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Population dynamics and the evolution of sex-determination in lemmings

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SUMMARY

The hypothesis is advanced that the evolutionary stability of the unusual sex-determining mechanisms of the Wood Lemming (*Myopus schisticolor*) and of the Varying Lemming (*Dicrostonyx torquatus*) is a direct consequence of certain characteristic features of their population dynamics, and that these include phases of unrestrained population growth and of mass dispersal. Computer simulations confirm the feasibility of such an explanation. Predictions of this hypothesis are found to differ in a potentially testable manner from those of the 'inbreeding' hypothesis of Stenseth (1978). The demonstration of such a direct link between population ecology and evolutionary genetics would, if substantiated, be exceptional in mammals.

1. INTRODUCTION

The recent discovery of unusual sex-determining mechanisms in two species of lemming poses interesting questions for evolutionary genetics. The mechanisms themselves have been described by Fredga *et al.* (1976, 1977) for the Wood Lemming, *Myopus schisticolor*, and by Gileva & Chebotar (1979) for the Varying Lemming, *Dicrostonyx torquatus*, and although some details remain to be clarified (for example, whether the *Y* chromosome in *D. torquatus* exists as a separate entity or as a translocation), the facts given in these papers are sufficient for the present purpose. Briefly, the essential feature is the existence of a gene, or gene complex, the effect of which is to produce a female phenotype in those individuals that carry it and that would otherwise be male. Three types of female occur, all phenotypically indistinguishable and of equal fertility. Type 1 are *XX* as are all their daughters. Type 2 are also *XX* but their daughters may be either *XX* or of Type 3. Type 3 are karyotypically indistinguishable from males (*XY* in *M. schisticolor*, probably *XO* in *D. torquatus*). The species differ in the degree of meiotic drive for *X*-chromosomes in Type 3 females. In *M. schisticolor* these females produce few if any sons, implying almost complete meiotic drive in favour of *X*-bearing gametes; whereas in *D. torquatus* about one third of their progeny are male, implying random segregation and absence of meiotic drive.

Bengtsson (1977) has explored some of the evolutionary consequences of this

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system. In particular, by calculating the genotypic frequencies for each generation in terms of those for the preceding one, he has demonstrated that a stable equilibrium is possible if the 'male-suppressor' gene is X-linked, but not if it is autosomal. Stability is also possible with Y-linkage, but only under rather special and unlikely conditions. He has shown further that, under the assumptions of X-linkage, random mating, equal fitness of all females and complete meiotic drive in favour of X-gametes, the equilibrium frequencies of males and of Type 1, Type 2 and Type 3 females are in the ratios 1:1:1:1, giving a sex ratio (males:females) of 1:3. These values are close to those observed in *M. schisticolor* both in the wild and in breeding studies (Kalela & Oksala, 1966; Fredga *et al.* 1977). Bengtsson's model can be extended to *D. torquatus* by assuming absence of meiotic drive, in which case the equilibrium frequencies of the 4 genotypes are respectively in the ratios 25:21:7:7, giving a sex ratio of 5:7.

These results are of great interest, but do not in themselves explain why, of all mammals examined to date, lemmings alone possess such a mechanism. It is the purpose of this paper to address this wider question.

2. THE PROBLEM

From the evidence provided by breeding studies (Fredga *et al.* 1977; Gileva & Chebotar, 1979) and from the results of Bengtsson's analysis, I shall assume X-linkage of the 'male-suppressor' gene and denote an X-chromosome carrying it by X^*. Further circumstantial evidence for X-linkage comes from Herbst *et al.* (1978) who recently reported that the postulated X and X^* chromosomes are cytologically distinguishable from each other in *Myopus*. For simplicity, subsequent remarks will be confined to a system with complete meiotic drive for X^* over Y in Type 3 females, although most of the qualitative conclusions can be taken to apply to a greater or lesser extent to a system with any degree of meiotic drive. Table 1 therefore represents the genotypes assumed to be present and their possible offspring, and corresponds to the hypothesis of Fredga *et al.* (1977) for *M. schisticolor*.

The difficulty of explaining the existence of such a system in nature can be appreciated by considering the relative genetic contributions of the 4 genotypes to generations far in the future – that is, their 'reproductive values' as defined, for example, by Bodmer & Edwards (1960). The Appendix contains a proof that at equilibrium the reproductive values of males and of Type 1, Type 2 and Type 3 females are respectively in the ratios 6:3:2:1. This is a surprising result, since it implies that an autosomal gene leaves on average six times more copies of itself to posterity if it is carried in an XY individual than if it is carried in an X^*Y one. Thus if such a gene were a mutation with the ability to neutralize the male-suppressing effect of X^*, it would have a considerable selective advantage over its silent allele. A more intuitive approach is to consider the fate of such a mutation in the immediately following generation. Since at equilibrium males have three times as many offspring as females, the mutant will pass on three times as many

copies of itself to the next generation as will its silent allele. If the mutation is Y-linked (denoted by Y^*; see Table 2) the advantage is even greater since the silent allele, if carried in an X^*Y individual, would not be passed on to the next generation at all. Finally, even if the mutation is X-linked it can be shown at equilibrium to pass on 50% more copies of itself to the next generation as does

Table 1. *The progeny of each mating in the system proposed for* Myopus *by Fredga* et al. *(1977), and assumed here.* X* *denotes a mutant* X*-chromosome carrying a 'male-suppressor' gene*

(The system assumes complete meiotic drive for X^* over Y in Type 3 females.)

	Female parent					
	Type 1: XX		Type 2: XX^*		Type 3: X^*Y	
Male parent	♂	♀	♂	♀	♂	♀
XY	XY	XX	XY	XX XX^* X^*Y	—	XX^* X^*Y

Table 2. *As Table 1, but with the addition of a mutant* Y *chromosome, denoted by* Y*, *which neutralizes the male-suppressing effect of* X*

	Female parent							
	Type 1: XX		Type 2: XX^*		X^*X^*		Type 3: X^*Y	
Male parent	♂	♀	♂	♀	♂	♀	♂	♀
XY	XY	XX	XY	XX XX^* X^*Y	—	XX^* X^*Y	—	XX^* X^*Y
XY^*	XY^*	XX	XY^* X^*Y^*	XX XX^*	X^*Y^*	XX^*	X^*Y^*	XX^*
X^*Y^*	XY^*	XX^*	XY^* X^*Y^*	XX^* X^*X^*	X^*Y^*	X^*X^*	X^*Y^*	X^*X^*

the silent allele. We must therefore conclude that there is strong pressure from the rest of the genome to neutralize the 'male-suppressor' effect of X^*, and thus restore the conventional X/Y system. This provides a possible explanation for the extreme rarity of the lemming system, since it implies that only in the presence of strong selective forces opposing this pressure can such a system survive. Stenseth (1978) has put forward the hypothesis that the system is maintained by recurrent inbreeding occurring with particular intensity at periods of minimum population density. Simulation studies by Maynard Smith & Stenseth (1978) provide some support for this theory inasmuch as certain mating schemes, involving increased probabilities of mating between close relatives, are found to lead to a stable frequency of X^* even when Y^* is initially present. Stenseth's

hypothesis is considered in more detail below, but first I wish to suggest an alternative explanation.

3. AN ALTERNATIVE TO STENSETH'S HYPOTHESIS

Lemmings are not unusual among mammals because of their method of sex-determination alone. They are also renowned for their unusual population dynamics, characterized by 3–4 year cycles of great increases in numbers followed by sudden declines (see e.g. Marsden, 1964). Field studies suggest that the increase in density during a population cycle may amount to 2–3 orders of magnitude (Shelford, 1943; Chernyavsky, 1978). Although many other, particularly arctic, species also undergo periodic fluctuations in density it is doubtful whether any attain increases of this magnitude, and it is therefore natural to speculate whether the two features are related and whether the method of sex-determination is in some sense a consequence of the population dynamics. That this may indeed be the case is suggested by the following argument.

Consider a population with a conventional X/Y sex-determining mechanism and a sex ratio of one, and suppose that a particular individual acquires a mutant X^* chromosome. Suppose further that X^* avoids immediate extinction and spreads a certain distance to define a local 'pocket' in which the male:female sex ratio is less than one. Intuitive reasoning suggests that the ultimate fate of X^* will depend critically on the conditions of population growth. For simplicity, two such conditions may be distinguished: (1) 'stable': the females within an area produce sufficient offspring of reproductive age to maintain a constant population density within that area; thus, if the proportion of females is low the average number of viable offspring produced by each female is high, and *vice versa*; (2) 'unrestrained': the average number of offspring of reproductive age produced by each female is constant from one area to another, and independent of the local population density.

In either condition we may assume that every female mates and hence, since all males have the same genotype, that random mating occurs. In addition, in the 'unrestrained' condition all female genotypes have equal reproductive fitness. As a result, all the assumptions of Bengtsson's simplified model are satisfied and the frequency of X^* will increase until the 1:1:1:1 equilibrium is established throughout the population. In the 'stable' condition, however, females within the pocket have lower reproductive fitness (in the evolutionary sense) than those outside, because they each produce fewer viable offspring in order to maintain a constant local population density. As a result, and since X^* is carried only by these females, X^* is at a selective disadvantage and will eventually be either established at a lower equilibrium frequency than in the 'unrestrained' condition or eliminated completely. Furthermore, reasons are given below (Section 5) for concluding that another unusual feature of lemming population dynamics – that of periodic mass dispersal – also contributes to the long-term persistence of X^*. Based on these considerations I therefore propose the following hypothesis: *that*

the evolutionary stability of the unusual sex-determining mechanisms of Myopus *and* Dicrostonyx *is a direct consequence of certain characteristic features of their population dynamics, and that these include phases of unrestrained population growth and of mass dispersal.*

Because intuitive arguments in evolutionary genetics are frequently controversial, I have used computer simulation in an attempt to support or refute the argument presented above and, further, to examine the consequences of introducing the Y-linked mutation, Y^*, that neutralizes X^*. In the latter case, intuition is of little help since the number of possible mating types is greatly increased, and random mating can no longer be assumed. The role of dispersal is not examined in the simulations since it can, I believe, be considered as a separate issue, and can be deduced by a purely intuitive approach. A full discussion of this point is therefore deferred until Section 5.

4. SIMULATION

In the simulated model, the generations are assumed to be discrete and non-overlapping, and the members of each generation are envisaged as occupying a fixed, pre-assigned number (denoted by NP) of positions equally spaced around the circumference of a circle. The rules for producing the $(i+1)$th generation from the ith are as follows: (1) each female in the ith generation mates; in the $X/X^*/Y/Y^*$ system (Table 2) the male partner is specified to be the nearest one or, if two are equidistant, one of the two is chosen at random; in the $X/X^*/Y$ system (Table 1) the identity of the male partner is immaterial since all males have the same genotype; (2) the number of offspring of each female depends on the conditions of population growth: in the 'stable' condition each individual, male or female, is given a score of 1, which can be thought of as representing, in a general sense, its ecological requirements or 'living-space'; each male then donates his score to the nearest female (or if two are equidistant to one of the two chosen at random); the number of offspring of each female is then equal to her total score, since this represents the amount of 'living-space' locally available to her for the use of her offspring; this rule satisfies the requirement that the average number of viable offspring of each female is inversely proportional to the local frequency of females, and hence ensures that the local population density remains constant; in the 'unrestrained' condition each female produces a fixed, pre-assigned number (denoted by NCON) of offspring; (3) the genotypes of the offspring of each female are then selected independently, at random and with equal probability from among those possible for the given mating (as indicated in Tables 1 and 2), and placed consecutively around the circumference of a new circle so that the ordering of the sibships in the $(i+1)$th generation is the same as that of their mothers in the ith; (4) in the 'unrestrained' condition the $(i+1)$th generation will in general be larger than the ith; hence, to avoid problems of computer space and time, individuals are removed at random in order to reduce the size of the $(i+1)$th generation to NP; since this is a random process it has

no effect on the expected gene frequencies; in the 'stable' condition the $(i+1)$th generation is necessarily the same size as the ith.

All simulations were run with NP = 250. Two values of NCON, 3 and 5, were used in the 'unrestrained' condition, but since the conclusions were in all important respects identical, the two sets of results were combined. The 'stable' and 'unrestrained' models were each run under each of three different sets of initial conditions, which are now described in detail.

(*a*) The initial generation was assumed to be in 1:1:1:1 equilibrium. That is, each individual was assigned, randomly and with equal probability, one of the genotypes XY, XX, XX^* or X^*Y.

Results: (1) 'stable' condition: in 20 simulations X^* became extinct in every case, after a mean of 465 generations (minimum 196, maximum 770); (2) 'unrestrained' condition: in 20 simulations there were no extinctions; taking mean frequencies over generations 451–500 inclusive, the lowest and highest values for any genotype in any simulation were 0·23 and 0·28 respectively; thus the equilibrium was maintained for at least 500 generations and would probably have continued indefinitely; as explained above, this result could have been predicted from Bengtsson's model.

(*b*) The initial generation was assumed to be in 'conventional' equilibrium but with a number of X^* chromosomes subsequently introduced. Specifically, each individual was initially assigned, randomly and with equal probability, one of the genotypes XX or XY. Subsequently, the 50 individuals occupying the odd-numbered positions between 1 and 99 inclusive were replaced by individuals with the genotype XX^*.

Results: (1) 'stable' condition: in 20 simulations X^* became extinct in every case, after a mean of 386 generations (minimum 97, maximum 747); (2) 'unrestrained' condition: in 20 simulations there were no extinctions; taking mean frequencies over generations 951–1000 inclusive, the lowest and highest values for any genotype in any simulation were 0·24 and 0·27 respectively; the mean number of generations until X^* first exceeded its equilibrium frequency of 0·25 was about 40 (minimum 11, maximum 90); thus the 1:1:1:1 equilibrium was attained after about 40 generations and thereafter sustained apparently indefinitely; this result could also have been predicted from Bengtsson's model.

(*c*) The initial generation was assumed to be in 1:1:1:1 equilibrium, but with a number of Y^* chromosomes subsequently introduced. Specifically, each individual was initially assigned, randomly and with equal probability, one of the genotypes XY, XX, XX^* or X^*Y. Subsequently the 50 individuals occupying the odd-numbered positions between 1 and 99 inclusive were replaced by individuals with the genotype X^*Y^*.

Results: (1) 'stable' condition: in 20 simulations Y became extinct in 19 and X^* in 1; the mean number of generations to extinction was 459 (minimum 156, maximum 1442); note that the effects of extinction of Y and of X^* are indistinguishable, since in either case the remaining genotypes all have a 1:1 sex ratio among their progeny, and the 'conventional' system is established; (2) 'un-

restrained' condition: in 40 simulations Y^* became extinct in every case, after a mean of 30 generations (minimum 10, maximum 72); it is a reasonable inference from the results of (*a*) and (*b*) that the 1:1:1:1 equilibrium would have been attained after the disappearance of Y^*.

Simulation thus provides unequivocal support for the intuitive argument – the $X/X^*/Y$ system was able to survive *only* in the 'unrestrained' condition, and the 'equal sex ratio' system *only* in the 'stable' condition. Evidently these conclusions extend even to the more complex system containing Y^*.

5. DISCUSSION

These results demonstrate that the evolutionary fate of certain sex-determining mechanisms may depend on the dynamics of the population in which they occur. They also suggest a stronger conclusion, namely that the *particular* sex-determining mechanism of lemmings is favoured by their *particular* population dynamics, while the conventional X/Y mechanism is favoured by a type of population dynamics characteristic of many, if not most, other mammalian species. This is, of course, a greatly simplified picture. The reality is probably that all species exhibit both types of population dynamics to a greater or lesser extent, but that only in lemmings (of those examined to date) is the balance tipped so far in the direction of unrestrained growth to permit the long-term survival of mutant X^* chromosomes and/or to prevent the spread of mutant Y^* chromosomes. If this is so, it suggests a number of potentially testable predictions. Thus, if lemming populations undergo alternating phases of stability, followed by phases of rapid increase characterzied by unrestrained reproduction, then the male:female sex ratio should increase during the former phase and decrease during the latter. Obviously it would be essential to discount the effect of differential mortality between the sexes, most simply by determining the sex ratio at birth. In just such a study, Chernyavsky (1978) reported that the sex ratio in *D. torquatus* declined from near equality at the population minimum to about 10:17 after a period of increase. Again, if in fact a population contains some Y^* chromosomes, they too would be expected to increase in frequency during the former phase and decrease during the latter. The existence of such chromosomes could be inferred from the presence in the population of males whose offspring have a 1:1 sex ratio irrespective of the genotype of their mates.

It might be argued that the effects of autosomal and X-linked neutralizing mutations should have been examined in addition to those of Y^*, but in practice this is unnecessary. We have already seen that a neutralizing mutation would be expected to exert its greatest effect when Y-linked. Thus if Y^* disappears from the population it follows that an autosomal or X-linked neutralizing mutation should disappear even more rapidly. On the other hand, if Y^* remains in the population and either Y or X^* disappears, then the effects of an autosomal or X-linked mutation become irrelevant. Since these were the only types of outcome observed in the simulations then, provided we assume that a Y-linked

neutralizing mutation is always potentially present in the population, it follows that it is sufficient to consider the effects of such a mutation alone.

So far we have not considered the effects of mortality and dispersal. Provided the population density is everywhere stable in the long-term, mortality will in general act to inhibit long-term changes in genotypic frequencies, since those genotypes that increase most rapidly during the phase of population growth (in this context the Types 2 and 3 females) will be precisely the ones to suffer the highest mortality later. However, the degree of inhibition depends on the dispersive behaviour of the population. At one extreme, in a completely sedentary population the inhibition is complete, since any change in genotype frequency during the growth phase is exactly compensated during the phase of population decline. At the other extreme, we might envisage a population that became randomly redistributed throughout its range following the growth phase. In this situation there is no inhibition, since the burden of mortality is shared equally by all genotypes and the genotype frequencies therefore remain the same throughout the phase of population decline. In the present context, it follows that the latter situation is the more favourable to the spread of the Types 2 and 3 genotypes, and hence of X^*. Where, then, do lemming populations stand between these extremes? It is perhaps significant that, so far from being sedentary, they display arguably the most dramatic dispersive behaviour of any terrestrial mammal. Characteristic features of these dispersals (Marsden, 1964) are the large numbers involved, the tendency of individuals to move outwards in all directions from a central area (rather than moving *en masse* from one point to another), and the fact that they tend to occur at times of peak density. Such conditions would be expected to enable the Types 2 and 3 females, by emigrating into areas of lower density, to consolidate some of the increase in frequency they have achieved during the growth phase. This would still be the case even after allowing for the increased mortality associated with dispersal, provided such mortality is less than these genotypes would experience by remaining in an area of high density. At the risk of some repetition, the argument can perhaps be clarified by reference to the 'pocket' example given above. Because of the higher proportion of females there, the population within the pocket increases more rapidly during a period of unrestrained growth than does the population outside. If, following such a period, dispersal takes place from high density to low density areas, it follows that there will be a net movement of population outwards from the pocket. As a result some Type 2 and Type 3 females will occupy the area outside the pocket. If mortality now operates to reduce the population everywhere to what it was before the period of growth, the situation within the original area of the pocket will be restored to its earlier state, whereas outside this area some Type 2 and Type 3 females will survive where originally there were none. Thus, taking the population as a whole, there will be a net increase over the whole cycle in the proportions of Type 2 and Type 3 females and hence of X^*. If, on the other hand, no dispersal occurs, mortality simply restores the population everywhere to its earlier state and the genotype frequencies remain unaltered.

If this view is correct it leads to a further testable prediction – that among dispersing groups the proportion of females, in particular of Types 2 and 3, should be higher than among non-dispersing groups (though again a note of caution is necessary concerning the possible effects of differential mortality between the sexes).

The models presented here and those of Maynard Smith & Stenseth (1978) have in common the not unrealistic assumption of an increased probability of mating between individuals born in close physical proximity. This explains why some of the simulations reported in the earlier study gave similar results to those reported here. However the hypotheses proposed to explain these results differ in at least one important respect. According to Stenseth's hypothesis, the forces maintaining X^* in the population operate most intensively during periods of minimum density, whereas according to the present hypothesis they do so during periods of population growth. As a result, Stenseth's hypothesis predicts that the proportion of females will be highest at the end of a period of low density (i.e. after a relatively long period of intensive inbreeding), and lowest after a period of population growth when inbreeding is reduced. As we have seen, however, the present hypothesis predicts exactly the converse. The possibility therefore exists of testing the validity of these hypotheses by field studies. On theoretical grounds, Stenseth's hypothesis is open to the objection that, unless Y^* occurs at a relatively high frequency (a situation that has not been shown to occur in practice), almost all males have the identical genotype, XY, and thus the degree of relatedness of a female to her mate would be expected to have only a minimal influence on genotype frequencies.

Finally, it would be of interest to see whether either hypothesis can account for the differences between the sex-determining mechanisms of the various species of *Myopus*, *Dicrostonyx* and *Lemmus* in terms of appropriate features of their population ecology. It is therefore to be hoped that further field studies will be undertaken since the demonstration of a close link between population ecology and evolutionary genetics is unusual in mammals.

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APPENDIX

The reproductive values of the 4 genotypes under Fredga *et al.'s* (1977) hypothesis are derived from the sequence of rows in Table 3. The following points require clarification.

Since each individual receives equal genetic contributions from its male and female parents, the total reproductive values of males and of females must be equal. In Row 2 the reproductive value of each male is arbitrarily set to 1. It then follows that those of the individual females of Types 1, 2 and 3 can be represented respectively by a, b and $1-a-b$. The mean value of the offspring of each genotype can then be calculated from Table 1 under the assumption

of random mating. Thus, the offspring of Type 1 females consist of equal numbers of XY (reproductive value = 1) and XX (reproductive value = a). Hence the mean value of the offspring is $(1+a)/2$. Type 2 females produce equal numbers of all 4 genotypes (as do the males) and hence the mean reproductive value of their offspring is $(1+a+b+1-a-b)/4 = \frac{1}{2}$. Type 3 females produce equal numbers of XX^* and X^*Y offspring and hence their mean reproductive value is $(b+1-a-b)/2 = (1-a)/2$.

Table 3. *Derivation of reproductive values at equilibrium in the system represented in Table 1*

	Males	Type 1	Type 2	Type 3
Row 1: Equilibrium frequency	0·25	0·25	0·25	0·25
Row 2: Reproductive value/individual	1	a	b	$1-a-b$
Row 3: Mean reproductive value of offspring	$\frac{1}{2}$	$\frac{1}{2}(1+a)$	$\frac{1}{2}$	$\frac{1}{2}(1-a)$
Row 4: No. of offspring at equilibrium	4	$\frac{4}{3}$	$\frac{4}{3}$	$\frac{4}{3}$
Row 5: Total reproductive value of offspring	1	$\frac{1}{3}(1+a)$	$\frac{1}{3}$	$\frac{1}{3}(1-a)$

Row 5 is derived as half the product of Rows 3 and 4, the factor of a half being introduced to compensate for the fact that each offspring has been counted twice, once each as the offspring of its male and female parents. Finally, since Rows 2 and 5 must be equal at equilibrium, the values of a and b must be $\frac{1}{2}$ and $\frac{1}{3}$ respectively. Hence the reproductive values of the genotypes are respectively in the ratios 6:3:2:1.

A conceptually simpler, but computationally more tedious, method is to calculate the relative genetic contributions of each genotype to the 1st, 2nd, 3rd, etc. subsequent generations. The values for the first three generations are respectively 6:2:2:2, 6:(2·7):2:(1·3) and 6:(2·9):2:(1·1) and converge rapidly to the asymptotic value of 6:3:2:1.

These results may appear counter-intuitive unless the distinction between 'reproductive value' and 'reproductive fitness' is borne in mind. The former refers to the genetic contribution to posterity (i.e. to generations far in the future), while the latter refers to the genetic contribution to the subsequent generation. The former is of course the crucial quantity for evolutionary studies. Thus, although all 3 types of female are assumed to have equal reproductive fitness, their genetic contributions to the grand-progeny and subsequent generations differ as a result of the differences in the proportions of males among their offspring. For example, since males have more offspring than females, Type 3 females (who have no male offspring) have fewer grand-progeny than Type 1 females (half of whose offspring are males), while Type 2 females (a quarter of whose offspring are males) have an intermediate number.

REFERENCES

BENGTSSON, B. O. (1977). Evolution of the sex ratio in the Wood Lemming, *Myopus schisticolor*. In *Measuring Selection in Natural Populations* (ed. T. Fenchel and F. Christiansen). Berlin: Springer-Verlag.

BODMER, W. F. & EDWARDS, A. W. F. (1960). Natural selection and the sex ratio. *Annals of Human Genetics* **24**, 239–244.

CHERNYAVSKY, F. B. (1978). Population cycles of Siberian and Varying Lemmings on Wrangel Island. Transactions of the Second Congress of the Theriological Society of the U.S.S.R., Moscow, 210–212. (In Russian.)

FREDGA, K., GROPP, A., WINKING, H. & FRANK, F. (1976). Fertile XX- and XY-type females in the Wood Lemming, *Myopus schisticolor*. *Nature* **261**, 225–227.

FREDGA, K., GROPP, A., WINKING, H. & FRANK, F. (1977). A hypothesis explaining the exceptional sex ratio in the Wood Lemming (*Myopus schisticolor*). *Hereditas* **85**, 101–104.

GILEVA, E. A. & CHEBOTAR, N. A. (1979). Fertile *XO* males and females in the Varying Lemming, *Dicrostonyx torquatus* Pall. (1779). A unique genetic system of sex determination. *Heredity* **42**, 66–77.

HERBST, E. W., FREDGA, K., FRANK, F., WINKING, H. & GROPP, A. (1978). Cytological identification of two X-chromosome types in the Wood Lemming (*Myopus schisticolor*). *Chromosoma* (*Berl.*) **69**, 185–191.

KALELA, O. & OKSALA, T. (1966). Sex ratio in the wood lemming, *Myopus schisticolor* (Lilljeb.), in nature and in captivity. *Annales Universitatis Turkuensis*, Ser. AII, **37**, 1–24.

MARSDEN, W. (1964). *The Lemming Year*. Chatto & Windus.

MAYNARD SMITH, J. & STENSETH, N. C. (1978). On the evolutionary stability of the female biased sex ratio in the wood lemming (*Myopus schisticolor*): the effect of inbreeding. *Heredity* **41**, 205–214.

SHELFORD, V. E. (1943). The abundance of the collared lemming in the Churchill area, 1929–40. *Ecology* **24**, 472–484.

STENSETH, N. C. (1978). Is the female biased sex ratio in wood lemming *Myopus schisticolor* maintained by cyclic inbreeding? *Oikos* **30**, 83–89.