Identification of the factors that determined fecundity in early Afrikaners

By

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under the supervision of

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Summary

Fecundity determines the magnitude and direction of evolutionary change. By analyzing traits correlating to human fecundity we may understand our own evolutionary roots, and the behaviour and choices that define modern reproduction. For this reason, studies that look at historical populations prior to the demographic inversion and the use of contraceptives have become popular. Very good genealogical records exist for the early Afrikaner population (1652-1820), but only a few studies have utilized this wealth of information, primarily because it is not available electronically.

My aim was to determine which factors affected fecundity in this historical population. The Afrikaner population had a very high growth rate, presumably because of ameliorated environmental conditions. Under high growth rates we can expect that the determinants of fecundity may differ from those found to be important in European populations.

I recorded fecundity and a number of predictor variables for 1138 first generation offspring (referred to here as B generation) of 517 founder individuals (A generation), as well as the B generation spouses. Data was compiled on nationality of origin; number of marriages (when sequential remarriage occurred); dates of birth and dates of various reproductive events (date of first and last child's birth/christening, wedding dates).

I analysed the data with a variety of approaches to estimate the effects of a number of predictor variables known to affect fecundity as well as a couple of new ones we propose. A few of the key findings are: a gender specific role for the effect of multiple marriages; a discrepancy in fecundity for the various nationality groups suggesting cultural inheritance of fecundity; a new fertility measure, the time lapse between marriage and the birth of first child, explained most of the variation in fecundity; and we found very limited evidence to support the idea that fecundity is heritable.

Declaration:

I, Heike Koberstein, declare that the dissertation, which I hereby submit for the degree MSc (agric) Genetics at the University of Pretoria, is my own work and has not previously been submitted by me for a degree at this or any other tertiary institution.

Signature:...

Date:...

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Chapter 1: Literature Review

1.1: Determinants of Human Fecundity

1.1.1: Introduction

Individual fecundity indicates the maximum potential genes that can be transmitted to subsequent generations. The distribution of fecundity therefore dictates the prevalence of phenotypic traits over time, and subsequently the direction and magnitude of evolution (Buss, 2007).

It would seem intuitive that, considering the above, selection over evolutionary time should result in human fecundity stabilizing at a maximal sustainable level, such that we become reproductively homogenous, with the maximum number of offspring produced that a couple can feed and support. Traits that negatively impact reproduction should be selected out of the population, and fecundity increasing traits should permeate. Reproductive traits such as age at first reproduction and interbirth interval should be fairly uniform across communities.

However, the complex interplay of factors influencing human reproduction, and our constantly changing environment complicate fecundity determination (Anderton *et al*., 1987; Pluznikov *et al.*, 2007) and ensures that evolutionary and behavioural biologists retain employment. Fecundity is a quantitative trait with a large behavioural element, strongly influenced by cultural and social norms, and its expression is further impacted by the effect of free choice.

Human fecundity varies greatly among individuals; from the highly prodigious (for example Ismail the Bloodthirsty, alleged to have fathered 888 offspring during his 40 year reproductive lifespan (Einon, 1998)) to the completely infertile (it is estimated that approximately that at least 5% of heterosexual couples are infertile (Belsey, 1976), with upper estimates in the range of 20-30% (Pinnelli, Di Cesare, 2005)).

Human choice drives the expression of human fertility. Fertile individuals may choose not to express their reproductive potential, aided by contraceptive innovations (Anderton *et al.*, 1987; Da Vanzo, Starbird, 1991), and supported by social and cultural views (Boone, Kessler, 1999; VanLandingham *et al.*, 1991). Subfertile couples may choose to reproduce utilizing the numerous breakthroughs in fertility treatments (Pinnelli, Di Cesare, 2005). While the innate fertility of couples is important in determining final offspring number, cultural, social and behavioural factors will determine how (and if) this fertility is expressed.

We are interested in the interplay of those factors that influence both the natural fertility and the will of the individuals to reproduce. The size and directionality of their effects can give us a greater understanding of the evolutionary history of human reproduction (Bribiescas, 2006), as well as an indication of future and current trends in reproductive biology. Our aim is to quantify a number of the key traits in order to create a statistical model. In figure 1 the directionality and interaction of various traits influencing human fecundity has been illustrated. It sets a framework for the analysis of human fecundity and the factors influencing both the ability and the will to reproduce.

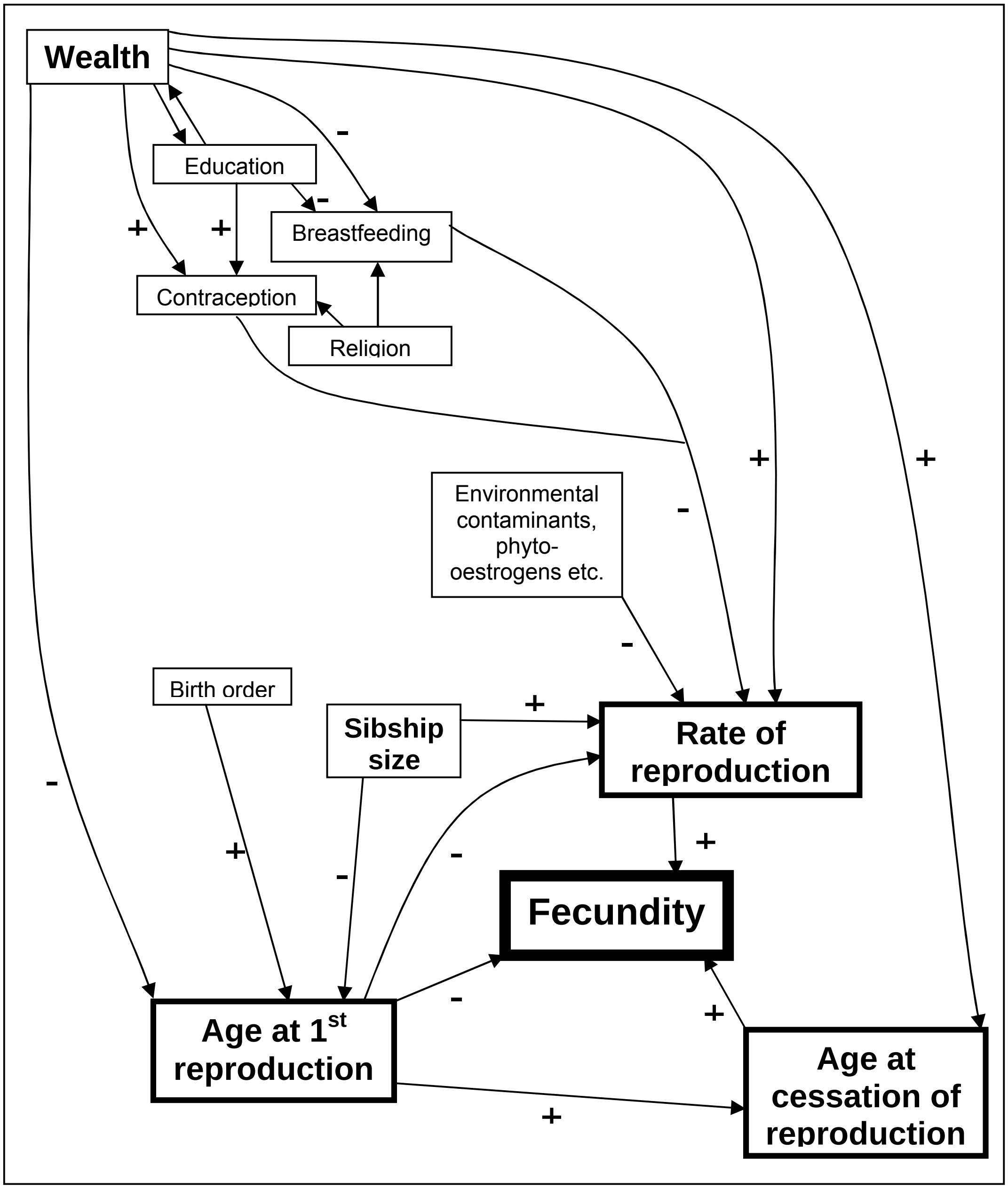

Figure 1: Flow diagram of factors influencing human fecundity and the direction of the influence

Various aspects of reproductive behaviour (Bereczkei, Csanaky, 1996) and their effect on human fecundity have been catalogued to date. These include practices like family planning and contraception (Da Vanzo, Starbird, 1991; VanLandingham *et al.*, 1991), the transmission of cultural ideals, for example religious beliefs (Sheps, 1965) and the role of family within society (Apostolou, 2007; Brown, Pimm, 1985), wealth transmission (Cronk, 1995; Reynolds, 1984; Weir, 1995) and social ideals (Bisin, Verdier, 1998).

Additionally attempts have been made to discover the heritability of fecundity (Fisher, 1929) and to identify and classify genes that regulate human reproduction (Achermann *et al.*, 1999; Bondy *et al.*, 1998; Habiby *et al.*, 1996) have increased our understanding of the biology of human fertility.

The impact of environmental factors, for example diet (Arakawa *et al.*, 2006; Mendiola *et al.*, 2008), nutrition (Anderson, McCabe, 1977; Castro, Alvina, 2002; Norman, Clark, 1998), activity level (Rich-Edwards 2002), and exposure to various compounds (Buck *et al.*, 2002; Mendola *et al.*, 2008; Waring, Harris, 2005) have also been investigated.

All of these factors have been utilized alongside anthropological studies into human evolution in order to uncover the evolutionary history of modern reproductive behaviours (Buss, 2007; Hawkes *et al.*, 1997; Holland Jones, 2009)

1.1.2: Human Fertility

1.1.2.1: Heritability of Fecundity

Selection and Fitness traits: Can Fecundity be heritable?

Life history traits, like fecundity, longevity and morbidity, tend to exhibit trends across generations within individual families (Kruuk *et al.*, 2000; Pettay *et al.*, 2005).

Heritability estimates for human fecundity range in value from low to highly heritable (see table 1). As fitness traits, such as fecundity, would assumedly be under severe selection, it may be argued that this selection should eventually diminish the genetic variation present for these traits (Kruuk *et al.*, 2000). This implies that eventually all observed phenotypic variation in these traits would be the product of environmental variation (discounting for rare genetic mutations), with heritability therefore equal to zero (Falconer, 1989; Kruuk *et al.*, 2000). This supports the findings of Gustafsson (1986) and Falconer (1989), who both found that traits determining fitness had the lowest heritability estimates.

Conversely, some researchers argue that genetic variation for these fitness traits is not entirely lost, for example Reed and Frankham (2003) found genetic variation to be responsible for 19% of the observed phenotypic variation in fitness traits (measured traits were total number of offspring surviving to adulthood, or population growth rate) in a number of different target species.

Heritability estimates in humans tend to average at between 0.3 and 0.4. This estimate is far higher than heritability estimates for other fitness traits, values not significantly different from zero were obtained in other studies for adult lifespan (Gustafsson, 1986 (in the collared flycatcher); Kruuk *et al.*, 2000 (in male red deer); Pettay *et al.*, 2005 (in humans)) and offspring survival (Pettay *et al.*, 2005).

Factors Influencing Heritability Estimates

Research into the heritability of human fecundity is complicated by the various factors that can influence overall fecundity. Observed correlations in intergenerational fecundity may be due to direct genetic inheritance of fertility, but could equally be the effects of shared environmental influences (for example wealth), cultural and social views regarding fecundity influencing factors (for example age at marriage (Anderton *et al.*, 1987), ideal family sizes (Anderies, 1996; Berent, 1953; Kantner, Potter, 1954), mate choice (Apostolou, 2007) and social status and education (Anderton *et al.*, 1987; Bisin, Verdier, 1998; Boone, Kessler, 1999; Ihara, 2008).

Modern studies of human fecundity are further confounded by measures employed to control natural fertility, for example the use and efficacy of contraception, and induced abortions (Anderton *et al.*, 1984; Anderton *et al.*, 1987; Da Vanzo, Starbird, 1991). Heritability estimates will therefore differ depending on the significance of such shared environmental factors. (For further discussion on these topics see sections on cultural inheritance and behavioural and social factors).

Methods to Determine Heritability

The phenotypic trait of fecundity is a reflection of the genotypes and environmental influences acting on two independent individuals. Sub-optimal fertility of either partner will negate the potentially high fecundity potential of the other. This means that heritability of fecundity of a single individual cannot be determined without including the partner's fertility.

The most common regression utilised for this purpose is maternal-line correlations (Austerlitz, Heyer, 1998; Helgason *et al.*, 2003; Imaizumi *et al.*, 1970; Pluzhnikov *et al.*, 2007) where female fecundity is correlated to the fecundity of their mothers. The maternal contribution of fecundity has been shown to be more significant than paternal, but father-son regressions are also often performed in addition to mother daughter studies.

A few studies have utilised pedigree information of more than two generations to estimate the heritability of fecundity (Pettay *et al.*, 2005; Pluzhnikov *et al.*, 2007), comparing up to four generations' fecundity. Their findings are similar to values estimated using simple offspring on parent regressions, implying that accurate estimates of heritability can be obtained from two generation pedigrees.

Table 1: Heritability Estimates of Human Fecundity

Estimate	Reference
0.4*	(Fisher, 1929)
0.34	(Madrigal *et al.*, 2003)
0.27[a]	(Pluzhnikov *et al.*, 2007)
0.38	(Berent, 1953)
0.22	(Kantner, Potter, 1954)
0.12	(Imaizumi *et al.*, 1970)
0.306±0.097[b*]	(Pettay *et al.*, 2005)
0.563±0.206[c*]	
0.161	(Austerlitz, Heyer, 1998)

[a] Value averaged using values calculated for males and for females
[b] Calculated using all individuals of the study
[c] Calculated using a censored sub group
* Values determined for females

1.1.2.2: Genes Affecting Fertility

Projects such as the human genome project (Cooper, 1994) have increased our awareness of the genetic origins of various characteristics of human life. Through the elucidation of specific gene functions, a large number of genes have been shown to have fecundity related functions (see table 2 for examples). This supports the idea of, at least partial, genetic heritability of fecundity. Clearly any genotypic trait that leads to complete infertility will not be transmitted to subsequent generations, unless it is a recessive gene.

It is estimated that approximately 5% of couples are infertile, although infertility rates in certain regions may be as high as 30-50% (Belsey, 1976). A large contribution to human infertility may be ascribed to environmental influences, for example venereal diseases and chemical exposure may drastically reduce fertility within communities (see section on environmental influences). Still the effect of the numerous genetic variants causing infertility or sub-optimal fertility illustrates that unfavourable alleles may persist in a population. These traits should be selected against, and should therefore be at a low frequency within the population. It may then be asked, why would mutations that decrease fecundity persist in a population (Westendorp *et al.*, 2001)? In the following sections we shall discuss potential explanations.

Table 2: Single Genes Affecting Human Fertility

Gene/haplotype	Position	Effect/role	Reference
Haptoglobin (Hp)		Hp*1/*1 genotype reproduce at younger age and have higher natural fertility potential, and had greater number of multiparous births	(Bottini *et al.*, 1999; Gloria-Bottini *et al.*, 2007)
ACP1 (cytosolic low-molecular-weight phosphotyrosine phosphatase		Carriers of low-activity ACP1 polymorphism better adapted to cold climates, able to reproduce in adverse conditions	(Gloria-Bottini *et al.*, 2007)
RIP140 (receptor interacting protein, 140kDa)		Essential for ovulation	(Parker *et al.*, 2003; White *et al.*, 2000)
INSL3		mutations result in undescended testis (rare cases)	(Ferlin *et al.*, 2003)
DAZL (deleted in azoospermia (DAZ) - like)	chromosome 3	role in spermatogenic failure in infertile man	(Teng *et al.*, 2006)
DAZ gene family		Azoospermia or oligozoospermia	(Ferlin *et al.*, 2004)
5 α reductase		male pseudohermaphroditism	Ivarsson *et al*. 1987 (Cohen-Kettenis, 2005)
FSHB		Haplotype associated with conception in females	(Grigorova *et al.*, 2007)
Oviductin gene (*MUC9*)		Protection of embryo and fallopian tube	(Lapensee *et al.*, 1997)
Azoospermia factor region (AZF) a, b & c		Deletions resulting in loss of spermatogenesis	(Li *et al.*, 2008)
KAL1	Xp	Kallman syndrome: recessive idiopathic hypogonadotrophic hypogonadism (IHH) in males. No effect in females	(Franco *et al.*, 1991; Legouis *et al.*, 1991)
GnRHR gene		Hypogonadism, chryptoorchidism (bilateral), absence of puberty, amenorrhea	(Caron *et al.*, 1999; de Roux *et al.*, 1999; de Roux *et al.*, 1997)
DAX1	X	Hypogonadotrophic hypogonadism	(Guo *et al.*, 1995; Habiby *et al.*, 1996; Iyer, McCabe, 2004)
FSHb gene		Males with delayed puberty and low testosterone	(Phillip *et al.*, 1998)
Leptin gene		Absence of puberty, hypogonadism	(Clement *et al.*, 1998; Ozata *et al.*, 1999)

LHb gene		pubertal delay, bilaterally small descended testes, low testosterone, and raised gonadotrophins, infertility in females	(Liao *et al.*, 1998; Lofrano-Porto *et al.*, 2007)
PROP1		Combined pituitary hormone deficiency. Absence of puberty or delayed puberty	(Cogan *et al.*, 1998; Fluck *et al.*, 1998; Fofanova *et al.*, 1998)
Diaphanous 2 (DIAPH2) gene	X	Ovarian failure	(Bione *et al.*, 1998)
FMR1	X	Fragile X syndrome. Carrier females at increased risk of premature ovarian failure	(Marozzi *et al.*, 2000)
DMRT1 & 2	9p	Sex-reversal (XY females)	(Faurie *et al.*, 2006)
SRY	Yp	Female phenotype despite male genotype, lack of puberty	(Jager *et al.*, 1990)
LHCGR		Infertility in both males and females: undermasculinisation or pseudohermaphroditism of males and amenorrhea of females	(Kremer *et al.*, 1995; Martens *et al.*, 1998; Misrahi *et al.*, 1997)
FSHR		Hypergonadotrophic ovarian dysgenesis	(Aittomaki *et al.*, 1995)
CYP17		Incomplete sexual development of males and females	(Costa-Sontos *et al.*, 2004)
NR5A1 gene		Sex reversal (XY females)	(Achermann *et al.*, 1999)
GALT (galactose1phosphate uridyltransferase) gene		Galactosaemia. Causes premature ovarian failure in 2/3 of affected woman, no testicular failure in affected men.	(Kaufman *et al.*, 1979)
CYP19		Incomplete sexual development of males and females	(Ito *et al.*, 1993)
FOXL2		Premature ovarian failure	(Crisponi *et al.*, 2001)
CFTR (cystic fibrosis) gene		Infertility in males due to congenital bilateral absence of the vas deferens	(Mercier *et al.*, 1995)
WT1/SOX9		Sexual ambiquity in males	(Barbaux *et al.*, 1997; Foster *et al.*, 1994; Pelletier *et al.*, 1991)
HOXA13		Uterine abnormalities resulting in pregnancy loss	(Mortlock, Innis, 1997)

Mutation/Selection Model

One possible explanation for the persistence of fitness reducing genes within the population is the mutation/selection equilibrium, whereby genetic traits that decrease fitness are selected against at a rate matching their influx into the population through de novo mutations (Jain *et al.*, 2009). Mutation rates in humans vary across regions of the genome, but most studies utilize the value determined in the haemophilia gene (the rate of 1 mutation in 50 000 life cycles (Haldane, 1935)) as a fair estimate. Mutation rates tend to be higher in males than in females (Crow, 1997), as a result of the greater number of cell divisions that occur during male gamete formation.

If selection does not exceed reported mutation rates, these deleterious genes will remain in the population. Additionally deleterious recessive alleles can remain “hidden” in the heterozygote, decreasing the efficiency of selection.

Inbreeding leading to inbreeding depression will bring previously unexpressed recessive alleles to the fore. This may result in fecundity reducing mutations being expressed in the offspring that are not expressed in either parent, with a resulting decrease in fecundity of highly inbred individuals (Ober *et al.*, 1999). Recessive alleles reduce the selection coefficient, allowing mutations to persist, and inbreeding acts to bring these mutations to the fore.

However, a degree of inbreeding has been shown to be favourable for fecundity (Schull *et al.*, 1970; Schull, Neel, 1972), with degree of consanguinity correlated to increased number of live births. It has, in fact, been shown that consanguineous reproduction between third or fourth cousins results in the greatest reproductive success (Helgason *et al.*, 2003). However, this recorded increase in fecundity may be explained by cultural and social effects, for example decreased age at marriage and education (Bittles *et al.*, 2002).

Additionally, some genetic disorders exhibit incomplete penetrance. It may be the case that while a parent has a dominant fecundity reducing genetic trait, he or she may not manifest the phenotypic effect, and therefore may pass a fecundity reducing gene on to his or her offspring, while remaining fecund. An example of a penetrance dependent genetic disorder is the congenital bilateral absence of the vas deferens associated with mutation of the cystic fibrosis transmembrane conductance regulator gene (Groman *et al.*, 2004). This mutation is found in approximately 10% of the population, and approximately 40% of men with the mutation are healthy and exhibit normal fertility due to incomplete penetrance. In the case of incomplete penetrance, the selection coefficient is reduced, and although the mutation is selected against when full penetrance is expressed, it persists due to incomplete penetrance occurring.

Antagonistic Pleiotropy

A number of the genes proven to have deleterious effects on human fecundity may be maintained within populations due to positive effects conferred by the alleles on other life history traits, for example a gene that extends longevity and decreases morbidity, at the cost of a decline in fecundity (Westendorp, Kirkwood, 1998). This is an example of antagonistic pleiotropy. Antagonistic pleiotropy may also result in genetic traits conveying different phenotypes in different individuals, leading to increased fitness when present in one individual, and decreased fitness in another (Jain *et al.*, 2009). For example differential expression of genes between the sexes may result in genes increasing fitness in one sex, while decreasing fitness (or possibly resulting in infertility) in the other sex.

An example of antagonistic pleiotropy has been suggested to explain male homosexuality, where for example, genetic markers on the X-chromosome (Hamer *et al.*, 1993) associated with increased probability of homosexuality in males (with subsequent low fecundity), have been indicated to potentially increase the fecundity of female relatives (King *et al.*, 2005).

Genotype by Environment Interactions

Environmental interactions may result in specific genotypes increasing fitness in one environment, and decreasing, or having no discernible effect on phenotype, in a different environment (Bottini *et al.*, 1999; Gloria-Bottini *et al.*, 2007). This may lead to deletions being maintained under specific environmental conditions, which would have otherwise been selected against.

Genetic Interactions

Deleterious genes may also be maintained in the population by the action of overdominance, i.e. where genes that reduce fitness when present in a homozygotic state, may lead to a fitness gain if present as a heterozygote (Jain *et al.*, 2009).

1.1.2.3: Environmental Influences on Human Fertility

Environmental factors have long been known to influence fecundity (Louis *et al.*, 2008), as well as other fitness traits. The environmental factors cover a broad range, from climate and natural phenomena, to chemical contaminants, mutagens and teratogens. Human beings are continuously exposed to fertility affecting compounds, and daily lifestyle choices (for example smoking, alcohol consumption, the use of recreational and medical drugs, diet and career choice) impact on individual ability to produce viable offspring.

Nutrition and Diet

Nutrition has a significant influence on the ability to conceive and carry offspring to term, as well as impacting the likelihood of perinatal mortality (Anderson, McCabe, 1977; Norman, Clark, 1998). The influence of nutritional status on fecundity is under stabilizing selection in that individuals at either end of the spectrum (both over- and under-weight) experience a decline in fecundity, and an increase in reproductive disorders. This effect was demonstrated by Rich-Edwards (2002) where a U-shaped curve shows an increase in reproductive disorders in both underweight (BMI<20) and overweight (BMI>25) women.

Body fat reserves play a role in the endocrine system, converting steroid hormones to active derivatives, androgens to oestrogens, acting in the metabolism of androgens and oestrogens and storing steroid hormones (Kirschner *et al.*, 1982; Norman, Clark, 1998). A minimal amount of fat tissue is therefore necessary for the onset of puberty in young girls, and for the maintenance of fertility (Frisch, McArthur, 1974). Additionally, if the body fat reserves become too large hormonal imbalances may result (Abbott *et al.*, 2005; Kirschner *et al.*, 1982; Norman, Clark, 1998; Pasquali, 2006; Pasquali, Casimirri, 1993; Pasquali *et al.*, 2003; Rich-Edwards *et al.*, 2002).

Underweight individuals do not have sufficient fat stores to produce and metabolize the steroid hormones required for fertility, and may cease ovulation. Low calorie intake, even in individuals with fat levels over the minimal requirement to maintain ovulation, results in poor reproductive outcomes, indicating that for optimal reproductive success a female should be in a positive energy balance. Underweight women (with body mass index of less than 19) have been shown to take four times as long to fall pregnant as women with a healthy body mass index (Hassan, Killick, 2004). They are also 3.5 fold more likely to suffer from subfertility (Hassan, Killick, 2004). Sufficient nutritional reserves are essential not only for ovulation and conception, but also for maintenance of pregnancy and survival of the offspring.

The results of studies analyzing the impact of obesity on female fertility are varied, with a number of studies claiming no fertility loss for over-weight women (Howe *et al.*, 1985). However, many studies have indicated numerous reproductive disorders that are prevalent in over-weight women (Fedorcsak *et al.*, 2004; Kirschner *et al.*, 1982; Lake *et al.*, 1997; Pasquali, 2006; Pasquali *et al.*, 2003), and that overweight (body mass index over 25) and obese (body mass index over 30) women suffer from more menstrual problems than their average weight counterparts, have less success with assisted reproduction, and are at a greater risk for miscarriage (Norman, Clark, 1998). Reproductive disorders may result from the action of the leptin protein (produced by fat cells), 16-hydroxylated oestrogen (product of the metabolism of oestrogen, active in obesity) or sex-hormone binding globulin (Norman, Clark, 1998). Increased hormone production in obese individuals has been associated with an increased incidence of reproductive cancers (Kirschner *et al.*, 1982). Obesity in women has been associated with an increased incidence of polycystic ovary syndrome (PCOS) (Fedorcsak *et al.*, 2004; Pasquali, Casimirri, 1993), resulting in decreased fertility and masculinization of these women, as well as increased incidence of miscarriage. Overweight and obese women have greater difficulty with assisted reproduction, in a study it was shown these women required higher doses of FSH (up to 14% longer treatment), had a higher proportion of early pregnancy losses (7.8% compared to 4.6% for normal weight women), and a lower live birth rate (3.9% lower compared to normal weight women) (Fedorcsak *et al.*, 2004).

Additional health impacts of obesity, for example diabetes and hypertension, further decrease reproductive success, and women who were overweight before falling pregnant have a greater risk of suffering from various pregnancy related diseases, e.g. hypertension, toxaemia, gestational diabetes, sleep apnea, asthma, urinary infection and macrosomia (Castro, Alvina, 2002; Galtier-Dereure *et al.*, 2000; Norman, Clark, 1998), as well as greater offspring mortality and incidence of still-birth (Sebire *et al.*, 2001).

Obesity increases the incidence of erectile dysfunction in males (ED) (Derby *et al.*, 2000; Shiri *et al.*, 2004) with a rate ratio of 1.7 (Shiri *et al.*, 2004), as well as leading to feminization as a result of over-production of oestrogen and reduced testosterone production (Pasquali, 2006). Sedentary lifestyle, commonly associated with obesity, further increases the risk of erectile dysfunction in men (Derby *et al.*, 2000). Obese men may improve their sexual function by losing excess body weight and instituting healthy lifestyle choices (e.g. exercise, balanced diet, non-smoking) (Esposito *et al.*, 2004), although for the best benefit these changes need to be implemented early in life (Derby *et al.*, 2000).

In addition to nutritional status, consumption of specific foods has been correlated with fecundity effects. High intake of animal products, e.g. meat and dairy, has been shown to decrease semen quality, and hence male fertility, while certain fruits and vegetables (for example peaches, apricots, tomatoes and lettuce) may facilitate normal reproduction in males (Mendiola *et al.*, 2008). Fish consumption by females has been implicated in possible decreased fertility, likely due to the presence of organochlorine in fish (shown to disrupt normal reproduction) (Buck *et al.*, 2002; Mendola *et al.*, 2008). However, results of studies are inconclusive (Arakawa *et al.*, 2006).

Phytoestrogens

That a number of plants contain oestrogenic compounds with the potential of interacting in the mammalian hormonal system has long been known (Bradbury, White, 1954). Compounds found in specific plants have been associated with induced infertility in a number of domesticated species (Kurzer, Xu, 1997), and have been implemented in human reproductive disorders (Kurzer, Xu, 1997; Skakkebæk *et al.*, 2006). Reproductive changes may result from the consumption of phytoestrogens, for example consumption of soy protein (containing isoflavones) by premenopausal women in a study delayed menstruation and suppressed the midcycle surges of LH and FSH and increased oestrogen concentrations (Cassidy *et al.*, 1994). Alternatively, exposure to phytoestrogens in utero may lead to changes in the reproductive system and subsequent fertility disorders, especially in male foetuses (Skakkebæk *et al.*, 2006).

Alcohol, Caffeine and Cigarettes

It is known that alcohol intake during pregnancy has teratogenic effects, leading to the spectra of foetal alcohol syndrome (Streissguth *et al.*, 1980). It has been postulated that alcohol may also dampen reproductive success prior to conception by reducing fertility. Alcohol consumption by women decreases the likelihood of conception per menstrual cycle by up to 50 % (Hakim *et al.*, 1998) and increases the risk of infertility by 50% (in high consumers) (Eggert *et al.*, 2004; Grodstein *et al.*, 1994). Infertility in these cases was as a result of ovulatory factor or endometriosis, the prevalence of which is elevated among women who consume 100g or more of alcohol per week (equivalent to approximately one drink per night) (Grodstein *et al.*, 1994). While a correlation was not discovered between alcohol consumption of males and infertility (Curtis *et al.*, 1997; Goverde *et al.*, 1995) excessive intake may exacerbate any existing fertility problems. Even though a fertility reducing effect has been found for alcohol in these studies, a number of studies have obtained contrary results, debating the role of alcohol in infertility (Curtis *et al.*, 1997; Olsen, Andersen, 1999). However, due to possible fertility reducing effects, as well as general health impacts, it is recommended that alcohol intake be limited when attempting to conceive (especially by females).

Excessive caffeine intake (seven or more cups of tea or coffee) is associated with a 1.5 fold greater probability of subfecundity (Hassan, Killick, 2004). Another study concluded that female coffee drinkers and males with a high tea intake had a slight reduction in fecundity (by 8% and 15% respectively-not associated with intake of other caffeinated beverages) (Curtis *et al.*, 1997). It is possible that the activity of caffeine on fecundity is to augment the effect of alcohol consumption (Hakim *et al.*, 1998).

Cigarette smoking has been linked to various health problems (Fageström, 2002), and also negatively influences fertility. Cotinine, a derivative of nicotine, is able to reach the ovarian follicles, where it reduces the ability of the oocyte to become fertilized (Hull *et al.*, 2000). Female smokers have an increased risk of infertility compared to non-smokers (odds ratio of 1.6 (Augood *et al.*, 1998)). Additionally smoking may lead to subfecundity, with increased time to conception intervals (this interval was doubled when women smoked more than 15 cigarettes per day, as compared with non-smokers (Hassan, Killick, 2004)). Even passive exposure to cigarette smoke has been linked to reduced fertility (Hull *et al.*, 2000), by delaying conception.

Industrial Contaminants

Environmental contaminants from industry and agriculture have become a major concern in recent decades, with the discovery of their role as carcinogens, teratogens, mutagens and endocrine disruptors. A number of compounds have been shown to reduce fecundity of wildlife (Guillette, Edwards, 2008; Gupta *et al.*, 2007; Harrison *et al.*, 1997; Sharara *et al.*, 1998; Waring, Harris, 2005). Studies on its reproductive effect in humans have been less clear, likely due to the complex mechanisms of these chemicals and inadequate study sample sizes (Sharara *et al.*, 1998), as well as the ethical implications that would arise from human experimentation. Certain environmental contaminants have been linked to significant decreases in fecundity, for example polychlorinated biphenyls (PCBs) exposure correlates to an increased time to conception interval (Buck *et al.*, 2002), and exposure to heavy metals (e.g. lead) is linked with pregnancy loss and delayed puberty (Mendola *et al.*, 2008).

The effect that chemical exposure has on the reproductive system varies between individuals, likely due to differences in susceptibility and the developmental points at which exposure occurs. This makes it difficult to quantify the effect size of environmental contaminants on fecundity. Endocrine disruptors have been blamed for various cancers of the reproductive systems of human males and females, reduced semen quality, and developmental impairments of children (Harrison *et al.*, 1997). Chemical contaminants that are present in the workplace environment in a number of occupations (for example the plastic and rubber industries, diethylstilbestrol formulation workers, dry-cleaners, the machine and metallurgic industry, agriculture, the textile industry, building industry and health services) negatively impact on the male reproductive system, lowering sperm count, causing abnormal sperm morphology, impotence and infertility, as well as being associated with increased miscarriage in their wives (Baranski, 1993). More research is required to determine the activity of various contaminants on the human reproduction, and efforts need to be made to limit environmental exposure.

Disease

The prevalence of disease, especially sexually transmitted disease, may result in wide spread infertility, or subfertility, in a community. In sub-Saharan Africa, for example, the prevalence of gonorrhoea infections in both males and females has been linked to high levels of infertility, with up to 40% of women exhibiting complete infertility in certain geographical regions (Belsey, 1976). Local climate may affect disease load in an area, as well as influencing the resources available for nutrition. Therefore in specific climatic conditions, reproductive rate will vary (Gloria-Bottini *et al.*, 2007), both as a result of disease load, and due to climatic adaptations.

Family Effects

Environmental factors may consist of more than simply the impact of the external environment on the individual. How environmental factors act on near-by individuals, especially those in the individuals' immediate family, may also influence their phenotype. The most influential of these is maternal effects, i.e. the intra-uterine environment during development and the nurturing provided during rearing. Numerous environmental influences may act upon the pregnant woman to reduce the fitness of her unborn child. Excessive androgen exposure in utero, as occurs with PCOS, causes female offspring to show hyperandroginism, masculinisation, and exhibit PCOS (Abbott *et al.*, 2005), the disease being transmitted from one generation to the next as an effect of the uterine environment rather than genes. Birth weight, largely determined by factors influencing maternal health, has also been attributed to later impacts on fertility. Low birth weight has been linked to numerous post-gestational health issues (Lummaa, Clutton-Brock, 2002; Ozturk *et al.*, 2001). In males, low birth weight has been associated with decreased fertility in adulthood due to hypergonadotrophic hypogonadism (Francois *et al.*, 1997). In females, low birth weight due to limited resources in utero, may induce metabolic and physiological changes that act to limit the size of her own future offspring (Barker *et al.*, 2000).

The family environment itself may also affect reproductive traits. For example the absence of a father in the family dynamic has been associated with precocious puberty in females, with the onset of menarche occurring almost one year earlier than the average for intact families, a more noticeable effect the younger the age of the female when the father left or died (Faurie *et al.*, 2006).

The age of onset of sexual activity was decreased, and the number of sexual partners increased (Figueredo *et al.*, 2005) in both sexes by the absence of a father figure (Faurie *et al.*, 2006). Mayan men raised without fathers are less willing to invest time and resources in nurturing a family (Waynforth *et al.*, 1998), forming unstable relationships and exhibiting promiscuous behaviour.

Birth order, and the individual's subsequent role in the family unit, correlates to certain personality and behavioural features. It has been shown to impact on the sexual strategy followed by individuals (Michalski, Shackelford, 2002). Firstborn offspring tend to take on more responsibility in the family, often helping to care for younger siblings (Brown, Pimm, 1985), and are more likely to mimic parental family dynamic (Michalski, Shackelford, 2002). According to research performed by Michalski and Shackelford (2002), firstborns tend to pursue long term sexual goals, commencing reproduction earlier and wanting a greater number of offspring, and later-born offspring follow short term sexual goals, seeking to amass greater numbers of sexual partners. Offspring may additionally exhibit fitness differences based on birth order and sex ratio. Offspring (regardless of sex) tend to be smaller when born after a brother as opposed to after a sister (Rickard, 2008). This is hypothesised to be due to the greater resources required to produce a son, and may have fitness impacts on the offspring as shorter individuals (of both sexes) exhibit lower fertility (Rickard, 2008).

Another possible consequence of birth order is the incidence of male homosexuality. It has been shown that later born males, with greater number of fraternal siblings, show increased incidence of homosexuality (Bogaert, 2004; McKnight, Malcolm, 2000), with every additional older brother increasing the probability of male homosexuality by 33% (determined from the population-wide probability of 2% chance of a male being homosexual, having no older brothers) (Bogaert, 2003; Bogaert, 2004).

1.1.3: Family Planning and Intent

1.1.3.1: Cultural Inheritance

Genes are not the sole unit of inheritance in human populations. Cultural traits, conditions and beliefs are transmitted from one generation to the next in human families, in a mechanism that resembles that of genetic inheritance, i.e. cultural inheritance. Cultural inheritance is defined by Cavalli-Sforza and Feldman as "the transmission from generation to generation of information", viewed as either all of the information that is transferred between generations, or the rules that control the mechanism of information transfer (Cavalli-Sforza, Feldman, 1973). The primary mechanism of cultural transmission is "vertical transmission" (Bisin, Verdier, 1998), whereby children develop cultural traits through observing and replicating behaviours presented by their family (immediate environment). Subsequent influences from non-relatives (e.g. peers, teachers and media) ("i.e. oblique transmission") (Bisin, Verdier, 1998) leads to broadening and cementing of individual views and preferences. Cultural inheritance leads to learnt behaviour and a change in future decisions, where the behaviour learnt is retained for future interactions, distinguishing it from copying (Brooks, 1998).

A number of non-genetic factors are commonly transmitted through families, for example the inheritance of wealth and assets, religious ideologies and views on the role of family and family values. These cultural factors influence various spheres of human behaviour and decision making, for example the assets of a family, along with transferred views on the role of family and the aspirations therein, will impact decisions regarding the number of offspring a couple wish to have, as well as intended timing of the births (Mace, 1996). As cultural factors such as wealth are transmitted down family lines through inheritance, reproductive strategy may be similarly transferred (as an effect of resource allotment and asset availability) (Mace, 1998), so that cultural inheritance may effectively inflate estimations of the genetic inheritance of fecundity. Anderton et al. (1987) expands on this by stating that the intergenerational correlations determined in offspring number may be "weak reflections of covariates of fertility such as status, education, and so on, which are also transmitted through the family" (Anderton *et al.*, 1987).

Status

Status (measured through numerous indicators (von Rueden *et al.*, 2008)) has been correlated to reproductive success in preindustrial human populations. The same correlation could be made for modern populations (were it not for the interfering effects of modern contraception) as it has been shown that high status males in industrial populations exhibit a greater potential fecundity due to a higher mating frequency (Hopcroft, 2006). Male status in pastoral communities affects the number of wives that a male may have (poygynous marriage), the number of offspring he produces, and the survival of these offspring (Borgerhoff Mulder, 2007).

Socio-economic status, e.g. the inheritance of wealth and/or social-status, is a culturally inherited trait shown to influence fecundity. It has been shown by previous studies that wealthier individuals (or landowners vs. non-landowners) have more children (in the study by Gillepsie et al. (2008) landless parents had on average 5.9 (±0.17) offspring, versus 7.53 (±0.19) for landowners ($p<0.01$)), and their children in turn are also more fecund (18.15 (±0.85) grand-offspring for landowners versus 12.25 (±0.7) for landless individuals ($p<0.01$)) (Gillespie *et al.*, 2008). Additionally, in this study, poorer individuals (non-landowners) exhibited a trade-off between offspring number and maternal fitness, with decreasing fitness corresponding to increased maternal fecundity (i.e. woman with higher offspring numbers tending to have fewer reproducing offspring, and hence fewer grand-offspring), a life history trade-off not evident among the wealthier land-owners in this study (Gillespie *et al.*, 2008). Poorer individuals commence reproduction later (determined in a Mayan sample), possibly as an effect of limited resources stunting reproductive development, or resource accumulation necessary in order for sexual selection (Waynforth *et al.*, 1998). In the study by Weir (1995) the fact that the wealthiest males produced the greatest number of offspring was attributed to the tendency of the wealthier males attaining younger wives.

In cases where the monetary or resource inheritance is a crucial aspect of offspring's future wealth (e.g. in pastoral societies where the inheritance of stock or land is necessary for offspring livelihood), parental wealth will be a primary determinant of viable offspring number, as beyond a sustainable number, these offspring will be unable to marry and reproduce themselves, effectively diminishing the parents' inclusive fitness (Mace, 1996). Additionally when offspring number exceeds the maximum carrying capacity of parental resources the growth and productivity of resources such as animal stock or produce will begin to decline as a result of consumption exceeding production, eventually causing the family to become destitute (Mace, 1996). Where kin support can be relied upon, this problem can be buffered to an extent (Sear *et al.*, 2003), but the optimal tactic to maximise fitness still depends on resource availability (Borgerhoff Mulder, 2000). This may ultimately not maximise reproduction, but rather ensure that the offspring produced have a greater chance of survival, and of producing grandoffspring (Mace, 1996). The degree of kin support available may be a factor of family wealth, where wealthier families are more likely to offer support in caring for young, increasing the number of surviving offspring (Borgerhoff Mulder, 2007).

The influence of monetary resources impacts the fecundity of males and females differently. Males in higher income categories have greater reproductive success, as they are better able to provide for a larger family, and sexual selection ensures that higher status males are considered more attractive mates (Hopcroft, 2006). Inversely, female reproduction tends to be highest in the lower income categories, possibly as a result of differences in education, as well the balance between career and reproduction that often leads career orientated women (hence in the highest income categories) to pursue career advancement at the cost of increased reproductive effort (Hopcroft, 2006).

The correlation between wealth and fecundity is clearest in populations with large class divisions, where wealth is a direct indication of availability of resources required to sustain increased reproduction. In societies in which the eldest son inherits the largest stake in his parents' estate (i.e. that practice primogeniture) it will be expected that he will also inherit the associated fecundity increase, but his siblings who do not inherit may opt for different reproductive choices. This means that older sons' reproductive behaviour may more closely correspond to parental fecundity.

The correlation between wealth and fecundity may be a consequence of phenotypic correlations of anthropometric traits, for example wealthier women tend to have higher BMIs (body mass indexes) as a result of additional resources (Philipson, 2001; Zagorsky, 2005), and higher BMIs in women (within normal weight ranges, see section on environmental influences) have been linked to increased fecundity (Madrigal, Relethford et al. 2003).

An exception to the general trend occurs during demographic transitions, when wealthy individuals decrease their offspring number more rapidly than poorer individuals (Borgerhoff Mulder, 1998; Botev, 1990; Szreter, 1993).

Age at Marriage

Biological factors may also act as culturally influencing factors, for example the size of family of origin (i.e. sibship size) may influence the planned family size (Berent, 1953). The age at which parents marry may influence the age at which their offspring marry (Anderton *et al.*, 1987; Kantner, Potter, 1954), therefore influencing the age at onset of reproduction, and subsequently lifetime fecundity. In the study by Anderton et al. (1987) daughter's whose mothers were married in the youngest age group were approximately 1.2 years younger at marriage compared to the daughters of those married in the older group.

1.1.3.2: Behavioural and Social Factors

Social trends are transmitted through peers by the process of oblique transmission (described above), resulting in demographic trends that permeate entire societies. Social ideals differ between different communities and among cultural groups, with distinct geographical regions often being culturally homogenous. Over generations, social norms and prescribed behaviour may change, leading to socially divergent cohort groups. The form of these social values differs between communities, from loosely structured guidelines, as present in more diverse cosmopolitan areas, to strict laws in homogenous isolated communities.

Individuals are aware of the socially stipulated code of conduct, and feel pressure to behave in a manner that is acceptable under the prevalent social etiquette. Due to our complex social needs, humans prefer to follow the social norms in order to gain acceptance and as a means to improve their view of self (Bereczkei, Csanaky, 1996). By meeting socially compiled goals, individuals may maximize their inclusive fitness (Irons, 1979). This is only the case when the social norms reflect the environment, meaning that social trends evolve over time in much the same way that organisms evolve biologically, allowing accepted behaviors to correspond to maximal fitness outcomes (Irons, 1979). A number of socially accepted norms are adaptive, and have been shown to increase fitness due to a greater number of surviving offspring (Bereczkei, Csanaky, 1996), for example trends that alter the rate of reproduction (i.e. influencing the interbirth interval) under varied environmental contexts. Behaviours such as breastfeeding and postnatal abstinence may be practiced through subsequent generations, and have been shown to impact inter-birth interval, and hence final offspring count (Da Vanzo, Starbird, 1991). Although these practices increase the inter-birth interval, and therefore may decrease the number of additional offspring produced, they maximise the survival and fitness of current offspring, and therefore increase inclusive fitness.

A number of seemingly maladaptive behaviors, with fitness reducing properties, are socially transmitted. For example, the quest for social status often leads to a decrease in fecundity (see section on life history trade-offs for more detail) and yet may permeate a society (Ihara, 2008). Social paradigms govern views and usage of methods of fertility control, i.e. the methods and efficacy of contraception usage as well as abortion. In most modern societies a social trend towards acceptance of contraceptive practices, and improvements in their efficacy, has contributed to population declines as seen during the demographic transition (Anderton and Bean et al. 1984). Health care practices and sexual education are additional social factors that may influence reproductive behaviour; decreasing fecundity as educations diminishes the number of unplanned pregnancies. Conversely, lack of sexual education and health care practices may decrease fecundity, due to an increase in venereal disease, as evidenced by the demographic survey of sub-Saharan African women (Belsey, 1976).

Gender Roles

Social norms also dictate the allocation of gender roles within a community. Mate choice will be influenced by these socially enforced roles, e.g. in societies where males must protect the family unit from predator threat, and females supply most of the food, physically fit and large males will be selected for, while the females will be selected based on foraging ability (Hawkes *et al.*, 1997). In societies where males supply resources to support the family, then wealthier males (often older) will be more successful at acquiring mates, and females will be selected on cues indicating fertility, i.e. youth and health (Hopcroft, 2006).

Whether a community practices polygamy or monogamy depends on the communities view of gender roles and environmental cues (Low, 2005), and have distinct consequences on offspring survivorship. Polygyny is much more prevalent than polyandry, although both may be present in some communities (Low, 2005). Polygyny enables males to maximise their fecundity in societies where fewer men are eligible for marriage (Borgerhoff Mulder, 1990). However it also causes a decrease in female fitness, as less offspring survive per wife due to competition for resources and conflict among co-wives (Low, 2005).

Role of Offspring

The role that offspring play within a community will strongly influence reproductive decisions. In societies where offspring carry the sole burden of caring for parents in old-age, it is considered essential to produce sufficient offspring surviving into adulthood. The costs involved in raising offspring will impact sex ratios and total number of offspring (Mace, 1996). In societies with a high degree of social or kin support (for example welfare states) this cost is buffered, and individuals often have a greater number of offspring than they would be able to care for unaided. Additionally, when offspring share in the family labour, as in agro-pastoral and hunter-gatherer tribes (Hawkes *et al.*, 1997), the cost of increased fecundity is, at least partially, buffered.

1.1.3.3: Life History Trade-Offs

Quantity versus Quality

Optimal life histories are those that are able to maximise the average fitness (or per capita growth rate) of the population (Caswell, 1982), where fitness can be described as the product of survival and reproduction traits (Caswell, 1982). The life history theory states that in order for fitness to be maximised within a species, there will be a trade-off between the number of offspring born, and the number that survive (Penn, Smith, 2007; Strassmann, Gillespie, 2002). This trade-off is analogous to that described by Lack using altricial bird clutch sizes (Lack, 1954). Simply stated this theory implies that reproduction above a certain fecundity level results in decreased lifetime reproductive output (i.e. offspring that survive to a specified age). High fecundity as well as decreased birth spacing is linked with increased maternal mortality and decreased offspring quality (Penn, Smith, 2007). The implication of this trade-off is that, in order to maximise fitness, an intermediate level of reproduction is favoured. The idea of limiting fecundity in order to maximise the fitness of offspring in human populations is a quality-quantity trade-off (Gillespie *et al.*, 2008).

Additionally, in instances of high fecundity, surviving offspring will often not reproduce. This has been shown in the study by Gillespie *et al.* (Gillespie *et al.*, 2008), where increased fecundity was associated with decreased offspring recruitment (i.e. fewer offspring themselves subsequently reproduced). Resource allocation in large families may be insufficient to allow for all offspring to marry and reproduce (Mace, 1996). For the same reason, parents may attempt to curb fecundity (through the use of contraception, reduced polygyny and abstinence) to ensure that they produce only as many offspring as they can afford to raise, so that additional offspring do not influence the fitness's of preceding offspring (Borgerhoff Mulder, 2000).

The explanations given for the comparatively low fecundity of offspring from larger families are varied. It may be assumed that fewer offspring survive to reproductive age, due to the increased morbidity and mortality associated with large families (Gillespie *et al.*, 2008; Mace, 1996). However, even discounting offspring who die before coming of age, not all surviving (and fertile) offspring choose to reproduce.

One explanation is that these offspring may increase their inclusive fitness through kin selection, helping to rear younger siblings as well assisting siblings in rearing their own offspring (Brown, Pimm, 1985). For this to hold true the siblings of these non-reproductive individuals should exhibit greater than average fecundity, compensating for their direct fitness loss (Voland, 1998).

Another controversial explanation could be the incidence of male homosexuality observed in later born sons of large families (see paragraph under environmental effects). An evolutionary theory is that females with high natural fecundity are associated with increased incidence of homosexuality among male relatives (McKnight, Malcolm, 2000). Homosexual men produce 80% fewer offspring than heterosexual men; this is a trade-off between increased reproduction of mothers with decreased reproduction of her sons, possibly due to genetic antagonistic pleiotropy. Seemingly unrelated is the increased incidence of males entering into priesthood (hence choosing enforced celibacy rather than entering the reproductive population) reported in families with a greater than average number of sons (Deady *et al.*, 2006). The evolutionary explanation for this trend may in fact closely parallel that given for male homosexuality, whereby these sons confer a kin selection advantage to their brothers by removing themselves as competition for a mate, and by increasing assets available for his brothers' offspring (Deady *et al.*, 2006; King *et al.*, 2005).

The trade-offs discussed above are between offspring number and offspring fitness (Borgerhoff Mulder, 2000). The quality-quantity trade-off may also be in the form of a trade-off between current and future reproduction, where the physiological costs of reproduction (i.e. its impact on longevity and on future reproduction) is considered by the mating pair (Borgerhoff Mulder, 2000).

Longevity versus Fecundity

Rapid and early reproduction depletes energy stores, and therefore may reduce maternal fertility, and ultimately be related to increased mortality and morbidity for mothers (Voland, 1998; Borgerhoff Mulder, 2000; Penn, Smith, 2007). The disposable soma theory implies that resources utilised for reproduction reduce the availability of resources for somatic aging, and hence longevity (Kirkwood, 1977). The Life History Theory (Figueredo *et al*., 2005) presupposes that individuals will devote their resources to two major efforts, the somatic effort (involving the growth, maintenance and survival of the individual) and the reproductive effort (involving all factors involved in the production and support of offspring).

Individuals (and entire species) may be divided into two groups, dependent on the emphasis they place on these two efforts. *r*-Selected individuals focus more on reproductive efforts, while *K*-selected individuals invest more in survival (Figueredo *et al*., 2005). Research has indicated that individuals that have lower than average fecundity, or who abstain from reproducing completely, tend to live to an riper age (i.e. *K*-selected individuals) (Westendorp, Kirkwood, 1998). However this study has been refuted by subsequent research (Gavrilova *et al.*, 2004; Mueller, 2004; Westendorp, Kirkwood, 1998) due to errors in database compilation and computation, and therefore this theory requires further research to be performed.

Social Status versus Fecundity

An effect of modern times has led to a number of unique trade-offs, not recorded in traditional societies. These are trade-offs between reproduction and social status, career advancement and education. Status has been shown to be linked with increased fitness (Hopcroft, 2006). However, efforts to increase status (through education, wealth amassment or networking attempts) have fecundity diminishing effects (Boone, Kessler, 1999). The long term effect of this may be an increase in inclusive fitness, as by lowering the number of offspring produced, they increase the status of the individual offspring (through more intensive education etc.), which should theoretically lead to increased reproductive success of these offspring (Boone, Kessler, 1999; Waynforth *et al.*, 1998). However, in modern society these “high status” offspring will still experience the social drive to increase individual status (Ihara, 2008), and in effect may not fulfil these long-term fitness goals.

1.1.4: Evolutionary Roots of Fecundity

By investigating the role of evolutionary biology in modern reproductive behaviour researchers may unravel the evolutionary and fitness consequences of specific personality and behavioural traits (Thornhill, Gangestad, 1996). This means that seemingly inherent and individualist views on reproduction and mate choice may have evolved to allow adaptation to environmental and social contexts.

Sexual Dimorphism

Reproductive behavior and mate selection is central to the evolutionary process. But these behaviors are themselves the product of evolution. A key example is the evolution of sexual selection. In humans, as in many other species, males and females differ physically to a greater extent than which could be explained by divergent reproductive systems. Human males are, on average, 12 % taller than females (Buss, 2007), and the sexes show different sexual traits not entirely restrained by physiological needs, for example facial hair in males and the size of female breasts (compared to those of other primates). These dimorphic traits are adaptive cues, as individuals exhibiting them tend to possess other traits that maximize their overall fitness. For example bigger and stronger males tend to possess high testosterone levels, and produce more offspring (von Rueden *et al.*, 2008), and specifically more sons (Kanazawa, 2006). Similarly physically attractive woman (an indicator of health, and consequentially fertility) are more fecund (Jokela, 2009, Thornhill, Gangestad, 1996).

The sexes follow distinct social cues and adapt different roles in producing and rearing of offspring, as well as exhibiting different behaviours and attitudes when seeking and obtaining a mate (Thornhill, Gangestad, 1996). These differences are due to sexual selection, characteristics that have evolved and are crucial to the selection of superior mates, with whom fecundity, and hence fitness, may be maximized. Traits that led to increased reproductive success would be selectively favoured during evolution, inducing an increase in the frequency of such traits. This implies that sexually dimorphic traits, such as large breasts in females (as compared to other primates) and facial hair in males, may have initially been present in low concentrations in the human population, but due to the increased reproductive success of individuals exhibiting these traits, and the selective advantage this conferred, they would have increased in abundance (Buss, 2007).

Sexual selection and the traits favoured may differ somewhat among communities, but the principles governing it are universal (i.e. maximizing achievable fecundity and sustainability of the subsequent family). Due to the roles imposed on the sexes, the traits that they find attractive in the opposite sex reflect the role for which the sex has “adapted”.

For example, females show preference for wealthier men (Bereczkei, Csanaky, 1996) (see section on behavioural and social influences) which should ensure the greatest survival for her offspring (as a consequence of the male's resources supporting the family's growth). Male post-coital support of offspring, rare among mammals, has evolved in our species as a consequence of the extended childhood developmental period and the increased demand on parental resources instigated by human infants (Marlowe, 2000; von Rueden *et al.*, 2008). The paternal support improves the survival and fitness of the offspring (Lawson, Mace, 2009).

By seeking out physically stronger and more aggressive males, females are utilising evolutionary cues that developed to ensure protection of the family from predators and competitors. Similarly, males who seek mates younger than themselves, concentrating on physical attractiveness, and especially typical feminine characteristics (e.g. large breasts, wide hips and small waist size) are seeking out those who display the highest cues of fertility (Thornhill, Gangestad, 1996).

An additional evolutionary artefact of these preferences for sexual selection is that these individuals are likely to display high immuno-competence (Thornhill, Gangestad, 1996). This is a consequence of the immunity suppressing quality of the hormones testosterone and oestrogen. Physical prowess in males results from increased testosterone levels, while in females higher oestrogen leads to more feminine appearance. It may be assumed that individuals exhibiting these characteristics would have high immune-competence to allow them to survive (Thornhill, Gangestad, 1996).

The Evolution of Mating Systems

The long-term monogamous mating system practiced by humans has evolved in order to support the requirements imposed by the extended nurturing period of human infants (Marlowe, 2000). However this is not the only mating system to have evolved in human populations, despite its obvious merits in human society.

Monogamy generally ensures that sufficient paternal support is given to improve offspring survival, and also allows more accurate paternal identification. Polygyny as a reproductive strategy is still comparatively common, occurring in approximately 75% of hunter-gatherer societies, while polyandry, less common, is still practiced in approximately 15% (Apostolou, 2007). These mating systems also have beneficial fitness consequences, for example, as discussed under the section on Behavioural and Social Factors, polygyny allows males to maximise their fitness, and polyandry may maximise fitness in societies where the resources of a single male are insufficient to support the offspring of a single woman, but where the pooled resources of two men may allow her to produce offspring at a higher level (Low, 2005).

Individuals may also elect to practice a short-term mating system. Short-term mating practices have greater evolutionarily benefits to males as opposed to females (Buss, 2007). A male participating in short-term mating behaviour has the potential to impregnate numerous women at a minimal cost to his resources (Buss, 2007). For a female however, the cost of pregnancy is far greater, and therefore she cannot produce as many offspring.

Alongside the evolution of stable mating strategies, practices such as infidelity, rape and mate poaching which may sabotage the benefits derived from monogamous mating, have evolved. These may have evolved simply as an offshoot of the universal quality of the traits governing sexual selection. More attractive individuals tend to attract more potential mates, and therefore have a greater likelihood of being in a relationship. These individuals will still attract the attention of those seeking a mate even when they are already in a relationship (Buss, 2007).

Menopause

One oddity of human life history is the extended post-reproductive life-span of women. Various hypotheses have been given to explain the evolution of this trait, the most popular of which is the grandmother hypothesis. The proposal is that post-menopausal women can increase their inclusive fitness by increasing the number of grandchildren that they have, after they are no longer able to reproduce themselves. They do this by assisting in the rearing and nurturing of their children's offspring (Hawkes *et al.*, 1998). By providing care and extra resources she allows her offspring the opportunity to produce more progeny than they could support alone.

Alternatively, the altriciality-lifespan hypothesis states that the high resource requirements of human infants, as well as the increased cost of pregnancy and lactation that is innate to our species (as a result of our erect stature and increased brain mass (Mace, 2000)) may favour early cessation of reproduction to allow continued investment in existing offspring (Scott Peccei, 1995a; Scott Peccei, 1995b). Later reproduction would entail too great a risk to be sustainable, the costs involved would likely deplete the mother before her offspring are sufficiently matured to care for themselves. Therefore this extended reproductive life would likely lead to reduced offspring survival and reduced total offspring number. This hypothesis differs from the grandmother hypothesis discussed above in that mothering as opposed to grandmothering is highlighted (Scott Peccei, 1995b).

The oocyte depletion hypothesis argues that women eventually run out of ova (Bribiescas, 2006). It is known that women begin life with a set number of oocytes, and therefore it is reasonable to consider that menopause may result from the depletion of these ova (Bribiescas, 2006).

The human lifespan has increased drastically due to the development of modern health care and the protection that our modern lifestyle has garnered us against natural threats, like predators and climate. During our evolution it was unlikely that women would have lived to the age of 50 years. As the post-reproductive lifespan would then be assumedly shorter, if women actually survived their entire reproductive life, it is suggested that the extended post-menopausal life-span of humans is simply an offshoot of our longer lifespan in general (Bribiescas, 2006).

Although our post-reproductive lifespan is unique to our species, reproductive senescence occurs in most mammalian species. In fact reproductive senescence as recorded for chimpanzees was shown to very closely coincide with that of humans (Thompson *et al.*, 2007). The core difference is that in most species reproductive senescence corresponds with general senescence, with the decline in fertility occurring simultaneously with a decline in survivability (Thompson *et al.*, 2007).

Optimal Reproductive Behaviour

As discussed in previous sections, evolutionary forces are expected to function such that optimal life histories result. However, as a result of the varying behavioural influences on fecundity, and the novel scenarios offered by modern society, it has become increasingly difficult for scientists to determine what these optimal life histories are. A possible option utilised by researchers to attempt to remove the modern influences from their analyses is to investigate reproductive traits of older communities, who were less exposed to modern influences, and socially homogenous communities, who share views on reproductive practices, and largely follow the same behavioural patterns (Eaton, Mayer, 1953). One way to do this is by investigating hunter gatherer communities, removed from modern social influences, who practice natural fertility.

These communities tend to share views on behaviours such as breastfeeding, post-partum abstinence, age at marriage (or first reproduction), birth spacing and preferences for family size. It has been hypothesized that without these external effects, communities should follow reproductive patterns that would maximise their overall fitness (Anderies, 1996; Borgerhoff Mulder, 2000). For example the !Kung community of Botswana exhibit a mean inter-birth interval of the women (± 4 years) (Anderies, 1996), shown to correspond to the interval that would maximise expected lifetime reproduction.

The advent of pastoral life is estimated to have occurred at most 10 000 years ago, before this point it is assumed that all communities were hunter gatherers (Hawkes *et al.*, 1997). Therefore hunter gatherer communities allow an understanding of our evolutionary past. However, modern day hunter gatherer communities have since culturally evolved from these initial civilizations, showing adaptation to modern conditions and influences, with some modern day hunter gatherer communities even having pastoral ancestors (Hawkes *et al.*, 1997).

1.1.5: Resolution between Different Views

Human fecundity is influenced by a large number of factors, both external and inherent, and the interplay of all these factors is responsible for the great variety of reproductive patterns exhibited by individuals. The key to understanding human fecundity is in the interplay of these factors.

For a couple to be fecund, both individuals require high innate fertility, resulting from advantageous genetic material, and lack of environmental contaminants. They need to both be of an optimal age, in good general health, and without fertility reducing vices. Their behaviour and culture needs to be such that they do not inhibit their natural fecundity through social practises such as contraception and abortion. They need to effectively time reproductive events to ensure maximal survival of their offspring, utilizing available resources. Additionally the couple should want to reproduce.

However, in modern society there may be a mismatch between optimal and actual behaviours due to genetic lag or the more rapid cultural evolution.

1.2: Inheritance of fitness in the Early South African settlers

The following was extracted from the books of Agar-Hamilton (1934), Giliomee (2003), Johnson (2004), Le May (1995), Ross (1993), Templin (1984) and Were (1974).

1.2.1: Historical Overview

The first Europeans to explore South Africa believed the land to be inhospitable, teeming with deadly wildlife and strange, hostile (possibly cannibalistic) natives.

A Portuguese ship led by Bartholomew Diaz alighted on South African shores in 1487, on route to the East to trade for spices. Another Portuguese ship carrying Vasco da Gama and his crew arrived in (and subsequently named) the province Natal In 1497. Neither of these voyages resulted in a permanent settlement, and it was not until April 1652 that South Africa accepted her first European colony.

The Dutch

The Dutch East India Trading Company (Vereenigde Oost-Indische Compagnie – VOC) chose to create a refreshment station in the Cape of South Africa, where traders could replenish their stocks on the long, arduous trip from Europe to the East Indian islands. This station was to be manned by Dutch company employees, led by Jan van Riebeeck. The settlement additionally had missionary aspirations, the faithful believing their journey to be blessed, and envisioning potential growth and expansion of the Christian Reformed church by spreading Christian beliefs into Africa.

The first permanent members employed to man the refreshment station were mostly single males, poorly educated peasants and labourers, who sought employment as sailors or soldiers. About eighty men arrived with van Riebeeck, a number which was gradually supplemented with fresh immigrations. The men soon became disheartened by their role as servants for the company, and it was decided that they would be emancipated from the company, and hence enjoy a degree of freedom in the new land, while remaining under the company's employ. Jan van Riebeeck officially requested the Company workers be considered free men and given their own plots of land.

In 1657 nine men were freed from servitude, and became the first "free burghers", with possession of their own farms. The transition to freedom was not as promising as it first appeared and these "free burghers" did not enjoy the sovereignty implied by this gesture. As employees they were still under the regimen of the company, forced to remain in the country under company employ for at least twenty years, expected to sell their produce to the company at fixed prices, and required to pay taxes to the company. Discontentment grew, but as the burghers' numbers were still too low, little could be done to fight the repressive control of the company and gain greater freedom.

Huguenots and Germans

In 1688 French Huguenots joined the Dutch settlers in the Cape, escaping from religious persecution in their own country following the revocation of the Edict of Nantes. They numbered almost two hundred, consisting mainly of families, and their arrival greatly boosted the colonists' numbers, giving them hope in their struggle against the company's regimen. However their arrival within the colony was not viewed as a completely positive development, as their numbers and differing cultural beliefs made the Dutch leaders anxious. The commander at the time, Simon van der Stel, became adamant that the settlement must remain Dutch. He ensured that Huguenot families were scattered throughout the existing colony so that they would be forced to adopt the Dutch language, and additionally outlawed the official use of French within the colony. Many of the French surnames were given a Dutch transformation, for example Colbert became Grobelaar and Villon became Viljoen.

In the 1700's German immigrants who had fled to Holland seeking employment, were recruited as soldiers and sailors by the VOC and joined the colonists in the Cape. The German colonists were seen as less of a threat to the Dutch heritage than the French had been as, being predominantly single males, they married Dutch (or French) women, and raised their children as Dutch.

The majority of the immigrants at this point were single males. The shortage of European women led to a number of formal and informal unions between European men and slave women (discussed further under "Genetic Contributors to the Afrikaner Race"). The company dispatched a number of orphaned Dutch girls to South Africa for these men to marry.

Slaves

In 1658, six years after the establishment of the Cape refreshment station, slaves began to be shipped in from West Africa. A slave market was established with slaves being shipped from Angola, Dahomy, Java, Madagascar, Mozambique, Batavia, Shri

Lanka and India. By the mid 18^{th} century at least 50% of burghers owned slaves, and the number of slaves in a household was an indication of social status.

Eventually slaves far outnumbered the European settlers. Whilst any indiscretion on the part of the slaves resulted in severe punishment, their vast numbers still made the Europeans nervous. A precarious balance was maintained between the incorporation of slaves as members of the master's "extended family", and mistrust coupled with a degree of contempt felt by both slave and master. Slave uprisings and financial instability did little to ease the minds of the burghers, and slavery was eventually abolished on 1 December 1838, Emancipation Day.

Afrikaners

The mid to late 18^{th} century was a comparatively peaceful time for the settlers, and allowed them to grow together as a community. The divergent European groups begun developing a more unified identity under the auspices of the shared Dutch language and the Dutch Reformed Church, and began to call themselves 'Afrikaners'. The merged culture that developed incorporated aspects of the different European nationalities involved, as well as traits adapted from the native Khoi San who lived in the area and the various slave nations who had become a part of Afrikaner family life.

The British

The peace was unfortunately not to last. In 1794 the Dutch East India Trading Company declared itself bankrupt, due in part to political and economic struggles brought about by the many lengthy wars in Europe. The following year British troops invaded, gaining a stronghold over the Cape Colony. There occupation was short lived, and by 1802 control of the East India Company was returned to the Netherlands, now the Batavian Republic.

After only four years British troops invaded once again, formally regaining control of the colony in 1814. The conversion from the Dutch system was slow, with few changes implemented until the 1820's, during which time currency based on British denomination was introduced and moves were made to change the official language from Dutch to English. Additionally they proposed allowing the slaves voting rights. The burghers were infuriated by the proposed language change and feared that, if given the right to vote, slaves would side with the British against them.

A number of British missionaries further infuriated the burghers by disturbing the racial hierarchy that had been laid in place. The Dutch settlers, following the teachings of their Reformed Church on predestination, believed that they (the Europeans) were members of the chosen race, and the non-believers (all non-Europeans) were members of the servant race.

British missionaries, led by Dr John Philip, fought for equality and fair treatment for the non-white population. Dr John Philip was considered accountable for the enactment of the 50th Ordinance, restoring civil liberties to non-whites. A number of Dutch missionaries joined in the fight for equality, to the disdain of the settlers. Foremost among these was Dr van der Kemp, a Dutch member of the London Missionary Society.

The Great Trek

Frustrated by political changes, and in search of a land that could be ruled under their own governance, where they could preserve their culture and believes, a group of burghers set off on a migration from the Cape region in 1835. The Great Trek, as it has since been called, led these trekboers through dangerous routes over unmapped territory, and brought them in contact with hostile factions. Many of the trekboers were killed, and a large number died from fever.

The travellers eventually found themselves in the province of Natal. After defeating the Zulu warriors in the Battle of Blood River, the Zulu leader Dingane granted the trekboers a large portion of Natal where they were able to establish their governance while retaining their unique cultural identity. Settlements were also established in the Provinces of Transvaal and the Orange Free State.

1.2.2: Social and Cultural Characteristics of the Early Settlers

Social and cultural factors experienced by the early Afrikaners were changing and developing alongside their history. During their colonization there would be different social factors affecting the various generations, and as an effect lifestyle choices varied with time. As the company's hold slowly diminished the servants of the company were able to form a social identify and unify as a people, and so the Afrikaner race was born.

Governmental influence was very weak, the company spending less and less time concerned with the policies of their new colony, and the people had developed a corresponding aversion to authority. Despite the scarce governance, order was strictly maintained within the colony and indiscretions severely punished. Corporal punishment was the prevailing trend, and, especially among the slaves, death by torture was a common penalty for the more severe crimes such as murder or rape.

The trekboer and the bourgeoisie

Expansion of the colony led to increased friction between the settlers and the native tribes, and violence was a familiar feature of everyday life on the frontier. The Free Burgher had at last found greater freedom, and those who remained as farmers (and didn't choose to become tavern owners or join the black market trading) expressed this freedom through his way of farming. The "trekboere" were born: cattle farmers who lived an almost nomadic life, disappearing on long hunting trips, and returning to a more simple way of life.

The society had diverged into two branches of people. On one side was the free-ranging "boer" of the plateau, on the other the settled bourgeoisie of Cape Town, whose lifestyle by comparison seemed almost aristocratic. The Cape Afrikaner lived very civilized lifestyles, building fine houses and grand churches, and filling their homes with beautiful furnishings, expensive finishing's and luxurious clothing.

On the other end of the spectrum, the "trekboer" lived in small, roughly built clay houses (often with just one small room for daytime use that would be shared with servants and animals) or else slept camped in their wagons. Due to their close contact with the Khoi servants the trekboer became in essence more "African" than the Afrikaners of the Cape, mimicking many of the mannerisms of the native Khoi.

Willem van der Stel introduced the concept of "loan farms", which facilitated the lifestyle of the nomadic boers. These farms could be rented for a small sum, and were hence easy to take occupancy in, and easy to once again leave. This allowed the trekboer to move easily to find new pastures, and also allowed all of the sons of the large boer families to settle on a loan farm (of approximately 6000 acres) for early marriage.

Religion and race

Despite the obvious differences in culture and lifestyle of the two Afrikaner groups, certain attributes united them in ideology. One was their strong religious conviction. All households had a family bible, which commonly would be the only book in the house. Their culture was built around the Dutch Reformed Church beliefs, and all of their social beliefs found credence in their religious teachings. The settlers in the Cape built grand churches, and the trekboer, despite his nomadic ways, brought a bible along on all their journeys, and sung psalms regularly. Visiting ministers helped facilitate marriages and christenings, which were often delayed for the minister's visit, and small, simple funerals would be conducted, when necessary, by the head of the household.

Another uniting factor was their racial awareness. Regardless of social standing and behaviour, the white settler remained an Afrikaner, and in this sense maintained a superior elevation in the social caste system. The European settlers believed that their role as the superior race had biblical support, seeing the native Africans as the "sons of Ham".

Family

Family life was of key importance to the burghers, and strong ties bound family members to one another. Children held enormous respect for their parents, following their advice and requests with an easy obedience, even as they matured into adults themselves. Parents could maintain control over the lives and choices of their children by retaining possession of their estate to death.

Compared to other colonies, the women of the Cape colony enjoyed a high status and the respect that went along with it. The wives of officials shared in the status of their husbands, and women could own their own farms, or acquire positions as a guest housekeeper or publican. Divorce was allowed within their church, and women could bring charges against their husbands for indiscretions, such as adultery, committed against their marital duties. On a husband's death the wife was bequeathed half of the estate, while the children shared the other half, thus allowing widows to gain riches through wise choices of remarriage.

1.2.3 Fecundity of the Early Settlers

The early Afrikaners were highly fecund, with their high reproductive rate contributing to steady population growth. This is illustrated by the increase in population from 2 540 "free burghers" in 1703 to 42 975 by 1820. This rapid growth was supplemented by new immigrations, but was also a consequence of the tendency to marry early and reproduce rapidly, with short birth intervals and reproduction extending later in life.

According to work performed by Ross (1993), the mean age at marriage for women was 21 years, with 53% marrying under the age of 20 years. Men tended to be older than their wives, although there was no clear observed age pattern for the men at marriage. These marital unions were almost always maintained until the death of one of the partners, divorce being very rare, but the remaining partner would often remarry.

Reproduction generally occurred rapidly after marriage, with the baptism of the first child occurring on average 18 months after marriage. The mean interbirth interval was recorded as 26 months. The age at cessation of reproduction is a trickier concept to analyze, as the shortage of recorded deaths means that it is not possible to determine the cause for the cessation. However the data suggests that these women continued to reproduce into their 40s.

A number of the social practices at the time were able to support the rapid reproduction of the settlers. For example, wet nurses were common in the settler households, and allowed a decreased interbirth interval due to the avoidance of postpartum lactational amenorrhea. Remarriage after the death of a spouse allowed the settlers to utilize the reproductive lifespan to a greater extent, as new marriages often mandated additional offspring as the cementing of the union.

A number of very prolific figures in early Afrikaans history exemplify the great fecundity of the people. Willem Schalk van der Merwe had 135 grandchildren from his seventeen children, and ex-president Paul Kruger amassed 156 descendents (if the number of his children, grandchildren and great-grandchildren were totalled).

1.2.4: Genetic Contributors to the Afrikaner Race

The initial population at the Cape refreshment station experienced a very clear sex-ratio skew, due to the predominantly male migrants arriving. This led to a form of social Darwinism, where only the wealthiest, most affluent men were able to acquire wives, and hence contribute significantly to the population. Economic status was an important factor in determining a male's eligibility for marriage, as only wealthier males were able to marry "white" women in their community. The poorer men, of inadequate social standing for marriage to the scarce European women, frequently formed unions (with or without marriage) with the female slaves within their households (Table 3).

Some of the mixed race offspring of these unions would be baptized into the Christian Church. Christianity was viewed as a distinguishing characteristic of the European settlers, who viewed themselves as, above all else, Christians. They believed this to be the main factor distinguishing them from heathen savagery and barbarism. Therefore, the baptism of “non-white” individuals earned them a higher status among the European settlers, allowing them to marry in their church and to present their grievances in court. Additionally, fair skinned children would be accepted as white, especially the very pretty “coloured” girls.

Table 3: Immigrants Marrying into the South African "White" Population (Ross, 1993)

Period	Men	White Women	Non-white Women
1657-1687	63	53	8
1688-1717	300	156	50
1718-1747	317	91	72
1748-1777	565	49	145

New immigrations meant that over time the gender imbalance became less extreme, with an increased availability of European women. The result was an acute decrease in the prevalence of mixed marriages, such that men who could not attain a European wife would rather remain a bachelor than endure the social stigma attributed with inter-racial marriages. Inter-racial offspring also found it more difficult to be accepted as part of the European society, and the colour-lines signifying Afrikaner society as a “white” community were laid.

Over time, the mixed origin of the Afrikaner race has been largely forgotten or denied due to growing racial stigma. However in studying the Afrikaner people and culture, the role played by the slaves and the Khoi San is clearly entangled in their history.

1.3: Aim of this study

Human fecundity patterns will determine who will eventually inherit the earth by dictating the shape and extent of our evolution. For this reason it is crucial to our understanding of humanity's future that we can identify those factors that influence fecundity, and investigate how their unique effects may be felt. Our understanding of the future of human evolution depends on our knowledge of our evolutionary past, with studies of historical populations allowing us insight into the driving forces of human reproduction. Modern society has introduced copious features that disrupt the expression of innate fertility, leading us to contemplate whether human fecundity can still be regarded as a natural trait, removed as it is from the expression of innate fertility. The introduction of effective contraception, in vitro fertilization and radically different cultural and social influences suggest that we may have become out of sync with our natural inclinations.

In an attempt to circumvent these novel issues researchers have focused their demographic studies on historical communities prior to the demographic transition. Our target population, the early European settlers in South Africa, may fulfil these criteria. They were a very fecund population and underwent rapid growth, their numbers growing from 2 540 to 42 975 in just under 120 years (Ross, 1993). Although this growth was supplemented by new immigrations, fecundity was very high. This suggests that they were not actively making use of any form of effective contraception, and additionally that they were not likely experiencing a demographic transition.

The settlers were exposed to unique social, political and cultural influences, which may have shaped their reproductive strategy. They developed a community from diverse backgrounds and heritage during intense political and historical turmoil. They were very fecund, producing offspring at a rapid rate (average interbirth interval was 26 months (Ross, 1993)) and beginning reproduction early (approximately 53% of women were married before they were 20 years old (Ross, 1993), and the first child was born on average 18 months thereafter). This implies that resources may not have been limiting in this community, and that recorded fecundity reflects innate fertility.

Our aim with this study was to identify factors considered to influence human fecundity for which sufficient data is available from this community, and to measure their effect on the fecundity of the Early South African Settlers.

We chose to study this group because of their high fecundity, diverse cultural background and the availability of data. The diverse origins of this group may reveal interesting social and cultural influences. Comprehensive genealogical records documenting South African families are available (De Villiers, Pama, 1966; Heese, 1999-2008), containing data on the initial European settlers in the Cape and their families, with records kept on birth and/or christening dates, wedding dates, as well as names of spouses and offspring. From this data it is possible to extract reproductive information such as total fecundity, age at commencement of reproduction and inter-birth interval. Additionally information could be gathered on the occurrence and frequency of remarriage.

With this data we were able to infer a number of subsidiary aims and hypotheses:

- Heritability of fecundity: numerous estimates have been obtained for the genetic inheritance of human fecundity. The value of 0.4 as obtained by Fisher (1929) is commonly used as a reliable estimate, although a number of studies have obtained different results when investigating the heritability of fecundity within distinct communities. This implies that communities with unique reproductive traits may also possess unique genetic variability for fertility traits, and therefore exhibit different heritability of fecundity. This warrants further studies to be performed in communities with unique reproductive traits.

As our population had a high reproductive rate it is possible that innate fertility may have resulted in more defined differences in fecundity and therefore may lead to higher estimates for heritability. Alternatively, as is commonly observed in animal trials under ad lib feeding conditions, high population growth rate may imply reduced selection and hence result in reduced heritability estimates.

Our aim was to calculate a heritability estimate for this population and see how it compares to previously calculated estimates. Some previously determined estimates may be inflated due to shared environmental factors that were not taken into account. We try to control for this by including data corresponding to cultural and social inheritance in our calculations.

Fecundity is controlled by the action of three life history traits, these being the age of onset of reproduction, the length of the interbirth interval, and the age at which reproduction ceases. As these factors shape fecundity, it may be beneficial to control for their actions when determining heritability of fecundity. However if all three factors are simultaneously controlled for heritability would have no avenue to function in dictating fecundity. For this reason we opted to utilize a model controlling for other extraneous factors, for example nationality and cohort, but leaving the effects of both age at first reproduction and interbirth interval (we did not have an accurate measurement of age at cessation of reproduction, and therefore could not control for its effect). In the event that heritability in this case functioned solely, or primarily, through only one of the two measured life history traits we included additional models controlling for both terms, and for each term independently.

- Cultural inheritance: A major focus of studies has been whether resource amassment imparts a limit on reproduction. In pre-transition communities the wealthiest individuals were the most fecund (Gillespie *et al.*, 2008) as they had more resources available to support reproduction. Reproductive strategy therefore involved a clear trade-off between quantity and quality of offspring, with wealth placing a fundamental limit on fecundity. The demographic transition led to an inversion of these trends, as wealthier individuals decreased their fecundity more efficiently than their poorer counterparts (Borgerhoff Mulder, 1998), and in time became the least fecund.

  From historical data it is evident that the various nationality groups comprising the early South African settlers, would have experienced unique cultural and social factors, and differed as well in measures of wealth (e.g. the French immigrants were historically wealthier than mixed race individuals or the German immigrants).

  We will investigate how the nationality groups differed in terms of fecundity, and observe how our data correlates with the referenced studies. In order to corroborate the work of Gillespie *et al.* (2008) we would expect the French immigrants to be the most fecund, and those recorded as of mixed race to be the least fecund of the nationality groups.

- Sexual antagonistic pleiotropy: we wanted to test for the presence of sexual antagonistic pleiotropy. If present then the hypothesis that different sexed siblings of individual families exhibit opposite trends in fecundity would prove true, i.e. an increased fecundity among brothers would result in a decline in their sisters' fecundity, and visa versa.

The high reproductive rate of the settlers may, once again, act to bring such discrepancies to the fore as individuals were able to reproduce to their maximal innate capacity. Alternatively the high overall fecundity may mask individual cases of antagonistic pleiotropy. We feel it would be edifying to test for the presence of antagonistic pleiotropy in this population as it could offer additional perspective into the genetics of human fecundity, and potentially explain the maintenance of genetic variation in fecundity.

- The effect of time dependent cohorts and possible demographic transition: Social influences and the views of one's peers may act to alter reproductive behaviours (Austerlitz, Heyer, 1998; Boone, Kessler, 1999). Social ideals often change between generations, such that separate generations will be under diverse social influences (Van Landingham *et al.*, 1991; Szreter, 1993; Pinnelli, Cesare, 2005). This may act to deflate estimates of genetic inheritance, as behaviour evolves to suit divergent environments.

  Birth years for our target individuals ranged from 1661 to 1844. This allows us to examine how trends changed over time, and what impacts the varied cohort groups experienced. We wish to investigate whether fecundity of this group changed over time, and if so, in what way. Additionally we wish to test for the presence of a potential demographic transition by ascertaining whether average fecundity decreased over the years.

- The influence of age on reproduction: earlier commencement of reproduction has been linked with increased lifetime fecundity (Pinnelli, Di Cesare, 2005). Males and females differ in their reproductive aging (Penn, Smith 2007) and therefore should be affected differently by the influence of age at first reproduction.

Our sample tended to begin reproduction at an early age, although a few individuals were older. We plan on investigating the influence of age at first successful reproduction event (i.e. the birth of the first offspring) on the total offspring number, and see how this effect varies between the sexes.

- The effect of inter-birth interval: we wish to test the hypothesis that shorter inter-birth intervals lead to greater final offspring count. Rapid reproduction may result in an increase in offspring number through maximizing reproductive effort. However if reproduction proceeds at too rapid a pace, a fitness cost is incurred, and the offspring produced may have higher morbidity and mortality rates, or the mother may have insufficient resources to support this level of reproduction, increasing her morbidity and mortality and decreasing her reproductive lifespan.

  As the settlers exhibited high overall fecundity it is of interest to discover at what rate they were actively reproducing, in order to see how this value compares to those determined in previous studies of populations purportedly reproducing at a rate to maximize their lifetime reproduction (Anderies, 1996).

- The influence of remarriage: we wish to investigate how the number of monogamous marriages affects an individual's fecundity. Serial monogamy is common in Western societies, where remarriage often occurs following the death of a spouse, or divorce. Remarriage is more common among males than females (Buckle *et al.*, 1996), suggesting a greater variance in mating success in males, consistent with Bateman's second principle (Brown *et al.*, 2009). Bateman's first principle states that this variance in mating success would be reflected by a variance in offspring number (Brown *et al.*, 2009), i.e. remarriage should increase male (but not female) fecundity

(Kaar *et al.*, 1998). Our hypothesis is that additional marriages increase male fecundity by potentially increasing his reproductive lifespan (through subsequent marriages to younger, fertile women). In women this is not expected to be the case.

In this population the occurrence of remarriage (following the death of a previous spouse) was sufficiently high (approximately 14% of males and females in our dataset were married twice, and 4.5% of males and 2.2% of females had three or more marriages) to allow us to analyze the impact of number of marriages on total lifetime fecundity.

- The influence of birth order on reproductive strategy: Birth order has been associated with differing personality types as well as different reproductive strategies (Michalski, Shackelford, 2002). Firstborns play different roles within the family to their later born siblings, which can lead to the development of unique reproductive behaviours. Firstborns and their younger siblings fulfil different niches in the family structure, and hence tend to develop different views on family and reproduction.

  We will compare the fecundity of first born individuals to their later born siblings to compare strategies adopted by each. We expect that firstborns will practice a more stable strategy, and tend to have a greater number of offspring compared to their younger siblings.

- Our final aim is to create a simple model explaining the effects of various significant factors on the fecundity of the early European South African settler.

Chapter 2: Database compilation and statistical analysis

2.1: Introduction

Genealogical books of the early European South African settlers and their families have been compiled for a number of generations, starting with the first European arrivals. These books provide insight into the reproductive behaviour of the early colonists, and permit us the opportunity to study the correlations of various factors affecting their lifetime fecundity.

We are interested in the effects of cultural inheritance, social pressures and genetic predispositions on reproduction of the early Afrikaners. This population was determined to be highly fecund (Ross, 1993) and may have experienced unique fecundity influencing cultural and social aspects.

2.2: Materials and methods

2.2.1: Genealogical database compilation

The books of De Villiers and Pama (1966) and Heese (1999-2008) (figure 2) were used to compile a genealogical database in Excel ® 1997 & 2007. Data was collected on 517 founders (including all founders listed from A-H in the alphabetical listing of De Villiers and Pama (1966) for whom sufficient data could be obtained).

(a)

van AARDE

Gerrit van Aarde, v. Delft, x, in Holland, Levina Theunis van Genz, v. Rotterdam.
b1 Barendina, x 1.4.1714 Gerrit Pietersz Vredenburg, v. Amsterdam
b2 Theunis, x 7.12.1731 Hilletje Kerver
c1 Leonora Catharina, ≈ 25.10.1733, x Casper J.N. Steenkamp
c2 Sara Maria, ≈2.7.1735
c3 Anna, ≈ 24.3.1737, x B. Vrey
c4 Maria, ≈5.3.1741
c5 Catharina, ≈20.9.1744, vgl. J. Strydom en W.A. de Klerk
c6 Theunis Gertsen, ≈5.5.1748
c7 Christiaan, ≈10.10.1750, burger Stellenbosch, x 9.5.1773 Catharina Lavina Nel,
[xx 19.3.1775 Johanna Francina Kruger
d1 Willem Adriaan, ≈25.12.1774, burger Graaff-Reinet, x 7.2.1796 Maria
[Susanna Triegard
e1 Willem Adriaan, ≈26.11.1797
e2 Christiaan, ≈16.9.1798
e3 Anna Elizabeth, ≈24.11.1799
e4 Willem Adriaan, ≈18.11.1801
e5 Carel Johannes, ≈18.11.1801
e6 Catharina Lavina, ≈19.2.1804
e7 Frans Johannes, ≈4.5.1806
e8 Theunis Gerritse, ≈ 19.2.1807
e9 Louis Jacobus, ≈22.1.1809
d2 Hilletje Maria, ≈23.2.1777, x W.F. Kruger
d3 Frans Johannes, ≈31.1.1779, burger Graaff-Reinet, x 21.10.1798 Susanna
[Wilhelmina Triegard
e1 Christiaan, ≈22.12.1799
e2 Carel Johannes, ≈25.11.1801
e3 Anna Elizabeth, ≈19.8.1804
e4 Theunis Gerrit, ≈20.5.1805
e5 Johanna Francina, ≈30.3.1807
e6 Frans Johannes, ≈2.10.1809
e7 Susanna Wilhelmina, ≈4.11.1810
d4 Maria Catharina, ≈28.4.1782, x Daniel Nel
d5 Christina Elizabeth, ≈21.9.1783, x Christiaan Botha

(b)

VAN AARDE/VAN AARDT

Gerrit van Aarde van Delft x Holland, Levina Theunis VAN GENZ, van Rotterdam.

b1 Barendina x 1.4.1714 Gerrit Pietersz VREDENBURG, van Amsterdam
b2 Theunis x Kaapstad 7.1.1731 Hilletje KERVEL
c1 Leonora (of Levina) Catharina ≈ Kaapstad 25.10.1733 x Casper J.N. STEENKAMP
c2 Sara Maria ≈ 2.7.1735
c3 Anna ≈ 24.3.1737 x B. VREY
c4 Maria ≈ 5.3.1741
c5 Catharina ≈ 20.9.1744 (vgl. J. Strydom en W.A. de Klerk)
c6 Theunis Gertsen ≈ 5.5.1748
c7 Christiaan ≈ Swartland 10.10.1750 x Swartland 9.5.1773 Catharina Levina NEL ≈ 15.7.1753 xx Tulbagh 19.3.1775 Johanna Francina KRUGER ≈ 23.12.1795
d1 Willem Adriaan ≈ 25.12.1774 † Graaff-Reinet 25.8.1847 x 7.2.1796 Maria Susanna TRIEGARD * 1778
e1 Willem Adriaan ≈ 26.11.1797, jonk †
e2 Christiaan ≈ 16.9.1798 x Cradock 9.10.1825 Johanna Francina VAN AARDT * Kookhuisdrif, Jagersfontein 1.12.1806 ≈ Graaff-Reinet 30.3.1807
f1 Susanna Wilhelmina * 13.12.1827 ≈ Somerset-Oos 14.12.1827
f2 Willem Adriaan * 22.2.1829 ≈ Somerset-Oos 29.3.1829, verdrink Smoordrif 26.11.1858 x Susanna Elizabeth DE KLERK
g1 Christiaan * 16.4.1854
g2 Maria Aletta * 3.11.1855
g3 Johanna Francina * 6.5.1858

Figure2: Examples of the format of the reference genealogies
(a) Extract from De Villiers and Pama (1966)
(b) Extract from Heese (1999-2008)

We refer to the founders as the A generation individuals and their offspring as the B generation (i.e. in the extracts in figure 2 Gerrit van Aarde is from the A generation, and his offspring Barinda and Theunis are from the B generation). The A generation data we collected included country of origin (indicative of social status – see section 1.2) and offspring number. Of the 1138 B generation individuals for whom data was available, we recorded the following: Date of birth/christening; Gender; Number of siblings; Position in the birth order; Date of marriage (multiple dates if sequentially remarried); Date of birth/christening of first born offspring; Total number of offspring; Date of birth/christening of last recorded offspring. The B generation data covered a large time-frame of Afrikaner history, with their years of birth ranging from 1661 to 1844 (figure 3).

This data was similarly compiled for the spouse/s of these B generation target individuals.

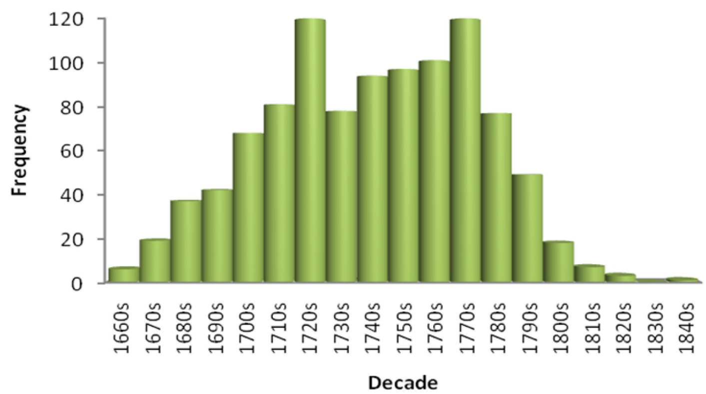

Figure 3: Histogram showing the decade of birth of B individuals included in the database

Nationality was recorded for married pairs as the husband's nationality. In incidences where females remarried men of different nationalities, her nationality was recorded as "other". The remaining nationality classes were Dutch, German, French, European mixed and race mixed (i.e. individuals recorded as having non-European ancestry). The majority of individuals were of German or Dutch origins (Figure 4), corresponding to the historical information (Gillespie *et al.*, 2008) We used the male's nationality as a married couple's socio-economic status was dependent on the male's status, and socio-economic status could be linked to nationality.

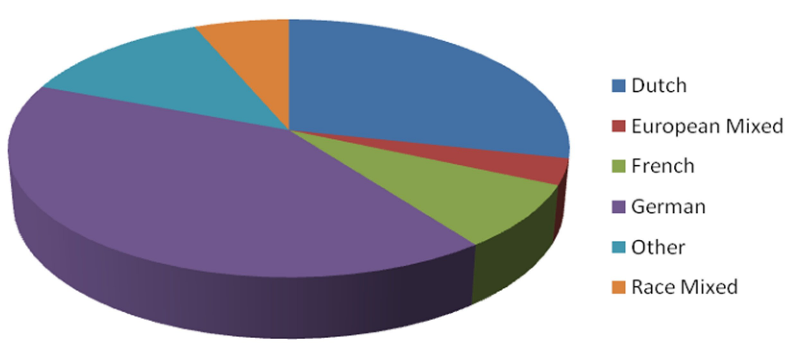

Figure 4: Pie chart of the proportion of individuals in the database belonging to each of the listed heritage groups

All recorded dates were converted to Julian dates using the following formula:

$$\textit{Julian date} = \textit{day} + \frac{[153 \times \textit{month} + 2]}{5} + 365 \times \textit{year} + \frac{\textit{year}}{4} - 32083 \qquad (1.1)$$

(Miller *et al.*, 2009)

Collected data was then used to calculate age at first reproduction (for husbands and wives), age at marriage (for both sexes) and average inter-birth interval of B generation individuals and spouses.

In instances where target individuals or their spouses remarry, offspring data was independently recorded for each marriage.

For statistical analysis, the database was divided into two datasets, one containing only couples where both individuals were married only once, and the other that included multiple marriages.

2.2.2: Tree Analysis

The statistical package R 1.2.1 © 2005 was used to analyze the once-married database. A tree model was constructed using all included terms to explain fecundity. Binary recursive partitioning was used, splitting the data at each node by the threshold value of the explanatory variable. The output of the tree model was then analyzed to highlight which factors were most influential in determining fecundity, and to indicate potential interactions to consider.

2.2.3: Statistical Analysis of database

The Tree model output was analyzed, and terms indicated as influencing fecundity in this model were utilized along with factors listed in the following table (table 4) to determine a minimal adequate model (MAM) that best explains the discrepancies in offspring number (the dependent variable) of the once married database (Crawley, 2005).

Table 4: Predictor Variables Analysed to Determine MAM

Factors included for analysis	Information to be gained from factor
A generation family Sex	} Used in antagonistic pleiotropy study
Date of birth of first offspring	Cohort effect
Age at 1st reproduction (AFR) male AFR female	Onset of reproduction, important in determining final offspring number
Birth order (factor)	Cultural & social impact (may differ between first and later born individuals)
Sibship sizes for male Sibship sizes for female	Indicate heritability of fecundity
Number of times married	Role of remarriage in final offspring number
Age at marriage male Age at marriage female	Similar to AFR. Start of reproductive attempts, Indicates possible delays in reproduction
Interval from wedding to birth of 1st child	Indicator of innate fertility
Offspring number	Indicator of fecundity
Heritage (nationality)	Cultural influence
Interbirth interval (IBI)	Indicator of fertility

2.2.4: Specific studies performed:

2.2.4.1: Cultural factors

Cultural inheritance was investigated using the influence of nationality of origin on the fecundity of the target individuals. Nationality served as an appropriate proxy for cultural inheritance due to its varied effect on traditional practices and religions originating from specific geographic regions. Additionally, the wealth of founding individuals varied with their country of origin, and wealth has been shown to impact on fecundity.

2.2.4.2: Cohort effect

The date on which couples entered the reproductive population (i.e. the Julian date of birth of their first offspring) was used to determine social cohort groups, as individuals reproducing during the same time period would assumedly face the same or similar social influences. This data was used to study changing fecundity levels over time, showing changes in social ideals and reproductive practices.

2.2.4.3: Influence of age

Age (in years) was recorded at the time of marriage (first marriage in cases of multiple marriages) and at the first successful birth for each individual, for husband and wife. To avoid colinearity of predictors we chose to include age at first reproduction (for males and females), but not age at marriage, when determining our linear model.

2.2.4.4: Interbirth interval

The average interbirth interval was calculated by dividing the period of time between the first and last offsprings' births (in years), by the total number of offspring born less one. The effect of this factor on fecundity was then investigated.

2.2.4.5: Antagonistic Pleiotropy

Possible sexual antagonistic pleiotropy was investigated by comparing male and female siblings of individual founding families in terms of their offspring number, in order to identify possible disparities in the fitness of male versus female offspring of the same family. Only families where B generation siblings of both genders were recorded were used, and averages were calculated for brothers' and sisters' fecundity. Lack of information on, for example longevity, limited the investigative routes for analyzing other forms of antagonistic pleiotropy.

This analysis was performed including all the factors included in the MAM, as well as excluding the factors directly determining offspring number, i.e. age at first reproduction, interbirth interval and the period from wedding to first reproduction. These factors were excluded as, if they were included in the model their effect on fecundity would be effectively removed, and antagonistic pleiotropy would have no pathway to express its action.

2.2.4.6: Inheritance of Fecundity

The heritability of fecundity was analysed separately for males and females. Number of offspring regressed on the husband's sibship size was used to calculate male inheritance of fecundity, and on the wife's sibship size to calculate female inheritance of fecundity.

Terms for female and male sibship sizes were included in the initial proposed model. Additionally heritability was investigated using a model that does not include terms for interbirth interval or age at first reproduction. This was done as interbirth interval and age at onset of reproduction (along with age at cessation of reproduction, which was not recorded) are the pathways through which fertility may act to influence fecundity. If these terms are included in the model then the inheritance of fecundity through these factors may not be obvious. Additional analyses were also performed including one or the other of the above terms in order to investigate the environmental pressures these aspects may be under.

2.2.4.7: Influence of Remarriage

The MAM developed for the once married database was then utilized in the multiple marriage database, to calculate residuals remaining after application of the determined factors' coefficients. These residuals were used to investigate the effect of the number of marriages of males and of females on their final offspring number, accounting for other known factors. Linear models were run to determine the effect of number of marriages on the residual, for each gender.

The multiple marriage database was additionally analyzed to determine a minimal adequate model that best described fecundity in a similar fashion as applied to the once married database, in order to evaluate whether the coefficients are relatively stable. All of the factors considered for the once married database MAM were included, as well as a term for the number of marriages. Models were determined for each gender.

Chapter 3: Results

3.1: Tree Analysis

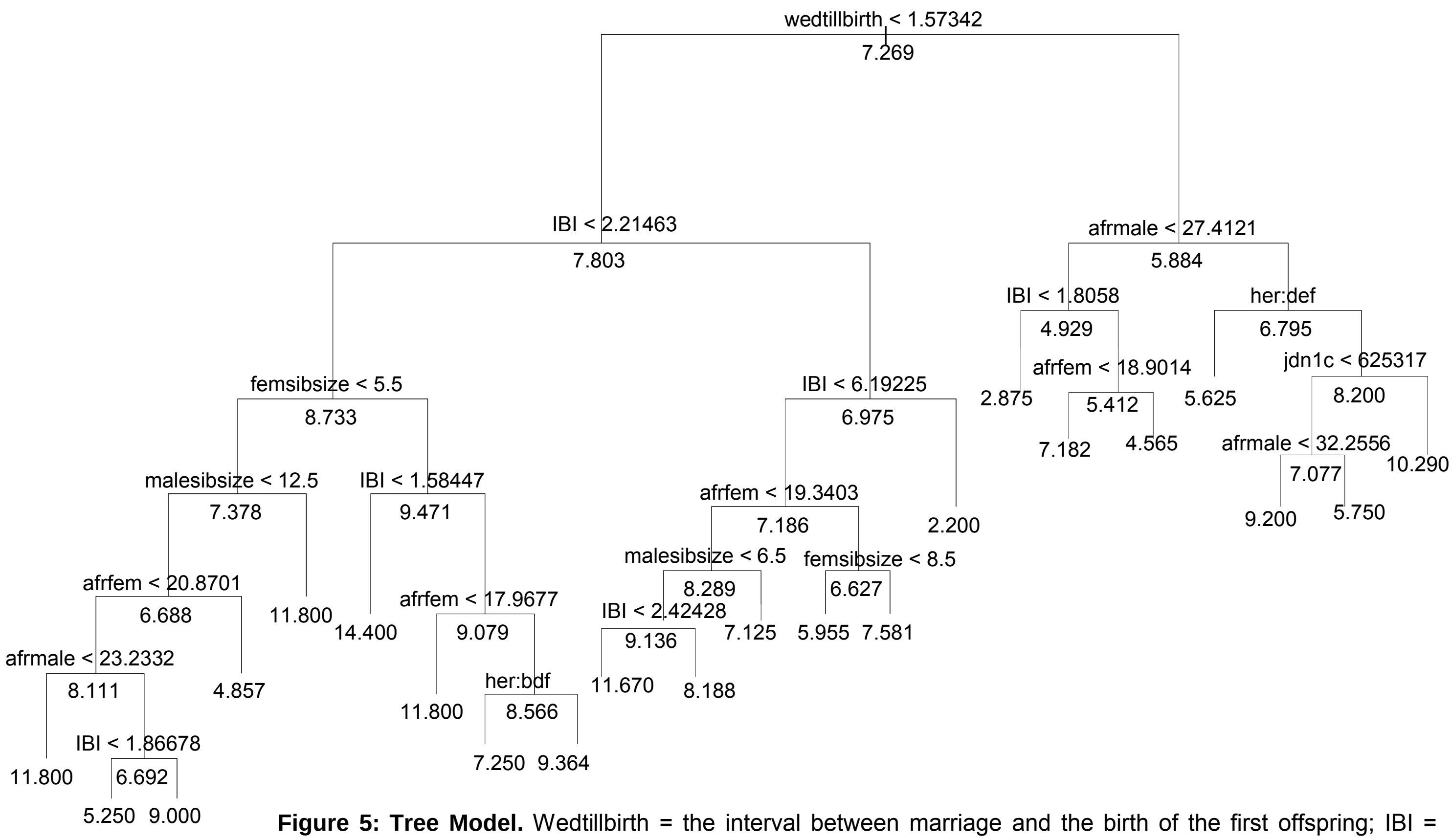

Figure 5: Tree Model. Wedtillbirth = the interval between marriage and the birth of the first offspring; IBI = interbirth interval; afrmale = age at first reproduction (male); afrfem = age at first reproduction (female); her = nationality (a = Dutch; b = European mixed; c = French; d = German; e = other; f = mixed race); femsibsize = wife's sibling number + 1; malesibsize = husband's sibling number + 1; jdn1c = first child's Julian date of birth

The mean fecundity for the database was determined as 7.269 (Figure 5).

The initial tree split was created by the wedding till birth interval (the period, in years, between the wedding date and the christening of a couple's first child). Individuals with shorter intervals (< 1.57 years) were the most fecund, producing approximately 2 offspring more than those with greater intervals.

Among the group with the shortest wedding till birth interval the next most important variable is interbirth interval. As expected, individuals with the shortest interbirth interval (< 2.2 years) were more fecund. However, the data indicates that an intermediate rate of reproduction is optimum, with the individuals with an interbirth intervals greater than 2.2 but less than 2.4 years having greater completed fecundity than individuals at either extreme.

Among the group with the greater wedding till birth interval, the effect of interbirth interval is reversed, with individuals with short interbirth intervals (but long wedding till birth intervals) having the lowest fecundity.

Female sibship size (the fecundity of the wife's parents) forms the next split, with women from larger families (more than 5 siblings) producing more offspring. Women from smaller families whose husbands come from very large families (more than 12 siblings) also produce more offspring, although when interbirth interval is greater and the first child is born while the wife is younger than 19.3 years of age, males from larger families produce fewer offspring. Interestingly, sibship size does not appear to play a role in determining offspring number for couples who wait more than 1.5 years after marrying before producing their first child.

Males and females who commenced reproduction at a younger age produced larger offspring counts in all circumstances.

Individuals of French and Dutch origin were shown to have the greatest fecundity of the nationality groups.

3.2: Minimal Adequate Model

The tree model determined above was utilized to identify important factors influencing fecundity. This data was combined with the specific questions we had regarding reproduction in this population to propose a model. Terms were sequentially dropped from the model by evaluating their p values at each stage (Crawley, 2005) to obtain a minimal adequate model. The order in which terms were dropped is given by table 5. Note the degree of freedom of the model increases as each term is dropped as more data rows have data for all of the predictor values.

The initial proposed model read as follows:

$$\boldsymbol{O \sim WtB + AFR♀ + AFR♂ + h + IBI + jc + jc^2 + sib♀ + sib♂ + famA[sex] + bo + bo{:}\ h + sib♀{:}sib♂ + AFR♀{:}AFR♂ + jc{:}bo + jc{:}h} \qquad \textbf{(1)}$$

Where O is the total number of offspring had by the target B generation individual; WtB is the interval from wedding until birth of first child (in years); AFRf♀ and AFR♂ refers to the age at first reproduction (in years) of the female and male respectively within each target couple; h is the nationality; IBI is the average interbirth interval in years; jc is the Julian date of birth of the oldest child (see equation 1.1); sib♀ and sib♂ refer to the sibship sizes for the wife and husband respectively; famA[sex] is the effect of gender nested in the A generational family, used to indicate correlations between opposite gender siblings; bo refers to the birth order of the target B generation individual (binomial term, first or later born); and interaction terms are indicated by the use of colons.

Table 5: Dropped terms during construction of the MAM (Model1)

Term	Order Dropped	p Value when Dropped	Degree of Freedom
jc:bo	1	0.6142594	1,265
famA[sex]	2	0.5258771	1,269
AFR♀:AFR♂	3	0.3736904	1,271
AFR♂	4	0.8337969	1,272
h:bo	5	0.590409	1,306
bo	6	0.9911394	1,311
sib♀:sib♂	7	0.4842607	1,312
sib♂	8	0.5406765	1,313
h:jc	9	0.157362	5,384

The following minimum adequate model was obtained (Table 6 Model 1). A second model was run excluding terms that directly determine fecundity, i.e. the interbirth interval (and wedding to birth interval), and age at onset of reproduction (see figure 1 in chapter 1 for illustration of this). By not including these terms in the model, the effect of other factors which influence fecundity through, for example more rapid (shorter interbirth intervals) or earlier (younger age at first reproduction) reproduction. The results of this analysis are given under Model 2 in Table 6.

Table 6: Coefficients of analyzed factors utilizing MAM

Factor		Model1 Coefficients	Model1 p-value	Model2 Coefficients	Model2 p-value
Wedding to birth interval		-0.5197	0.00617	-	
AFR (female)		-0.1883	5.23×10^{-06}	-	
Nationality	Dutch	13.5725	0.085791	7.3273	0.0198882
	European mixed	15.0276		9.1170	
	French	14.6294		8.4654	
	German	13.4303		7.3073	
	Other	13.2648		6.7455	
	Race mixed	13.2648		6.5803	
IBI		-0.7909	1.31E-06	-	
1st child dob		-0.01163	0.04213	-0.01904	0.000210
$(\text{1st child dob})^2$		-0.00052	0.00225	-0.000532	0.000276
Female sibship size		0.07774	0.08305	NS	
Male sibship size		NS		NS	
Model adjusted R^2		0.1528		0.06051	

NS: non-significant factor

3.3: Influence of Nationality

Families from different nationality groups differed in their average fecundity (Table 6). This term was significant in influencing fecundity in both models (p = 0.085791; p = 0.0198882), although the degree of significance increased when the effect of interbirth interval, wedding till birth interval and age at first reproduction were removed

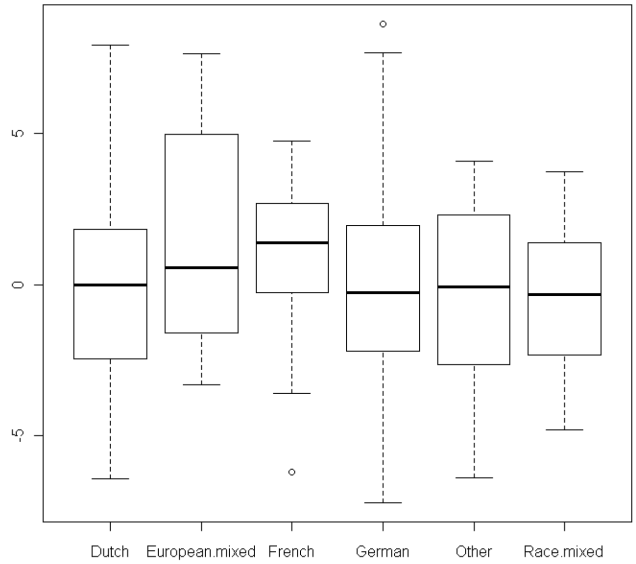

Figure 6: The effect of nationality on fecundity. Figure illustrates the effect of nationality on residuals of a model including all the terms of the MAM but excluding nationality. Residuals plotted on the Y axis

from the model (table 6) Individuals with mixed European ancestry had the greatest fecundity, followed by the French who had on average 1.1 more offspring than the Dutch. Mixed race individuals were least fecund, and had between 1.6 and 2 offspring less than the French (Figure 6).

This effect was only marginally significant, and indicates that further investigation is recommended to fully define the impact of cultural inheritance in this group.

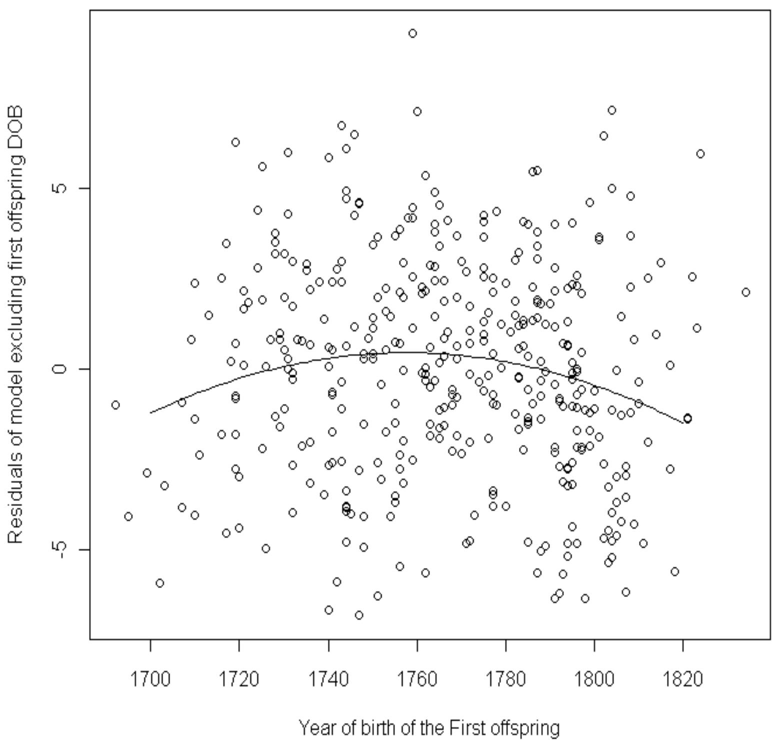

Figure 7: Offspring number as determined by the year of birth of the first offspring. Figure illustrates the effect of first offspring year of birth on residuals of a model that included all the terms of the MAM except for date of birth of first offspring

3.4: Cohort effect

A decline in fecundity over time (after about 1760) was evident (Figure 7) equivalent to approximately 0.01 fewer offspring per year. This is equivalent to approximately 0.2 less offspring per generation (if each generation is considered to equal approximately 20 years).

3.5: Age at First Reproduction and at Marriage

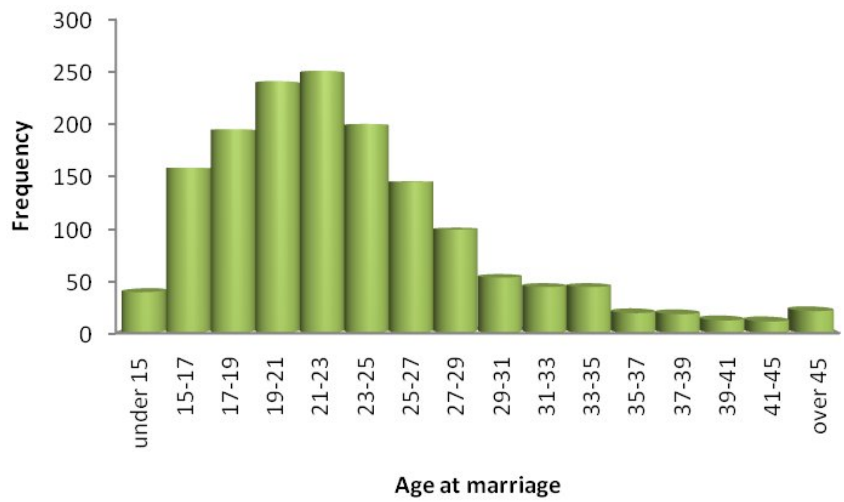

Figure 8: Histogram showing the distribution of age at marriage

Average age at first reproduction among our database was 22.1501 years for females and 26.79714 years for males, corresponding well with the data of Ross (1993). Age at marriage and age at first reproduction closely corresponded, both events commonly occurring in the early twenties. The majority of B generations (approximately 88% of females and 57% of males) were married before the age of 25, most between 21 and 23 (Figure 8).

Female age at first reproduction was significant ($p = 5.2 \times 10^{-6}$) in influencing total offspring number (table 6). Every ten year interval before commencing reproduction correlated to approximately two fewer offspring produced (Figure 9). Age at first reproduction of males was not significant in determining lifetime fecundity. As older men tended to have correspondingly older wives (Table 7), it may be assumed that any effect of male age on fecundity would largely be the result of his wife's age, and therefore by including both terms in the model, any effect of male age would be masked by colinearity. However when the model was run excluding female age at first reproduction but including male age, male age was still not significant in influencing final offspring

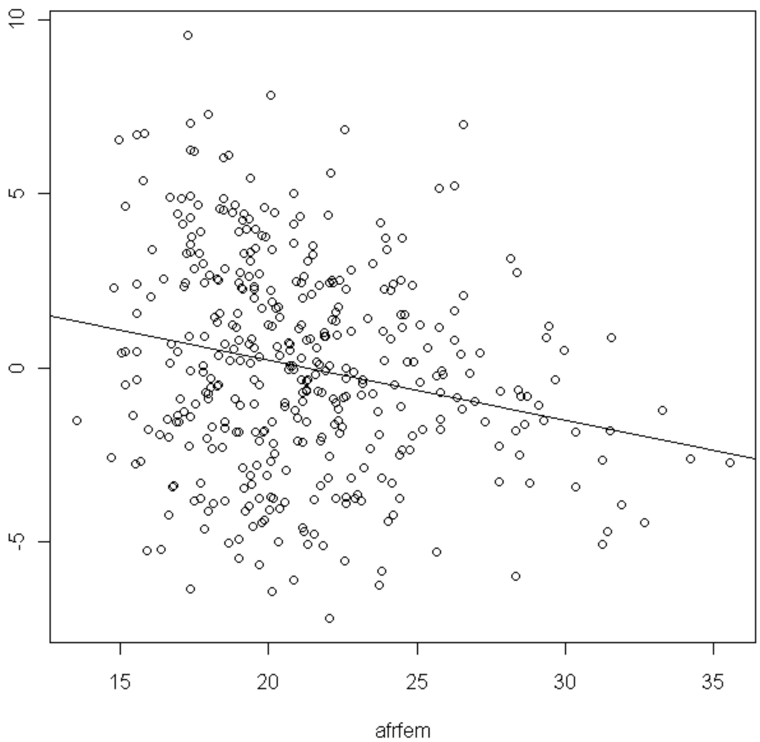

Figure 9: The effect of female age at first reproduction on fecundity. Figure illustrates the effect of female age at first reproduction on residuals of a model that included all the terms of the MAM excluding female age at first reproduction. Residuals of the model are plotted on the Y axis

number, suggesting that the two terms were not strongly correlated, and that female age was the appropriate choice for inclusion in the model.

The rate of decrease of fecundity with increasing female age at first reproduction shows a sharp drop off after the age of thirty (table 8) corresponding to data indicating the greatest declines in female fecundity occur after the age of thirty (Nasseri, Grifo, 1998; te Velde, Pearson, 2002; van Noord-Zaadstra *et al.*, 1991).

Table 7: Age at marriage as a function of spouse age.
Percentages of the population marrying in each age category

Age of Husband	Age of Wife: Under 20	20-24	25-29	30-34	Over 35	Total
Under 20	6.398104	1.421801	0.7109	0	0.473934	9.004739
20-24	24.40758	19.66825	3.317536	0.7109	0	48.10427
25-29	13.50711	8.056872	3.080569	0.473934	0	25.11848
30-34	6.161137	2.606635	1.421801	0.7109	0	10.90047
Over 35	3.791469	1.895735	0.947867	0.236967	0	6.872038
Total	54.2654	33.64929	9.478673	2.132701	0.473934	100

Table 8: Influence of age at first reproduction (for wife) on final offspring count

Age of female at 1st Reproduction	Average offspring number	Standard Deviation	Median Offspring number	Range
under 15	7.666666667	3.44480284 9	6.5	5:14
15-19	7.153110048	3.79720322 1	7	1:18
20-24	6.034188034	3.25307810 4	6	1:14
25-30	5.17721519	3.22950934 5	5	1:15
over 30	3.34375	2.02579136 3	3	1:8

3.6: Interbirth interval

The rate at which reproduction took place significantly influenced the total fecundity of individuals, with 0.7901 fewer offspring for each year the interbirth period was extended ($p = 1.34 \times 10^{-6}$).

3.7: Interval between wedding and first reproduction

The longer couples waited to commence reproduction following marriage, the lower their completed lifetime fecundity (Figure 10). Every year without a birth resulted in approximately 0.5 fewer offspring being born ($p = 0.00628$).

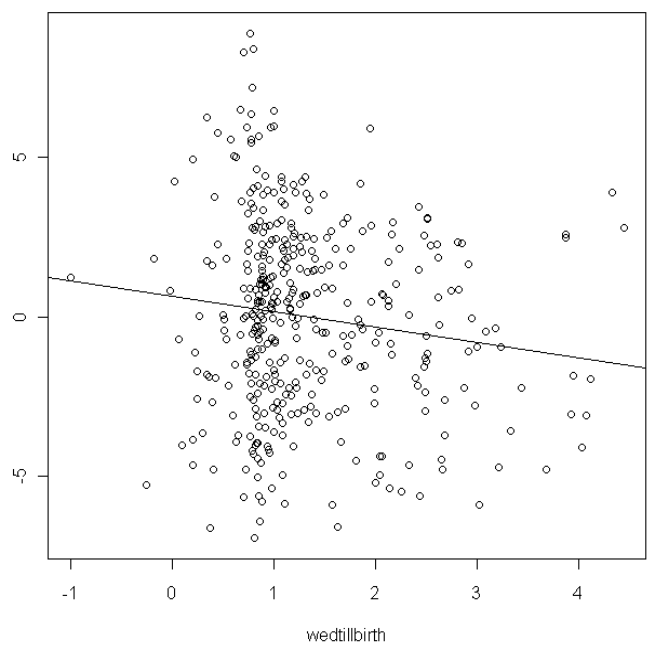

Figure 10: The effect of wedding till reproduction interval on fecundity. Figure illustrates the effect of this interval on residuals of a model that included all the terms of the MAM excluding wedding till birth interval. Residuals of the model are plotted on the Y axis

3.8: Inheritance of fecundity

Paternal sibship size was not given to be significant in determining fecundity in the MAM. Maternal sibship size was marginally significant, with each additional sibling increasing a woman's fecundity by 0.08. This indicates a role for maternal inheritance of fecundity, with a heritability estimate of approximately 0.16 (Figure 11). However, when the model was run excluding the terms directly determining fecundity, i.e. interbirth interval; wedding till first birth interval; and age at first reproduction, female sibship number was no longer significant (Table 6, model 2). This implies that care needs to be taken in interpreting this result, and more research is needed as to the mode of influence of parental fecundity in this population.

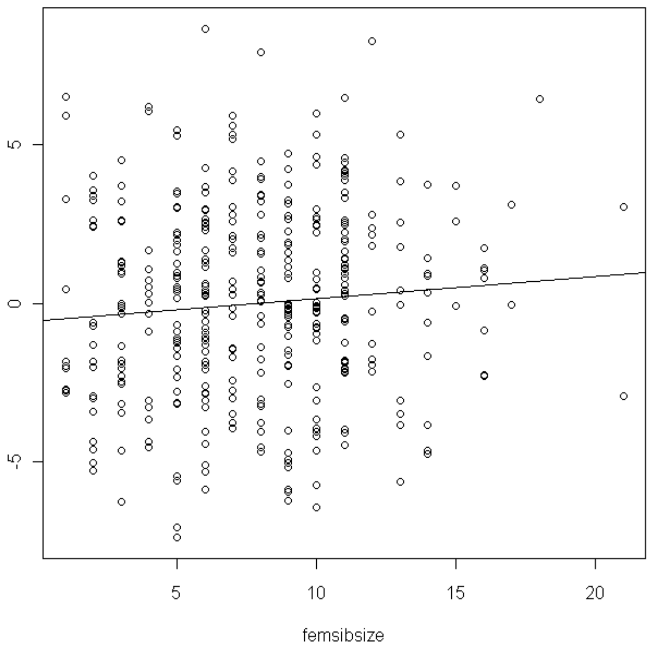

Figure 11: The effect of maternal sibship size on fecundity. Figure illustrates the effect of female sibship on residuals of a model that included all the terms of the MAM excluding female sibship size. Residuals of the model are plotted on the Y axis

Interestingly, when a model was run that included age at first reproduction but not interbirth interval or wedding till first birth interval the effect of female sibship size on fecundity was significant (Table 9), and its effect was comparable to that obtained by model 1 (Table 6). Once again no significant effect was determined for sibship size on male fecundity.

Table 9: Coefficients for sibship size and age at 1st reproduction

Factor	Coefficient	p value	Degrees of freedom	Adjusted R squared
Sibship size	0.08284	0.0381	1,556	0.07874
Age at 1st reproduction	0.21069	$2.2e^{-11}$	1,556	

3.9: Antagonistic pleiotropy

No significant sexual antagonistic pleiotropy was observed. Graphs of male versus female fecundity within families show that increased fecundity in one sex leads to increased fecundity in the other sex (Figure 12). No significant relationship between the reproductive performance of opposite sex siblings could be elucidated, implying that genes influencing fecundity had similar effects on both gendered siblings.

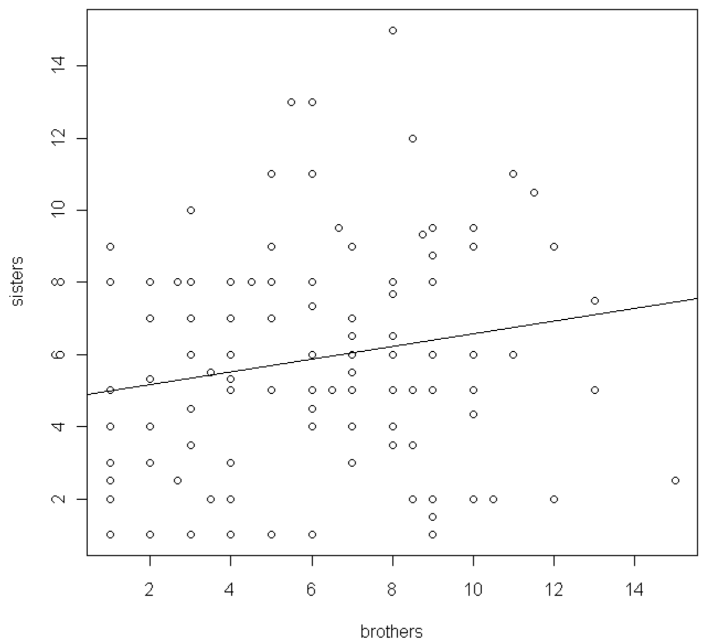

Figure 12: Average fecundity of sisters versus brothers within families

As limited demographic traits were recorded in the genealogical records, the presence of antagonistic pleiotropy for other traits could not be determined.

3.10: Birth Order

No significant interaction was found between parental birth order and final offspring count.

3.11: Role of remarriage

Remarriage was common enough in this population (Table 10) to allow us to examine its effect on fitness. The number of marriages was shown to be significant in influencing these residuals (and hence offspring number) in both sexes (Table 11). In females each additional spouse resulted in 0.892 additional offspring and in males each additional wife resulted in 2.1973 additional offspring.

Table 10: Frequency of remarriage in our sample
Values in parenthesis give percentages in each marriage class

		Males	Females
Number of marriages	1	920 (80.84)	946 (83.13)
	2	167 (14.67)	167 (14.67)
	3 or more	51 (4.48)	25 (2.20)
		1138	1138

Table 11: Results for the linear models of the influence of number of marriages on the model residuals for each gender

	Male	Female
Coefficient	2.1973	0.8920
p Value	$<2 \times 10^{-16}$	0.000911
Multiple R^2	0.1229	0.01724
Degree of Freedom	1,633	1,633

MAM for multiple marriage dataset

Female sibship size dropped out of the MAM determination, for both the male and the female datasets (with p = 0.269488, and degree of freedom $F_{12,622}$ for the female dataset and p = 0.2516957, $F_{12,622}$ for the male dataset).

All of the other terms included in the single marriage database MAM remained significant in the male and female multiple marriage dataset MAMs (Tables 12 and 13).

Although the effects of each term were less than in the once married database, the direction of the trends remained the same.

Table 12: Coefficients of factors affecting females of the multiple marriage dataset
Adjusted $R^2 = 0.1876$; Degrees of Freedom $F_{11,625}$

Factor		Coefficient	p value
Wedding to birth interval		-0.1849	0.00247
Nationality	**European mixed**	5.8244	0.035531
	French	6.1545	
	German	5.0473	
	Other	5.0427	
	Race mixed	4.2664	
	Dutch	5.3864	
IBI		-0.5848	4.06×10^{-14}
AFR		-0.1646	6.7×10^{-9}
1st child dob		0.003564	0.00206
(1st child dob)^2		-2.932×10^{-9}	0.00191
Number of Marriages		0.8105	0.00303

Table 13: Coefficients of factors affecting males of the multiple marriage dataset
Adjusted $R^2 = 0.2044$; Degrees of Freedom $F_{11,625}$

Factor		Coefficient	p value
Wedding to birth interval		-0.2685	0.008952
Nationality	**European mixed**	6.055479	0.0312446
	French	6.350149	
	German	5.379049	
	Other	4.972649	
	Race mixed	4.753649	
	Dutch	6.020649	
IBI		-0.4699	2.34×10^{-10}
AFR		-0.1175	0.000145
1st child dob		0.00336	0.007839
(1st child dob)^2		-2.767×10^{-9}	0.007358
Number of Marriages		2.006	$< 2 \times 10^{-16}$

Chapter 4: Discussion

Our analyses suggest that cultural inheritance, maternal genetic inheritance, female age at first reproduction, wedding to first birth interval, interbirth interval, cohort and number of marriages were integral to the determination of fecundity of the early South African settlers. We did not find evidence indicating a sexual antagonistic pleiotropy effect, an effect of age of male at first reproduction, or a significant male contribution to the heritability of fecundity.

Given a lack of contraception, the interval between a pair's wedding date and the birth of their first child is dependent on their innate fertility. Beyond a threshold of approximately 1.75 years a couple could be diagnosed as infertile, as conception would not have occurred after 12 months of intercourse without contraception (Augood *et al.*, 1998). This interval has not received a great deal of attention in human fecundity studies to date, with the interval between successive births more often utilized in demographic studies, which is also an indicator of fertility. The wedding till birth interval has been used to determine fecundability, i.e. the probability of conception during a menstrual cycle (Bongaarts, 1975; Knodel, Wilson, 1981). However they did not directly analyze the influence of the length of this interval on lifetime fecundity.

We included both wedding till birth and interbirth intervals in our analyses, but feel that the wedding till birth interval may provide a more reliable proxy for innate fertility. Interbirth interval is potentially more influenced by social practices, for example contraception and breast-feeding induced amenorrhea (Knodel, Wilson, 1981; VanLandingham *et al.*, 1991). The use of wetnurses by the target population (Giliomee,

2003) may have resulted in some unexplained variation in this interval. Peri-natal mortalities may additionally alter the interbirth interval (Bhalotra, van Soest, 2008) so that obtaining a reliable estimate of average interbirth interval may not be possible. Additionally interbirth interval calculation requires the birth dates of multiple offspring, whereas wedding till birth interval can be determined for single offspring. The two terms were not significantly correlated (Pearson's correlation = 0.0177, p-value = 0.7237), meaning that they could be included together in the MAM determination.

Our analysis indicates that an increase in the interval from wedding to first birth resulted in a decline in total fecundity, as would be expected. Every year that this period was extended resulted in 0.5 less offspring (table 6). Similarly each year increase in the interbirth interval resulted in 0.8 fewer offspring being born (table 6). In the multiple marriage database these effects were less, with every year from wedding to first birth resulting in 0.18 fewer offspring in the female subset (table 12) and 0.27 less offspring in the male subset (table 13). Interbirth interval corresponded to a decrease of 0.6 offspring in the female and 0.6 in the male subset for each additional year between births.

Cultural inheritance was identified by the typical fecundity traits of the nationality groups. Nationality was used as a proxy for cultural inheritance, as behaviours and ideas inherent among individuals from the various countries could transmit through successive generations of individual families within this new heterogeneous society. Additionally country of origin may have influenced wealth and social status. Differences in cultural factors, such as wealth and religiosity, between the various nationalities may explain observed differences in the fecundity of these groups.

Historical data indicates that the early Dutch and German arrivals were impecunious males, seeking employment and subsistence. In contrast, the Huguenots arrived at the colony as intact family groups, and despite early prejudice against their minority, they were in general from a higher social class. Mixed race Afrikaners received the greatest social stigma, and formed the lowest social class.

Our analysis indicated that the French (historically the wealthiest subgroup) had on average 1.6 offspring more (table 6) than the inter-racial group (historically the poorest subgroup). This supports the results obtained by Gillespie *et al.* (2008). The mixed European ancestry group are recorded as having the greatest fecundity, but this effect may result from their limited number (only 64 individuals recorded as European mixed in the once married database).

Fecundity was shown to decline over time, at a rate of approximately 0.5 offspring per generation. This decline indicates changing social ideals regarding reproduction. This decline is not likely indicative of a demographic transition (Borgerhoff Mulder, 1998) as no significant evidence was found for the richer subpopulation curtailing reproduction either sooner or at a greater intensity compared to the rest of the population (the interaction term for cohort effect and nationality was not significant). Rather it may be the result of a quantity versus quality trade-off developing due to limited resources (Gillespie *et al.*, 2008).

Female (but not male) age at onset of reproduction significantly influenced final fecundity, with a decline in fecundity observed over time as reproduction was delayed. The greatest decline was observed in women who delayed child bearing until after the age of 30 years (Table 8), corresponding with the previously determined age of decline (Nasseri, Grifo, 1998; te Velde, Pearson, 2002; van Noord-Zaadstra *et al.*, 1991). This effect was as would be expected due to the limitations of the female reproductive lifespan (van Noord-Zaadstra *et al.*, 1991). Any influence of male age is most likely to be a reflection of his wife's age, as male reproductive lifespan is far less rigid (Pettay *et al.*, 2005). Women are unable to produce additional offspring after they complete menopause, while men, despite experiencing a decline in fecundity with andropause, the male counterpart to menopause, can often produce viable sperm into old age. The oldest woman known to have naturally conceived was 59 years old at the time of birth (The Telegraph UK, 20 August 2007), while the oldest father was 90 (Daily Mail UK, 20 September 2010). These were highly unusual cases, and generally couples are limited to complete reproduction when the wife reaches menopause, at approximately 40 to 50 years of age. This explains why the effect of male age at reproduction would be less signifcant, and trends observed are better considered as an effect of the correlation of spousal ages.

Maternal inheritance of fecundity was marginally significant, but not paternal inheritance. Our analysis indicates a heritability estimate in the range 0.14 – 0.16. Our estimate was low in comparison with previous studies (Berent, 1953; Fisher, 1929; Pettay *et al.*, 2005), which may be due to the changeable environment and the numerous cultural and social influences involved. Our result did however correspond with the findings of Imaizumi *et al.* (1970) and Austerlitz and Heyer (1998).

Sequential multiple marriage increased fecundity for both sexes, although the effect was greater for males. Our estimates exceed those of Kaar *et al.* (1998). Their results may have been lower due to the limited sample size for multiple marriages included in their study. Remarriage was more common in our population, allowing for better analysis of its effect. The effect in males would likely be due sequential marriages occurring with younger wives, in effect extending his reproductive lifespan.

That a similar effect could be found for women seems less intuitive, as reproductive period would remain essentially the same between subsequent marriages, and she may in fact lose fertile years in the period between widowhood and remarriage. As insufficient data on dates of death was available, it is possible simply that women who outlived their spouses, and hence remarried, also outlived their counterparts who only married once, and may have died while still in their fertile years. If this was not the case, this data may suggest a novel reproductive trade-off that developed in this population. It is possible that a woman entering into a new marriage with offspring from a previous marriage would have attempted to produce at least one additional child fathered by her new husband, in order cement the new union. In this sense she may have more offspring than might normally have been ideal in order to ensure that her new husband accepts her and her current offspring. Additionally through sequential remarriage she may amass greater assets (Giliomee, 2003), and as such be better equipped to support a larger family.

We did not find significant evidence for sexual antagonistic pleiotropy in this population with regards to fecundity. However genes with this form of antagonistic pleiotropy may be present in individual families, but not be dispersed across the general population. Therefore antagonistic effects may be masked when considering the sample as a whole. To further investigate this problem families who show discrepancy between sons' and daughters' fecundity could be identified, and then additional generations of these families analyzed to examine whether the pattern persists. This was the first study that looked for evidence of sexual antagonistic pleiotropy in this manner, and further research may identify genes that have influenced the fecundity of historical populations in this manner. The low (marginally significant) estimates for heritability in this population imply that genetic factors were not pivotal in determining fecundity. This may be due to a greater availability of resources and a small initial population. The negligible action of genetic factors may explain the lack of significant pleiotropy.

Evidence that reproductive status was influenced by birth order was found. Firstborns tended to produce larger offspring numbers than their later born counterparts (table 10), supporting Michalski and Shackelford's (2002) study which found that firstborns pursued a more stable reproductive strategy than laterborns. Additional analysis indicated that in larger families firstborns tended to curtail their fecundity, possibly as a kinship effect as they played a role in supporting their parents in rearing their younger siblings (Brown, Pimm, 1985).

In conclusion, we have shown that the interval between wedding and first birth significantly influences lifetime fecundity by acting as a reliable proxy for fertility; that both genders may experience a fitness gain through sequential remarriage; and that evidence supports the role of cultural inheritance in influencing fecundity. Additionally we were able to calculate a heritability estimate for fecundity in this population and illustrate how the changing social climate coincided with changes in fecundity. The wedding to first birth interval is of import as it indicates that a new approach may be called for in demographic fertility studies. The potential of this term over interbirth interval would need to be considered. The increase in female fecundity with sequential remarriage is a novel discovery, and while the extra child may serve a social role of cementing the family, the actual number of children surviving to adulthood still needs to be investigated.

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Appendix A

Factors influencing human fecundity and their effect sizes as shown by previous studies

Table A1: Factors found to affect fecundity and references

	Examples		Odds ratio for subfertility or infertility	Pearson r correlation	Reference
Heritability	Maternal line		1.4	0.23	(Kruuk *et al.*, 2000)
Environment	Nutritional status	Malnourishment	1.36[1]	0.326	(Anderson, McCabe, 1977)
		Obesity	1.8[1]		(Lake *et al.*, 1997)
			1.7[2]		(Shiri *et al.*, 2004)
	Smoking	Active	1.98[1]		(Howe *et al.*, 1985)
			1.6[1]		(Augood *et al.*, 1998)
			1.5[2]		(Shiri *et al.*, 2004)
		Passive	1.14[1]		(Hull *et al.*, 2000)
	Alcohol		1.08		(Hakim *et al.*, 1998)
	Caffeine		1.09		(Hakim *et al.*, 1998)
	Chemical Contaminants	PCB congeners		0.155	(Buck *et al.*, 2002)
Cultural inheritance	Wealth		1.3*		(Gillespie *et al.*, 2008)
	Remarriage		1.3*[2]		(Kaar *et al.*, 1998)
			1.1*[1]		(Kaar *et al.*, 1998)

* Odds Ratio given for fecundity
[1] Value for female
[2] Value for males