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Demographic Behaviour and Behaviour Genetics

Atam VETTA* and Daniel COURGEAU**

With the race to decode the human genome, the prestige of research in biological genetics is higher than ever. Its incursion into demography has long been viewed with enthusiasm but also with reservations. Atam VETTA *and* Daniel COURGEAU *here set out the problems associated with the heritability analysis promoted by behaviour genetics. Going back to the work of Fisher (1918), the authors examine the principles of this analysis and criticize the mathematical formulas it uses, which are assimilated by demographers despite being marred by algebraic error. Their argument also rests on the belief that individual behaviour is explained largely by the social, political and economic conditions in which individuals live. In conclusion they argue that this current of genetics, which emerged in the early twentieth century, is outdated in the age of the genome and thus cannot provide a legitimate model for the study of human behaviour.*

Recently, a number of researchers have published articles in major demographic journals (Kohler et al., 1999; Foster, 2000; Morgan and King, 2001; Rodgers et al., 2001), arguing that the methods of quantitative genetics based on Fisher (1918), and the model fitting approach used by behaviour geneticists in particular, should be used to study demographic behaviour. Other demographers support this view, because they believe that it is necessary to consider the impact of behavioural genetics on demographic behaviour (Coleman, 2002; Hobcraft, 2002). The links between genes and human reproduction (fertility and other fitness traits) are also the subject of interdisciplinary studies. New books (Rodgers et al., 2000; Rodgers and Kohler, 2003) consider various questions in this field. Previously, the behaviour genetics approach has been used in a number of social science areas. It has been used for over 30 years in psychology (Herrnstein and Murray, 1994; Dunne et al., 1997; Segal and McDonald, 1998; for more references, see Capron et al., 1999), geronto-

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logy (McGue et al., 1993), sociology (Lichtenstein et al., 1992), psychiatry (Kender et al., 2000), and so on. The Behavior Genetics Society and its journal *Behavior Genetics* are dedicated to research using this methodology. The central point of these studies is the claim that there is a genetic component in behavioural traits, and that the contribution of this component to the variance of the traits in the population can be measured. Demographic traits for which this claim is now being advanced include fertility, mating success, longevity, juvenile survival, divorce, etc. The psychological and medical traits include "intelligence" as measured by IQ scores (Pedersen et al., 1992), personality (DiLalla et al., 1996), alcoholism (Blum et al., 1990), smoking (Kender et al., 2000), homosexuality (Eckert et al., 1986), femininity (Bouchard and McGue 1990), morningness-eveningness (Hurr et al., 1998), aggression, hostility and anger (Gustavson et al., 1996), obesity (Brookman and Bevoral, 2002), soda or fruit juice intake (de Castro, 1993), etc.

Galton's (1869) *nature–nurture* division is illusory. The two effects cannot be separated for any human trait. We explain the genetic concepts used in behaviour genetics model fitting and the concept of heritability, as well as their deficiencies. We suggest another concept to study inheritance of a trait. Most human traits involve assortative mating, but behaviour geneticists use incorrect formulae when they fit models involving assortative mating (Capron et al., 1999). We explain why Fisher's (1918) formulae are wrong and discuss the algebraic error in Jinks and Fulker (1970). We enumerate some factors that affect fertility and give examples of how molecular biology and genomic research, and specifically the unravelling of the species' genome, are increasing our knowledge of demographic behaviour.

I. Definitions and genetic terminology

The name "behaviour geneticist" is used by two distinct groups of researchers. One group specializes in laboratory experiments on animals. Their experiments are well designed and well executed. We acknowledge their contribution to science and this paper does not relate to their work. The other group of "behaviour geneticists" acknowledge their debt to Jinks and Fulker (1970). They do not conduct experiments and fit *statistical* models of the components of variance type to *observed* data. They could be described as *observational* behaviour geneticists. The parametric values obtained from fitted models, they believe, enable them to solve the nature–nurture problem. Examples in the Introduction relate to them and we are, primarily, concerned with questions and problems associated with their work. As not all readers of *Population* are specialists in population genetics, we define the genetic terms and concepts used in this article. Those familiar with the genetic terminology may go directly to Section II.

The basic unit of human heredity is a "chromosome". The name arises from the fact that chromosomes have an affinity for certain stains (*chroma* = colour and *soma* = body) and is due to the nineteenth century German biologist Walther Flemming. The fundamental hereditary material in a chromosome, DNA (deoxyribonucleic acid) is composed of a double-stranded helix of sugar phosphate held together by pairs of nucleotide bases, that carry information by means of the linear sequence of its nucleotides. Humans have 23 pairs of chromosomes (46 chromosomes). In females all 23 pairs are identical. In males 22 pairs are identical but the 23rd pair, called the sex chromosome, is not identical. A gene is a molecule of DNA situated on a chromosome. It can have many forms that are known as "alleles". The exact position at which a gene is situated is called a "locus". On each homologous chromosome, an allele of the gene will be situated at the same position. The whole set of genes carried by a species is called the "genome" of the species. If a person has two identical alleles at a locus, he or she is "homozygous", and otherwise, "heterozygous". Among humans, germ cells (eggs and sperm) are produced by a process called *meiosis*. It is a type of cell division that reduces the amount of genetic material. Thus, each egg and sperm has only 23 chromosomes. When a sperm impregnates the egg, each of the 23 chromosomes in the sperm joins its counterpart in the egg and the process of forming a human begins with 23 pairs of chromosomes. An individual's "genotype" is the complete set of all alleles at all loci. The human genome has about 25,000 genes.

Mendel was the first to study a qualitative trait. A Mendelian or qualitative trait is under the control of one gene residing on a chromosome pair. Let's assume that this gene has two alleles, *A* and *a,* one on each chromosome of a pair. As we receive one allele each from mother and father, the population will consist of three genotypes *AA*, *Aa* and *aa* with respect to this gene (we do not distinguish between *Aa* and *aA*). When we can distinguish between the genotypes, the trait is known as a qualitative trait and we can study the effect of the gene. Blood groups are an example of a qualitative trait. A Mendelian trait may exhibit dominance. If, for example, allele *A* is completely dominant over allele *a*, then *Aa* looks like *AA*. If dominance is partial, then *Aa* will be closer to *AA* than to *aa*.

Behaviour genetics is not concerned with qualitative traits. It is concerned with *quantitative* traits. A quantitative trait is determined by a large number of genes. Consider a second gene *B*. It will also have three genotypes *BB, Bb* and *bb.* Thus, two genes will give rise to 9 genotypes (each of the three *A* genotypes combining with each of the three *B* genotypes, i.e. *AABB, AABb, AAbb, …aabb*). For *n* genes, the number of genotypes will be 3^n. A quantitative trait, e.g. height, is measured on a continuous scale. Some genotypes may give rise to similar phenotypes and we may not be able to distinguish between these genotypes. Thus, there is no one-to-one correspondence between genes and their effect. Environment may

also affect the trait, in which case an individual's phenotype may not be a true reflection of the genotype.

A behaviour geneticist collects data on the phenotype of a trait and then tries to make inferences about the genotype. A phenotypic value needs to be associated with the underlying genotypic value or with the genotype. Without association, no genetic inference can be made. Therefore, new concepts not used in Mendelian genetics are needed. "Genotypic value" is one of these new concepts. Regrettably, it can be defined for one gene only and then, inappropriately, "generalized". The genotypic values of the three genotypes *AA, Aa* and *aa* are defined as the *regression* of their phenotypic values on genotypic frequencies. As genotypic values are hypothetical and their exact values are unknown, this line of regression cannot be found. Another new concept, that of "additive value", is needed. The same trick is played, and additive values are defined as the *regression* of genotypic values on genotypic frequencies. Additive values are also hypothetical, and may or may not exist. The deviations from this hypothetical regression of genotypic values on genotypes are called "dominance values". In Mendelian genetics, dominance effects are real. Fisher (1918) assumed that dominance values are random fluctuations from the hypothetical line of regression of genotypic values on genotypes and this convention is retained in Quantitative Genetics (Falconer, 1972). This distinction is not generally understood. To explain the concept of additive values, textbook writers give genotypes *AA*, *Aa* and *aa* hypothetical values a, d and –a (please note that equally spaced values for the three genotypes would not reflect "dominance"). This, however, does not mean that they are "real". We emphasize that genetic, additive and dominance values are hypothetical statistical constructs and may or may not exist.

II. Current methodology and hypotheses of behaviour genetics analysis

The methods of quantitative genetics assume that measurements follow a ratio scale. Such a scale has a zero point, and ratios of numbers reflect ratios of magnitude. Regrettably, this is not the case with some psychological and behavioural measurements, e.g. IQ (McInerney, 1999; Capron et al., 1999). In demographic studies where childless families are ignored, the distribution is truncated. We do not discuss the genetic analysis of truncated distributions or "folded" distributions. We assume that the data are, in fact, in ratio scale. Fisher (1918) proposed the hypothesis that a continuous trait is determined by a large number of genes, each having a small summary effect (the name "polygenic" was coined by Mathur, a student of Fisher, in 1946). He obtained formulae for kinship correlations on this hypothesis assuming (1) random mating and (2) assortative mating.

We do not explain his theory here but note that most human traits involve assortative mating. Regrettably, his theory of assortative mating is not easy. Nonetheless, behaviour geneticists need to master it.

When dealing with effects of more than one gene, assumptions concerning the effects of combining two or more genes are required; for example, are they multiplicative or additive? Fisher assumed that the effects of all genes are additive, i.e. there is no covariance or interaction between genes. Hence, his model is known as the additive model. He defined "environment" as "arbitrary external causes independent of heredity". This implies that environment is independent of genes and random with a mean of zero and unknown variance. We note that the assumption of random environment is not valid for most human behavioural traits. Thus, the behaviour genetics model is additive in two respects: (1) the effects of genes are added and (2) genetic and environmental effects are added. We sketch the theory as used by behaviour geneticists.

Fisher and behaviour geneticists make the following assumptions to develop a quantitative genetics model. These assumptions are generally not clearly stated.

(i) Polygenes act additively.

(ii) Polygenes segregate independently.

(iii) Environment is independent of genes and random.

(iv) The population is in Hardy-Weinberg equilibrium.

(v) To simplify the algebra, Fisher assumed that the number of polygenes is infinite.

Please note that when alleles *A* and *a* at one locus and *B* and *b* at another locus segregate independently in a population, the probabilities of the four combinations *Ab*, *AB*, *aB* and *ab* are equal, i.e. 0.25. If, however, probabilities of some combinations are greater than other combinations, for example due to assortative mating, these alleles are said to segregate "together". Hardy-Weinberg equilibrium means that there is no change in gene or genotypic frequencies from one generation to the next, i.e. no migration, assortative mating, mutation or selection of any type. Assume that the genetic effect of the gene on the *i*th locus is given by the equation, $g_i = a_i + d_i$, where a_i is its additive effect and d_i is its dominance deviation. Fisher assumed that dominance deviations are random. Using assumption (i) and (ii) for all genes:

$$\sum_i g_i = \sum_i a_i + \sum_i d_i$$

This equation is generally written as:

$$G = A + D \qquad [1]$$

Using the statistical concept of *expectation*, under Fisher's assumptions, we get the equation:

$$Var\,G = Var\,A + Var\,D \quad [2]$$

where *Var G* is the "genotypic variance". *Var A* and *Var D* are additive and dominance variances respectively. In [2] it is assumed that there is no covariance or interaction between genes.

Assuming an independent environment (iii), we can write:

$$Var\,P = Var\,G + Var\,E \quad [3]$$

where *Var P* and *Var E* are, respectively, the phenotypic and environmental variances.

Note that if assumption (iv) is violated, e.g. if the frequency of an allele changes from one generation to the next, then both the additive and dominance variances and, consequently, genetic variance will change and the simple structure given above will not exist.

For a population mating at *random*, Fisher found the genetic sib covariance and genetic parent-child covariance, which in modern terminology are written as:

$$Cov(sibs) = 0.5VarA + 0.25VarD$$

and

$$Cov(parent - offspring) = 0.5VarA$$

Note that:

$$Cov(sibs) > geneticCov(parent - offspring)$$

Fisher did not consider monozygotic (*MZ*) twins and did not find their covariance. As all of their genes are common,

$$Cov(MZ) = VarA + VarD$$

In setting up these equations we ignored the contribution of environment. Assuming random environment, the phenotypic covariance between *MZ* twins reared together will be:

$$Cov(MZ) = VarA + VarD + VarCe$$

where *Var Ce* is the variance due to common environment. It is not necessary to use covariances and sometimes a correlation matrix is used. Thus, a behaviour geneticist collects data, calculates covariances (or correlations), equates them to theoretical correlations, and solves these equations to find estimates of parameters such as *Var A* and *Var D*. The amount of genetics used is minimal.

There is, however, a mathematical restriction on the number of equations required if a unique solution is to be found. This restriction is that the number of equations must equal the number of parameters to be estimated. This is known as the *minimal* set. In the absence of a minimal set, a unique solution is not possible. If estimates of only three parameters e.g. *Var A*, *Var D*, and *Var Ce* are required, a minimal set of three equations is needed. This set could have the format:

$Cov(MZa)$ (phenotypic covariance between monozygotic twins reared apart) $= VarA + VarD$

$Cov(DZt)$ (phenotypic covariance between dizygotic twins reared together) $= 0.5VarA + 0.25VarD = VarCe$

and

$Cov(DZa)$ (phenotypic covariance between dizygotic twins reared apart) $= 0.5VarA + 0.25VarD$

Given these three phenotypic covariances, unique values of the parameters can be found. As we are using correlations and not covariances, the values of the parameters will, in fact, be proportions of the phenotypic variance. Please note that behaviour geneticists use a statistical package LISREL for model fitting.

For a trait, the mathematical uniqueness of the solution refers only to the set of equations used and has no other significance. Capron et al. (1999) emphasize this point by estimating the values of behaviour genetic parameters in two different minimal sets of three equations, with the correlations used by Jinks and Fulker (1970). Their sets had two common equations but differed in the third. One of the sets gave $VarD = -0.22$. A negative value for a proportion of the variance is not acceptable The estimates of parameters obtained from fitting a behaviour genetics model should, therefore, be treated with caution as the results obtained using a different covariance or correlation matrix are likely to be different. It is not generally realized that an acceptable solution to a minimal set is available when numerical values of correlations differ and that the source of these correlations is immaterial. For example, a set of three correlations on height for three kinships from three different countries — e.g. correlation (English *MZ* twins reared apart), correlation (Samoan *DZ* twins reared together) and correlation (Indian sibs reared apart)—is a minimal set. A behaviour genetics model can be fitted to these correlations. It would, however, be difficult to interpret the parametric values as genetic constitutions and environments of the three populations differ.

III. Heritability

Heritability is the most widely used concept in behaviour genetics. Heritability analysis aims to divide the phenotypic variance on a trait into smaller components, e.g. genetic and environmental components, in order to find estimates of heritability. There are two types of heritabilities. "Heritability in the broad sense" is the proportion of *Var P* accounted for by all forms of genetic variance, i.e.:

$$H^2 = VarG/VarP$$

"Heritability in the narrow sense" is the proportion of *Var P* accounted for by additive genetic variance (Jacquard, 1983):

$$h^2 = VarA/VarP$$

This term, which evokes the image of transmission from parents to children is, in fact, an F statistic and its main drawback is that its numerator is the variance of a hypothetical statistical construct.

Sewell Wright used the symbol h^2 in the 1920s. Holzinger (1929) used the symbol H^2 to find the "nature" component. Their use of h^2 and H^2 should, however, not be confused with the "heritability" concept as used by geneticists or behaviour geneticists. The concept of heritability is due to Lush who in 1936 devised it in the context of plant breeding experiments. Later, Lush said "I think I must have been systematically avoiding the use of a single word, lest the readers oversimplify it and apply it too widely to conditions to which it was not suited" (Bell, 1977). Fisher (1951) said: " ...co-efficient of heritability, which I regard as one of those unfortunate short-cuts, which have often emerged in biometry for a lack of a more thorough analysis of the data ". Regrettably, the use of heritability by behaviour geneticists shows that their fears were justified.

The definition of heritability of a trait in a population is based on assumptions stated in Section II. Numerous geneticists have explained why heritability cannot be used for human traits and we commend Feldman and Lewontin (1975), Jacquard (1983), and Sarkar (1998) for doing so. When Lush first used the term heritability, chromosomes had not been deciphered. Now we know that all genes on a chromosome, except for mishaps, segregate together and not independently. Fisherian genetics and heritability analysis are based on the false assumption that genes segregate independently. Human populations mate assortatively for many traits. Fisher (1918) showed that under assortative mating, genes with similar effects tend to segregate together. Thus, additivity of effects is destroyed. Moreover, environment is not random for any human trait. Indeed, if the concept of evolution by adaptation is accepted, then "environment" moulded the genetic constitution of a species. Heritability analysis of a behavioural trait is based on false assumptions.

1. Heritability is not inheritance

Behaviour geneticists confuse heritability with "inheritance". The fact is that heritability tells us nothing about the inheritance of a trait. The basic principle of genetics is that genes are transmitted only through children and if a genotype has no progeny, its genes will die out. Fitness is the most important concept in genetics. Fisher (1930) loosely defined it as the "number of children". Fitness (loosely defined as the "number of children") is the most important concept in genetics. To put simply, the formula for the inheritance of a trait has the form: Intensity of inheritance = h^2 of fitness × h^2 of the trait × genetic correlation (fitness, trait) (Capron and Vetta, 2001b). Thus, a trait is propagated only if it is positively correlated with fitness. We know that IQ has a negative phenotypic correlation with family size (Herrnstein and Murray, 1994; Capron et al., 1999). Its genetic correlation, if any, must also be negative. This was the reason for the popularity of the Eugenics movement in the early twentieth century. It is generally known that higher social classes also had, in the past, low fertility. For example, Fisher (1958, p. 248) said:

> "In his book on Hereditary Genius, published in 1869, Galton considered the problem presented by the generally acknowledged fact that the families of great men tend, with unusual frequency, to die out."

Galton attributed this dying out to a tendency for peers to marry heiresses who were single children. If the correlation between parental IQ and the number of children is negative, as observed by some researchers, then intensity of inheritance of IQ will be also be negative. Similarly, the intensity of inheritance of a hypothetical homosexuality gene will be negative. The future of IQ and homosexuality genes, if they exist, is bleak indeed.

2. No support from behaviour genetics model fitting

Behaviour genetics research spanning the last 30 years does not provide any support to heritability analysis. Nor has such analysis advanced our understanding of how to "improve" a behavioural trait. After more than 30 years of research on IQ, all that behaviour geneticists can say is that its narrow heritability h^2 has changed from 0.6 to 0.36 (Devlin et al., 1997, McGue, 1997). Behaviour geneticists who make such a claim show profound ignorance of evolutionary genetics. A change of this magnitude in h^2 would occur only if there were a drastic change either in human genotype or environment. Neither seems to have occurred.

The simple explanation for the claim of a decrease is that it is "politically" motivated. When Jensen (1969) wanted to argue against the money allocated to the Head Start programme for black children in the USA, he produced estimates of 0.6 and 0.8 for h^2 and H^2 of IQ. He claimed that as IQ has a high genetic component and is highly correlated with educational achievement, the programme would not result in higher

achievement by black children and money was being wasted. Herrnstein and Murray (1994) in their widely publicized book *The Bell Curve*, argued that given assortative mating and these high heritabilities, a cognitive elite (a sort of western Brahmin class) would emerge. The reason is that the distribution of IQ will eventually become bimodal: high IQ genotypes on one side and low IQ genotypes on the other, with a small sprinkling of other genotypes in between. The emergence of a cognitive elite is likely to frighten liberals in the West. Devlin et al. (1997) produced lower estimates for h^2 and H^2 of IQ at 0.36 and 0.48 and McGue (1997) triumphantly informed us that "Devlin and colleagues' findings will lead to a reconsideration of the dire conclusions from *The Bell Curve*". He is wrong. Actually, if IQ were a genetic trait, the lower estimate would only delay the "dire" event. We need not, however, fear the arrival of the cognitive elite. We noted in the last section that IQ genes are doomed if their genetic correlation with fertility is negative.

The fact, however, is that average IQ has been increasing. It is called the Flynn effect (from Flynn, 1984). Herrnstein and Murray (1994, p. 308) in *The Bell Curve* acknowledge this effect: "In some countries the upward drift since World War II has been as much as 1 point a year for some span of years. The national averages have in fact changed by amounts that are comparable to the 15 or so IQ points separating whites and blacks in America". Here a word of caution is necessary. A polygenic trait is normally distributed. An important property of this distribution is that the mean and variance are independent. An increase in the mean IQ does not necessarily imply a change in its variance.

3. Calculation of the heritability of fertility

We discuss Kohler et al. (1999) and Rodgers et al. (2001) here, because many fertility researchers use the same methods to analyse fertility data.

Kohler et al. ask two specific questions: (1) do genetic dispositions influence fertility and fertility– related behaviour; and (2) does the relative magnitude of nature vs. nurture shift over time or with demographic regimes? Neither question can be answered with their data. Both can, however, be answered theoretically. Our view that a genotype is a reproduction machine answers part of the first question. We do not, however, wish to comment on fitness– related behaviour unless such behaviour is specified. The nineteenth century nature–nurture question can be answered only by experiments in which the levels of the two factors, genotypes and environment, are varied. For ethical reasons such experiments are not possible on humans. Concerning the "shift over time", we agree with Fisher that over the evolutionary period *nurture can become nature*.

Kohler et al. took two samples of Danish twins, the first born between 1870-1910, and the second between 1953-64. They divided the first sample into two groups: those born between 1870-89 and between 1890-1910. After subdividing all groups into males and females, they had six groups. The variance of the number of biological children for females decreased from 9.986 to 1.145 and that for males from 8.94 to 1.188 in the three groups. Changes of this magnitude in the variance of a trait require an explanation; they did not look for it. Moreover, the "twin fertility differs somewhat from the fertility of their singleton cohort mates" (p. 264). It is difficult to justify the fitting of a component of variance model to these data.

They noted that the average number of children changed significantly through time but failed to ask why. According to the genetic hypothesis, this could happen only if the genotypic frequencies had changed drastically in that short span of time. If, on the other hand, it reflects a change in environment, then the genetic hypothesis will be suspect. They note the rise of deliberate fertility control in marriage but still claim that their sample allows them to resolve the nature–nurture question. They ignore assortative mating because they claim that they have no information on it! Actually, the coefficient of assortative mating between husband and wife for the trait "number of children born in a family" is approximately 1. Demographers need to note an important difference between the traits that have been the subject of heritability type of analysis, e.g. IQ and height, and the trait "number of children in a family". IQ and height are the attributes of an individual with no contribution from the spouse. The trait "number of children" is not such an attribute because the spouse also contributes to it. Indeed, if the spouse of an individual is infertile, he or she will have no child. The fertility of the absent spouse needs also to be taken into account. The standard twin model used by heritability analysts cannot be used for fertility analysis because the fertility of each of the *MZ* twin depends on the fertility of his or her spouse. Spouses of *MZ* twins are rarely themselves *MZ* twins.

Numerous methods for estimating heritability from twin data are available. Capron and Vetta (2001b) discuss some of them critically. Kohler et al. (1999) were aware of behaviour genetics methods that use *minimal* set (Section II above) and called them Structural Equation Modelling, but they did not use them. They used "the (statistical) regression approach" of DeFries and Fulker (1985) and regressed "the number of children" of one twin on that of the other (they also use bivariate probits). They assert that the coefficients in their regression equation provide them with estimates of heritability. Thus, they confuse statistical regression with heritability. We explain the difference between genetic and statistical regression in Section VI. Statistical regression does not estimate heritability.

Kohler et al. (1999) claimed that the "aforementioned pattern of an increasing relevance of genetic effects reverses itself with the cohorts born in the late 1880s"(p. 269). A big change or reversal of a genetic trend in a population can occur only if there is a substantial change in the genetic constitution of a population. They provide no evidence of such a change. They obtained negative estimates for the variance component they call "shared environment". Their interpretation is "that the assumptions of the additive genetic model do not hold" (p. 266). Actually, estimates from a regression or heritability model depend on the explanatory variables used (Capron et al., 1999). A different set of variables might help them to obtain positive but still worthless estimates.

Rogers et al. (2001) also use DeFries and Fulker (1985) analysis, but they have a section on Structural Equation Modelling. We explained this method in Section II. They do not state the equations they used for their heritability analysis but we suspect that these were based on random mating. They claim that their perspective would lead us to expect relatively low heritability for low fertility, and high heritability for high fertility for females. Heritability analysis of a trait is based on Fisher (1918) and Jinks and Fulker (1970) and on the assumption that the trait is determined by a large number of factors. In this case, the distribution of the trait should be normal. If their "high" and "low" fertility refer to average fertility, then we know from normal distribution theory that it should be independent of the variance or standard deviation of fertility. If, on the other hand, they have high and low variance in mind, their inference is wrong. Heritability is the ratio of genetic and phenotypic variances and can be high when the phenotypic variance is low, or vice-versa. There is no genetic or statistical reason for their assertion. They said: "Past research suggested that the answer to the question 'Do genes influence human fertility?' is simply 'No'" (p. 40). This answer, however, is much too simple to be correct. Our findings suggest that the answer should be "Sometimes they do, and sometimes they don't". Actually, genes are responsible for fertility. Gene "mutation", miscopying or "environmental" factors can create conditions that result in infertility. We discuss some of the causes of infertility in Section XII.

4. Regression due to an underlying third factor

It has been known for a long time that correlation (regression) between two variables could be caused by a third underlying variable. Jacquard (personal communication, 1999) gives the example of a genetic trait, skin colour, and a behavioural trait, employment. Skin colour "is directly linked to genes. In a country like France where unemployment is a major problem, finding work is harder for someone with a dark skin. So the fact of being unemployed is 'influenced' by the person's genetic endowment. The same is true for all characteristics of whatever kind. Even

religious convictions and political opinions are types of behaviour subject to these genetic influences, through the action of processes of varying complexity (and notably those involving the genes that determine skin colour)". We may conclude that a causal analysis may show a relationship between characteristics but the reason is not genetic but social.

5. The blind alley of heritability

Heritability analysis leads to a dead end. Once one has found the heritability of a trait in a population nothing more can be said. Can one escape from this blind alley? Yes. Human behaviour is the brain's response to an external stimulus. Thus, the brain is the source of behaviour. It is therefore surprising that behaviour geneticists rarely, if ever, mention the brain. We are not yet in a position to answer many questions relating to the human brain but we know enough to ask questions. What then do we know about the human brain? One of the most remarkable differences between us and our closest simian cousins is that our brains undergo astonishing postnatal growth, doubling in size during our first two years, finally increasing by nearly 400%, from 450 cc to a maximum of some 1700 cc by the time we are sixteen years of age. We are born with most of the neurons we need. What changes is "the connections between them". The extensions that grow out of neurons "can be diverted and steered by chemicals. These extensions, some of which are established in the womb, remain highly dynamic. They are constantly strengthened by experience or atrophy through lack of it" (Greenfield, 2000, p. 61). What are these experiences that strengthen or atrophy the extensions of human neurons? Here is an alternative to the blind alley of heritability analysis for the study of human behaviour.

We hope that the new generation of researchers will take up the challenge of "experiences" that strengthen the extensions of neurons and of non-experiences that atrophy them. This will advance our understanding of human behaviour. Should it be found that some of the new "connections" appear in the progeny, then Vetta's hypothesis that the brain evolved by "problem solving" (Vetta and Capron, 1999) may deserve serious consideration.

IV. Some issues concerning the polygenic model

The polygenic model hypothesizes that a trait measured by a continuous variable is determined by a large number of genes, each having a small summary effect, and that they segregate independently. If the traits mentioned in the Introduction and others are determined by polygenes, then the number of genes in the human genome should be more like a million. Since the deciphering of the human genome, we know that their number is about 25,000. It is likely that some genes contribute to a few quantitative traits. Such traits may therefore not be independent.

Fisher (1918) showed that a polygenic (quantitative) trait will have a normal distribution. The late Professor Thoday and some of his students believed that a normal distribution could be generated by a small number of genes (Thompson, 1975). One of the referees of this paper also drew our attention to this fact. In the seventies there was a discussion in the columns of *Nature* on this question (Thompson, 1975; Vetta, 1976b). Vetta accepted Thompson's contention that a quantitative trait could be determined, in association with environment, by a few "major" genes but insisted that heritability type of analysis could not be conducted on such a trait. The reasons are: (1) in presence of assortative mating, complex correlations between the additive values and dominance values of these genes will develop and independent segregation of genes will be destroyed; (2) correlation between genetic values and environmental variables will also develop; (3) the simplification obtained by Fisher assuming a large number of polygenes will no longer be available and covariance formulae will contain a number of covariances and interactions between genes. A mathematical theory would be difficult to develop. Currently, there is no such theory.

We now know that it is not genes that segregate independently, but chromosomes. Normally, all genes on a chromosome segregate together. (A number of things happen during meiosis and they are beyond the scope of this paper.) There is no chromosomal model of a quantitative trait and it would be difficult to devise one. We now that all chromosomes have neither the same number of genes nor equal effect.

There is another problem that is rarely, if ever, discussed. We noted earlier that a polygenic trait must have a normal distribution. From the statistical theory of normal distributions, we know that their means and variances are independent. This implies that the factors which affect the variance do not affect the mean. Consider a simple example. If we set up a machine to produce 10cm long nails, not all nails produced in a day will be *exactly* 10cm long. Some could be 10.0001 and others 9.9999 cm, etc. The reason for the variation in the trait, nail length, is that the production is affected by a large number of factors. The distribution of nail length will be normal with the mean 10cm. The causes that introduced variation

around the mean, 10cm, did not affect the mean itself. We were the "cause" for setting up the mean. The variation was, however, introduced by causes that were not under our control. In this case, "causes" for the mean and variance are independent. The obvious implication is that if the mean of a polygenic trait is determined by genes, its variance may not be. This would cause a serious problem for any genetic hypothesis for a quantitative trait.

V. Why are Fisher's kinship correlation formulae wrong?

Let us first examine Fisher's (1918) kinship correlation formulae under assortative mating. Vetta (1976a) showed that these formulae are wrong. The reasons are still not properly understood and Fisher's kinship correlation formulae are still reproduced in textbooks on genetics. The formulae used by behaviour geneticists when they fit realistic models involving assortative mating are also invariably wrong (Capron et al., 1999). We explain the reasons briefly.

In Section I, we discussed the concept of dominance. Fisher assumed that dominance deviations contribute to sib correlation but make no contribution to parent–child correlation. This view is generally accepted (Falconer, 1972a; Kempthorne, 1969) and can be verified mathematically for one locus. Therefore, sib correlation, in the presence of dominance, is greater for a genetic trait than parent–child correlation. This is not the case with Fisher's formulae. Why did Fisher get it wrong?

Fisher assumed that additive values are the only cause of correlation between parent and child. Wright (1921), on the other hand, believed that "assortative mating introduces correlation between dominance deviations of parents and offspring and between dominance deviations of either and additive deviations of the other". This is, indeed, the case in Fisher's model of assortative mating but Fisher took no account of these correlations.

To obtain his sib correlation formula, Fisher discarded his model of assortative mating and used random mating. He said "the mean variance of the sibships must be taken for our purposes to have the value appropriate to random mating". As the proportions of different types of matings differ in random mating and assortative mating, this assumption is not correct.

Fisher assumed that the terms of third and higher degrees of smallness were negligible as compared to the terms of second degree of smallness, i.e. variances. This assumption is incorrect. The terms of third degree of smallness are not negligible but terms of fourth and higher order of smallness are. Correct formulae are obtained by taking terms of third degree of smallness into account.

VI. Confusion between statistical and genetic regression

Some researchers still confuse statistical regression with Galton's (1869) "filial law of regression". Galton's concept predates statistical regression. He noted that sons of tall fathers were, on average, less tall and thought that filial regression in a trait indicates that the trait is under genetic control. Fisher (1924) among others found it necessary to distinguish between statistical regression and filial regression. Vetta (1975) explained the reason for the latter. We will explain here the difference between the two concepts.

It is generally known that the regression coefficient of Y on X measures the expected change in Y for a unit change in X. Thus, if one finds that the regression coefficient of the number Y of children of twin "A" on the number X of children of twin "B" is 0.5, this simply means that if the average number of children of twin B increases by 1, then the expected increase in the average of twin A will be 0.5. (Here we ignore the problems of discontinuity and truncation.) Obviously, a statistical regression coefficient should not be confused with a genetic parameter like heritability.

The genetic explanation of Galton's filial regression is different. Consider a quantitative trait with no dominance and no environmental effects. Fathers whose trait value is x units above the assumed population mean = 0, will on average, have children whose average is $x/2$ units from the mean. This is what Galton noticed. The reason for this regression is that we considered fathers only. As mothers are chosen at random, their mean value is 0. The average of progeny is, therefore, $(x+0)/2 = x/2$. If there is perfect assortative mating, then the mothers' value on the trait will also be x. There will, now, be no regression to the mean as progeny average is $(x+x)/2 = x$. Thus, genetic regression occurs in the absence of perfect assortative mating. Inclusion of more genes, dominance or random environment will not affect the argument.

The coefficient of assortative mating for fertility must be nearly 1. For a population in equilibrium there should be no filial regression. The genetic study of a population far from genetic equilibrium is a complex problem beyond the scope of this paper.

VII. Difficulties in making a complete genetics model of a behavioural trait

As noted previously, Jinks and Fulker (1970) made the first serious attempt to use Fisher's model to analyse human behavioural traits. Their paper is probably the most cited paper in behaviour genetics. Eysenck (1979) said his "book is the first to base itself entirely on these new methods". Martin, Boomsma and Neale (1989, p.5) regard the paper as "seminal". Neale and Cardon (1992, p. 31) describe it as a "landmark" paper. The late Professor Jinks, however, granted that "the model is ridiculously oversimplified" (personal communication, May 1974).

One of the problems in behaviour genetics is the likely existence of genotype-environment ($G \times E$) interaction. There can also be *GE* covariance. More generally, formula [3] may be written, with *G&E* covariance and interaction but ignoring covariance between genes, as:

$$Var\ P = Var\ G + Var\ E + 2Cov\ (G,E) + Interaction\ (G,E) \qquad [4]$$

In the Fisherian model, environment is assumed to be random, and therefore $Cov\ (G,E) = 0$. We are still left with the interaction term. There was no method for estimating *GE* interaction. Jinks and Fulker (1970) devised one. This was hailed as a breakthrough and was immediately used by Jensen (1970) to show that there is no *GE* interaction for IQ. Eaves (1972) extended the method to the multivariate case. Fulker and Eysenck (1979) claimed: "We can test directly for some form of genotype-environment interaction". Vetta has pointed out that there was an algebraic error and when this error is corrected their method is useless. In the rest of their paper Jinks and Fulker used Fisher's (1918) incorrect formulae (Vetta, 1976a) to analyse data on some behavioural traits. It is therefore difficult to accept the claims made on behalf of Jinks and Fulker.

VIII. Does the coefficient of genetic variation have any value?

According to Rodgers et al. (2001), "In a final analysis, we compute coefficients of genetic variation to supplement the information provided by heritability estimates". Hughes and Burleson (2000) have also used this coefficient. Considered superficially, it is attractive as it gives a number that is independent of the unit of measurement. The formula they give is: Coefficient of additive variance, $CVa = 100 \times$ additive standard deviation / phenotypic mean. When looked at closely, it turns out to be rehash of an old and discarded formula in statistics. In the first quarter of the twentieth century, statisticians noted that they could compare variances in different populations only if units of measurement were identical (The *F*-test had

not yet been invented). To overcome the problem of differing units, several coefficients were proposed. Of those coefficients only two, namely Karl Pearson's Coefficient of Variation and Gini's Coefficient of Concentration, were extensively used. The formula for the Coefficient of Variation is $V = 100\frac{\sigma}{\mu}$, where μ = population mean and σ = population standard deviation. The reason for discarding it was that statisticians realized that the reverse of $\frac{\sigma}{\mu}$ is far more useful, particularly if one uses its deviation from μ i.e. $(x-\mu)/\sigma$, i.e. standardisation. This formulation now permeates statistics.

Rodgers et al. (2001) went on: "Heritabilities are proportions, and thus 'wash out' the information about overall phenotypic or genetic variance". *CVa* is also a ratio and will wash out some information. It has a further drawback. It involves parameters from two different distributions, namely, the phenotypic distribution of a trait and the hypothetical distribution of additive values. The latter parameter cannot be measured. Their coefficient is heavily affected by the mean. We see no merit in it.

IX. Genetic and environmental effects on behaviour cannot be separated

We summarize our views on separation of genetic (*G*) and environmental (*E*) effects on a human behavioural trait: (1) Behaviour genetics model-fitting methodology is useless for "cause and effect" research (Gottlieb 2001, Capron and Vetta, 2001b). To separate the *G* and *E* effects on a trait, one would need to select genotypes of the trait and environments at random. Genotypes will need to be raised in different environments. We know neither the environment completely, nor the genotypes. Moreover, such an experiment is not possible. (2) The genetic behaviour patterns of a species are the product of battles to adapt to the environment during the long evolutionary period. The simple rule was "adapt or die". We are the progeny of those who adapted. Environments to which our ancestors adapted are long gone. It is, therefore, impossible to design an experiment to separate *G* and *E* effects or interactions, as we cannot recreate that long gone environment.

X. Isolating genetic variation in fitness is difficult

In a letter to Kempthorne in 1955, Fisher defined fitness as "the capacity to leave a remote posterity" (Bennett, 1983). This makes sense in the evolutionary context. Fitness, defined in this way, can only be measured long after a person is dead and cannot be used in model fitting of behaviour genetics. We discuss some reasons that make behaviour genetics analysis inappropriate for fertility data.

1. Differences in fitness between men and women

In heritability analysis of a behavioural trait, no difference between male and female phenotypes is assumed. Statistically speaking, the mean and variance of the sexes with respect to the trait are equal. This is not true of fertility. A number of factors, such as length of pregnancy or demands of the infant, restrict female fitness. These factors do not limit the fitness of a male genotype. Age limits for having children also differ among the sexes. Thus, the fitness of a female genotype is more limited. In some populations that practice polygyny, and men have more children than women, this difference is visible.

2. Mutations and sterility

According to Kimura and Ohta (1971, p.144), "An interesting additional finding is that most mutants causing sterility do so in only one sex. A majority of such mutant genes may be kept in the population by mutation-selection balance". Initial analysis of the Human Genome Project confirms this view as "Most mutations occur in males" (BBC News/Sci/Tech, 11 February 2001).

3. Assortative mating for fitness

Rodgers et al. (2001) use "the number of children" as a measure of fertility and/or fitness. They ignore assortative mating. As previously stated for the trait "number of children", the coefficient of assortative mating between husband and wife in most monogamous societies is nearly 1 (the "nearly" takes care of infidelity, etc.). To use the behaviour genetics model, fitness has to be considered a multifactorial trait and there is no theoretical model for a trait with such a high degree of assortative mating. We explain the reason. If the coefficient of assortative mating for a behavioural trait is nearly one, then, eventually, the population will come to equilibrium with a "high" genotype *ABCD*… and a "low" genotype *abcd*… At equilibrium, matings will be within each group and heritability of each

group will be zero. If, however, the trait were fertility (fitness), the genotype *abcd*… would have no children and would cease to exist. As the genetic make-up of each member of the population is the same *ABCD*…, the variation is entirely environmental. Variation is non-genetic and heritability will be 0. Thus, the behaviour genetics model is not appropriate for the study of fertility behaviour.

4. Number of children is not an appropriate measure of fitness

A researcher wishing to use the "number of children in a family" as a genetic variable should first remove the effects of factors that are known to affect this trait. Indeed, this is precisely what demographic researchers do when they use event history and multilevel models. They try to take account of factors affecting the number of children from several levels of aggregation e.g. individual, family, economy, environment, etc. (Courgeau and Lelièvre, 1992; Courgeau, 2002).

XI. The future of Population Genetics

R. A. Fisher's contributions to genetics and evolutionary theory are immense. He worked at a time when our knowledge of chromosomes was negligible. He devised a new type of mathematics to explain the inheritance of a polygenic trait and used similar mathematics to solve evolutionary problems. In the last few years, genomes of some species have been mapped and we need to evaluate the role of Fisherian genetics within the current state of knowledge.

The credit of being the first genome to be analysed in 1998 goes to the small nematode worm *Caenorhabditis elegans* (see Section XIII). Next was the humble fruit fly, *Drosophila melanogaster*. It has four pairs of chromosomes and 13,600 genes. About 60% of its genes are also found in humans and 70% of the genes known to cause human malignancy exist in similar form in the fruit fly. Then the genome of the small weed thale cress (*Arabidopsis thaliana*) was decoded. It has five chromosomes and 25,000 genes. Rice and yeast have also been decoded.

As noted earlier, the human genome has 23 pairs of chromosomes and about 25,000 genes. In the gene count race we are below rice whose genome has 50,000 genes. The mouse genome has 20 pairs of chromosomes and about 30,000 genes. According to Dr. Hubbard, "Their [man and mouse's] genomes are so similar that you can just compare the two directly. If there are mouse genes we know something about, we can now find genes that look the same in humans" (BBC News/Sci/Tech., 6 May 2002). What then is the genetic difference between Mickey Mouse and the

master of the universe, Adam? We suspect that the difference lies in the "control" genes and the interaction between genes. With the recognition that there are so many common genes among species, the "nature" of genetics will change.

We may have to invent a new type of genetic mathematics where heritability will have no place. We will need to borrow concepts and methods from other branches of mathematics. If *C. elegans*, mouse, man and other species have a common gene, then recent research suggests that the *species value of a gene* will become an important concept in genomic mathematics. We are familiar with the concept of place value of a number in arithmetic. For example, the number 2 has a value 2 but in 245, its value is 200. Similarly, the worth of a gene may depend on the species in which it appears. In different species the *same* gene acting in concert with other genes may give rise to a different genetic expression.

XII. Genomic research and demographic behaviour

Molecular genetics is now being used to study demographic behaviour. Recent research in molecular biology or gene substitution shows the complexity of factors involved in male and female fertility. We enumerate some of them. In the next section we discuss a new method to study the role of genes that differs from behaviour genetics model fitting.

1. Advances in gene technology

In the last 20 years great advances in gene and embryo technology have taken place. The easy access to fertility clinics provided to infertile couples is only one aspect of this. We can now freeze both egg and sperm for later use even after the death of the donor. We can clone animals. Geneticists now experiment with genes that are shared between humans and other species. For example, to address the problem of human infertility, experiments for replacing faulty fertility genes can be perfected on mice. The gene therapy would enable researchers to test how gene substitution affects the future generations of mice and could provide safeguards against introducing harmful genes in the human genome.

2. Some factors associated with male infertility

Huynh et al. (2002) show that genetic factors are associated with male fertility e.g. autosomal and sex chromosomal abnormalities, disorders associated with impaired gonadotrophins' secretion, etc. Silber and

Repping (2002) show that the most frequently documented cause of male infertility is a *Y* chromosome deletion. We know that in the production of an egg, the male sex chromosome *Y* plays no part. However, Page and Hughes reported that in the production of sperm the female chromosome *X* appears to play a part. They said "all genes related to earliest stages of sperm production reside not on the male sex *Y* chromosome as expected but on the *X* chromosome, universally considered the female sex chromosome" (*The Dawn*, 31st March 2001). Xu et al. (2003) focused on the gene known as BOULE that is found in humans, fruit flies and other species. In male fruit flies it regulates meiosis. Its loss leads to meiotic arrest, and, hence, infertility. They inserted the human BOULE gene in infertile fruit flies, and development of the sperm resumed. This has obvious implications for human male infertility.

At the February 2003 Conference of the American Association for the Advancement of Science, chromosome *Y* was discussed. It is generally thought that it is passed unchanged from father to son. This does not appear to be the case. David Page of the Whitehead Institute (Cambridge, Massachusetts) is involved in decoding chromosome *Y*. He said that the primary function of the *Y* chromosome seems to be acting as a master switch that turns on male development and sperm production. He has discovered that the *Y* chromosome has found a way of evolving new gene "complexes" of its own accord. In women the two *X* genes swap genetic material so that children inherit very different gene combinations to those of their parents. David Page also discovered that the *Y* chromosome changes slightly over the generations and, in his view, this provides good evidence that genes along the length of *Y* chromosome are evolving. He said that when a useful new gene combination is produced, the *Y* chromosome appears to duplicate it — often hundreds of times — so that it cannot be lost again. Skaletsky et al. (2003) have decoded one man's *Y* chromosome and confirm some of the statements made by Page.

3. Female infertility

The causes of female infertility differ from those of male infertility. Fertility researchers at the U.S. National Institute of Child Health and Human Development (NICHD) have discovered a gene in the human egg that may be essential for early embryo development (Tong et al., 2002). The gene may also play a role in premature ovarian failure (this is a mysterious condition in which the ovaries stop functioning years, and sometimes decades, before natural menopause). "This finding could lead to new insights into the causes of unexplained infertility in women", said Duane Alexander, Director of NICHD. It may also "lead to a better understanding of the possible role that the immune system may play in some cases of premature ovarian failure" (NICHD website).

XIII. Molecular genetic and genomic approaches

In Section XI, we mentioned the nematode worm *C. elegans*. It grows to about one mm in length and has six chromosomes. It develops complex tissues and organs. It has a nervous system that can detect odour and taste, and responds to temperature and touch. It is, in fact, like a "miniature human being". "By looking at the genes that are needed to make worm muscles, we can learn quite directly about the genes that make human muscles — because they are the same" (John Sulston, Head of the Sanger Centre team of the UK HGP programme, on BBC News/Sci/Tech. 7 May 2002). Schwartz et al. (2000) have suggested that the human central nervous system has controls for intake of food. Experiments on human beings are not possible but we can draw some conclusions from research on the nematode and other species. De Bono et al. (2002) suggest that the gene *npr*-1 in nematode may be responsible for individual vs. social feeding. It represses social feeding; when it is deleted, solitary feeders congregate. A few other genes also play a role in the feeding habits.

Ashrafi et al. (2003) devised a method to find out very quickly what a gene does, thus shortening the time needed to study a genome. They created thousands of strains of genetically engineered bacteria. Each strain was designed to *block* a specific gene using RNAi (RNA interference). By feeding each strain to nematodes, they were able to block the function of individual genes selectively. They found that there are 417 genes involved in metabolism. 305 of these genes *reduced* body fat (–genes) and 112 *increased* it (+genes). Not all genes are responsive to RNAi and there may be more genes regulating body fat. According to Ashrafi et al. (2003, p. 268):

> "Many of the newly identified worm fat regulatory genes have mammalian homologues, some of which are known to function in fat regulation. Other *C. elegans* fat regulatory genes that are conserved across animal phylogeny, but have not previously been implicated in fat storage, may point to ancient and universal features of fat storage regulation, and identify targets for treating obesity and its associated diseases."

Behaviour genetics methodology cannot take account of genes having opposite effects because (1) it is based on the additive model and (2) it is concerned with analysis of variance and not effects of genes. It has no contribution to make in the genomic era. Indeed, we need to move away from Fisher's (1918) idea of the *effect of a gene* that is the basis of quantitative genetics and embrace a new concept, namely, the *regulatory role* of a gene. It is likely that most human traits are regulated by genes, some of which have +effect and some others have –effect. Behaviour genetics methodology cannot take account of both types as it is based on the concept of the additive effects of genes.

Conclusion

Heritability analysis of behaviour genetics rests on three legs: (1) The nineteenth century nature–nurture ideas of Galton, (2) Fisher's (1918) genetics and (3) Jinks and Fulker (1970). If one accepts the concept of evolution by adaptation, then many of our behavioural traits evolved when our ancestors tried desperately to adapt to the environment. That environment is gone. Therefore, Galton's idea of separation of nature and nurture effects is not realistic. Moreover, the effects of two factors can be separated only by properly designed experiments in which the levels of the two factors are under the control of the experimenter. Such experiments are not possible on human beings. Fisher's genetics predates our understanding of chromosomal inheritance. His basic assumption that genes segregate independently is not correct because all genes on a chromosome segregate together. Moreover, his kinship correlation formulae are wrong (Vetta, 1976a). Vetta also pointed out the algebraic error in Jinks and Fulker (1970). Thus, none of the three legs would support anything.

Most human traits involve assortative mating. Whenever behaviour geneticists fit a genetic model involving assortative mating, they use incorrect formulae (Capron et al., 1999). Heritability analysis would, at best, tell us that *x*% of the variation of a trait is "genetic". It cannot tell us anything about the factors that affect the trait or how to improve it. It is a blind alley. Moreover, behaviour genetics confuses statistical concepts with genetic concepts. It is better to study the inheritance of a trait using the concept of the intensity of inheritance. Demographic behaviour, e.g. fertility, differs from other behavioural traits. Fertility and the factors that cause infertility *differ* in the two sexes. Heritability analysis should not be used for such a trait.

Molecular and genomic sciences provide better avenues for research in demographic behaviour. Molecular research suggests that human traits could be *regulated* by genes. These genes can be "+genes" or "–genes", depending on their effect. Thus, the Fisherian concept of genes having only additive effects may be outdated. The concept of the *species value of a gene* that is similar to the concept of "place value of a number" may play an important role in the study of behaviour.

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VETTA Atam, COURGEAU Daniel.– **Demographic Behaviour and Behaviour Genetics**

The use of behaviour genetic heritability analysis to study demographic behaviour is fraught with problems. We explain the concepts and methods used by behaviour geneticists, which are based on Fisher (1918) and Jinks and Fulker (1970), point out their deficiencies, and show that the basic assumptions of the behaviour genetic model do not hold. A behavioural trait should be analysed not by using heritability but by using the coefficient of intensity of inheritance. Confusion between statistical concepts and heritability abounds. Fertility differs from other behavioural traits in many respects. It is affected by many known environmental factors. Male and female fertility are affected by different factors and should be studied using different techniques. Galton's 19th century idea of nature-nurture or Fisher's early 20th century genetics have little use in the genomic era. We need new concepts. One of these could be the species value of a gene, another is regulatory genes i.e. + or – genes that regulate a behavioural trait. The latter poses a serious challenge to the Fisherian concept of additive genes and this concept has to be discarded. Molecular genetics is the key to the understanding of human and animal behaviour.

VETTA Atam, COURGEAU Daniel.– **Comportements démographiques et génétique du comportement**

L'utilisation de l'héritabilité proposée par la génétique du comportement pose de nombreux problèmes. Celle-ci repose sur des concepts et des méthodes basés sur les travaux de Fisher (1918) et de Jinks et Fulker (1970) : nous indiquons les questions qu'ils soulèvent et montrons que les hypothèses à la base de la génétique du comportement ne tiennent pas. Un trait de comportement ne doit pas être analysé en utilisant le concept d'héritabilité mais en utilisant le coefficient d'intensité de l'hérédité. Les confusions dans l'interprétation statistique du concept d'héritabilité abondent. La fécondité diffère des autres traits de comportement sous de nombreux aspects. Elle est influencée par de nombreux facteurs d'environnement qui sont en partie connus. La fécondité des hommes et des femmes est affectée par des facteurs différents selon le sexe et doit être étudiée en utilisant des techniques différentes. L'opposition entre *nature* et *culture* introduite par Galton au XIX^{e} siècle ou la génétique proposée par Fisher au début du XX^{e} siècle n'ont pas d'utilité à l'ère de la génomique. Nous avons besoin de concepts nouveaux. Un de ceux-ci pourrait être la *valeur d'espèce d'un gène*, un autre est celui de *gènes régulateurs,* c'est-à-dire de gènes à effet positif ou négatif qui régulent un trait de comportement. Ce dernier concept pose un défi sérieux au concept fisherien de gènes additifs qui doit être abandonné. La génétique moléculaire est aujourd'hui la clé pour mieux comprendre les comportements humains et animaux.

VETTA Atam, COURGEAU Daniel.– **Comportamientos demográficos y genética del comportamiento**

En genética del comportamiento, el uso del concepto de heredabilidad crea numerosos problemas. Los conceptos y métodos relativos a la herencia se basan en los estudios de Fisher (1918) y de Jinks y Fulker (1970). En este artículo formulamos las preguntas que tales estudios suscitan y mostramos que las hipótesis sobre las que se basa la genética del comportamiento son insostenibles. Los rasgos del comportamiento no deben analizarse a través del concepto de heredabilidad sino utilizando el coeficiente de intensidad de tal herencia. La interpretación estadística del concepto de herencia suscita mucha confusión. La fecundidad se diferencia de otros rasgos de comportamiento en varios aspectos: numerosos factores del entorno, en parte conocidos, tienen una influencia significativa sobre el nivel de fecundidad. Los factores que influyen sobre la fecundidad de hombres y mujeres son distintos, y deben estudiarse utilizando técnicas distintas. La oposición entre naturaleza y cultura, introducida por Galton en el siglo XIX, o la genética propuesta por Fisher a principios del siglo XX, no son útiles en la era de la genómica. Necesitamos conceptos nuevos. El valor de especie de un gene podría ser uno de estos conceptos; el de genes reguladores, es decir, genes que tienen un efecto positivo o negativo sobre un tipo de comportamiento determinado, sería otro. Este último concepto supone un reto importante a un concepto creado por Fisher que debe abandonarse, el de genes aditivos. La genética molecular es la clave para comprender los comportamientos humanos y animales.

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