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THE EVOLUTION OF *XY* FEMALES IN MAMMALS

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SUMMARY

Some lemmings have a large proportion of *XY* females in the population, as well as *XX* females and *XY* males. In this paper we first review the biological literature on *XY* females in mammals, with particular emphasis on the genetics and cytogenetics of this trait. We then consider population genetics models of the behaviour of this trait, and we show that there are serious discrepancies between predictions and observations regarding the sex ratio and the frequency of *XY* females; in several lemming populations the reported sex ratio is lower and *XY* females are more common than expected. Finally we consider evolutionary models to understand why *XY* females persist in these populations and how selection might act on other parts of the reproductive biology to modify the system.

1. INTRODUCTION

MAMMALS are renowned for their conserved sex determining mechanism. Male heterogamety characterises virtually the entire class (*XY* males, *XX* females), and the *X* chromosome is homologous throughout the marsupial and placental lineages (Ohno, 1967, 1979; Cooper *et al.*, 1975). It has therefore been of interest to discover that some species deviate from this pattern. In particular, some rodent populations contain a large proportion of *XY* females (Rausch and Rausch, 1972; Fredga *et al.*, 1976; Gileva and Chebotar, 1979).

The evolution of *XY* females in mammals poses several questions:

(1) Why are they apparently present in few species?
(2) What selective and ecological factors maintain the *XY* females in the population?
(3) What are the effects on the population sex ratio?

These questions are addressed in previous papers which consider natural selection for *XY* females (Bengtsson, 1977; Stenseth, 1978; Maynard Smith and Stenseth, 1978; Carothers, 1980), but for only a portion of the pertinent biology. This paper provides a more comprehensive treatment of the evolutionary biology of *XY* females in mammals.

2. *XY* FEMALES IN LEMMINGS AND OTHER MAMMALS

(i) *Wood lemmings* (Myopus schisticolor)

The following facts about sex determination are reported by Fredga *et al.* (1976), Gropp *et al.* (1976) and Fredga *et al.* (1977). All males are chromosomally *XY*, but some females are *XX* and others *XY*. The *Y* chromosome of *XY* females is indistinguishable from that of males. *XY*

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females are fully fertile but their offspring are nearly all female. (Among 50 XY females, 42 were mothers of daughters only, whereas 8 gave birth to at least one son.) The oocytes of XY females have an orthodox XX constitution at diakinesis of the first meiotic division. (This was found in all 29 diakinesis figures from 10 XY females.)

To explain these facts the above authors postulated a mutant X chromosome, X^*, which suppresses the male determining effect of the Y chromosome so that X^*Y individuals are female. (It has been shown by Wachtel *et al.* (1976) that these XY females are H–Y-negative, and we may suppose that X^* acts by somehow suppressing the production of the male-determining H–Y-antigen.) To explain the XX constitution of the germ line in X^*Y females they postulated non-disjunction in foetal oocytes, leading to the production of X^*X^* and YY cells, of which the latter died, so that X^*Y females produce only X^* ova.

The facts that XY females have XX oocytes and produce nearly all daughters, taken together, suggest strongly that the gene suppressing the male-determining effect of Y is X-linked rather than autosomal or Y-linked. The mutant X chromosome, present in all XY females and singly in some XX females, has been identified cytologically (Herbst *et al.*, 1978); it differs from the non-mutant X in the length and G-banding pattern of the short arm (fig. 1a).

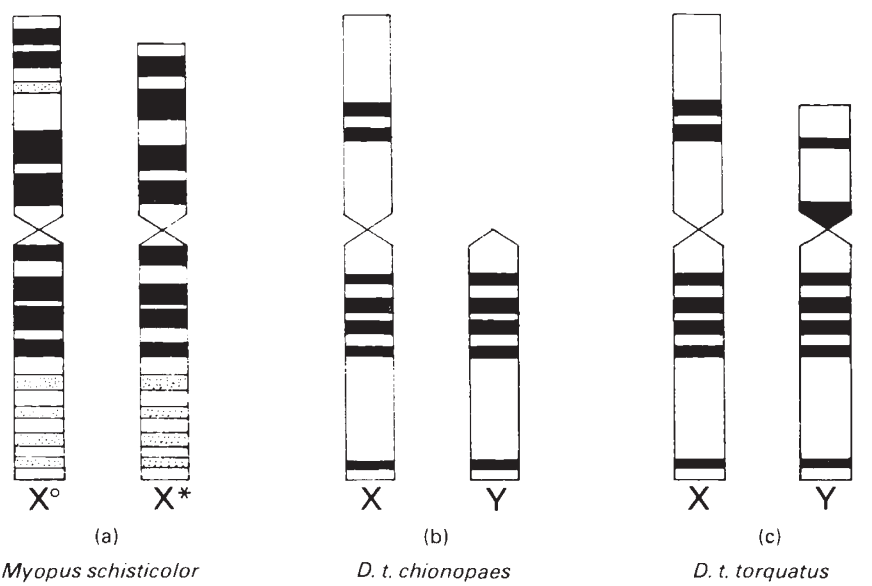

FIG. 1.—Diagrammatic representation of G bands of sex chromosomes in lemmings. (a) The two X types in the wood lemming (Herbst *et al.*, 1978); (b) and (c) X and Y in the two subspecies of *Dicrostonyx torquatus* (Gileva, 1975).

In this paper we use the symbol X^* for a mutant X chromosome and X^0 for the non-mutant, wild type X; we use X for either type when we do not wish, or are unable, to distinguish between them. On the above hypothesis males are X^0Y, while females may be X^0X^0, X^*X^0 or X^*Y. (X^*X^* cannot occur because X^* does not occur in males.) If non-disjunc-

tion in X^*Y oocytes is complete so that they produce only X^* ova, then X^*Y females should produce only daughters, half of them X^*X^0 and half X^*Y. In fact they produce a few sons. This can be attributed either to an occasional failure of X^* to suppress Y or to an occasional failure of non-disjunction so that a few Y ova are produced; the latter seems more likely.

If the three types of females are equally fertile, and if X^*X^0 females and X^0Y males have no segregation distortion while X^*Y females produce only X^* ova, the equilibrium frequencies of the four genotypes (three female and one male) should each be $\frac{1}{4}$, so that the sex ratio at birth (proportion of males) should also be $\frac{1}{4}$ (Bengtsson, 1977). The theory underlying these results and their accordance with the data will be discussed in the next section.

(ii) *Varying lemmings* (Dicrostonyx ssp)

XY females have been found in *Dicrostonyx groenlandicus* of North America, and in *D. torquatus* of Russia. (Some authors regard these taxa as synonymous.) Chromosomal studies show that a variety of Alaskan populations of *D. groenlandicus* contain *XY* females (Rausch and Rausch, 1972; their interpretation is polymorphism for different *X* chromosomes, but their excellent illustrations are highly suggestive of *XY* females.) There has otherwise been little work on the genetics of these populations.

The existence of *XY* females has been demonstrated in two subspecies of *D. torquatus* in Russia and the genetics has been carefully worked out in a series of papers (Gileva, 1973, 1975, 1980; Gileva and Chebotar, 1979). In this species *XY* females have sons as well as daughters and there is no evidence, either from segregation analysis or cytology, of non-disjunction in their oocytes. Gileva postulates a mutant suppressor chromosome, X^*, as in the wood lemming, but she supposes that X^*Y females segregate X^* and Y in equal numbers; hence they should have X^*X^0, X^*Y, X^0Y and YY offspring in equal numbers, but the YY zygotes are assumed to be inviable.

Segregation data which support this hypothesis are shown in table 1; data for the two subspecies have been combined since there is no significant difference between them. *XX* mothers were classified as X^0X^0 if they had at least eight daughters none of which were *XY*, and as X^*X^0 if they had at least one *XY* daughter; otherwise they were excluded from the analysis.

TABLE 1

Segregation data for Dicrostonyx torquatus (Expected numbers in brackets) (Gileva, 1980; Gileva and Chebotar, 1979)

Mother's genotype	X^0X^0	X^*X^0	X^*Y
Offspring phenotype			
XX female	77 (70)	182 (201)	134 (134)
XY female	0 (0)	121 (100·5)	142 (134)
XY male	63 (70)	99 (100·5)	126 (134)
Total	140	402	402
χ^2	1·40	6·00*	0·96
df	1	2	2

* Just significant at 5 per cent level; others not significant.

Expected numbers were calculated on the assumption of random segregation in all heterozygotes. Agreement with expectation is reasonably good. There is some evidence of an excess of XY daughters from X^*X^0 mothers, but this probably reflects a bias from the method of classifying these mothers as X^*X^0 only if they have had at least one XY daughter.

These data are consistent with either an X-linked or a dominant autosomal suppressor. Data on another generation are needed to distinguish between these alternatives. Under X-linkage the XX daughters of XY dams must all be X^*X^0 and will be capable of producing XY females, whereas under autosomal dominance only half of these daughters would be capable of producing XY females. Gileva and Chebotar (1979) record that, of eight XX daughters of XY dams who gave birth to young, seven produced both XX and XY females; the eighth gave birth to eight daughters, all XX. These data are unfortunately inconclusive since the status of the eighth daughter is ambiguous. (It is not stated how many sons she had; this information is relevant in calculating the relative likelihood of autosomal or sex-linked inheritance.) Gileva and Chebotar (1979) nevertheless conclude that most experimental findings favour X-linkage. The main difference from wood lemmings is the absence of meiotic drive for X^* in X^*Y females. In consequence a lower frequency of X^* chromosomes and a less distorted sex ratio are expected in varying lemmings than in wood lemmings (see later).

Figure 1 shows a diagrammatic representation of the sex chromosomes in the two subspecies of *D. torquatus*. In *D. t. chionopaes* (fig. 1b) the telocentric Y chromosome appears to be homologous with the long arm of the X chromosome and pairs with it at meiosis in males and XY females. It seems likely that the Y was originally an autosome which has been translocated onto the original X (represented by the short arm of the present X). The fate of the original Y is unknown. (In contrast the X and Y of *D. groenlandicus* pair end to end in males as is usual for mammals; see Rausch and Rausch, 1972.) In support of this suggestion, Gileva (1980) describes an isolated population in which the translocation has not occurred. In this population the X is unpaired in male meiosis; the X has a terminal centromere and lacks the long arm of fig. 1b, while an extra pair of autosomes is present, corresponding to the X-long arm and Y of fig. 1b. The mechanism of sex determination is not clear since all of seven animals examined (two males and five females) had a single X chromosome. (Gileva has a different nomenclature for what we call the Y in *D. torquatus*, but the present context does not warrant introducing a new terminology to distinguish them from more typical sex chromosomes. White (1978, Chapter 9) describes the sex chromosomes in this situation as "neo-X" and "neo-Y".)

A further complication has occurred in the second subspecies, *D. t. torquatus*, in that a second autosome has been translocated onto the Y of *D. t. chionopaes* (fig. 1c). A trivalent is thus formed at male meiosis, and there is on average one less chromosome in XY individuals, whether male or female, than in XX females (Gileva, 1973, 1975; we say "on average" because the diploid number is variable due to the presence of B chromosomes). It is not clear what bearing these facts have on the evolution of XY females but they have been reported here because they may turn out to be relevant.

(iii) *Man*

XY gonadal dysgenesis is a rare human abnormality. Affected individuals have a normal male 46, *XY* karyotype and they are phenotypic females, but they have streak gonads and are consequently sexually immature and sterile. Pedigrees are consistent with either *X*-linked or autosomal dominant inheritance of a gene which inhibits maleness in *XY* individuals, but it is not possible to distinguish between these modes of inheritance from pedigrees because affected individuals are sterile (Simpson, 1979).

Moreira-Filho *et al.* (1979) point out that some patients with this syndrome are H–Y-negative while others are H–Y-positive, and that the latter, but not the former, are prone to develop tumours in their streak gonads. They suggest that the syndrome is genetically heterogeneous, the H–Y-negative form resulting from a mutation that prevents H–Y-antigen synthesis and therefore testicular development, while the H–Y-positive form is due to a mutation at a different locus which is involved in the organisation of the testis at a different level. They also suggest that the H–Y-negative form may be homologous with the *X*-linked gene which makes *X*Y* wood lemmings into H–Y-negative females. A similar conclusion was reached by German *et al.* (1978), who suggested that the reason *XY* females are fertile in lemmings but not humans is due to the single *X* rather than to a difference in the affected *X*-locus. They note that *XY* and *XO* humans are both sterile and manifest similar gonadal abnormalities, while *XO* mice and *XY* lemmings are both fertile (though only partly so in mice). It is therefore of interest that Bernstein *et al.* (1980) describe a pedigree of H–Y-negative, *XY* gonadal dysgenesis accompanied by multiple congenital abnormalities, which is caused by a duplication on the short arm of the *X* chromosome, thus demonstrating *X*-linked inheritance of this form of the syndrome.

The possible homology of *XY* femaleness in lemmings and humans is strengthened by studies of Wolf *et al.* (1980*a*, *b*) identifying a part of the short arm of the human *X* chromosome which regulates expression of the H–Y-antigen. These authors postulate that the structural gene for the H–Y-antigen is autosomal, that there is a regulator gene on the *X* chromosome which represses the autosomal structural gene, and that the *Y* chromosome in turn represses the *X*-linked regulator, either directly or indirectly. They further suppose that a duplication of the *X*-linked regulator will avoid repression by the *Y* chromosome leading to H–Y-negative *XY* females. In support of this view they cite the pedigree of Bernstein *et al.* (1980) discussed in the previous paragraph and the fact that the *X** chromosome in wood lemmings appears to have a duplication in the third dark band of its short arm (fig. 1a). The existence of female *X*YY* wood lemmings, however, casts doubt on this interpretation (Gropp *et al.*, 1976).

(iv) *Akodont rodents*

Sex chromosome polymorphism suggestive of *XY* females occurs in some South American akodont rodents (Bianchi *et al.*, 1971). These authors interpret their findings as polymorphism for different *X* chromosomes, although in some species the karyotypes of males are indistinguishable from karyotypes of some females in their figures. Further observations

are warranted to resolve the nature of these sex chromosome polymorphisms.

(v) *Conclusion*

It is well established that the active part of the X chromosome, which includes many genes vital to both sexes, is homologous among marsupial and placental mammals (Ohno, 1967, 1979; Cooper *et al.*, 1975). The same genes are consistently observed to be X-linked in different species, suggesting that the origin of the X predates the common ancestry of these groups. Therefore the observations of X-linked mutations with apparently similar effects in rodents and man suggests that a major sex determining locus of this sort may be present on the X of all therian mammals (Ohno, 1979). If so, then a great many mammals are likely to have the potential to produce X^*Y females. With this foundation, we consider what selective forces are likely to determine the fate of such a mutation and why X^*Y females are not more commonly observed in mammals.

3. Ecological stability of XY females

Maynard Smith and Stenseth (1978) distinguish between "ecological" and "evolutionary" stability. By ecological stability is meant that a given genetic system is stable provided that no genetic modifiers are introduced; by evolutionary stability, that the genetic system is also capable of resisting the introduction of some defined types of genetic modifier. In this section we shall consider the ecological stability of XY females. Given the genetic system as it is, will an X^* mutation spread in the population, and if so what equilibrium frequency will it reach? The answer to this question depends largely on the fertility of XY females, as we discuss first.

(i) *Reproduction of* XY *females*

An XY female is unusual in that her oocytes are chromosomally XY rather than XX, and the XY oocytes may be adversely affected because of the single X. (Both X chromosomes are active in normal oocytes (Burgoyne, 1978).) For example, XO and X^*Y humans are usually sterile females and XO mice are subfertile females. The depressed fertility of XO mice might be thought to stem from loss of O ova fertilised by Y sperm, but Morris (1968) shows that fecundity is reduced by more than 25 per cent, and Burgoyne and Biggers (1976) observed that the majority of zygotes from XO females develop abnormally. There may be an early stage in oocyte fortification which benefits from two active X's, and a single X is inadequate. Burgoyne (1978) suggests that the reason XO mice are partly fertile while XO humans are not, is due to the mouse having a shorter critical phase in oogenesis when two active X's are required. There is the further complication that, at least in mice, XO females have only half the number of ovarian follicles as XX, so that XO menopauses prematurely (Burgoyne and Baker, 1981).

Besides the possibility of abnormal ovarian function, an XY female loses YY embryos, perhaps comprising a fourth of her zygotes. This may not be so serious, however, because she may be able to compensate partly

for their loss. If a mother normally produces more zygotes than she brings to term, with density-dependent mortality in the uterus, then loss of YY will enable other embryos to survive which would normally have died; there may likewise be lower postnatal mortality in smaller litters. Compensation may take other forms as well, such as increased longevity of the mother or a decreased interval between estruses. Compensation preferentially enhances X^*: while only half the zygotes have X^*, $\frac{2}{3}$ of the surviving embryos have X^*, and these are the ones used to supplement fecundity.

The X^*Y female wood lemming avoids both of these complications to fecundity. The X^* is doubled and the Y eliminated in early oocytes, avoiding the problem of oocytes with only one X chromosome. Consequently, all ova are X^* so that no YY zygotes are formed.

Data on litter size of the three female genotypes of the varying lemming (both subspecies of *Dicrostonyx torquatus* combined) are given by Gileva *et al.* (1980) and are shown below:

Female genotype	X^0X^0	X^*X^0	X^*Y
Mean litter size ± standard error	2·94 ± 0·11	3·23 ± 0·12	2·81 ± 0·11

There may be a slight upward bias in the values of X^0X^0 and X^*X^0 females due to the method of classifying these genotypes from their offspring, as described in the previous section. The litter size is slightly higher in X^*X^0 than in X^0X^0 females, but the difference is not significant. The combined value for these two types is 3·08 ± 0·08; the relative fertility of X^*Y females can be estimated as $2{\cdot}81/3{\cdot}08 = 0{\cdot}91$. Thus there seems to be appreciable compensation for the loss of YY zygotes.

It can be concluded that abnormal ovarian function and the loss of YY embryos in XY females present a barrier to the spread of the X^* mutation in most mammals. Wood lemmings and varying lemmings have overcome this barrier in different ways, the former by meiotic drive for X^* in X^*Y females and the latter by maintaining normal ovarian function and possibly by increasing the degree of compensation for the loss of YY embryos in X^*Y females. We shall discuss in the next section whether these ways of overcoming the fertility barrier represent an evolutionary response to the peculiar ecology of these species.

(ii) *Selection of* X* *in random-mating populations*

A common first step in understanding the evolution of a trait such as X^* is to investigate selection in an infinite, random-mating population. This provides a basis for interpreting selection in more complicated situations such as structured populations. Bengtsson (1977) analyses several models of XY femaleness under random mating. One important result is that XY femaleness is likely to be maintained only if caused by an X-linked mutation. (An elaboration of his formulae is presented in table 2, discussed below.) This increases our confidence that XY femaleness in lemmings is indeed due to an X^*, though only to the extent that these rodents are outbred. His model of X^* allows an arbitrary fertility and segregation for X^*Y females, and he shows that X^* is maintained only if the number of X^* daughters of X^*Y females exceeds the number of daughters of X^0X^0

TABLE 2

Equilibria for an X^0, X*, Y *sex determining system in mammals*

$\hat{y}_1 = 1 - \hat{y}_3/(1 - r_4)$

$\hat{y}_2 = r_4\hat{y}_3/(1 - r_4)$

$\hat{y}_3 = (1 - r_4)[r_2r_4f_2 + r_3(1 - r_4)Cf_3 - r_4]/[r_2r_4(1 - r_4) - r_4(1 - r_4f_2) + r_3(1 - r_4)Cf_3]$

sex ratio $= [\hat{y}_1(1 - r_4) + \hat{y}_2(1 - r_4)(1 - r_2)f_2 + \hat{y}_3r_4(1 - r_3)Cf_3]/[\hat{y}_1 + \hat{y}_2f_2 + \hat{y}_3f_3]$

where

$C = 1/(r_3 + r_4 - r_3r_4)$.

Notation:

$\hat{y}_1$, $\hat{y}_2$, $\hat{y}_3$ are the relative frequencies at equilibrium (summing to 1) of the three female genotypes X^0X^0, X^*X^0, X^*Y.

f_2 and f_3 are the fertilities of X^*X^0 and X^*Y females respectively, relative to X^0X^0, taken as 1.

X^*X^0 segregates r_2X^*, $1 - r_2X^0$
X^*Y segregates r_3X^*, $1 - r_3Y$
X^0Y segregates r_4X^0, $1 - r_4Y$

X^0X^0, X^*X^0, X^*Y are female; X^0Y is male; YY dies.

females (with normal fertility and segregation in X^*X^0). The frequency at which X^* is maintained increases with the excess of X^* daughters.

Bengtsson's results indicate that, perhaps surprisingly, the loss of YY zygotes does not select against X^*, and in fact enhances selection for X^* if there is any compensation. With 1 : 1 segregation of X^* and Y, an X^*Y female produces equal numbers of X^*X^0, X^*Y, X^0Y and YY zygotes. Without compensation she therefore produces as many X^* offspring as an X^0X^0 female produces daughters, even though YY is lost. With compensation, $\frac{2}{3}$ of the offspring which replace YY are X^*, so that any compensation whatsoever selects for X^*. Of course, if X^*Y oocytes are abnormal because of the single X, compensation may not be sufficient to yield a net advantage.

Bengtsson's formulae allow arbitrary fertility and segregation of X^*Y females, inspired by observations of wood lemmings. It is also of interest to know how the frequency of X^*Y females is affected by arbitrary segregation distortion in all heterozygotes and by arbitrary fertility in all female genotypes. The formulae are shown in table 2. It will be seen that the equilibrium frequency of X^*Y females is especially sensitive to the segregation from both X^*X^0 females and males. Although these segregation ratios are likely to be $\frac{1}{2}$ at the outset, as in Bengtsson's model, long-term evolution in populations with X^* may modify these segregation ratios (see next section). Consequently it is important to account for segregation ratios for all heterozygous genotypes in order to predict equilibrium frequencies.

We shall now consider the available data on genotype frequencies in lemmings, nearly all of it from captive colonies, in the light of these theoretical results. The simplest model for the wood lemming is to take $\hat{f}_2 = \hat{f}_3 = 1$, $r_2 = r_4 = \frac{1}{2}$, $r_3 = 1$, in the notation of table 2. The sex ratio should be $\frac{1}{4}$, and the three female genotypes should be equally frequent. In one captive colony the sex ratio based on 1073 births was 0·25, exactly as predicted (Kalela and Oksala, 1966). The correspondence between prediction and observation in this case was previously noted by Bengtsson (1977). However, in another colony on which most work has been done

there is a considerable discrepancy. In this colony Frank (1966) reported a sex ratio of 0·18 based on 513 births in the first few years after its foundation, and Gropp *et al.* (1978) reported a sex ratio of 0·19 between 1975 and 1977. Furthermore Gropp *et al.* (1976) report the sex chromosome types of 349 randomly chosen adult females from this colony; there were 180 *XX*, 163 *XY* and six abnormal karyotypes. Thus there are nearly as many *XY* as *XX* females, whereas there should only be half as many; the excess of *XY* females is highly significant ($\chi_1^2 = 31{\cdot}1$).

The simplest model for varying lemmings is to take $\hat{f}_2 = 1$, $r_2 = r_3 = r_4 = \frac{1}{2}$, and to leave $\hat{f}_3$ the fertility of X^*Y females, arbitrary. The predicted sex ratio is

$$(9+16f^2)/12f(1+4f)$$

and the female genotypes X^0X^0, X^*X^0 and X^*Y should be in the ratios $3:4f-3:4f-3$, where f stands for $\hat{f}_3$ throughout. With complete compensation ($f=1$) the sex ratio should be 0·42 and the three female genotypes in the ratios 3:1:1, but with partial compensation, which is more likely, the sex ratio and the proportion of X^0X^0 females will increase.

Gileva (1973, 1975) reports a sex ratio of 0·41 ($N=233$) at birth in a captive colony of *Dicrostonyx t. torquatus*, in good agreement with prediction for complete compensation, but a sex ratio of only 0·27 ($N=316$) in a captive colony of *D. t. chionopaes*; the latter figure is significantly less than 0·42 ($\chi_1^2 = 28{\cdot}4$). There is also evidence that *XY* females are more frequent than one would predict. Gileva and Chebotar (1979) and Gileva (1980) give the following data on the frequencies of the chromosome types in (presumably adult) animals in captive colonies of the two Russian subspecies:

Chromosome type and sex	*XX*♀	*XY*♀	*XY*♂
D. t. torquatus	398	210	233
D. t. chionopaes	395	242	221

The sex ratio is 0·28 in *D. t. torquatus* and 0·26 in *D. t. chionopaes*, both significantly less than 0·42, but there may have been a bias in selecting animals for chromosome typing, and in any case these are not sex ratios at birth. Of greater interest is the proportion of the *XY* karyotype among females, which is 0·35 for *D. t. torquatus* and 0·38 for *D. t. chionopaes*, both highly significantly greater than 0·2 ($\chi_1^2 = 80{\cdot}3$ and 128·8 respectively).

The same may also be true in North American varying lemmings. In a captive colony of *D. groenlandicus* from Alaska, Rausch and Rausch (1972) found a sex ratio of 0·42 ($N=213$), exactly as predicted, though they state that demographic data on the colony are not good; but among 22 females karyotyped they found 9 *XY* females, giving a proportion of *XY* among females as 0·41, significantly higher than 0·2 ($\chi_1^2 = 6{\cdot}0$).

There is thus quite strong evidence that the frequency of *XY* females is higher than one would predict in both wood lemmings and varying lemmings, and less consistent evidence that the proportion of males is sometimes lower than one would predict. The situation would seem to

warrant further investigation with the following possible explanations in mind:

(1) an artefact due to the way in which lemmings are bred in captivity;
(2) pleiotropic effects of X^* on aspects of fertility other than litter size;
(3) segregation distortion in X^*X^0 females or males.

Segregation distortion seems to be ruled out for the varying lemming by the data in table 1, but would be worth while investigating for the wood lemming. It is interesting in this context that the duplication of X^* in X^*Y females is apparently a property of the X^* chromosome rather than of X chromosomes in general. Gropp *et al.* (1976) observed that an XO female had a univalent X in meiosis. That this female was X^0O rather than X^*O was verified by her giving birth to a son. Thus it is not implausible that there be some segregation distortion in X^*X^0 females. Segregation distortion in males should also be investigated; it will be shown in the next section that selection for segregation distortion in males would lead to a sex ratio of 0·18 with about half the females being XY, which happens to fit closely with the observation from Frank's colony.

(iii) *Selection of* X* *in structured populations*

It is only in lemming populations that XY females are definitely known at appreciable frequencies, and these rodents experience periodic major changes in population size (Stenseth, 1978; population cycles are discussed by Finerty, 1980). Therefore population structure might be thought to influence selection for X^*. If the population is broken up into a large number of small demes, there might be some form of local mate competition within each deme which would select for a female-biased sex ratio and might therefore select for a higher equilibrium frequency of X^* and a more female-biased sex ratio than under random mating.

While it is true that many types of population structure may lead to a female-biased sex ratio through local mate competition and interdemic selection if autosomal variability for sex ratio is available (Taylor and Bulmer, 1980; Bulmer and Taylor, 1980; Colwell, 1981), it is not true that they will select for a higher frequency of X^*. Provided that the population structure does not affect the relative fertility or viability of the three female genotypes, then the equilibrium frequencies of these genotypes and the sex ratio are unaffected by the population structure. The reason is that all males have the same genotype, X^0Y. Hence the relative frequencies of different types of offspring of a female of specified type are unaffected by population structure, whence the recurrence relationships for the genotype frequencies in the whole population in successive generations will also be unaffected. In particular, the equilibrium genotype frequencies are the same as under random mating, and they remain constant during any periodic increase and decline in a fluctuating population, unless of course the fertility of the genotypes is differentially affected.

To illustrate this general result we shall consider the haystack model of Bulmer and Taylor (1980), which may serve as a crude model of the population cycles of lemmings. In this model a species of rodent lives entirely in haystacks. At the beginning of each breeding season a stack is colonised by a number of fertilised females, and their offspring breed there

for several generations until the following year, when females disperse, mate at random with males from the whole population and compete to colonise new haystacks which have become available. Suppose first that X^* is absent. If there is arbitrary autosomal genetic variability for sex ratio, this model will generate selection pressure for a female-biased sex ratio; for example, with one founding female in each stack and two generations of breeding in the stack before dispersal, the uninvadable sex ratio is 0·3125 (Bulmer and Taylor, 1980).

There is however no selection for X^* generated by the population structure. Consider for example the varying lemming situation with no segregation distortion and with equal fertilities of the three female genotypes, so that their equilibrium frequencies under random mating are in the ratios 3:1:1. Suppose that there is one founding female in each stack, and that each female has four offspring. Table 3 shows the numbers

TABLE 3

Absolute genotype frequencies under the haystack model for the varying lemming

Generation	0	1	2	3	
X^0X^0	1	2	4	8	Type
X^*X^0	0	0	0	0	1
X^*Y	0	0	0	0	stack
X^0X^0	0	1	3	8·33	Type
X^*X^0	1	1	2·33	5·44	2
X^*Y	0	1	2·33	5·44	stack
X^0X^0	0	0	1·33	5·78	Type
X^*X^0	0	1·33	3·11	7·26	3
X^*Y	1	1·33	3·11	7·26	stack
X^0X^0	3	7	16·33	38·11	3 × Type 1
X^*X^0	1	2·33	5·44	12·70	+ Type 2
X^*Y	1	2·33	5·44	12·70	+ Type 3
					= whole population

of females by type in the three different types of stack (those founded by X^0X^0, X^*X^0 and X^*Y females) in successive generations of breeding within the stack; it also shows the numbers in the whole population on the assumption that the first type of stack is three times as common as the other two. It will be seen that the frequencies in the whole population remain in the ratios 3:1:1, although the frequencies within each stack change.

It was assumed above that population structure does not affect the relative fertilities or viabilities of the female genotypes. There are two ways in which fertilities might be affected by population structure, both of which will select against X^*. In the wood lemming X^*Y females only have daughters. If a stack is colonised entirely by X^*Y females under the haystack model, in the next generation there will be only females and the colony will die out unless they are mated by a male from outside the stack. Unless this always happens there will be selection against X^*Y females and the equilibrium frequency of X^* will be less than under random mating. Bulmer and Taylor (1980) give an explicit expression for the equilibrium genotype frequencies under the haystack model. Maynard Smith and

Stenseth (1978) reach a similar conclusion under a model with inbreeding, determined by fixing the proportion of females who mate with their brothers.

The second way in which fertilities might be affected by population structure is through density-dependent factors operating within the stack. This will again select against X^* because density-dependent selection will act more severely on stacks containing X^* which have a higher intrinsic growth rate. Suppose that at the beginning of each season each stack is colonised by k fertilised females, and that density-dependent factors operate so that at the end of the season there are N females in each stack. A stack may by chance be colonised by k X^0X^0 females, in which case it will have N X^0X^0 females at the end of the season. Other colonies will have at least one X^* female (either X^*X^0 or X^*Y), and we suppose that there is a large number of generations of mating within the stack, so that at the end of the season there are $\hat{y}NX^*$ females and $(1-\hat{y})NX^0X^0$ females; $\hat{y}$ is the proportion of X^* females attained at equilibrium under random mating, given by $\hat{y} = \hat{y}_2 + \hat{y}_3$ from table 2.

Two extreme cases may arise, (i) all k founders of a colony are drawn from the same ancestral colony, or (ii) the k founders are drawn at random and independently from any ancestral colony. In case (i), once a colony loses X^* it never regains it. All colonies will eventually lose X^*, because all k founders may be X^0X^0 females. In case (ii), X^* may be maintained indefinitely (deterministically). If P_t is the proportion of colonies with X^* in year t, the Expected proportion of colonies with X^* the next year is

$$P_{t+1} = 1-(1-\hat{y}P_t)^k.$$

If only one female is drawn to found each colony ($k = 1$), X^* is eventually lost; if two females found each colony, X^* is expected to be maintained if $\hat{y} > \frac{1}{2}$, and so on. If $\hat{P}$ is the equilibrium value of P_t, such that

$$\hat{P} = 1-(1-\hat{y}\hat{P})^k,$$

the frequency of X^* females in the population is $\hat{y}\hat{P}$, which is certainly less than $\hat{y}$, the frequency under random mating. The above discussion offers some explanation of the simulation results obtained by Carothers (1980).

4. Evolutionary modification of the genetic system

If the X^* mutation has evolved to an intermediate frequency, its long-term persistence may depend on whether selection favours other genes which suppress its activity or modify its characteristics in other ways. From Fisher (1930), we may expect selection to eliminate X^* in order to restore a primary sex ratio (proportion male) of $\frac{1}{2}$ in random-mating populations. The evolution of X^* to an intermediate frequency occurs despite this selection for a sex ratio of $\frac{1}{2}$ because of the advantage X^* receives from compensation or segregation distortion in X^*Y females. Autosomal modifiers of X^* would not receive this segregational advantage and therefore might be selected in various ways to produce a sex ratio of $\frac{1}{2}$. On the other hand, in a structured population with local mate competition or interdemic selection favouring a female-biased sex ratio, there might be selection for autosomal modifiers which facilitate the spread of X^*. We

consider here three types of modification: suppression of X^* activity, modification of segregation ratios, and modification of compensation.

(i) *Suppression of* X* *activity*

One of the most straightforward means of eliminating XY femaleness is to suppress the X^* mutation so that the H–Y-antigen is expressed in X^*Y genotypes and they become male. There is no evidence that such suppressors exist, but few studies on these species are thorough enough to have detected them if they were present. Suppressors could be detected if they segregate in populations with XY females; otherwise, if a suppressor has arisen and been fixed, its presence would be detectable only by some biochemical assay or by crossing to individuals known to transmit X^*.

It is convenient to consider two locations in the genome for a suppressor of X^*, the Y chromosome and the autosomes. The reason for making this distinction is that selection of Y-linked genes differs from selection on autosomal genes as far as sex ratio evolution is concerned (Shaw, 1958; Hamilton, 1967).

It is much easier to model the evolution of a Y-linked suppressor (situated on the non-pairing segment of the Y chromosome), and consequently most analyses have been limited to this case; but we question whether a Y-linked suppressor of X^* is plausible. Maynard Smith and Stenseth (1978) justify this on the observation that Y-linked suppressors of meiotic drive occur in the mosquito, *Aedes aegypti* (Wood and Newton, 1976). This is not especially relevant to mammals, however, because the Y of *Aedes* is not highly degenerate like that of mammals. In lemmings, the Y is either degenerate with little active genetic material (wood lemming and *Dicrostonyx groenlandicus*) or is a neo-Y with only a pairing segment, which behaves like an autosome for genes far enough from the centromere (*Dicrostonyx torquatus*). The simplest possibility to consider as a suppressor of X^* is duplication of the male-determining part of the Y; the existence of female X^*YY wood lemmings, however, demonstrates that duplication of the Y would not suppress X^* (Gropp *et al.*, 1976).

Nonetheless, we now review briefly the previous models for selection of Y-linked suppressors of X^*. Maynard Smith and Stenseth (1978) investigate selection of a Y-linked suppressor (Y^*) in a wood lemming system (X^*Y females produce only X^* ova) with varying degrees of sib-mating and/or father–daughter matings. They found that Y^* spread to fixation under all but the most extreme inbreeding, and then only when there is father–daughter mating. In their discussion, however, they suggest that their results exaggerate the stringency of the conditions required to prevent the spread of Y^* because they disregarded forms of inbreeding other than brother–sister and father–daughter mating. This suggestion is confirmed by the study of Bulmer and Taylor (1980) on the spread of Y^* under the haystack model described in the previous section which takes into account all forms of mating between close relatives. Suppose that there is one founder female in each stack at the beginning of the season, that there are G generations of breeding within the stack before dispersal, and that in the first generation the females in a stack founded by an X^*Y female have a chance p of being mated by a random male from outside the stack. Bulmer and Taylor (1980) considered whether or not Y^* will invade when

it is rare. They found that Y^* will always invade when $G \leqq 2$, and that it will never invade when $G \geqq 5$; for $G = 3$ or 4, Y^* will invade if p is sufficiently small, but not otherwise. Thus it is quite plausible to suppose that the wood lemming system is evolutionarily stable against invasion by a Y^* suppressor under the appropriate form of structured mating. The reason why consideration of sib-mating alone gives a misleading idea of the effect of population structure for the wood lemming is the special circumstance that X^*Y females have only daughters who have no brothers to mate with.

We now turn to the more plausible case of an autosomal suppressor. Bulmer and Taylor (1980) investigate selection on a rare, dominant, autosomal suppressor of X^* in the wood lemming system (under the same model of population structure as in the above paragraph). Selection for autosomal suppression was observed to be weaker than for Y-linked suppression. The autosomal suppressor was selected against for $G \leqq 3$ and favoured for $G \geqq 10$; the evolution of the suppressor was sensitive to p for $4 \leqq G \leqq 8$.

This study of autosomal suppression of X^* leaves an important question unanswered. It is not clear whether polymorphism for the suppressor will result if it is favoured when rare, or instead, whether it will spread to fixation and so completely eliminate XY femaleness in the population. To study this question we have modelled a varying lemming population with a dominant, autosomal suppressor (A) such that aaX^*Y is female, but AaX^*Y and AAX^*Y are male; the sex of all other genotypes depends only upon whether they are XX (hence female) or XY (hence male). The population structure assumed was such that a fraction K of the daughters mate with their brothers (in proportion to their frequencies in the brood), $(1-K)$ of the daughters mate randomly in the population, and all sons mate randomly regardless of whether they inseminated a sister. Sib mating levels (K) from 0·1 in increments of 0·1 or 0·05 were studied, largely for the case of complete compensation ($f_2 = f_3 = 1$), and less extensively for the case of 50 per cent compensation ($f_2 = 1, f_3 = 0{\cdot}875$). Initial frequencies of the various mating types were varied somewhat. Only a few mating types were specified as having non-zero frequencies in the first generation, but nine generations of random mating ($K = 0$) were then allowed to create all mating types before the specific K value was assigned for the next several hundred generations. We feel that the frequency of sib mating is a valid indicator of the degree of selection for a female-biased sex ratio in the varying lemming system. The complication arising from the occurrence of all-female broods in the wood lemming does not normally occur in the varying lemming. (The exception is the mating $aaX^*X^*♀ \times aaX^0Y♂$; it was assumed that all the offspring mate at random in this case. aaX^*X^* females can occur, for example, from the mating $aaX^*X^0 \times AaX^*Y$.)

The results indicate that, at least for some values of K around 0·6, there is not a global equilibrium for the system. With complete compensation and an initial frequency of 0·01 for A, the suppressor was selected against for $K = 0{\cdot}55$, 0·60 and 0·65, but favoured for $K = 0{\cdot}75$ and above and for $K = 0{\cdot}45$ and below. With an initial frequency of 0·23, however, the suppressor was favoured for all K values tried and evolved to polymorphism for $K \neq 0$. (It seems as though the suppressor will be fixed for random mating, $K = 0$, but this cannot be established numerically.) When

TABLE 4

Equilibrium genotype frequencies in the varying lemming with full compensation and some sib mating (K) *with an autosomal suppressor*

	male		female			
	AAX^*Y	AaX^*Y	AAX^*X^*	AaX^*X^*	aaX^*X^*	aaX^*Y
$K=0{\cdot}2$	0·312	0·168	0·312	0·154	0·024	0·028
$K=0{\cdot}5$	0·238	0·213	0·238	0·184	0·057	0·071
$K=1$	0·174	0·222	0·174	0·179	0·107	0·144

only 50 per cent compensation was allowed, there was selection against the suppressor, when rare, in the region near $K = 0{\cdot}6$.

When the suppressor was favoured there was an accompanying increase in the frequency of X^*; equilibrium was reached with the loss of X^0 and with A common. An intuitive explanation of this result is that, in the absence of the suppressor, the equilibrium frequency of X^* is determined by a balance between selection for X^* from compensation and against X^* from the female-biased sex ratio. The spread of the suppressor reduces the female bias, which allows X^* to increase in frequency, which allows the suppressor to spread further, until eventually X^0 is eliminated. The equilibrium genotype frequencies are shown in table 4 and the equilibrium sex ratio in fig. 2. The sex ratio decreases with K, as expected, but it does not attempt to attain the evolutionarily stable sex ratio of $\frac{1}{2}(1-K)$ which it should take under this model if there is arbitrary autosomal variability for sex ratio modification (Maynard Smith, 1978). The likely reason is that, when X^* is fixed, A behaves more like a sex-determining gene with major effect than like a sex ratio modifier.

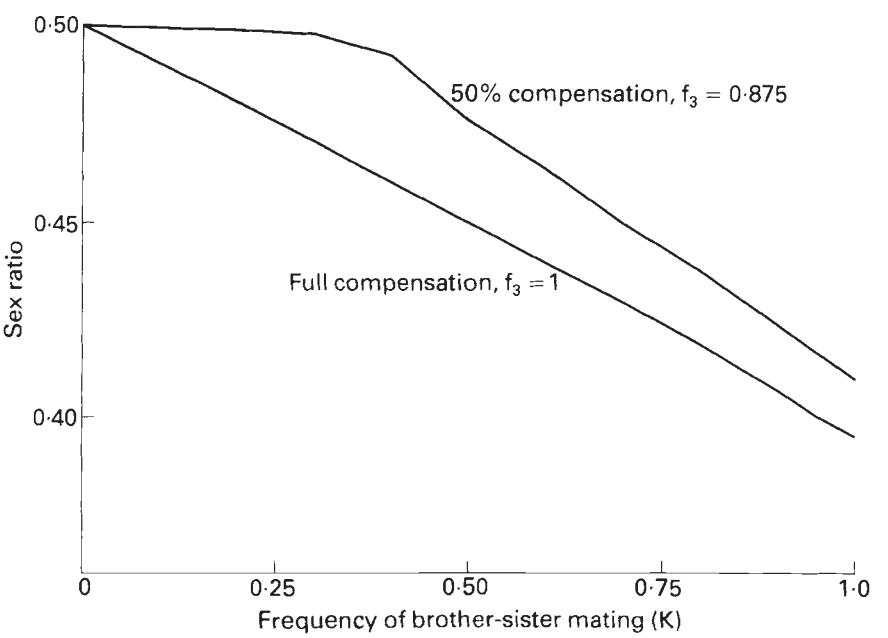

FIG. 2.—Equilibrium sex ratio in the wood lemming system with an autosomal suppressor.

(ii) *Modification of segregation ratios*

In the absence of suppressors of X^*, selection may change the proportion of Y gametes from X^*Y and X^0Y genotypes. The effect of modified segregation ratios on the sex ratio is evident from table 2. We investigate only two of the many possibilities here, and the especially interesting result in one case motivates further investigation of data from this perspective.

In wood lemmings, the X^*Y female produces only X^* ova, whereas in varying lemmings she produces X^* and Y ova in equal numbers. It seems quite plausible that the wood lemming system has arisen from the varying lemming system. An X-linked mutation giving meiotic drive for X^* would be favoured for obvious reasons, but an autosomal mutation might also be favoured since it avoids any loss of fertility due to oocytes with a single X and the production of YY zygotes, particularly in a structured population favouring a female-biased sex ratio. On the contrary, in a random-mating population an autosomal gene leading to the suppression of meiotic drive in wood lemmings might be favoured. If an X^*Y wood lemming segregates Y to some ova, then half of these ova would die as YY zygotes as Maynard Smith and Stenseth point out. However, the equilibrium fraction of males is otherwise $\frac{1}{4}$, so each son has three times as many offspring as a daughter (under random mating). Thus the loss of even 50 per cent of Y ova does not overcome the advantage of producing sons by this route. Of course, a wood lemming which produced Y ova might also suffer such a reduced fecundity from a single-X oocyte that there was no net advantage through sons (although XO ovaries in wood lemmings are at least not grossly abnormal (Gropp *et al.*, 1976)).

Selection might also act on males under random mating to increase the proportion of Y sperm and so produce more males. This has a paradoxical effect. In a wood lemming population in which most males segregate Y to half of their sperm, a male which segregates a greater proportion of Y to sperm indeed produces more sons than average. Hence, an autosomal gene which causes males to over-produce Y sperm (without loss of fertility) is favoured under random mating.

The paradoxical effect is that the population sex ratio becomes even more female-biased (tables 1 and 5). An increased proportion of Y sperm increases the frequency of X^*Y females and reduces the frequencies of X^0X^0 and X^*X^0 females, so that fewer X^0 ova are available. All males are affected equally, so that the advantage of overproducing Y sperm continues to apply as this trait starts to spread in the wood lemming population. We have studied this effect both analytically (solving for the uninvadable level of segregation distortion) and numerically, and both methods indicate that the uninvadable equilibrium level of segregation distortion in males is approximately 0·64 Y sperm to 0·36 X sperm (table 5). This is true even if the population is polymorphic, some males segregating randomly and others producing only Y sperm. This yields a population sex ratio of 0·18.

If the segregation distortion is accompanied by a reduced fertility in these males, the equilibrium level of segregation is shifted towards $\frac{1}{2}$, and the gene causing segregation distortion may not even be favoured (table 5). It is by no means clear that segregation distortion of large magnitude

TABLE 5

Equilibrium levels of segregation distortion in wood lemming males

Segregation of Y by male genotype			Fertility of A-relative		Mean segregation	Frequency of X^*Y among
aa	*Aa*	*AA*	to *aa*	Sex ratio	ratio $(1-\bar{r}_4)$	females $(\hat{y}_3)$
0·5	0·52	0·55	0·99	0·25	0·50	0·33*
0·5	0·55	0·60	0·99	0·20	0·60	0·50**
0·5	0·60	0·70	0·99	0·19	0·63	0·54
0·5	0·60	0·70	0·95	0·25	0·50	0·33*
0·5	0·70	0·90	1·00	0·18	0·64	0·56
0·5	0·70	0·90	0·90	0·25	0·50	0·33*
0·5	1·00	1·00	1·00	0·18	0·64	0·56
0·5	1·00	1·00	0·90	0·22	0·55	0·42

* A allele lost.
** A allele fixed.

can be achieved without some loss of fertility and the model must be interpreted with this caution in mind.

(iii) *Modification of compensation*

The main barriers to the initial spread of the X^* mutation are subnormal function in single-X ovaries and the loss of YY zygotes. The wood lemming has overcome these problems by duplication of X^* in the ovaries of X^*Y females. In the varying lemming there is no evidence of subnormal function of X^*Y ovaries; this may be a preadaptation in the species which facilitated the spread of X^*, or more likely it may have evolved together with the spread of the X^* mutation.

As an illustration of the mechanism by which compensation might work, suppose that a female's fecundity is due to two interacting processes, the number of eggs ovulated and the probability that an embryo will survive to term. The probability of survival of each embryo, $p(y)$, probably decreases with the number of embryos initially present. In this model, selection adjusts the level of compensation by changing the number of eggs ovulated; selection does not alter the form of the survival function, $p(y)$. If each female ovulates y eggs, then XX females have y embryos of whom $p(y)$ survive while XY females have $0{\cdot}75y$ embryos of whom $p(0{\cdot}75y)$ survive. To study the evolution of compensation in this situation, we consider one locus with a common allele a and a rare allele A which control fecundity as follows:

Female	No. Live Offspring	
aaX^*Y	$\frac{3}{4}yp(\frac{3}{4}y)$	$(\frac{1}{3}X^*X^0, \frac{1}{3}X^*Y, \frac{1}{3}X^0Y)$
aaX^*X^0, aaX^0X^0	$yp(y)$	
AaX^*Y	$\frac{3}{4}zp(\frac{3}{4}z)$	
AaX^*X^0, AaX^0X^0	$zp(z)$	

We have solved this for the value of y which is uninvadable (evolutionarily stable) both for the case with X^* absent or very rare and for X^* common (with the different *aa* genotypes in their respective frequencies according

to table 2 with all segregation ratios $\frac{1}{2}$). Numerical results for the two cases $p(y) = e^{-y}$ and $p(y) = e^{-\frac{1}{2}y^2}$ are presented below. For both of these functions, the presence of X^* has only a small effect on the level of compensation, which is in any case high:

$p(y)$	X^* absent or rare		X^* present	
	Litter size	Rel. fertility of X^*Y	Litter size	Rel. fertility of X^*Y
e^{-y}	1	0·963	1·044	0·974
$e^{-\frac{1}{2}y^2}$	1	0·933	1·032	0·947

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