Sexual selection, redundancy and survival of the most beautiful

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A model is described of a highly redundant complex organism that has overlapping banks of genes such that each vital function is specified by several different genetic systems. This generates a synergistic profile linking probability of survival to the number of deleterious mutations in the genome. Computer models show that there is a dynamic interaction between the mean number of new deleterious mutations per generation (X), the mean number of deleterious mutations in the genome of the population (Y) and percentage zygote survival (Zs). Increased X leads to increased Y and a fall in Zs but it takes several generations before a new equilibrium is reached. If sexual attraction is influenced by the number of deleterious mutations in the genome of individuals then Y is reduced and Zs increased for any given value of X. This fall in Y and rise in Zs is more marked in polygamous than monogamous mating systems. The model is specified such that deleterious mutations can occur without any observable or measurable effect on function. Thus sexual selection, in this organism, for low levels of deleterious mutations cannot be based on assessment of performance. Instead it is based on a simple symmetrical surface pattern that is flawlessly reproduced by organisms with no deleterious mutations, but is less than perfect, and therefore less attractive, if genetic systems have been deleted. A complex vital task requires a system with a high level of redundancy that acts so that the loss of one component has no observable effect and therefore cannot be used for sexual selection. The reproduction of a beautiful surface pattern also requires a low error, high redundancy genetic system; however, in this case there is advantage if a single deleterious mutation produces a recognisable change. This leads to the conclusion that sexual selection and sexual attraction should be based on beauty rather than utility, and explains the common observation in nature that it is the most beautiful that survive.

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1. Introduction

The information that specifies the complexity of biological organisms is stored in the genome and is subject to mutation with each cell division. In humans the number of new deleterious mutations entering the genome in each generation is at least one and could be as many as four (Nei 1987; Eyre-Walker and Keightley 1999). These are mutations that effectively delete a functional gene. There is a problem in understanding how the human race has survived in the face of this steady erosion of genomic information (Crow 1997).

A fundamental property of all systems that store and transmit information is that a high level of redundancy is required in order to maintain complexity (Shannon 1948; Tautz 1992). When a message is transmitted in any physical system there is background noise and therefore a possibility that information will be lost and an error of interpretation will occur. The possibility of error can be reduced by repeating the message, or by sending it in parallel, or by increasing its information content so that even if some is lost, enough gets through to specify its content (Green and Swets 1966). These are all forms of redundancy. As systems become more complicated the opportunity for error rises and there is a requirement for higher and higher levels of redundancy in order to maintain function and integrity.

To illustrate this idea consider an organism (organism A) with a complex system concerned with resisting a parasitic infection. The system has m components, each

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specified by r genes. A single deleterious mutation in one of the genes will delete a component. There are $m - i$ active components, i deleted components and j deleterious mutations (there can be more than one deleterious mutation per deleted component). The probability that a parasite evades an active component is p. If the parasite evades all $m - i$ active components organism A is killed. The probability of survival following an attack by q parasites is $(1 - p^{m-i})^q$. This is a redundant system in that the probability of error falls rapidly as m is increased. If $p = 0{\cdot}1$, $q = 1$, $i = 0$ and $m = 1$, the probability of error is $0{\cdot}1$, if $m = 2$ it is $0{\cdot}01$, if $m = 3$ it is $0{\cdot}001$ and so on. In organism A, if $m = 7$, then the probability of survival under parasitic attack is high but it will fall as deleterious mutations accumulate in the genome. The form of the survival profile is as follows: the rate of fall is small initially, but as i increases it becomes progressively more marked. This organism will tolerate a few deleterious mutations but thereafter it is likely to die (Morris and Morris 2003).

In previous publications a model has been proposed in which the human genome specifies N systems (Morris 1997, 1999, 2001; Morris *et al* 2002). The mean number of deleterious mutations in the germ line of adults is Y and the mean number of new deleterious mutations per generation is X. During sexual reproduction the deleterious mutations are distributed at random to gametes which then fuse at random to form zygotes. Thus mutations in zygotes follow an approximate Poisson distribution with a mean of $Y + X$. In the model it is assumed that if one of the N systems has four or more deleterious mutations it will fail and the zygote will not develop. The result is that a subset of the zygotes survive; the mean number of mutations in the subset is less than $Y + X$. The population comes into balance when the decrease in the mean number of mutations due to selection equals X. A further assumption of the model is that if one of the N systems has three deleterious mutations its performance will be impaired to some degree and this will manifest in a number of ways, including an increased risk of disease in later life (polygenic disease trait).

A second model has been used to investigate the same problem (Morris and Morris 2003). This model has N redundant systems each with the specification as in organism A above. There are, however, $Nmrz^{-1}$ genes in the genome because each gene contributes to z components. Thus the relationship between the genome and the functions specified is more complicated than in the previous model. The results, however, are broadly the same: there is a Poisson distribution in zygotes, a subset with a lower mean number of mutations survive, and with reasonable estimates of X and Y the surviving fraction of zygotes is close to 50% of the total. Thus in humans most of the selection against deleterious mutations occurs at the zygote stage, but there is still, in those that develop, a correlation between impaired performance on a wide range of tasks and the number of deleterious mutations in the genome. This has implications for health and disease in children and adults.

In this paper a third model is used in which functions are specified by overlapping sets or banks of genes. The mathematical methods are different but the results obtained are similar. The model is also used to explore the effects of sexual selection on the population load of deleterious mutations. Once again the predictions of the model are similar to those of previous models based on redundancy. This model is specified in such a way, however, that selection cannot be based on performance but it can be based on the attractiveness of a surface pattern. The significance of this for the common observation that sexual attraction seems to be more concerned with beauty than with function is explored.

2. The model

In this model the genome has n^2 functions to perform. Each function is represented by a unit square on a board "*n units by n units*" (figure 1). The board is covered by n blocks, each "*n units by one unit*". Each block is a bank of genes and a single deleterious mutation in any one of the genes will delete the entire block. The board is then covered a second time by n blocks, each "*n units by one unit*", placed at right angles to the first layer. A third complete layer consists of n blocks each "*(0·5)n units by 2 units*". Thus there are $3n$ blocks with each block covering a different set of n functions. Each function is covered by three different blocks.

If deleterious mutations cause any one of the n^2 functions to be uncovered then the organism will not survive. If all the functions are covered then the organism will survive and function normally regardless of the number of deleterious mutations in the genome. It is assumed that death will, in most cases, result from failure of early development leading to zygote loss (Moller 1997; Morris 1997).

Let j be the number of deleterious mutations in the genome. Let b, c and d be non negative integers such that $b + c + d = j$.
The number of deleterious mutations in one layer is b, in another layer is c and in the third layer is d.

If there is one deleterious mutation in each layer the probability that a function is uncovered is 1 in n, the probability that all functions are covered is $(1 - n^{-1})$. With $b + c + d$ deleterious mutations in the genome, the probability that all functions are covered is $(1 - n^{-1})^{bcd}$.

The probability of survival with j deleterious mutations is therefore the sum of the following function

$$\frac{\left(\frac{j}{b}\right)\times\left(\frac{j-b}{c}\right)\times\left(1-\frac{1}{n}\right)^{bcd}}{3^{j}}$$

for all values of b, c and d such that $b + c + d = j$:
Computer programmes written in Pascal are used to investigate random, monogamous and polygamous mating systems with this model. It is assumed throughout that $n = 10$.

For convenience we use the following conventions throughout:

j = the number of deleterious mutations in zygotes and in the germ line of a given adult.

i = the number of deleted components in zygotes and in the germ line of a given adult.

Y = the mean number of deleterious mutations in the germ line of parents.

$Y(k)$ = the mean number of deleterious mutations in the germ line of parents in generation k.

X = the mean number of new mutations arising in spermatogenesis and oogenesis in each generation and added to zygotes.

It is assumed that the number of deleterious mutations in zygotes following random mating in adults forms a Poisson distribution with mean $X + Y$.

The computer programme starts with the assumption of some value of X between 1 and 8 and some value of $Y(1)$ between 1 and 12. A Poisson distribution in zygotes is formed with mean $X + Y(1)$. The distribution of deleterious mutations in the surviving subset is then calculated by multiplying the Poisson distribution by the probability of survival for each value of j. The mean of this distribution is $Y(2)$. The process is then repeated forming a Poisson distribution with mean $X + Y(2)$ and then calculating the distribution in the next generation of parents and the value $Y(3)$. Eventually an equilibrium position is reached in which $Y(k) = Y(k + 1)$. If X is increased the value of Y increases over several generations to a new equilibrium. Equally if X is decreased the value of Y falls over a number of generations to a new equilibrium. We have not proven that an equilibrium position will always occur, but we found one always did in practice in the range of values explored (table 1).

In monogamous and polygamous mating systems there is some form of sexual selection. In the model described so far there is nothing on which selection can be based. Within the set that survives there is no relationship between function or behaviour and the number of deleterious mutations. The model is therefore modified as follows: each organism has a pattern on its surface and each functioning bank of genes contributes to a component of the pattern (figure 2). If there are no deleterious mutations the pattern is symmetrical and attractive. Each time a bank of genes is deleted part of the pattern is lost and it is less attractive. Sexual attraction is then based purely on the surface pattern.

In the monogamous mating system it is assumed that males and females form stable faithful unions. Each organism is motivated to find the most attractive partner, in practice this means most will mate with a partner of similar attractiveness to themselves. The calculations are performed assuming that each member of a union has the same number of deleterious mutations. (In fact attractiveness depends on the number of deleted components, but in both the monogamous and polygamous mating systems it is assumed, in order to simplify calculations and programming that $i = j$.) The programme starts with a random mating system at equilibrium as determined above. This

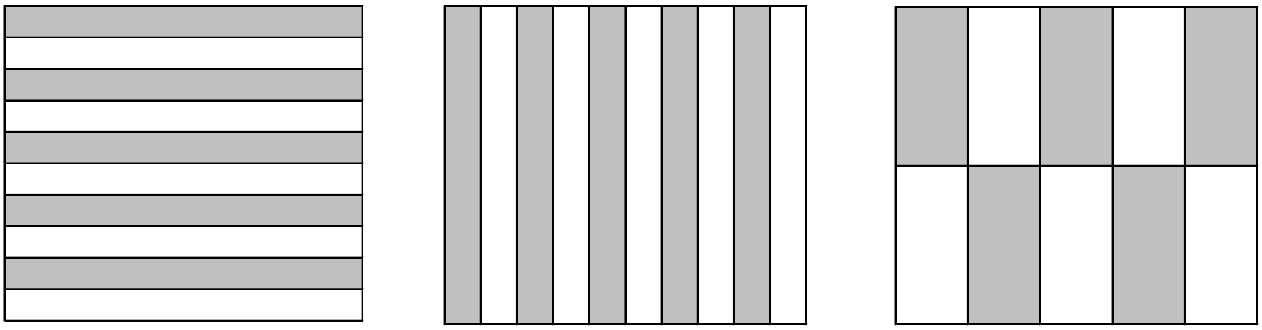

Figure 1. In this example a board "10 units by 10 units" has 100 unit squares each of which represents a single vital function. The board is covered, in the first layer by ten horizontal components, each "10 by 1 unit". The second layer consists of ten vertical components, each "10 by 1 unit". In the third layer there are ten components, each "5 by 2 units". Thus each function is covered three times and each component covers ten different functions.

specifies the initial value of X and Y. The distribution of deleterious mutations in adults is calculated and monogamous pairs for each value of j produce zygotes in which there is a Poisson distribution with mean $X + j$. The distribution in the total population of zygotes is the weighted sum of the distributions for all unions; this distribution has a greater variance than would a Poisson distribution with the same mean. This distribution is in turn multiplied by the survival function for each value of j to produce a new distribution in adults and the process of monogamous mating is repeated. The process goes through a number of iterations until an equilibrium is reached. Once again we have not proven that there must be an equilibrium but one was found in practice in all the simulations performed. It is possible to modify the programme by assuming that below a certain level of attractiveness the organisms will not find a partner. This means that only a subset of adult survivors mate and this reduces the population load further (tables 2 and 3).

The polygamous model is created by assuming that there is a threshold value of j for males and a higher threshold value for females, such that those above the threshold never find a partner. The eligible males and females then mate at random. The computer programme starts with an equilibrium position for a random mating population and then the polygamous rules are imposed. The distribution in adults is calculated (the mean of this distribution is termed A), then the mean for those adults below the threshold is calculated to give Y (males) and Y (females). The distribution in zygotes is Poisson with mean of $X + (0{\cdot}5) \times [Y \text{ (males)} + Y \text{ (females)}]$. The process is then repeated calculating Y (males) and Y (females) for the next generation. The programme iterates until an equilibrium is reached.

3. Results

Table 1 shows the equilibrium value of Y and the percentage of zygotes that survive at equilibrium in random mating for values of X from 1 to 8. When the value of X is greater than 4, less than 10% of zygotes survive. In humans, by comparison, zygote survival (Zs) exceeds 25% (a couple who engage in regular unprotected sexual intercourse, that is two to three times per week, have a 25% chance of a conception that goes to term (Juul *et al* 1999)).

Table 1. Values of Y and Zs at equilibrium for values of X from 1 to 8 when mating is random i.e. the number of deleterious mutations does not influence mate choice.

X	1	2	3	4	5	6	7	8
Y	4·72	6·01	6·97	7·83	8·69	9·63	10·79	12·40
Zs	66·1	40·6	23·6	13·0	6·8	3·3	1·4	0·5

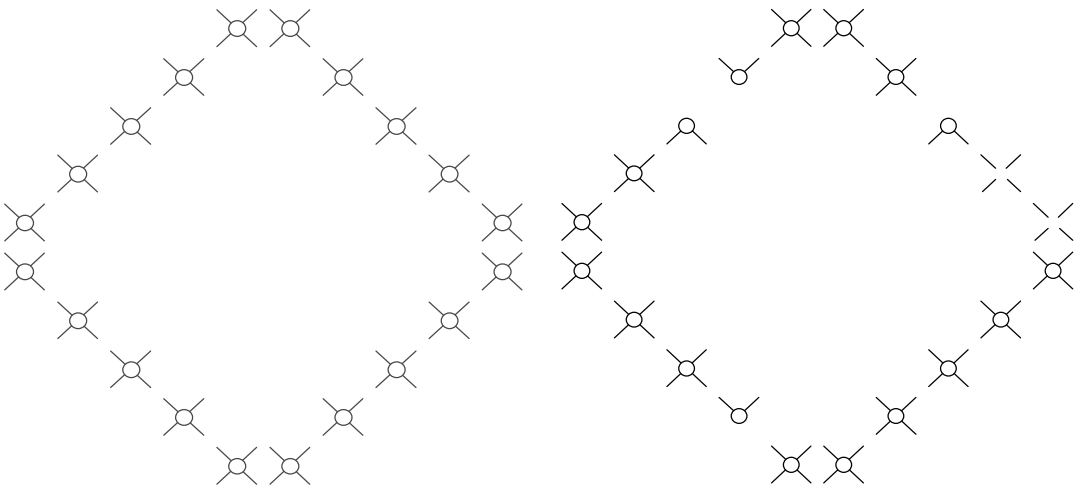

Figure 2. All 30 components in figure 1 contribute to a simple symmetrical surface pattern shown on the left. The upper diagonals are specified by the first layer, the lower diagonals by the second layer, and the circles by the third layer. In the pattern on the right one component from each layer has been deleted by mutation. The effect of mutation is to impair symmetry and render the pattern less attractive.

Table 2 shows values of *Y* and Zs at equilibrium for values of *X* from 1 to 8. In this case mating is monogamous and all animals find a mate. A comparison of tables 1 and 2 shows that *Y* is decreased and Zs increased for each value of *X* in monogamous mating compared with random mating.

Table 3 shows the results of a monogamous mating system in which organisms with more than 7 deleterious mutations fail to find a mate. In this model *Y* is the mean number of mutations in zygotes that survive and eventually mate. The overall mean in the zygotes that survive is *A*. This system of mating reduces *A* and *Y* compared with *Y* in table 2. Zs is also increased. The percentage of the population that find a mate falls as *X* rises.

The results of a polygamous mating system are shown in table 4. In this example females with more than seven mutations and males with more than two mutations are never selected as mating partners. Mating within the eligible subset is random. This system reduces the population mutational load and increases Zs compared with the monogamous systems. The ratio of eligible females to eligible males rises as *X* increases. In table 4 Zs is above 25% when $X = 6$ and above 10% when $X = 8$. This system provides an effective survival strategy when the mutation rate is high.

Table 5 shows a polygamous system in which eligible females have no more than four mutations and eligible males have no more than two. This causes a further fall in mutational load and a further rise in Zs.

4. Discussion

Sexual selection confers viability benefits on the offspring of females (Fisher 1930; Andersson 1994). But this creates a strong selection pressure against "bad genes" and for "good genes" which should eliminate genetic variation in the absence of a high mutation rate (Charlesworth 1987; Kirkpatrick and Ryan 1991). In humans, for instance, approximately one new mutation is introduced into the genome per generation (Nei 1987; Eyre-Walker and Keightley 1999); this is equivalent to a mutation rate per gene locus of 1 in 30,000. The latter rate will not maintain sufficient genetic variation for sexual selection but the former rate could (Kondrashov 1988; Rice 1988). Thus it would appear that for sexual selection to work females must be able to recognize deleterious mutations in the entire genome, not just a small part of it. A further problem is that if the mutation rate exceeds one per generation there is difficulty in understanding how a species can avoid the progressive build up of mutations in the genome even with sexual selection (Crow 1997; Agrawal 2001; Siller 2001). In this paper a model is presented, based on the concept of redundancy, that overcomes these problems. The initial discussion, below, is concerned with the way in which redundancy can obviate a progressive build up mutations. The difficult question of how a female can recognize low levels of deleterious mutations in males is then explored.

If females can recognize males with low levels of deleterious mutations then, in theory, sexual selection will decrease the overall level of mutations in the next generation. This idea has been explored, using quantitative methods, in a number of recent articles. One approach is to assume that deleterious mutations act independently to reduce fitness or survival (Agrawal 2001; Siller 2001): thus fitness is proportional to $(1 - hs)^j$, where j is the number of deleterious mutations and $1 - hs$ is the proportional decrease in viability in the heterozygote for a single deleterious mutation. Assuming that deleterious mutations in zygotes follows a Poisson distribution with a mean of t, it is possible to show that the mean after selection is $t(1 - hs)$ and the difference between t and $t(1 - hs)$ is equal to the new mutation rate per generation if the population is in equilibrium. The models then assume that selection is more severe for males than for females and the progeny of the subsequent unions have an intermediate number of mutations. In these models mean viability is approximately

Table 2. Values of *Y* and Zs at equilibrium for values of *X* from 1 to 8 when mating is monogamous. In this example all organisms find a mate and the individuals in mating pairs have the same number of deleterious mutations.

X	1	2	3	4	5	6	7	8
Y	3·24	4·70	5·71	6·53	7·26	7·93	8·59	9·29
Zs	78·8	55·9	37·1	23·6	14·4	8·6	5·0	2·8

Table 3. Values of *Y*, *A* and Zs at equilibrium for values of *X* from 1 to 8 in a monogamous mating system in which organisms with more than 7 deleterious mutations do not find a mate. Once again partners have the same number of mutations.

X	1	2	3	4	5	6	7	8
Y	3·02	4·16	4·80	5·21	5·49	5·70	5·86	5·98
A	3·18	4·57	5·52	6·26	6·90	7·49	8·04	8·57
Zs	90·0	60·8	44·0	31·1	21·5	14·7	10·0	6·6
Mating (%)	97·1	90·8	82·2	72·4	62·3	52·6	43·6	35·6

e^{-X} where X is the average new mutation rate per generation. If X is much greater than 1 the population will not survive.

The alternative approach is to assume that the genome is highly redundant, as in this and previous articles (Morris *et al* 2002). This leads to the prediction that deleterious mutations interact synergistically and viability falls slowly and then more rapidly as the number of deleterious mutations is increased. This synergistic survival profile is similar to that of truncation selection. Kondrashov (1988) showed that truncation selection against deleterious mutations could preserve a species from extinction even if the new mutation rate per generation exceeds one. Assuming, as before, that deleterious mutations follow a Poisson distribution in zygotes, the proportion of zygotes that survive for any given value of X is increased in the redundant system. Indeed in humans values up to $X = 8$ are consistent with population survival. Furthermore, although the model assumes a Poisson distribution of deleterious mutations in zygotes produced by random mating, the distribution following selection is not Poisson. In a monogamous mating system with sexual selection the variance of deleterious mutations in the progeny is greater than a Poisson distribution with the same mean and following selection this leads to a fall in the mean mutation load in survivors compared with random mating.

The idea that there is redundancy in the genome is a prediction from information theory, which was developed following Shannon's seminal publication in 1948. It was not until the 1990s, however, that there was direct evidence: there are highly conserved genes in mice and in yeast which can be deleted with no measurable or observable effects (Tautz 1992; Erickson 1993; Oliver 1996). If there is redundancy in genetic control and in biological systems then the survival profile is synergistic i.e. the probability of survival falls slowly and then more rapidly as i or j is increased (Morris and Morris 2003). This contrasts with the multiplicative or independent profile described above in which the probability of survival is proportional to $(1 - hs)^j$.

The results obtained with the model in this paper are consistent with those obtained previously with models based on the concept of redundancy. The number of deleterious mutations in zygotes follows a Poisson distribution and then a subset are selected based on a synergistic survival profile. The difference between the means of the Poisson and selected subset is equal to X when the population is in equilibrium. The process of sexual selection can reduce the mean mutational load in the population. A system of monogamous mating in which everyone finds a partner will decrease the mean mutational load by a small amount. The fall occurs because the distribution in zygotes has greater variance than Poisson and therefore there are a few more zygotes at the lower end that survive and a few more at the upper end that do not. A system of monogamous mating in which a proportion of the population,

Table 4. Equilibrium values for a polygamous mating system. Females will not accept males with more than 2 mutations, males will not accept females with more than 7 mutations. Eligible males and females then mate at random.

X	1	2	3	4	5	6	7	8
Y (male)	1·31	1·54	1·65	1·71	1·75	1·78	1·81	1·83
Y (female)	2·66	3·76	4·45	4·92	5·25	5·50	5·69	5·84
A	2·69	3·92	4·82	5·55	6·17	6·74	7·26	7·77
Zs	91·4	77·4	62·3	48·2	36·1	26·4	18·9	13·3
Male (%) mating	49·5	23·4	11·5	5·9	3·1	1·7	0·9	0·5
Female	99·4	96·5	91·1	83·6	74·8	65·5	56·4	47·7
Female: male	2·01	4·12	7·90	14·3	24·2	39·0	60·1	89·3

Table 5. Equilibrium values for a polygamous mating system in which males have at most 2 and females at most 4 mutations.

X	1	2	3	4	5	6	7	8
Y (male)	1·28	1·51	1·62	1·69	1·73	1·77	1·79	1·81
Y (female)	2·10	2·68	2·99	3·18	3·31	3·41	3·48	3·54
A	2·48	3·58	4·41	5·11	5·72	6·27	6·78	7·27
Zs	93·6	83·1	70·7	57·9	45·8	35·1	26·2	19·1
Male (%) mating	54·4	28·8	15·5	8·5	4·7	2·7	1·5	0·9
Female	88·8	71·0	53·6	39·1	27·9	19·7	13·8	9·6
Female: male	1·63	2·46	3·46	4·62	5·94	7·41	9·03	10·8

at the upper end of the distribution, fails to find a partner has a larger effect. Polygamous systems have an even greater effect. If the value of X is small it could well be that the economic advantages of monogamy outweigh the genetic advantages of polygamy, but if X is large only polygamy can protect against extinction.

A wide range of different factors can influence the mutation rate in cells and therefore the value of X will vary with geography, with social factors and over time. Chemicals, radiation and infection can cause mutation and although there are protective factors in the diet, these will vary with the availability of food. It is likely (although there is no direct evidence) that the value of X has fallen in humans in the Western World over the last 250 years. Social and economic advance gives protection from the environment with shelter, clean water and warmth and an all year round plentiful supply of food. In these circumstances the mutation rate (X) is likely to fall and part of the improvement in general health over this period will be a consequence of a decrease in the population mutational load (Y). We should not, however, be complacent. Technological advance can also pollute and it is possible that the mutational rate could increase in the future with profound consequences for health. There is a need to develop methods to measure and monitor the values of X and Y.

Sexual selection can only succeed in reducing the population mutational load if males and females can recognize members of the opposite sex with low levels of deleterious mutations and be attracted to them. How could this work? Let us consider a very clever robot, specified by a highly redundant information system. It can play chess, do the housework, perform double somersaults and mow the lawn. These are just a few of its many accomplishments. If several instructions are deleted at random from its information store its performance will be impaired and that impairment can be recognized. In general the degree of impairment will correlate with the number of deletions. In the same way in humans, impairment of the performance of a complex system will correlate with the number of deleterious mutations in the genome and that impairment can be recognized. Our ability in tests of intelligence, our athletic prowess, our ability to fight disease and maintain health: these are all complex traits and they will be impaired by deleterious mutations in the genome. These traits are also relevant to sexual attraction.

Another very important aspect of sexual attraction in humans is beauty of physical form, particularly facial beauty. Experiments carried out in the nineteenth century showed that a composite face, produced as an average of faces in the general population, is more attractive than the faces used to create it (Galton 1878; Etcoff 1994). The suggestion that beauty is average, however, was never regarded as a very exciting idea. The process of creating a composite produces a symmetrical structure and there is a great deal of evidence that symmetry is attractive (Moller 1990; Moller and Thornhill 1998). The development of a symmetrical body form is a complex task and arguing from first principles we would expect that deleterious mutations would impair the process and impair symmetry. Since symmetry is attractive this means that attractiveness will correlate negatively with the number of deleterious mutations in the genome. Thus the beautiful face is not just an average face; it is an exceptional face, it is the product of a flawless process of development leading to perfect symmetry, testimony to a high level of redundancy and a low level of deleterious mutation.

Sexual attraction can be based on the assessment of function or on the beauty of physical form. We would tend to regard the former as sensible and the latter as superficial. The success of a biological organism depends on its ability to perform various tasks and we ought to be attracted to those who perform the tasks to a high standard. There is, however, a problem in discriminating on the basis of function. Consider the highly redundant system described in the introduction: a system with m components resisting parasitic infection. If $p = 0{\cdot}1$, $q = 1$, $m = 7$ and $i = 0$, the probability of failure is 1 in 10,000,000. If $i = 1$ the probability of failure is 1 in 1,000,000. This is a ten-fold rise but in absolute terms the difference is negligible. This is an important property of a redundant system: for the most important functions the difference between m and $m - 1$ should be so small that it cannot be measured or observed. But if this is the case, how can it form the basis of sexual attraction?

In the model used in this paper it is not possible to distinguish between functions covered once, twice or three times on the basis of performance. It is possible to tell, however, from the surface pattern. There are many examples in nature of attractive surface patterns in butterflies, intense feather colouration in birds or elaborately decorated patterns as in peacocks that influence sexual attraction (Gould and Gould 1989).

If the information to specify an intricate surface pattern, a beautiful symmetrical physical structure or an elaborate ornament is present in the genome, a highly redundant structure will be required to translate the information without error. Any errors that do occur will impair beauty and the degree of impairment will correlate with the number of deleterious mutations in the genome. If only part of the genome is concerned with specifying beauty the correlation with the genomic mutational load will be weak, but if most of the genome is concerned with the specification of beauty the correlation will be strong. It has been argued previously that in the course of evolution the size of the genome grows slowly because genes are used in new combinations to increase complexity (Morris and Morris 2003). If this is correct the genes that specify beauty are the same genes that specify other functions and

therefore a large part of the genome could be involved in coding for attractiveness.

There is direct evidence that an increased rate of mutation will impair sexual attractiveness (Moller and Mousseau 2003). Barn swallows from Chernobyl that had suffered an increased rate of mutation acquired phenotypic changes similar to those that differentiated mated swallows from unmated swallows in other localities; thus an increased rate of mutation made the Chernobyl swallows less attractive. A prediction of the models presented in this paper is that if a species suffers an increased rate of mutation then a polygamous pattern of mating will become more common. Equally if the new mutation rate falls there will be a gradual change to a monogamous pattern. In birds extra-pair mating occurs in otherwise monogamous couples and the model predicts this will increase with increased mutation. There is some support for this in that Moller and Cuervo (2003) showed in a comparative study of birds that there was a positive correlation between mutation rate at minisatellite loci and extra-pair paternity.

If the above analysis is correct, then once sexual reproduction had evolved and multicellular organisms were established, an important force in evolution would be the appreciation of beauty. Organisms that had acquired a concept of beauty and were attracted to beautiful things would be at a survival advantage. This makes sense in relation to much that is known about sexual attraction (Maynard Smith 1978, 1998; Gould and Gould 1989). The capricious peahen is attracted to the male with a beautiful train. This ornament is not only without function it is an encumbrance, but it is also a marker, when flawlessly produced, of a genome low in deleterious mutations. It is better, in many cases, to assess a redundant system on its beauty than on its utility. In evolution it is the most beautiful that will survive.

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