	~16
		Family (1)	3489	.071(.000)	.0000(.01)			602.056	
		IBD(3)	3489	.071(.000)			0.000(0.03)	600.643	
	PO_FS	FS family (1) PO family (2) IBD(3)	13918		.46(.397)	.009(.018)	.094(.69)	-6940.83	
		FS family(1) IBD(3)	13918		.454(.396)		.1143(.692)	-6940.94	.11
		PO family(1) IBD(3)	13918		.0079(.018)		.639(.334)	-6941.79	1.84
Height	Full sibs	Family(1) IBD(3)	6159	.377(.000)	0.00 (.13)		.71 (.25)	-3624.81	
		Family(1)	6159		.358 (.013)			-3628.75	7.88
		IBD(3)	6159				.704(.025)	-3624.81	
	Parent-offspring	Family(1) IBD(3)	5029	.397(.000)	.3625(1.07)		.0000(2.119)	-2399.906	
		Family(1)	5029		.359(.015)			-2399.8	
		IBD(3)	5029				0.71(0.03)	-2400.02	
	PO_FS	FS family(1) PO family(2) IBD(3)	11441		.135(.348)	.359(.015)	.247(.557)	-5642.58	
		FS family(1) IBD(3)	11441		.0000(.333)		.8774(.598)	-5828.05	185.4
		PO family(1) IBD(3)	11441		.358(.015)		.417(.296)	-5642.66	

Table 2.6: ** RS, longevity, and height are all standardized by birth year, sex, and geographic region.

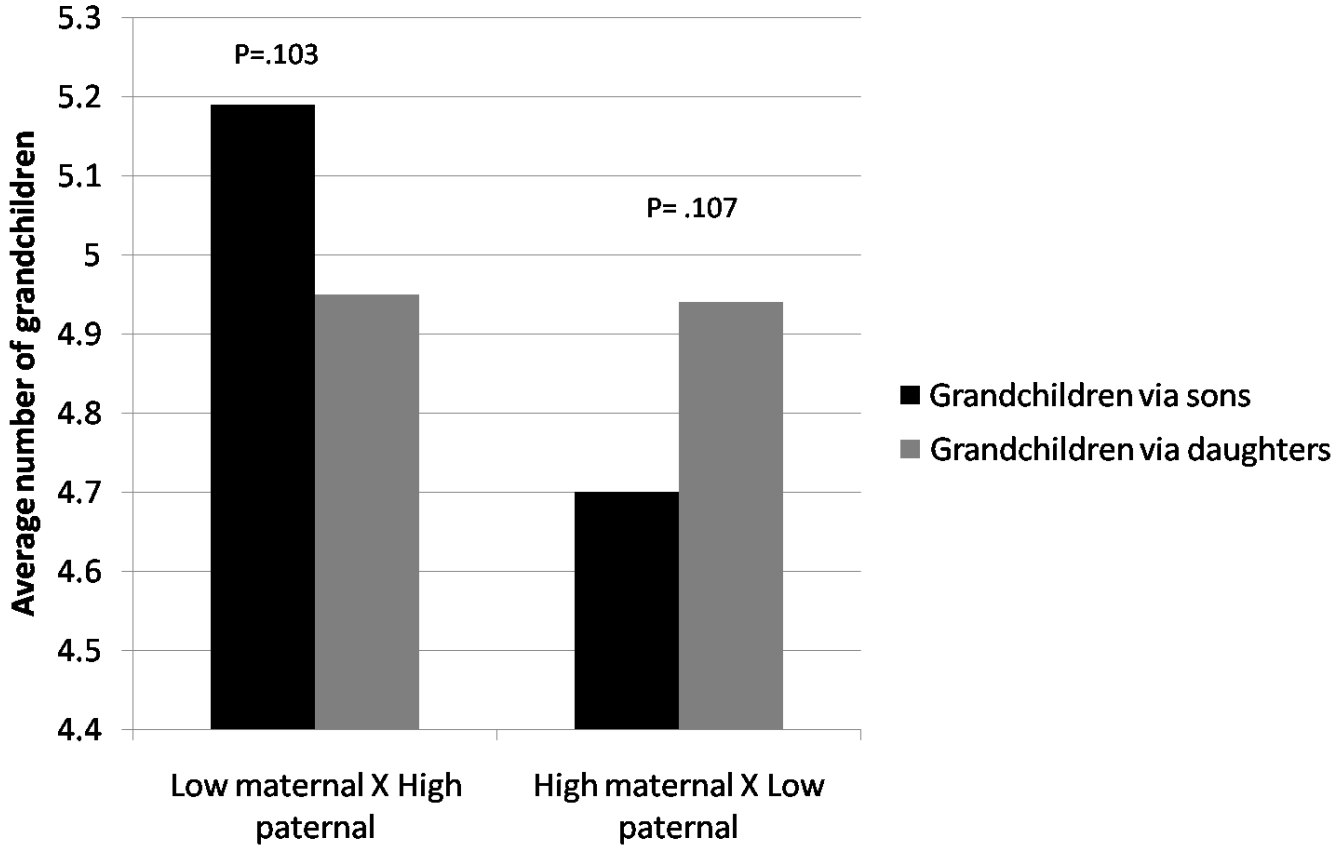

Figure 3.1: Suggestive evidence of sexual conflict in Iceland. Testcrosses between low RS grandmothers (2 or less children) and high RS grandfathers (5 or more children). Low maternal (2 or less children) X high paternal (5 or more children) crosses produce low RS fathers and high RS mothers while test-crosses between high RS grandmothers and low RS grandfathers (high maternal X low paternal) produce high RS mothers and low RS fathers as hypothesized by sexual conflict theory.

Chapter 4A: Reproductive Success is differentially inherited by sex: Data support a benefit to biasing the sex ratio in a manner suggested by the Trivers-Willard hypothesis

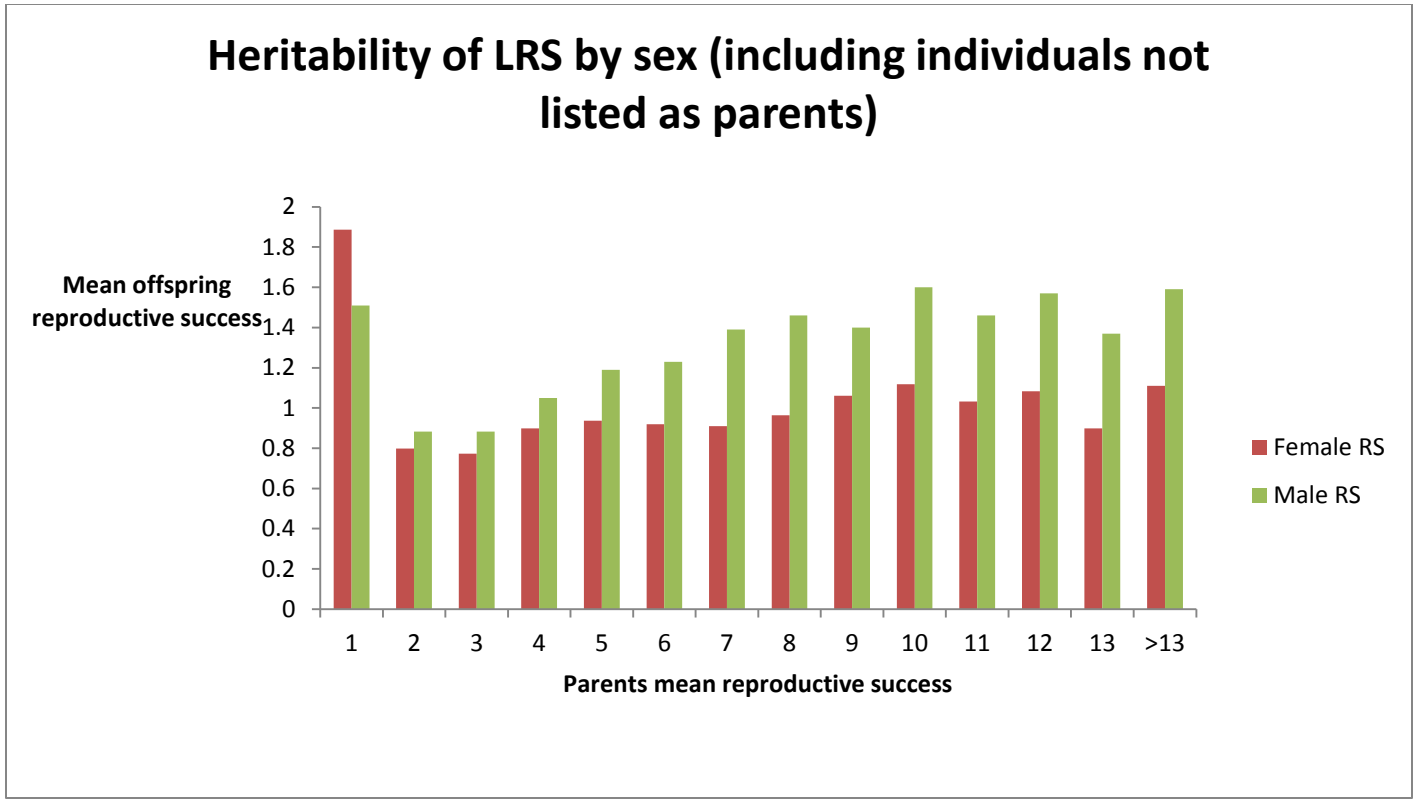

Figure 4A.1: Sample includes all individuals in which parents are known. Parents with high lifetime reproductive success (2+) produce sons with higher lifetime reproductive success than daughters while only daughters outperform only sons.

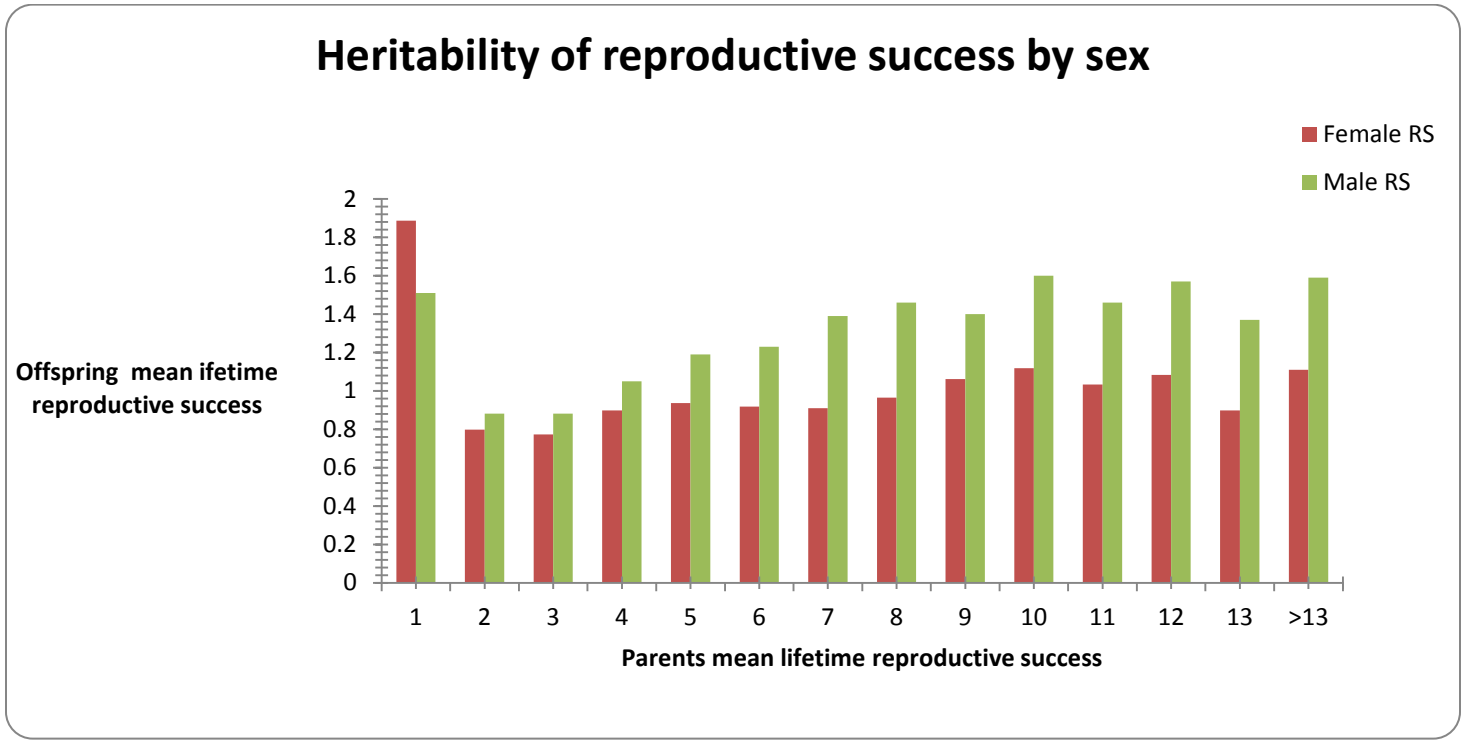

Figure 4A.2: Lifetime reproductive success of offspring based on parental reproductive success. Higher parental LRS produces increasingly more successful sons in comparison to daughters. The only case in which daughter outperform sons is when they are only children (parental LRS=1). Results are obtained from a publicly available genealogy with 235,000 individuals included in this sample. Only individuals with both parents and offspring listed are included. So this is likely the most accurate sample.

Individuals without identified parents	R=.294	F=2563, p<.0001
Individuals without parents or offspring identified	R=.166	F=562, p<.0001

Table 4A.1: Regression statistics for offspring sex and LRS on parental Lifetime Reproductive Success.

:

	males	females	total sex ratio	only sons to only daughter sex ratio
Entire sample	111,145	101,335	1.097	
Individuals w/o parents excluded	77,801	67,996	1.144	.798
Individuals w/o parents or offspring excluded	26,987	29,956	1.35	.913

Table 4A.2: Sample sizes and sex ratios: the group 'Individuals without parents excluded' consists of all individuals except those for which parents were unknown. These individuals were excluded in both samples. The group 'Individuals without parents or offspring excluded' consists of all individuals, except those for which parents were unknown and offspring were, either unknown or the individuals did not have any. The second sample gains in accuracy but suffers by excluding all individuals who did not report having any children.

	Mean LRS males	Mean LRS females	Variance in male LRS	Variance in female LRS
Individuals w/o parents excluded	1.25	1.05	6.36	5.2
Individuals w/o parents or offspring excluded	3.6	3.6	9.8	8.7

Table 4A.3: descriptive statistics on reproductive success. Sample sizes for parents mean lifetime reproductive success are as follows: males: females with 1 child: 6787: 4961; with 2 children: 2834: 2191; with 3 children: 2603: 2087; with 4 children: 2265: 1874; with 5 children: 2265:1524; with 6 children: 1830: 1326; with 7 children: 1770: 1524; with 8 children: 1830: 1326; with 9 children: 1407: 1071; with 10 children: 1299: 849; with 11 children: 953: 663; with 12 children: 705:490; with 13 children, 369: 238; with more than 13 children.

Total (N)	4058814	2076969	1981845	Sex ratio	fathers average age	secondary sex ratio
Under 15	364	183	181	1.01105		1.011049724
15-19	138070	71197	66873	1.06466	17	1.064659878
20-24	613672	315123	298549	1.055515	22	1.055515175
25-29	902017	462141	439876	1.050617	27	1.050616537
30-34	938365	480326	458039	1.048657	32	1.048657429
35-39	588626	301553	287073	1.05044	37	1.050440132
40-44	227479	116416	111063	1.048198	42	1.04819787
45-49	65940	33661	32279	1.042814	47	1.042814214
50-54	19287	9831	9456	1.039657	52	1.03965736
55-98	7927	4034	3893	1.036219	58	1.036218854
Not stated	557067	282504	274563	1.028922		1.028922324

Table 4A.4: Center for Disease Control data, 2008. All births in the United States in 2008. Father's age predicts sex ratio. Older fathers produce more daughters. R=-.95125, $p< 10^6$.

Chapter 4B: No Advantage Found for Biasing the Sex Ratio as a Function of Family Size Found in Iceland

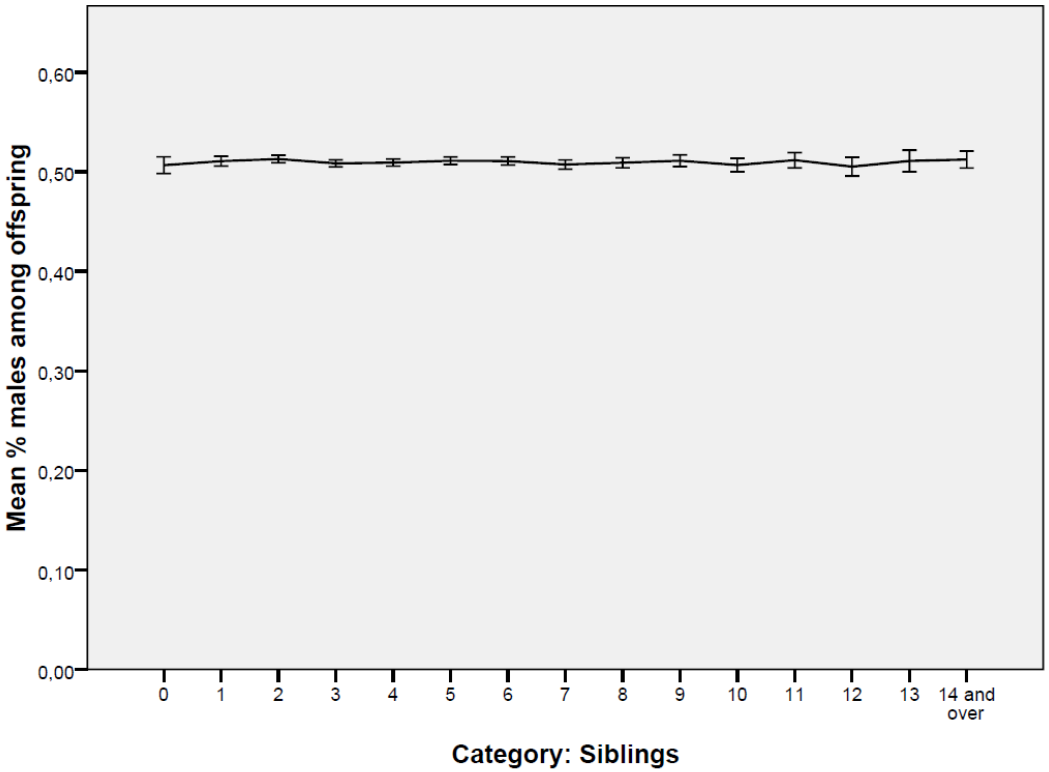

Figure 4B.1: Large families are one proxy for good condition (or high LRS) parents. This figure depicts a broad graphical overview of the relationship between parental reproductive success (represented on X axis by full siblings) - the more siblings, the better the condition of the parents- and sex ratio (percentage males on the Y axis). There is no discernible pattern between the two traits. R =.000, p=.772.

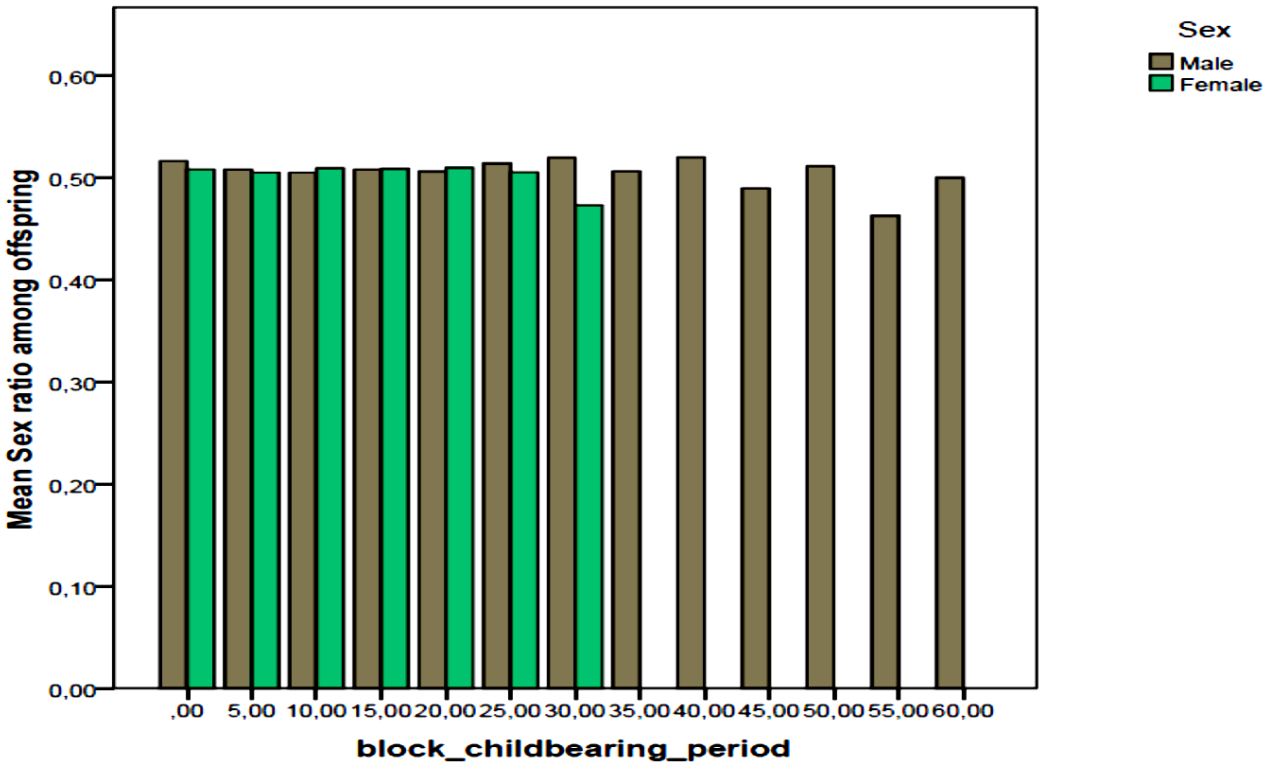

Figure 4B.2: Sex ratio plotted against reproductive duration (the time in years between first and last birth). There is no relationship.

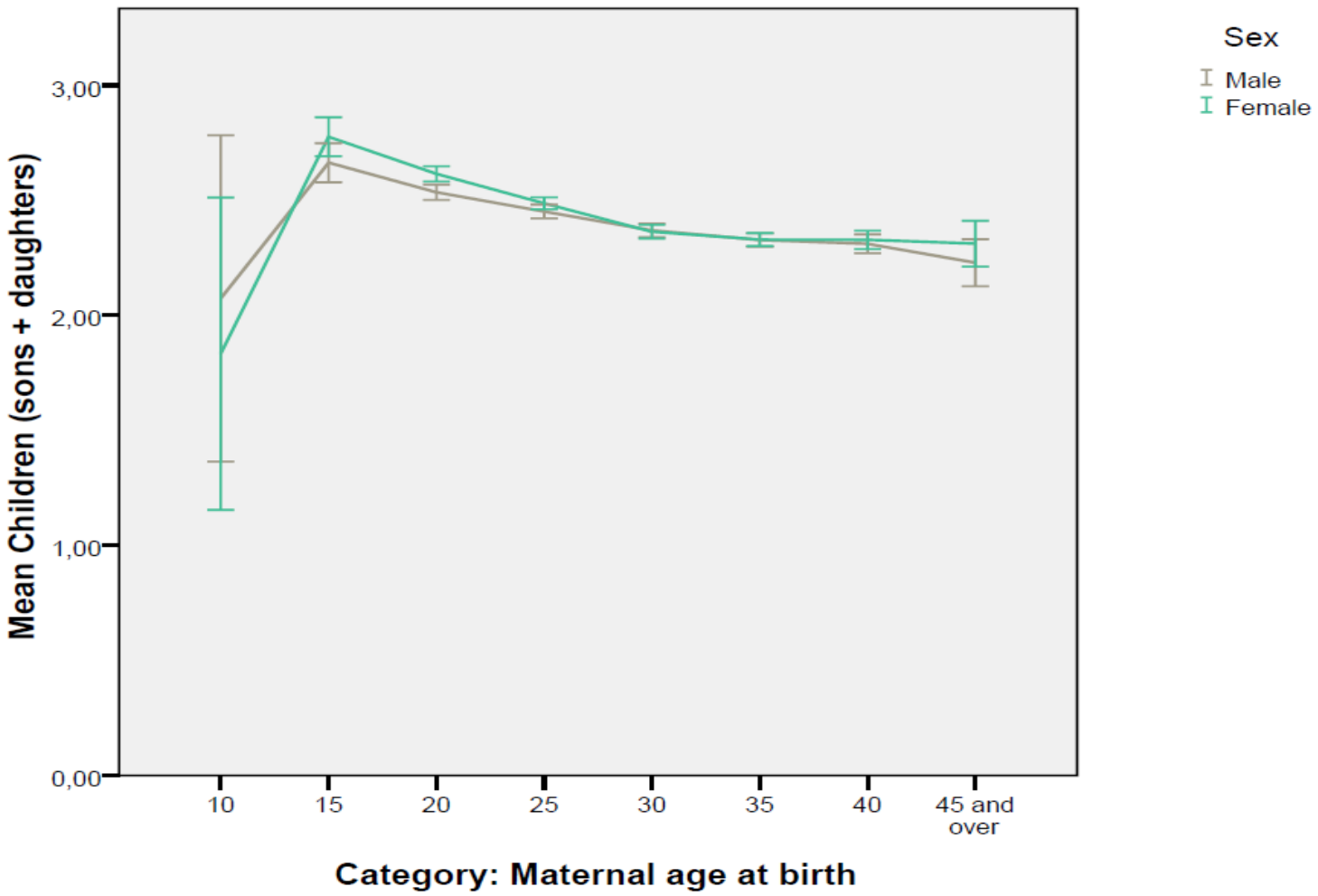

Figure 4B.3: No relationship between mother's age at birth and sex of child (1650-1950 Iceland). R=.04, p=.210

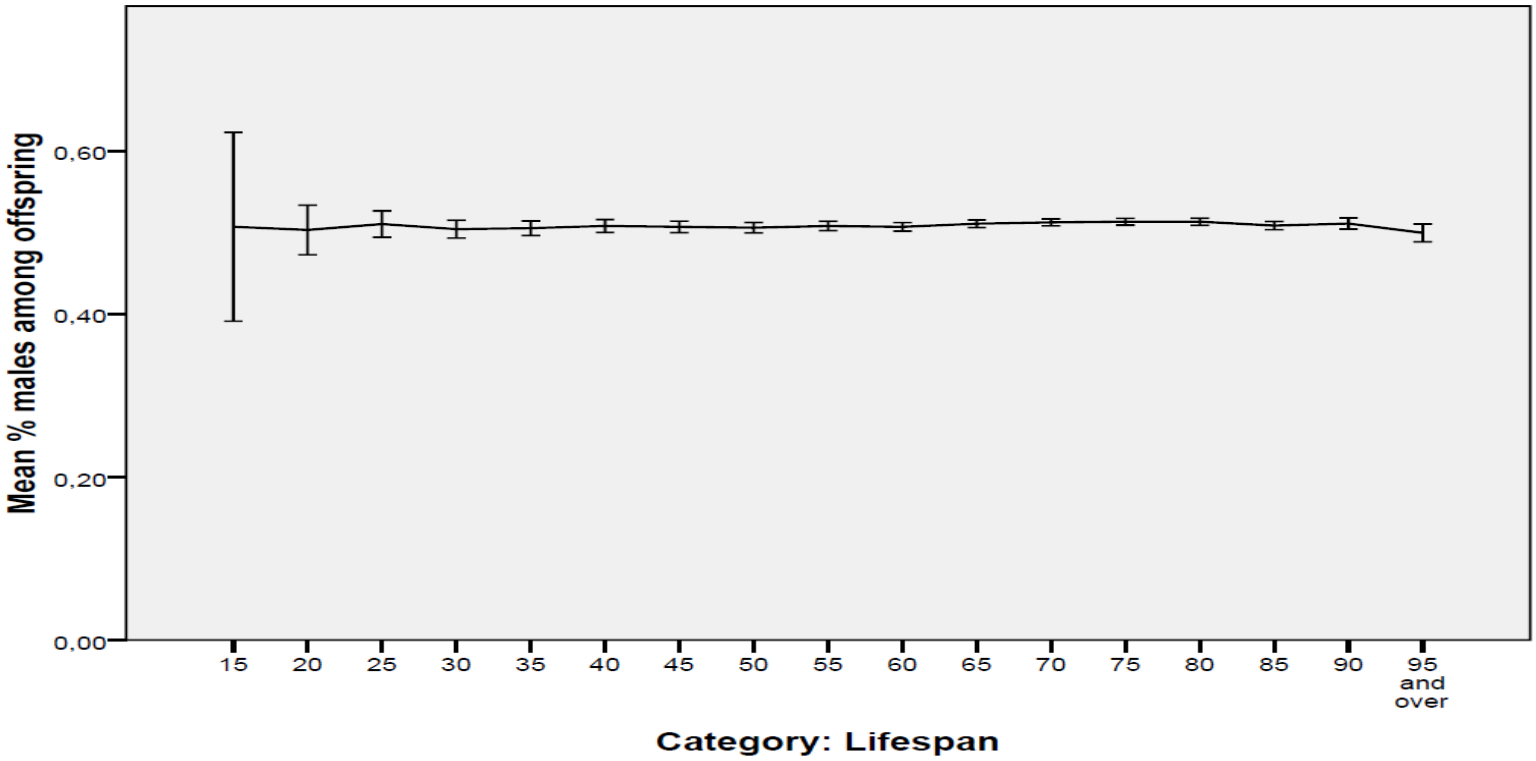

Figure 4B.4: Parental lifespan (X-axis) as a function of sex ratio (Y-axis), No relationship.

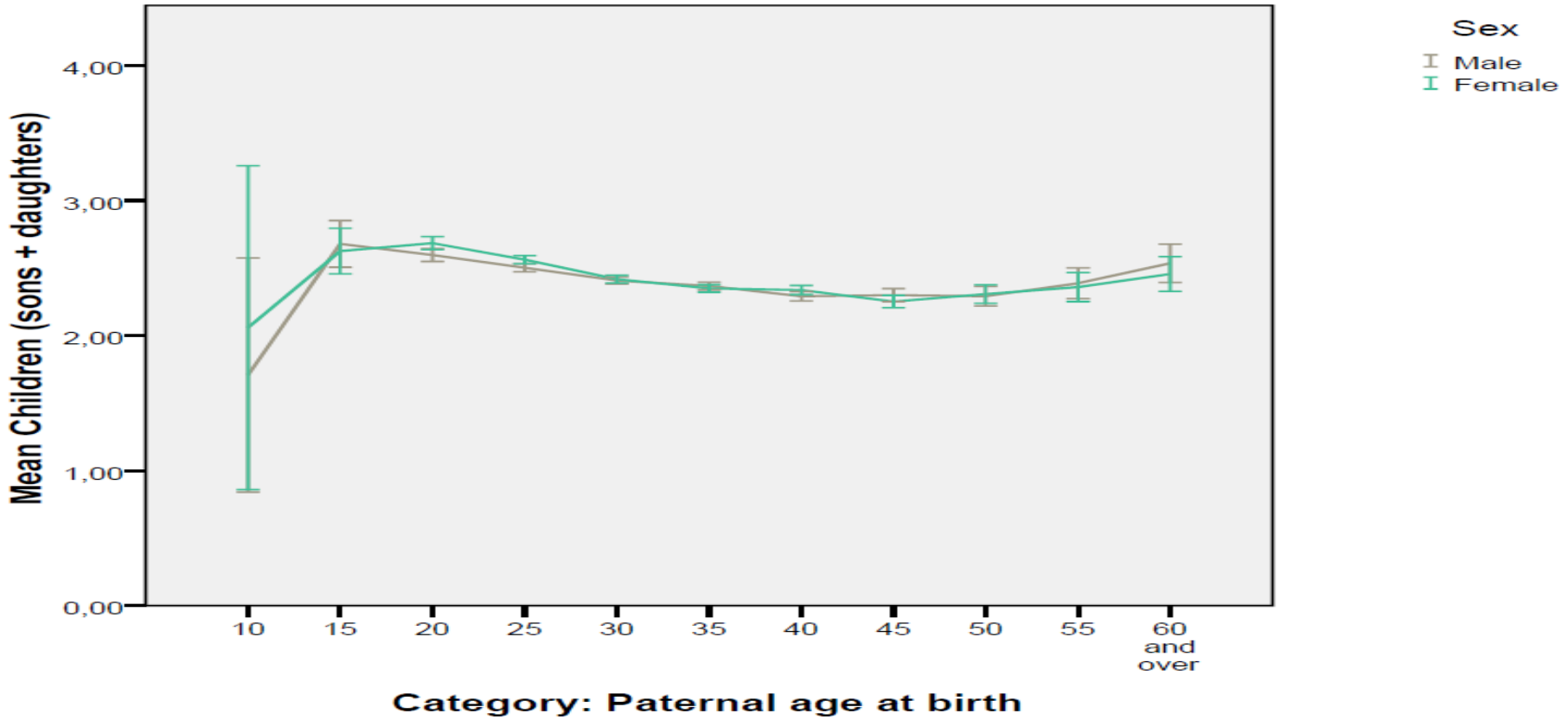

Figure 4B.5: Sex ratio and paternal age at birth; younger fathers are more likely to give birth to sons and older fathers are more likely to give birth to daughters. (r=-.004, p=.029).

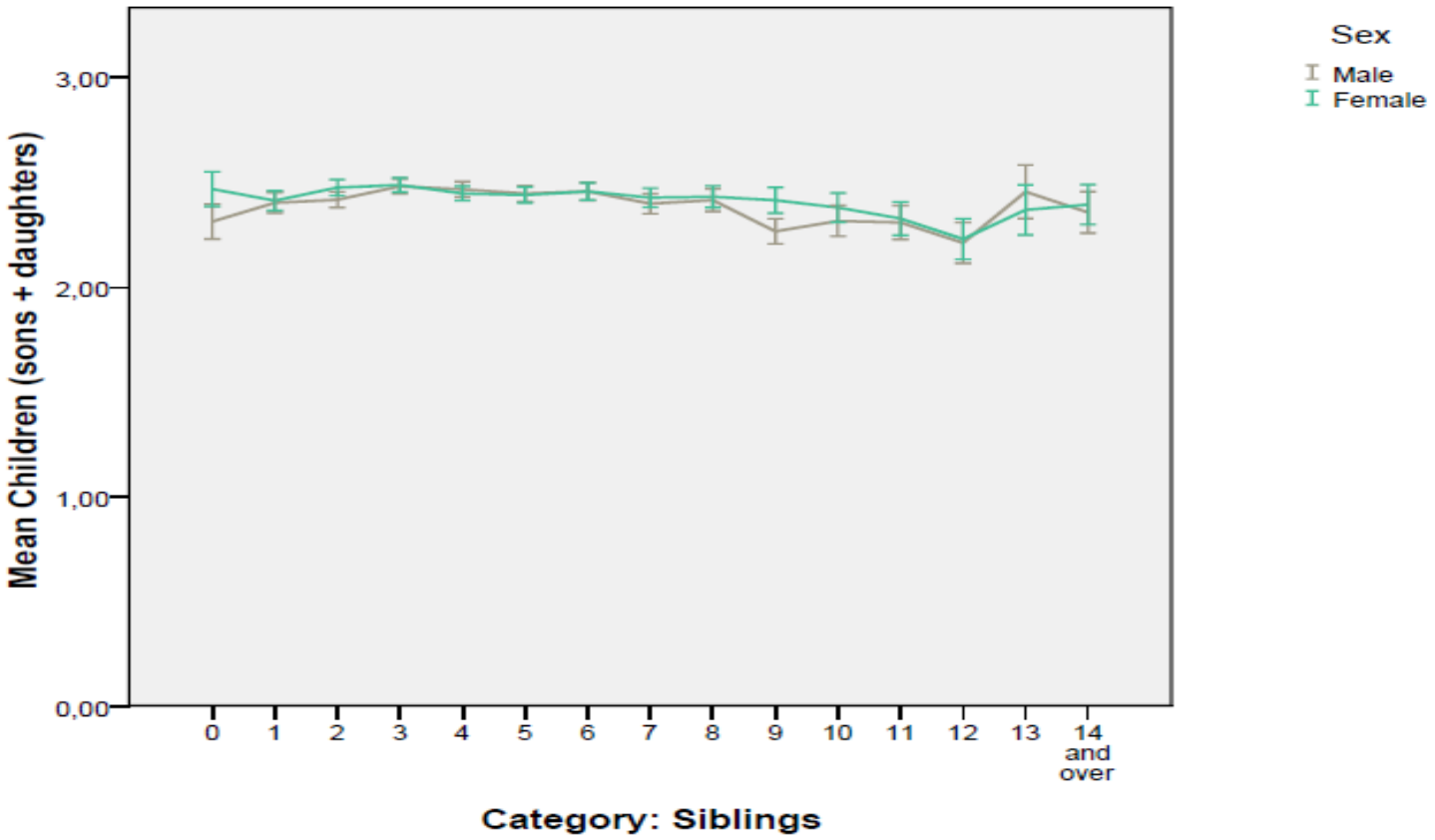

Figure 4B.6: No relationship between sex of offspring and parental reproductive success (depicted as siblings on X- axis). Regression (r=-.01, p=.784).

Correlations

		Full siblings	Siblings (full siblings + 1/2 half siblings)	% males among offspring
Sex	Pearson Correlation	,000	,000	-,002
	Sig. (2-tailed)	,869	,923	,445
	N	378138	378138	251354
Year of birth	Pearson Correlation	,018**	,023**	,012**
	Sig. (2-tailed)	,000	,000	,000
	N	378138	378138	251354
Children (sons + daughters)	Pearson Correlation	-,016**	-,012**	,001
	Sig. (2-tailed)	,000	,000	,626
	N	378138	378138	251354
Full siblings	Pearson Correlation	1	,929**	,000
	Sig. (2-tailed)		,000	,772
	N	378138	378138	224155
Siblings (full siblings + 1/2 half siblings)	Pearson Correlation	,929**	1	,000
	Sig. (2-tailed)	,000		,737
	N	378138	378138	224155
% males among offspring	Pearson Correlation	,000	,000	1
	Sig. (2-tailed)	,772	,737	
	N	224155	224155	251354
% males among full siblings	Pearson Correlation	-,001	,000	,005*
	Sig. (2-tailed)	,428	,657	,033
	N	345450	345450	203354
Birth order within sex	Pearson Correlation	,571**	,533**	,004
	Sig. (2-tailed)	,000	,000	,062
	N	378138	378138	224155
Age when first child born	Pearson Correlation	,065**	,059**	-,006**
	Sig. (2-tailed)	,000	,000	,003
	N	216414	216414	242884
Duration of reproductive period	Pearson Correlation	,029**	,040**	,003
	Sig. (2-tailed)	,000	,000	,098
	N	216414	216414	242884
Repro. rate (offspring per year of reproductive duration)	Pearson Correlation	-,043**	-,048**	,000
	Sig. (2-tailed)	,000	,000	,716
	N	338267	338267	202028
Repro. effort (offspring ^ 2 per year of reproductive duration)	Pearson Correlation	-,007**	-,002	-,003
	Sig. (2-tailed)	,000	,212	,220
	N	338267	338267	202028

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Table 4B.1: Correlation matrix for key variables expected to affect sex ratios of offspring. Iceland 1650-1950.

Chapter 5: Conclusion and Future Directions

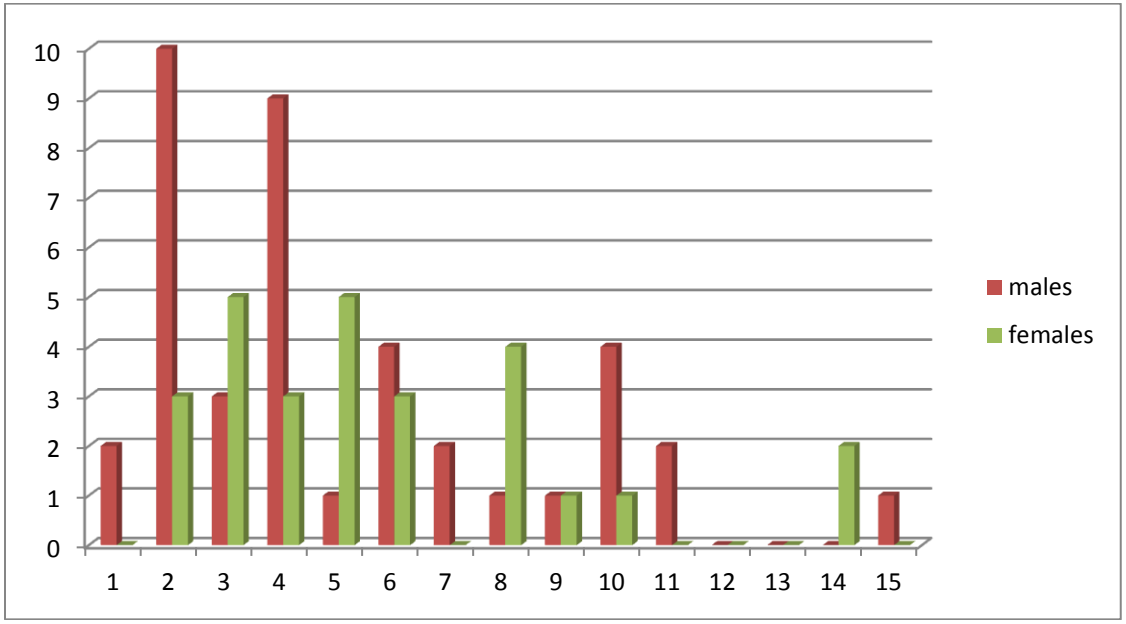

Figure 5.1: Age of divorce based on ages of sons and daughters: Data from New Brunswick Family Court.

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APPENDIX A: Visual Basic Code for Simulations

'SIMULATION CODE GENETIC AND RESOURCES

```
Type ParentVec
'Type organizes the following variables as subcategories of ParentVec
'ParentVec is a actegory of variable we have inveneted, not an actual varibale
  Resources As Double
  Geneticfitness As Double
  Allele1 As Integer
  Allele2 As Integer
  ChildCount As Integer
  GrandChildCount As Integer
  Grandchildren() As Integer
  'empty parentheses indicate a dynamic (flexible) array
```

```
End Type
Type ChildVec
  ParentID As Integer
  Resources As Double
  Geneticfitness As Double
  Allele1 As Integer
  Allele2 As Integer
  SibCnt As Integer
  SibsChildCnt As Integer
  SibsAvgChildCnt As Variant
  ChildCnt As Integer
  MateID As Integer
  Mated As Boolean
End Type
Sub model()
 'this starts the routine
Dim xx() As Double
Dim yy() As Double
'xx and yy are dynamic arrays
Dim pearsoncorr As Double
Dim PearsonPval As Double
Dim pearsonZ As Double

 Dim i As Integer
 Dim j As Integer
 Dim k As Integer
 Dim dummyCount As Long
 Dim MeanChildren As Double
```

```
Dim ParentCount As Integer

Dim ResourceMean As Double, ResourceStDev As Double

 Dim ParentCount_firsthalf As Integer

 Dim TotChildren As Long


  'You must give your variables a type of number (e.g. integer, decial etc..)

  'Dim is short for dimension.  Use 'variant for variables where you are unsure what dimensions they will have


 Dim Parents() As ParentVec

   'This tells the computer that Parents is a dynamic array holding the variables under ParentVec


 Dim Children() As ChildVec

 'This tells the computer that Children is a dynamic array holding the variables under ChildVec



'Next 7 variables that follow should be the only things you change in simulations

  Simulations = 20

  ParentCount = 10

  ResourceMean = 1

  ResourceStDev = 0.05

  AlleleAfrequency = 0.5

  'the population frequency of allele 0, th rest of the time it has a value of 1

  Allelicimpact = 0.9

  'the effect certain combinations of alleles have on fitness

  MeanChildren = 2


  For k = 1 To Simulations
```

```
   'This is the number of simulations (e.g. correlations) you want to run/ get
ParentCount_firsthalf = ParentCount / 2

 TotChildren = 0
 ReDim Parents(ParentCount)
  'The Redim statement tells the computer the size of the array - here it is saying that this array
  'should be the same size as the number of simulations you run (e.g. 1000)

For i = 1 To ParentCount

  If Rnd < AlleleAfrequency Then
  Parents(i).Allele1 = 0
  Else: Parents(i).Allele1 = 1
  End If

 'assign Allele1 to parents

  If Rnd < AlleleAfrequency Then
  Parents(i).Allele2 = 0
  Else: Parents(i).Allele2 = 1
  End If


  'assign Allele2 to parents

  Parents(i).Resources = WorksheetFunction.NormInv(Rnd, ResourceMean, ResourceStDev)
  Parents(i).ChildCount = InversePoisson(Rnd, MeanChildren)
  'Under the category 'parents'take ChildCount(and for whatever run it's on (i)) assign to the 'Childcount'
```

```
    'subvariable a random number taken from the possion distribution with a mean of 2


    TotChildren = TotChildren + Parents(i).ChildCount
    'keeps an accumualting running total of the number of children produced through sim


    ChildCount = Parents(i).ChildCount
      'The ChildCount variable is now defined here as the same thing as above


Next i
     'Do the next parent
For i = 1 To ParentCount


    If Parents(i).Allele1 = 0 And Parents(i).Allele2 = 0 Then
    Parents(i).Geneticfitness = 0.5
    End If
    If Parents(i).Allele1 = 0 And Parents(i).Allele2 = 1 Then
    Parents(i).Geneticfitness = 1.5
    End If
    If Parents(i).Allele1 = 1 And Parents(i).Allele2 = 0 Then
    Parents(i).Geneticfitness = 1.5
    End If
    If Parents(i).Allele1 = 1 And Parents(i).Allele2 = 1 Then
    Parents(i).Geneticfitness = 1.5
    End If
Next i
    'Give parental genotypes genetic fitness number
 dummyCount = 0
 ReDim Children(TotChildren)
```

```
'tells the computer that the size of the array Children is now the cumulative number of children
'produced by the simulation
 For i = 1 To ParentCount
   'do the runs again
   ReDim Parents(i).Grandchildren(Parents(i).ChildCount)
   'size the grandchildren array (previously undefined) from the parents category
   'for parent i to parent i's child count.
   'basically just make the grandchildren category (filing cabinet) the
   'same size (or give it as many slots as the child count for parent i
   '(e.g. if parent 3 has 6 children, give the array grandchildren fot that parent 6 slots

   For j = 1 To Parents(i).ChildCount
    'this begins the subloop that will run (j) times for each parent (i) which is
    'telling the computer to run this loop as many times as children were produced for parent i
     dummyCount = dummyCount + 1
     'starts the dummy count or the total number of children count
     'this loop is a running summary of children produced and runs during both j and i loops
     Children(dummyCount).ParentID = i
     'assigns the parent id (i) to each successive j loop which is of course based on the i loop
     'this is a clever way to to give the kids parents ids in order
     Parents(i).Grandchildren(j) = InversePoisson(Rnd, (MeanChildren * (Parents(i).Resources /
Parents(i).ChildCount)))
     'assign grandchildren j from parents i a number of kids from the poisson dist
     Parents(i).GrandChildCount = Parents(i).GrandChildCount + Parents(i).Grandchildren(j)
     'defines grandchild count for each parent as the number of grandchildren produced by each
     'child produced in the j loop
     Children(dummyCount).ChildCnt = Parents(i).Grandchildren(j)
     'ChildCnt is equal to the number of gandchildren slots
```

```
Children(dummyCount).SibCnt = Parents(i).ChildCount - 1
'calculates sibs which are equal to the ChildCount of parent i minus 1
If Rnd < 0.5 Then
Children(dummyCount).Allele1 = Parents(i).Allele1
Else: Children(dummyCount).Allele1 = Parents(i).Allele2
End If
'this gives children a 50% chance of getting allele1 from their parents and a 50%
'chance of getting allele 2.  This should give relatedness of parents to offspring
'as r= 1/2
If Rnd < AlleleAfrequency Then
Children(dummyCount).Allele2 = 0
Else: Children(dummyCount).Allele2 = 1
End If
 'this gives the other allele to the children from a random mate with allele
 'frequencies defined by the population or here by AlleleAfrequency
'Might want to modify this by some random mendeliian factor (e.g. multiply by 1/2)
Children(dummyCount).Resources = (Parents(i).Resources) / (Parents(i).ChildCount)

If Children(dummyCount).Allele1 = 0 And Children(dummyCount).Allele2 = 0 Then
Children(dummyCount).Geneticfitness = 0.5
End If
If Children(dummyCount).Allele1 = 0 And Children(dummyCount).Allele2 = 1 Then
Children(dummyCount).Geneticfitness = 1.5
End If
If Children(dummyCount).Allele1 = 1 And Children(dummyCount).Allele2 = 0 Then
Children(dummyCount).Geneticfitness = 1.5
End If
If Children(dummyCount).Allele1 = 1 And Children(dummyCount).Allele2 = 1 Then
```

```
    Children(dummyCount).Geneticfitness = 1.5
    End If
   Next j
  'do j or the children and grandchildren loops again
 Next i
  'do the i or parents loop again
 dummyCount = 0
 'reset dummyCount or total kids to 0
 For i = 1 To ParentCount
 'run the i loop through all the parents
  For j = 1 To Parents(i).ChildCount
  'run the j loop through all the kids
   dummyCount = dummyCount + 1
  'start counting the kids
  Children(dummyCount).SibsChildCnt = Parents(i).GrandChildCount - Children(dummyCount).ChildCnt
  'calculate sibs kids as parent i's grannkids minus whatever kid the computer is on's kids
  If (Children(dummyCount).SibCnt) <> 0 Then
   Children(dummyCount).SibsAvgChildCnt = Children(dummyCount).SibsChildCnt /
(Children(dummyCount).SibCnt)
    Else: Children(dummyCount).SibsAvgChildCnt = -1
   End If
   Next j
 Next i
   'fill in the two vectors I want (the dependent and independent variables)-
   'here they will always be ChildCnt and either SibsChildCnt or SibCnt
   ReDim xx(dummyCount)
   ReDim yy(dummyCount)
 'this makes dummyCount the number of categories for xx and yy variables
```

```
 dummyCount = 0
 'reset to 0
 For i = 1 To TotChildren
   'Totchildren is the same as dummyCount
   dummyCount = dummyCount + 1
   'start a running tally
 xx(dummyCount) = Children(dummyCount).ChildCnt
 yy(dummyCount) = Children(dummyCount).SibCnt
    'stick the variables into the arrays
 Next i
  'fills in the xx and yy variables for the corrleation
Call Pearson1(xx, yy, dummyCount, pearsoncorr, PearsonPval, pearsonZ)
  'this tells computer to go to the function called Pearson1 and do what it says
Cells(k + 2, 1) = pearsoncorr
Cells(k + 2, 2) = PearsonPval
Cells(k + 2, 3) = pearsonZ
Cells(k + 2, 4) = dummyCount
 'fill in the two vectors I want (the dependent and independent variables)-
    'here they will always be ChildCnt and either SibsChildCnt or SibCnt
    dummyCount = 0
 ReDim xx(TotChildren)
 ReDim yy(TotChildren)
 For i = 1 To TotChildren
   If Children(i).SibsAvgChildCnt >= 0 Then
     dummyCount = dummyCount + 1
     xx(dummyCount) = Children(i).ChildCnt
     yy(dummyCount) = Children(i).SibsAvgChildCnt
   End If
```

```
  Next i
 ReDim Preserve xx(dummyCount), yy(dummyCount)
    'stick the variables into the arrays, still don't know what preserve means
  'fills in the xx and yy variables for the corrleation
Call Pearson1(xx, yy, dummyCount, pearsoncorr, PearsonPval, pearsonZ)
 'this tells computer to go to the function called Pearson1 and do what it says
Cells(k + 2, 5) = pearsoncorr
Cells(k + 2, 6) = PearsonPval
Cells(k + 2, 7) = pearsonZ
Cells(k + 2, 8) = dummyCount
 Next k
  MsgBox "pause"
End Sub
Function InversePoisson(rand As Double, lambda As Double) As Long
 Dim i As Long
 Dim Fx As Double
 Dim px As Double
On Error GoTo ErrorEndSub
 px = Exp(-lambda)
 Fx = px
 If rand < Fx Then
  InversePoisson = 0
  Exit Function
 End If
 For i = 1 To lambda * 4
  px = px * lambda / i
  Fx = Fx + px
  If rand < Fx Then
```

```
    InversePoisson = i
    Exit Function
  End If
 Next i

ErrorEndSub:
  InversePoisson = 0
  Exit Function
End Function
'This just provides the number of children based on
'numbers drawn randomly from a possion distribution with a mean of X
Sub Pearson1(xx() As Double, yy() As Double, n As Long, rp As Double, probrp As Double, z As Double)
'#include <math.h>
'#define TINY 1.0e-20 Will regularize the unusual case of complete correlation.
'void pearsn(float x[], float y[], unsigned long n, float *r, float *prob,float *z)
'Given two arrays x[1..n] and y[1..n], this routine computes their correlation coe.cient
'r (returned as r), the signi.cance level at which the null hypothesis of zero correlation is
'disproved (prob whose small value indicates a signi.cant correlation), and Fisher's z (returned
'as z), whose value can be used in further statistical tests as described above.
'float betai(float a, float b, float x);
'float erfcc(float x);
Dim j As Long, yt As Double, xt  As Double
Dim syy As Double, sxy As Double, sxx As Double, ay As Double, ax As Double
Dim TINY As Double
Dim k As Integer, m As Integer
Dim t As Double, df  As Double
syy = 0
sxy = 0
```

```
sxx = 0
ay = 0
ax = 0
TINY = 1E-20
'Find the means.
For j = 1 To n
  ax = ax + xx(j)
  ay = ay + yy(j)
Next j
ax = ax / n
ay = ay / n
' Compute the correlation coefficient.
For j = 1 To n
  xt = xx(j) - ax
  yt = yy(j) - ay
  sxx = sxx + (xt * xt)
  syy = syy + (yt * yt)
  sxy = sxy + (xt * yt)
Next j
rp = sxy / (Sqr(sxx * syy) + TINY)
  z = 0.5 * Log((1 + rp + TINY) / (1 - rp + TINY))
  df = n - 2
  t = rp * Sqr(df / ((1 - rp + TINY) * (1 + rp + TINY))) 'Equation (14.5.5).
  probrp = betai(0.5 * df, 0.5, df / (df + t * t)) 'Student's t probability.
'/* *prob=erfcc(fabs((*z)*sqrt(n-1.0))/1.4142136) */
'For large n, this easier computation of prob, using the short routine erfcc, would give approximately
'the same value.
End Sub
```

```
Function ProbNorm(X As Double) As Double
    ' upper one sided tail probability of the normal distribution
    ' for a given normal deviate, x, by 26.2.16 in Abramowitz and Stegun.
    Dim z As Double, t As Double, p As Double, xa As Double
    xa = Abs(X)
    If xa > 12 Then
      p = 0
     Else
      z = 0.39894228 * Exp(-0.5 * xa * xa)
      t = 1 / (1 + 0.33267 * xa)
      p = z * t * (0.4361836 + t * (0.937298 * t - 0.1201676))
    End If
    If X >= 0 Then ProbNorm = p Else ProbNorm = 1 - p
End Function
Function ProbChi(X2 As Double, ndf As Integer) As Double
  ProbChi = gammq(CDbl(0.5 * ndf), 0.5 * X2)
End Function
Function PoissonCumProb(Success As Long, Mean As Double) As Double
 PoissonCumProb = gammq(CDbl(Success + 1), Mean)
End Function
Function gammq(a As Double, X As Double) As Double
 Dim gamser As Double, gammcf As Double, gln As Double
  If X < 0 Or a <= 0 Then MsgBox "Invalid arguments in routine gammq"
  If X < a + 1 Then
     Call gser(gamser, a, X, gln)
     gammq = 1 - gamser
    Else
     Call gcf(gammcf, a, X, gln)
```

```
    gammq = gammcf
  End If
End Function
Sub gcf(gammcf As Double, a As Double, X As Double, gln As Double)
 Dim ITMAX As Integer, EPS As Double, FPMIN As Double
 Dim i As Integer
 Dim an As Double, b As Double, c As Double, d As Double, del As Double, h As Double
 ITMAX = 100
 EPS = 0.0000003
 FPMIN = 1E-30
   gln = gammln(a)
   b = X + 1 - a
   c = 1 / FPMIN
   d = 1 / b
   h = d
   For i = 1 To ITMAX
      an = -i * (i - a)
      b = b + 2
      d = an * d + b
      If Abs(d) < FPMIN Then d = FPMIN
      c = b + an / c
      If Abs(c) < FPMIN Then c = FPMIN
      d = 1 / d
      del = d * c
      h = h * del
      If Abs(del - 1) < EPS Then Exit For
   Next i
   If i > ITMAX Then MsgBox "a too large, ITMAX too small in gcf"
```

```
    gammcf = Exp(-X + a * Log(X) - (gln)) * h
End Sub
Sub gser(gamser As Double, a As Double, X As Double, gln As Double)
 Dim ITMAX As Integer, EPS As Double
 Dim n As Integer
 Dim sum As Double, del As Double, ap As Double
 ITMAX = 100
 EPS = 0.0000003
  gln = gammln(a)
   If X <= 0 Then
      If X < 0 Then MsgBox "x less than 0 in routine gser"
      gamser = 0
      Exit Sub
     Else
      ap = a
      del = 1 / a
      sum = 1 / a
      For n = 1 To ITMAX
         ap = ap + 1
         del = del * (X / ap)
         sum = sum + del
         If Abs(del) < Abs(sum) * EPS Then
            gamser = sum * Exp(-X + a * Log(X) - (gln))
            Exit Sub
         End If
      Next n
      MsgBox "a too large, ITMAX too small in routine gser"
      Exit Sub
```

```
    End If
End Sub
Function gammln(xx As Double) As Double
 Dim X As Double, y As Double, tmp As Double, ser As Double
 Dim cof(6) As Double
 Dim j As Integer
 cof(0) = 76.1800917294715
 cof(1) = -86.5053203294168
 cof(2) = 24.0140982408309
 cof(3) = -1.23173957245015
 cof(4) = 1.20865097386618E-03
 cof(5) = -5.395239384953E-06
   y = xx
   X = xx
   tmp = X + 5.5
   tmp = tmp - (X + 0.5) * Log(tmp)
   ser = 1.00000000019001
   For j = 0 To 5
     y = y + 1
     ser = ser + cof(j) / y
   Next j
   gammln = -tmp + Log(2.506628274631 * ser / X)
End Function
Function betacf(a As Double, b As Double, X As Double) As Double
 Dim MAXIT As Long, EPS As Double, FPMIN As Double
 MAXIT = 100
 EPS = 0.0000003
 FPMIN = 1E-30
```

```
'Evaluates continued fraction for incomplete beta function by modified Lentz's method (§5.2).
'void nrerror(char error_text[]);
Dim m As Integer, m2 As Integer
Dim aa As Double, c As Double, d As Double, del As Double, h As Double
Dim qab As Double, qam As Double, qap As Double
qab = a + b 'These q's will be used in factors that occur in the coefficients (6.4.6)
qap = a + 1
qam = a - 1
c = 1 'First step of Lentz's method.
d = 1 - qab * X / qap
If Abs(d) < FPMIN Then d = FPMIN
d = 1 / d
h = d
For m = 1 To MAXIT
  m2 = 2 * m
  aa = m * (b - m) * X / ((qam + m2) * (a + m2))
  d = 1 + aa * d ' One step (the even one) of the recurrence.
  If Abs(d) < FPMIN Then d = FPMIN
  c = 1 + aa / c
  If Abs(c) < FPMIN Then c = FPMIN
  d = 1 / d
  h = h * d * c
  aa = -(a + m) * (qab + m) * X / ((a + m2) * (qap + m2))
  d = 1 + aa * d 'Next step of the recurrence (the odd one).
  If Abs(d) < FPMIN Then d = FPMIN
  c = 1 + aa / c
  If Abs(c) < FPMIN Then c = FPMIN
  d = 1 / d
```

```
 del = d * c
 h = h * del
 If Abs(del - 1) < EPS Then Exit For ' Are we done?
Next m
If m > MAXIT Then
 MsgBox "a or b too big, or MAXIT too small in betacf"
End If
betacf = h
End Function
Function erfcc(X As Double) As Double
 'returns the complementary error function erfc(x) with fractional error
 'everywhere less than 1.2 x10^7
 Dim t As Double, z As Double, ans As Double
 Dim tmp As Double, tmp2 As Double, tmp3 As Double
 z = Abs(X)
 t = 1 / (1 + 0.5 * z)
 tmp = (1.48851587 + t * (-0.82215223 + t * 0.17087277))
 tmp2 = (-0.18628806 + t * (0.27886807 + t * (-1.13520398 + t * tmp)))
 tmp3 = (1.00002368 + t * (0.37409196 + t * (0.09678418 + t * tmp2)))
 ans = t * Exp(-z * z - 1.26551223 + t * tmp3)
 If X < 0 Then ans = 2 - ans
 erfcc = ans
End Function
Function betai(a As Double, b As Double, X As Double) As Double
'Returns the incomplete beta function Ix(a, b).
'float betacf(float a, float b, float x);
'float gammln(float xx);
'void nrerror(char error_text[]);
```

```
 Dim bt As Double
 If X < 0 Or X > 1 Then
   MsgBox "Bad x in routine betai"
   Exit Function
 End If
 If X = 0 Or X = 1 Then
    bt = 0
   Else
    bt = Exp(gammln(a + b) - gammln(a) - gammln(b) + a * Log(X) + b * Log(1 - X))
   'Factors in front of the continued fraction.
 End If
If X < (a + 1) / (a + b + 2) Then 'Use continued fraction directly.
   betai = bt * betacf(a, b, X) / a
  Else 'Use continued fraction after making the symmetry transformation.
   betai = 1 - bt * betacf(b, a, 1 - X) / b
End If
End Function
Function Min(v1 As Variant, v2 As Variant) As Variant
 If v1 > v2 Then Min = v2 Else Min = v1
End Function
Function Max(v1 As Variant, v2 As Variant) As Variant
 If v1 > v2 Then Max = v1 Else Max = v2
End Function
Function StDev(sum As Double, SS As Double, n As Long) As Double
   StDev = Sqr((SS - ((sum ^ 2) / n)) / (n - 1))
End Function
Function Variance(sum As Double, SS As Double, n As Long) As Double
   If n > 1 Then
```

```
    Variance = (SS - ((sum ^ 2) / n)) / (n - 1)
  Else
    Variance = 0
 End If
 End Function
```

APPENDIX B: Excel Results for Simulations

Excel sheet attached

FOOTNOTES

1
v
1

[1] As part of my agreement with deCode Genetics and its' C.E.O., Kari Stephanson, all of the analyses had to be completed in Iceland. No data is allowed to leave the deCode system. As a result, most of the charts and graphs were saved as .pdf files in Iceland and could not be altered once I left the island. The only exceptions are some charts and figures that do not contain data from Íslendingabók, such as simulations, or those that I copied the essential numbers into Excel and recreated the graphs myself.

Appendix C:

Iceland Fast, Perl script extracting variables from genealogy

---

```
#! /usr/bin/perl

use strict;

my @columns = qw ( person_id mother_id father_id sex yob yod lifespan fullsibs );

#this is our list of people. each one is indexed by his ID
my $people = {};
#this is our families list. this is indexed by mother_id+father_id.
#each entry is just a list of the kids.
my $families = {};
#the current person
my $person;
#the current family
my $family;
#our list of IDs
my @id;

sub buildgenealogy {
        # we take our input and try to build a genealogy.
        # 1) we add the indi ID to @id
        # 2) we create a hash, which we put in the $people hash, indexed by $id, above
        # 3) we populate that with values. we also add the sons_list and daughters_list arrays
        # 4) if this person has both a father_id and mother_id, we add him/her to a list in the "families" hash

        my @cols;
        my $familyid;

  #for each row in the file.
        while ( <> ) {
                chomp;
                @cols = split ",";

                $person = { person_id => $cols[0],
                        sex => $cols[1],
                        yob => $cols[2],
                        yod => $cols[3],
                        father_id => $cols[4],
                        mother_id => $cols[5] };

                $people->{ $person->{person_id} } = $person;

                push ( @id, $person->{person_id} );
                if ( $person->{father_id} && $person->{mother_id} ) {
                        $familyid = $person->{father_id}."+".$person->{mother_id};
```

```
                        $family = $families->{$familyid};
                        if ( $family ) {
                                #add this individual to his list of sibs
                                push ( @$family, $person->{person_id} );
                        } else {
                                $family = [ $person->{person_id} ];
                                $families->{$familyid} = $family;
                        }
                }

                $person->{lifespan} = $person->{yod} - $person->{yob} if ( $person->{yob} && $person->{yod} );
        }
}

sub countfullsibs {
        my $familyid;
        foreach ( @id ) {
                $person = $people->{$_};
                if ( $person->{father_id} && $person->{mother_id} ) {
                        $familyid = $person->{father_id}."+".$person->{mother_id};
                        $family = $families->{$familyid};
                        $person->{fullsibs} = @$family;
                }
        }
}

sub printresults {
        #print our header
        print join ( ",", @columns ) ."\n";

        #print each individual
        foreach ( @id ) {
                $person = $people->{$_};
                map { print $person->{$_}."," } @columns;
                print "\n";
        }
}

sub main {
        buildgenealogy ();
        countfullsibs ();
        printresults();
}

main();
```