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EVOLUTIONARY BIOLOGY

Genetics and bisexuality

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A population-genetic model indicates that if there is a gene responsible for homosexual behaviour it can readily spread in populations. The model also predicts widespread bisexuality in humans.

For human societies at large, homosexuality is a sensitive issue. For biologists it is an intriguing one[1,2]. How can genes influencing homosexual — and so non-reproductive — behaviour be favoured by natural selection? An answer is offered by Gavrilets and Rice, in a paper that has just appeared in *Proceedings of the Royal Society*[3]. They provide a population-genetic analysis that explains why, in theory, a gene predisposing an individual to homosexual behaviour would spread in a population, and that predicts its widespread occurrence in humans and other sexually reproducing species.

No predisposing gene for homosexual behaviour has been identified, but there is evidence that genetic controls are involved: for example, human twins are more likely both to be gay compared with non-identical brothers; and male homosexuality is more often inherited maternally, indicating that heritable maternal effects and/or genes linked to the X chromosome are in operation[2,3]. However, unlike heterosexuals, who devote a significant amount of time to reproductive sex, homosexuals are involved in non-reproductive sex, so hampering the direct transmission of any gene underlying this behaviour. Homosexuality has a cost to fitness — that is, the ability of an individual to produce offspring that survive and reproduce — and it can only evolve if it otherwise provides indirect benefits to reproduction.

Three main mechanisms have been proposed in which variety in genes controlling homosexuality could be maintained in a population: overdominance, sexually antagonistic selection, and kin altruism[2–4]. For simplification, we will consider here male homosexuality, but these mechanisms also apply to female homosexuality. They also apply no matter how many genes contribute, but Gavrilets and Rice's analysis deals with a single theoretical gene and its two variants (alleles).

First, in the case of overdominance, a 'gay allele' would result in homosexual behaviour in an individual who has received this allele from both parents (homozygous), but would provide an advantage to the heterozygote (where only one parent has transmitted the gay allele). This situation would be similar to the renowned example of sickle-cell anaemia in Africa, a genetically inherited disease controlled by a deficient allele. Homozygotes for this allele suffer severe disorders. But because this allele confers resistance to malaria when heterozygous, it is maintained in human populations exposed to malaria. Under this scenario, heterozygotes for the gay allele may have higher success in attracting females and/or their sperm may have some competitive advantage[5].

In the second case, sexually antagonistic selection, a gay allele would result in a cost when expressed in males ('feminization' and loss of fitness), which would be counterbalanced by a fitness advantage when it is expressed in females.

In the third hypothesis, kin altruism, homosexuals would help their own family members, increasing the fitness of their relatives and therefore the probability that a gay allele is passed on to the next generation[2,4].

These hypotheses have previously been speculative, but they have now been modelled and formalized by Gavrilets and Rice[3]. The authors adapted the classical population-genetic equations established by J. B. S. Haldane[6,7] and describe the evolution of the frequency of two alleles at one locus, in large populations for which each allele may result in sex-specific effects on fitness. Considering hypothetical straight and gay alleles, Gavrilets and Rice document the conditions of relative costs and benefits to fitness under which the gay allele can enter a population of straight alleles and be maintained subsequently. They establish the conditions under both the overdominance and sexually antagonistic selection hypotheses for a homosexual gene that would be located on autosomes (non-sexual chromosomes) or on the X chromosome. These conditions still remain to be evaluated in the kin-altruism hypothesis.

Crucially, in these population-genetic models, a gay allele will produce variable degrees of homosexual behaviour, which is equivalent to the fitness cost of that behaviour (which, for example, could be interpreted as the proportion of time devoted to homosexual rather than reproductive sex). If one homozygous individual is not at all involved in reproductive sex, then the cost of homosexuality is maximal and this individual's phenotype is obviously strictly gay; however, in all other combinations, homozygous individuals exhibit a degree of bisexual behaviour depending on the costs.

Gavrilets and Rice show that, for a large set of costs and benefits, the gay allele can invade a population. Under overdominance, once a gay allele has entered a population it will be maintained in a polymorphic equilibrium, and this is easier if the homosexual gene is autosomal rather than X-linked. Further, under sexually antagonistic selection, the gay allele may even go to fixation — that is, each individual will become homozygous for this allele — thus implying widespread bisexuality.

This theoretical framework[3] is an advance in evolutionary biology and studies of human behaviour because it generates several testable predictions: for example, if a gene influencing homosexuality is linked to the X chromosome, then it would support the sexual-antagonism hypothesis rather than overdominance. The framework will be used to guide research on the genetic basis of male and female homosexuality, and will help in resolving the 'Darwinian paradox of male homosexuality'[2]. But it is of course theory only. Tasks for the future are to establish more precisely the costs and benefits of such behaviour in natural populations[1]. Such knowledge will help fine-tune these models of sexual orientation and show whether overdominance or antagonistic selection has been operating in mammals and throughout human history. ■

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