Life-History Evolution, Human

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Glossary

Demographic transition The change from high birth and death rates to low ones as a country develops from a preindustrial to an industrialized economic system.
Genome-wide association study (GWAS) A study determining the genomic regions underlying traits of interest by assessing statistical associations between traits and the genotypes of many individuals at many thousands of loci across the genome.
Heritability The additive genetic variance in a trait, divided by the total phenotypic variance in that trait: $h^2 = V_A/V_P$. The additive genetic variance is the variance accounted for by additive effects on that trait that is due to the alleles an individual possesses.
Life-history theory A branch of evolutionary theory that explains aspects of anatomy and behavior by determining how patterns of development, growth, reproductive schedule, and lifespan have been shaped by natural selection.
Pleistocene period The geological epoch spanning approximately 2.5 million to 11 700 years before the present, when modern humans evolved.
Quantitative genetics The branch of genetics which studies how genes contribute to variation in complex traits determined by many genetic loci, such as size or fecundity.
Quantitative trait loci (QTLs) Regions of the genome that are linked to, or contain, genes which explain a part of the genetic variation in a complex trait.
Single nucleotide polymorphism (SNP) A DNA sequence in which a single nucleotide (A, C, T, G) differs between members of a species or between paired chromosomes.

Then and Now: The Unusual Life History of Humans

The human life history is characterized by several puzzling aspects. Compared to our closest relatives, the other great apes, humans have a delayed onset of reproduction; a short birth interval between each offspring; a complete cessation of reproductive capacity in females (menopause); and a long post-reproductive lifespan (Mace, 2000; Hawkes and Paine, 2006). Evolutionary biologists use 'life-history theory' to determine how these unusual patterns have evolved. Traditionally, studies on human life-history evolution have focused on the past, attempting to determine what genetic and environmental factors favored the evolution of these unusual life-history characteristics. Consequently, most studies on human life-histories have focused on contemporary 'traditional' societies, such as extant hunter-gatherer groups (see Hawkes *et al.*, 1997), whose lifestyle is thought to resemble that during the 'Pleistocene period' when many key human characteristics evolved.

This focus on a limited subset of human societies may hinder our attempts to determine how human life-histories evolved, since there is remarkable variation across cultural and ecological contexts. For example, average age at first reproduction can differ by 10 years even between populations with no access to medical care and contraception (Walker *et al.*, 2006); average family size ranges from little over one in contemporary Europe to around ten in the Hutterites, an Anabaptist group who practice communal living and shun birth control (Tietze, 1957); and the age when women have their last child differs by several years between populations. An increasing number of researchers have therefore begun to determine how this variation arises, including asking to what extent human life-history traits are still evolving in contemporary populations. This is particularly significant in the wake of the recent transition of many contemporary populations to dramatically reduced mortality and fertility rates (the 'demographic transition'), associated with industrialization and the introduction of effective contraception and medicine ('industrialized populations'). Determining how such rapid environmental changes may have altered selection on life-history traits and associations between them will be important for predicting how human life-histories may evolve in the future and the epidemiology of health and disease.

The question of whether or not contemporary human populations are undergoing evolutionary change has been a subject of debate in many disciplines. The major argument against the continuing evolution of human life-histories has been that, thanks to modern medicine and improved nutrition, nearly all children survive to adulthood in industrialized countries, which starkly contrasts with child mortality rates in preindustrial populations (Gagnon *et al.*, 2009; Rickard *et al.*, 2010; Gillespie *et al.*, 2013). Thus, natural selection through survival is weak in industrialized populations, but survival alone does not guarantee descendants in future generations. Only individuals that reproduce gain fitness, and while survival is a prerequisite to reproduce, natural selection ultimately acts on variation in reproductive success. Therefore, even if a set of individuals all survive to reproductive age, they may vary substantially in their ability to find a mate and produce and raise children, and this indeed is the case in industrialized populations (Byars *et al.*, 2010; Stearns *et al.*, 2010). This observation is crucial to the study of human life-history evolution, since any trait associated with variation in lifetime fitness can be under natural selection.

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Phenotypic Correlations between Human Life-History Traits and Fitness

The traditional approach to studying life-history evolution has been to quantify phenotypic correlations between life-history traits and fitness. This approach has revealed that both the opportunity for selection (variation between individuals in fitness) and strength of selection on life-history traits, such as age at first and last reproduction, can be substantial in industrialized populations (Stearns *et al.*, 2010) and that selection pressures can change rapidly over time (Moorad, 2013). For example, in a contemporary Gambian population going through the 'demographic transition,' selection reversed from favoring decreased height and increased BMI before the transition to favoring increased height and decreased BMI after the transition, over a period of less than 60 years (Courtiol *et al.*, 2013). This change probably arose at least partially from improvements in the predictability of food supplies, which led to reduced need to carry energy reserves that allowed girls and women to survive past the annual hungry season.

A Crash Course in Quantitative Genetics

Although studies of phenotypic selection on human life-history traits are informative, selection on phenotype alone does not lead to evolution. The 'breeder's equation' (**Box 1**) shows that only traits that have 'heritable' genetic variation can show an evolutionary response to selection (Falconer and Mackay, 1996). Only recently have studies on humans followed the lead of studies of natural populations of animals (Kruuk, 2004; Wilson *et al.*, 2010; Charmantier *et al.*, 2014) in applying methods that estimate selection, heritability, and evolutionary responses. Applying these methods would allow us to predict how traits under selection can evolve over time, which is entirely feasible, given recent evidence for phenotypic selection operating in modern societies (Byars *et al.*, 2010; Stearns *et al.*, 2010; Courtiol *et al.*, 2013; Moorad, 2013), and evidence that many of these traits are heritable (de Bruin *et al.*, 2001; Kirk *et al.*, 2001; Pettay *et al.*, 2005; Milot *et al.*, 2011; Vink *et al.*, 2012; Bolund *et al.*, 2015). Life-history traits show considerable among-individual variation, which may be driven in part by social (e.g., age at marriage) or cultural factors (e.g., contraception), but each trait also has a complex genetic basis and is influenced by many genetic loci. For example, age at first birth depends on social practices, but has a substantial genetic basis in modern populations, with an individual's genes explaining ~11% of the total phenotypic variation (Stearns *et al.*, 2010). These genes may influence characteristics underpinning an individual's age at first reproduction, such as behavior, personality, appearance, and reproductive physiology. The study of such complex quantitative traits is the subject of quantitative genetics.

Quantitative genetics is based on estimating the genetic contribution to phenotypic similarities between relatives. By measuring the phenotypes of many individuals and coupling this with their relatedness information, the phenotypic variation in traits can be divided into contributions from genetic and environmental factors (**Figure 1**). A traditional approach has been to compare sets of relatives, such as parents and their offspring, or different types of siblings. Based on information about the 'average' relatedness of these sets of relatives, the proportion of variation in a trait due to genetics can be

Box 1 Predicting Evolution: The Breeder's Equation

$$R = h^2 S$$

This simple equation states that the evolutionary response (R) to selection of a trait is a product of the selection differential S (the difference in mean trait value between individuals that reproduce and the mean trait value of the population), and the heritability h^2 of the trait. This univariate Breeder's equation was developed for predicting a response to artificial selection by animal breeders. However, in wild populations, where selection acts on many correlated traits simultaneously, the multivariate form of this equation is more effective:

$$\Delta \mathbf{z} = \mathbf{G}\beta$$

The multivariate breeder's equation calculates the expected change in several traits at once: the vector of changes of mean trait values $\Delta\mathbf{z}$ is the product of the genetic variance-covariance matrix $\mathbf{G}$ (the diagonal elements $\sigma^2_{1,1}$ and $\sigma^2_{2,2}$ are the genetic variances for traits 1 and 2, and the off-diagonal element $\sigma_{1,2}$ is the genetic covariance between the traits) and the vector of selection gradients β:

$$\begin{pmatrix} \Delta z_1 \\ \Delta z_2 \end{pmatrix} = \begin{pmatrix} \sigma^2_{1,1} & \sigma_{1,2} \\ \sigma_{1,2} & \sigma^2_{2,2} \end{pmatrix} \times \begin{pmatrix} \beta_1 \\ \beta_2 \end{pmatrix}$$

Here, the response of traits 1 and 2 to selection additionally depends on the genetic covariance between the traits and the strength of selection on the other trait:

$$\Delta z_1 = \sigma^2_{1,1}\beta_1 + \sigma_{1,2}\beta_2$$
$$\Delta z_2 = \sigma^2_{2,2}\beta_2 + \sigma_{1,2}\beta_1$$

Thus, the response to selection on one trait depends on the degree to which genes associated with that trait are also associated with related traits under selection. However, the Breeder's equation assumes that any association between a trait and fitness are causal, and that there are no unmeasured traits. This assumption may be unlikely to be met (Hadfield, 2008; Morrissey *et al.*, 2010), and so an alternative model has recently been advocated for the study of natural populations (Morrissey *et al.*, 2012). This model is known as the Robertson–Price identity, or Robertson's secondary theorem of natural selection (Robertson, 1966; Price, 1970), and states that evolutionary change can be calculated as the additive genetic covariance between a trait and fitness:

$$\Delta \bar{z} = \sigma_a(z, w)$$

This equation does not assume causality between trait and fitness: evolution is predicted by directly estimating the genetic aspect of the trait–fitness association. This change may be due to selection on an unmeasured, genetically correlated trait and so the equation does not identify the true form of selection, but unlike the breeder's equation it does provide a direct prediction of evolutionary change. These methods are vital for determining how life-history traits have evolved and how they may respond to selection in the future.

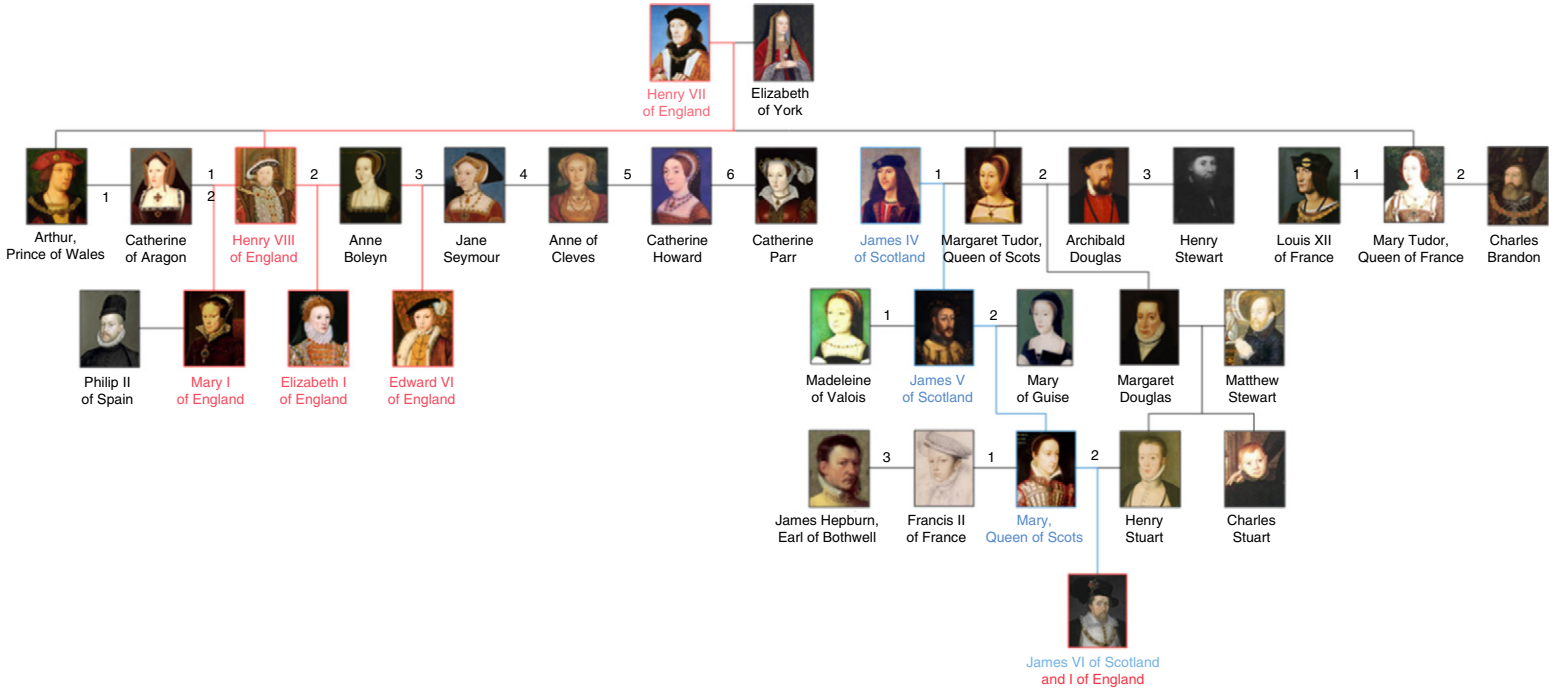

Figure 1 A pedigree showing the Tudor family tree, the dynasty of English monarchs, 1485–1603. Simple quantitative genetic analyses might calculate genetic parameters using trait values from parents and offspring (e.g., Henry VII and Henry VIII), or half-siblings (e.g., Mary I and Elizabeth I). The 'animal model' (see text) accounts for phenotypic information from all relatives: for example, James VI has 18 relatives in the pedigree depicted.

calculated: for example, monozygotic twins, full siblings and half-siblings share 100%, 50%, and 25% of their genes on average.

A drawback of this approach is that relatives share environments as well as genes: an individual's early puberty may arise because of genes inherited from their parents, or because they received a similarly good diet as their parents. In laboratory studies, this can be addressed by using breeding designs to accurately quantify genetic and environmental effects on specific traits (Falconer and Mackay, 1996; Lynch and Walsh, 1998). Such manipulations are usually not possible in studies of wild populations, which must rely on the often incomplete and unbalanced data that are available. The same is true of human populations: close relatives commonly share environments and many traits are affected by cultural transmission, which upwardly biases heritability estimates calculated using traditional methods.

Another nonexperimental situation is in populations of domesticated animals, which include many different levels of relatives, some of which share genes but not environments. A statistical approach to estimate heritability of traits in such situations, the 'animal model,' was developed over 50 years ago (Henderson, 1950, 1975). It estimates the heritability of quantitative traits by using pedigrees (**Figure 1**), and has been successfully applied to answer a wide range of evolutionary questions in wild animal populations (Kruuk, 2004; Kruuk and Hill, 2008; Wilson *et al.*, 2010; Charmantier *et al.*, 2014). The animal model is able to adjust for confounding factors, and therefore can account for aspects of cultural transmission measured in pedigreed humans, such as socioeconomic status, in the model structure. The heritability estimate from such a model quantifies the genetic contribution to phenotypic variance, accounting for the fact that individuals within one socioeconomic group are likely to be more similar to each other than to individuals from other socioeconomic groups.

Several multigenerational pedigree datasets, collected for clinical, medical, or demographic reasons, but also suitable for quantitative genetic analyses are now available. Historic pedigree data on humans use social information to determine parentage, though this should not be a limitation given the low extra-pair paternity (EPP) rates in humans (Anderson, 2006) and the robustness of quantitative genetic models to low EPP rates (Charmantier and Reale, 2005; Firth *et al.*, 2015). The availability of such pedigree datasets and statistical methods means that an evolutionary perspective on human life-histories is gaining momentum.

Case Studies on Using Quantitative Genetics to Understand Life-History Evolution in Humans

Life-History Traits are Heritable

A system of data collection originally designed to enforce stringent tax collection may seem an unlikely source of data on human life-history evolution, yet a system of Finnish church records dating back to the seventeenth century has increased our understanding of human life-history evolution (**Box 2**). This dataset and others like it, comprising information on relatedness, survival, reproduction, and socioeconomic standing, have been used to determine the genetic basis of key life-history traits and how selection acts upon them. A landmark study using the Finnish dataset estimated heritabilities and genetic correlations of several important life-history traits (Pettay *et al.*, 2005). Heritabilities of female life-history traits were largely significant (estimates ranged 0.18–0.47 for number of children, number of surviving children, the time interval between births, age at last reproduction, and lifespan). Interestingly, bivariate models also revealed evidence for genetic trade-offs: a genetic correlation between inter-birth interval and lifespan suggested that genes associated with producing children rapidly were associated with a shorter lifespan. These results showed that selection could have shifted the average birth interval over time, due to the additive genetic basis of this trait, but that a response to selection could have been constrained because of genetic correlations between traits (Pettay *et al.*, 2005). In this case, selection for faster birth rate

Box 2 The Finnish tax system during the eighteenth century, and how it unexpectedly aided the study of human life-history evolution

The King of Sweden, of which Finland was formerly a province, decreed in 1749 that the Lutheran church was obliged to document all births, marriages, deaths, and movements in the whole population for tax purposes. At the time, the Finns mostly depended on farming for their livelihood, supplemented with fishing and hunting. The standard of living was low with famine and disease being common, and with the main causes of death being infectious diseases associated with malnourishment (Turpeinen, 1978; Hayward *et al.*, 2012). Genealogists have used such records to compile multigenerational datasets with accurate information on individual survival, reproduction, family configuration, and dispersal for up to 15 generations. Industrialization began relatively late in Finland, and fertility and mortality rates were high until this occurred in the early twentieth century (Liu *et al.*, 2012). This dataset offers rare opportunities to follow the same genetic lineages from 'natural' mortality and fertility periods through the different phases of the demographic transition until the modern day. Finnish church records also provide information on occupations of adult men, allowing classification of individuals into different socioeconomic groups. Distinguishing different levels of resource availability is important as it is often associated with survival, reproductive success, and selection on different life-history traits (Pettay *et al.*, 2007; Courtiol *et al.*, 2012). Similar datasets on historical human populations are also available for many other countries, and they are increasingly used in evolutionary research.

An example of a church record page (left) detailing life-events of historical Finnish families (on the right).

would also lead to indirect selection for shorter lifespan, with the likely overall outcome of selection being a compromise between such alternative outcomes. Such trade-offs are one of the forces maintaining genetic variation in key life-history traits (Stearns, 1992).

Genetics of Life-History Traits in the Two Sexes – an Arena for Conflict

Genetic correlations between traits can constrain independent evolution in the two sexes because, while the sexes share much of their genome, their evolutionary interests are often not aligned (Parker, 1979). If the sexes have different phenotypic optima for a trait, different selection pressures act in the two sexes. Coupled with a shared genetic basis of the trait (a high cross-sex genetic correlation, r_{MF}), this leads to conflict over optimal trait expression, or intra-locus sexual conflict (Lande, 1980; Bonduriansky and Chenoweth, 2009). Studies on the Finnish population have revealed that age at first and last reproduction, reproductive timing and reproductive rate were all strongly genetically correlated with fitness in both sexes, and thus under selection (Bolund *et al.*, 2013). Further, there were no differences between the sexes in these genetic correlations between life-history traits and fitness. The genetic correlations between the sexes (r_{MF}) were also high, suggesting that, for example, genes associated with an early age at first reproduction in women are also associated with an early age at first reproduction in men. This indicates that the sexes cannot reach their sex-specific optima of trait expression even if selection pressures are different between males and females, as was indeed the case. These results likely reflect the strictly monogamous mating system in preindustrial Finland, which meant that an individual's reproduction was severely constrained by that of their partner. Thus, the sexes constrained each other's independent evolution. Given that humans exhibit many different mating systems (monogamy, polygyny, polyandry, polygynadry), they offer the opportunity to investigate how changes in mating systems affect selection pressures and genetic correlations. For example, over the reproductive lifetimes of Utahans born between 1830 and 1894, socially induced reductions in the rate and degree of polygamy corresponded to a 58% reduction in the strength of sexual selection (Moorad *et al.*, 2011). This illustrates the potency of sexual selection in polygynous human populations and the dramatic influence that societal changes can have on evolutionary processes. Intra-locus sexual conflict can also have important implications for health in modern populations, as shown by recent work using the ongoing Framingham Heart Study in the USA, which showed that evolution of a contemporary population may be constrained by genetic conflict between the sexes (Stearns *et al.*, 2012). In this population, selection favored shorter women and taller men, but female height was negatively genetically correlated with male cholesterol. Thus, selection for shorter women could lead to higher cholesterol levels in men. The possibility that complex traits associated with diseases can have a sexually dimorphic genetic basis which is maintained by disparate selection pressures in the sexes is an exciting emerging area of study.

Genetics of Life-History Traits over the Lifetime

As well as differing between the sexes, the expression of additive genetic variance is often observed to increase with age in diverse taxa (Réale *et al.*, 1999; Charmantier *et al.*, 2006; Von Hardenberg *et al.*, 2007; Wilson *et al.*, 2007, but see Brommer *et al.*, 2007). Evolutionary theories of senescence predict this is due to the accumulation of late-acting deleterious mutations, which are not selected against due to the declining force of selection with age (the mutation accumulation theory of ageing; Medawar, 1952). Senescence may also have evolved through antagonistic pleiotropy (Williams, 1957), or the effects of genes promoting fitness in early life at the expense of fitness in later life, a scenario which would lead to antagonistic genetic correlations between early- and later-life fitness. Quantifying age-specific changes in additive genetic variance is thus crucial if we are to determine the evolutionary mechanisms for senescence (Wilson *et al.*, 2008). In the preindustrial Finnish population, the additive genetic variance for female fecundity increased with age (Pettay *et al.*, 2008), due to changes in maternal and genetic variance for fecundity. Before age 31, maternal identity explained 25% of the variance in fecundity, and the additive genetic effect only 13%; after the age of 31, the maternal effect accounted for only 2% of the variance in fecundity, and the additive genetic effect increased to 25%. This suggests that early in reproductive life, mothers may have played a substantial role in enhancing their daughters' fecundity, perhaps by proving childcare through a 'grandmother effect' (Hawkes and Coxworth, 2013; Lahdenperä *et al.*, 2004). However, the Finnish study found no evidence for a negative genetic correlation between fecundity at early and later ages, and thus no evidence for the antagonistic pleiotropy theory of ageing. Further research using these approaches could determine whether the trade-off between fecundity and survival changes with age, testing the prediction that the menopause could have evolved as a result of diminishing benefits and increasing costs of reproduction with increasing female age (Williams, 1957; Hawkes and Coxworth, 2013).

Genetics of Life-History Traits Over Time and Across Environments

A major goal of evolutionary quantitative genetics is to predict evolutionary change over several generations: given current selection pressures and patterns of genetic (co)variance, how will traits respond to selection and be expressed in the future? To this end, the Framingham heart study measured the strength of selection, estimated genetic (co)variation, and predicted gradual evolutionary change in several life-history and health traits in the contemporary USA. The descendants of the study women were predicted to become on average slightly shorter and stouter; have lower total cholesterol levels and systolic blood pressure; have their first child earlier and reach menopause later than they would in the absence of selection (Byars *et al.*, 2010). Two important caveats to these predictions are that both the selection pressures and the genetic (co)variance may change rapidly. Changes in climate have been shown to affect selection and patterns of genetic (co)variation in wild animals (Garant *et al.*, 2008; Björklund *et al.*, 2013) and could

have profound effects on human populations, particularly with regard to changing patterns of disease spread (Lafferty, 2009; McMichael, 2014). Generally, the genetic (co)variance of traits (conceptualized as **G**, **Box 1**) are predicted to change over time and across environments (Roff, 2000) but little is known about how rapidly this happens (Steppan *et al.*, 2002; Arnold *et al.*, 2008). This has important implications for predicting evolutionary change, because such predictions rely on stability of **G** and β (**Box 1**). Thus, it is important to reveal how rapid changes in culture and technology are changing human biology. A study on the Finnish dataset found that the **G**-matrix of four key life-history traits (age at first and last reproduction, lifespan and lifetime reproductive success) remained largely stable over the demographic transition in Finland, but showed a trend for increased additive genetic variance (increased evolutionary potential of the population) after the demographic transition (Bolund *et al.*, 2015). Similarly, recent studies of other populations found changes in the additive genetic variance of age at first reproduction over a 140-year period (Milot *et al.*, 2011) and female fecundity over a 100-year period (Kohler *et al.*, 2002). Such changes in patterns of genetic (co)variance mean that projections of evolutionary change over more than a few generations are likely to be unreliable.

Studying Human Life-History Evolution in the Genomics Era

The last 20 years have seen rapid progress in genomic technologies. A major aim of medical genetics is to identify 'quantitative trait loci' (QTLs) associated with risk of developing diseases including cancer, heart disease, and mental illness (Plomin *et al.*, 2009; Visscher *et al.*, 2012). Sequencing the human genome has enabled the development of 'genome-wide association studies (GWAS),' which assess an individual's genotype at many thousands of 'single nucleotide polymorphisms (SNPs)' across the genome (Donnelly, 2008; Hindorff *et al.*, 2009). GWAS can also provide insight into the genetics underpinning life-history traits, as has been demonstrated in wild populations (Slate *et al.*, 2010; Jensen *et al.*, 2014). Combining genome sequence data with pedigree and life-history data could determine how individual genetic loci underlie important associations between life-history and health traits. A recent study found a negative genetic correlation between the lifetime number of children born to women and their lifespan, and then identified five SNPs that were associated with this relationship (Wang *et al.*, 2013). However, this result was not robust to changes in the sample, illustrating the difficulty in identifying QTLs that underlie variation in quantitative traits that are likely to be influenced by hundreds or even thousands of loci.

In summary, application of quantitative genetic techniques can help us to determine how cultural and environmental changes have led to new selection pressures and, importantly, what the evolutionary consequences of these changes could be. The observation that our biological nature continues to evolve has important implications for public health and demographic forecasts because it will allow us to predict human evolutionary responses to a rapidly changing world.

See also: Aging: Why Do We Age?. Evolutionary Genetics, History of. Human Life Histories, Evolution and. Life History Trade-offs. Schools of Classification

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