Facultative sex ratios and population dynamics

THE theory of R. A. Fisher[1] for the effect of natural selection on the sex ratio predicts the population to be in evolutionary equilibrium when half of the parental reproductive resources are devoted to sons, and half to daughters. Provided that sons and daughters are of equal cost, this implies a sex ratio of 1/2 males at or near conception, and the result is independent of differential male and female survival after the period of parental care[2-5]. Most models demonstrating these results assume discrete generations, but some show that they also follow with overlapping generations[6-8]. These latter models typically assume the population to be in stable age distribution. Here, we relax that assumption, and show that selection can favour genes which result in the temporary overproduction of one or the other sex, under certain general conditions. The conditions are; overlap in generations; different temporal changes in life history expectations for the two sexes; parental ability to vary the sex ratio in response to the life history changes. The results are of interest because stable age distributions are probably uncommon in nature, with fluctuations in survival more the norm.

To investigate sex ratio selection under these new conditions, two models were developed, a cyclical model and a perturbation model. In the cyclical model, life history expectations for males and females vary cyclically over time, perhaps because of seasonality. Consider the following simple example. Let individuals be born in the spring and the autumn. Males and females born in the spring survive until the autumn. They then reproduce and die. Of those born in the autumn, the females survive to reproduce in the spring (and then die), while the males survive to reproduce in the spring and some survive to reproduce again the following autumn. Life history expectations differ for individuals born in the spring versus the autumn, and some overlap exists in generations (Fig. 1). This example is extreme in that only one sex ($\male$) reproduces twice (so that spring-born males face reproductive competition from males born the previous autumn, while spring-born females face no such competition).

If r_f and r_s are the sex ratios (proportion males) born in the autumn and spring respectively, it is easy to show (using computer simulation or analytical population genetics) that the sex ratios favoured by natural selection are such that $r_f > 1/2$ while $r_s < 1/2$. As the amount of competition faced by spring-born males for mates in the autumn increases due to the presence of autumn-born males of the previous year, the deviations in sex ratios
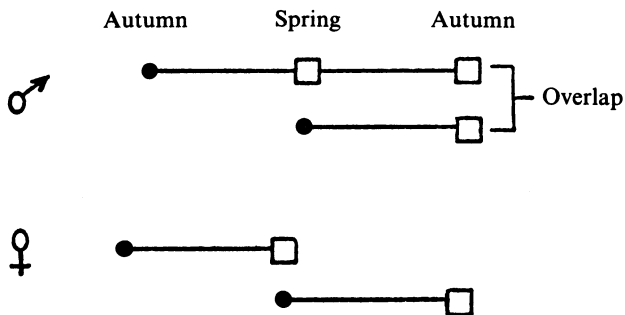

Fig. 1 Diagrammatic representation of the hypothetical set of life histories in a seasonal environment. ●, Born; □, mating period.
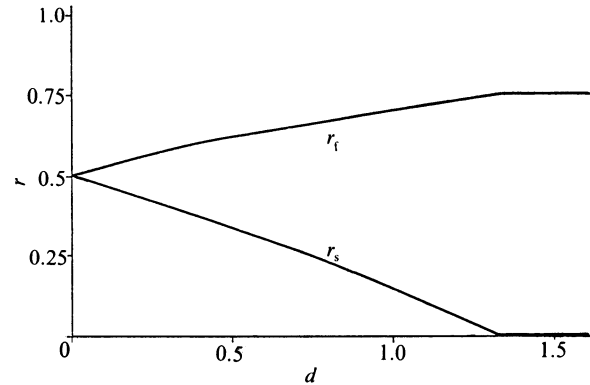

Fig. 2 The equilibrium solutions for autumn sex ratio (r_f) and spring sex ratio (r_s) as a function of d, a measure of the competition for mates faced by spring-born males in the autumn due to the presence of autumn-born males. Specifically, if N_f is the number born in the autumn, N_s the number born in the spring, S_f the survival of autumn-born males to the following autumn, S_s the survival of spring-born males to autumn and C the competitive ability of autumn-born males relative to spring-born males in gaining mates during the autumn, then
$$d = C \, (N_f \, S_f / N_s \, S_s)$$
As d increases, the reproductive success of spring-born males decreases.

increase from 1/2. Figure 2 shows this effect of increasing overlap upon the equilibrium sex ratios among spring-born and autumn-born, as derived from an analytical population genetics treatment of the life history outlined above.

A more extensive treatment of our model, with consideration of other seasonal life histories (such as a model where both males and females have overlap in generations) will be published elsewhere. However, the model just presented captures the basic points. Selection can favour seasonal shifts in sex ratio if males and females experience different life histories which vary seasonally, and, because of overlapping generations, those born at different times compete with each other for relative reproductive success. This creates a situation where a parent may 'derive' more fitness by shifting the sex ratio towards offspring of the sex with improved reproductive success relative to the other sex. We note here that if facultative sex ratios are not possible (so that $r_s = r_f$), our analysis shows that the equilibrium sex ratio returns again to 1/2 males.

The model just presented assumed the life history characteristics to vary seasonally. We now consider a perturbation model. Assume that a population with overlapping generations has a stable age distribution, and suppose that the population is occasionally perturbed by a period of exceptional mortality or survival, followed by a return to 'normal conditions.' These sorts of perturbations may be common in nature; they can be caused by dramatic weather changes, such as cold or heat spells and floods. In species subject to strong sexual selection, one sex can be affected considerably more than the other by such occurrences, due to behavioural and/or physiological differences. Males are known to be more vulnerable to environmental stress than females in many species[9,10]. The perturbations must be dramatic and fairly frequent if they are to select for facultative sex ratios. The question follows: does selection favour genes which allow an individual (mother or father) to respond to the change from stable age distribution conditions by overproducing the sex which suffered the greater mortality (in terms of deviation from normal survival)? We investigated this question using computer simulation.

Let us suppose, for example, that the perturbation caused young males to suffer twice their normal rate of mortality between birth and age one. A gene which biases sex ratio towards males in the next generation would be favoured because of the reduced competition faced by those males relative to their own expected mortality. On the other hand, if females suffered twice their normal rate of mortality, selection would favour biasing sex ratio towards females in the next generation. Again, if the facultative response is not possible (a single sex ratio is used all the time), our computer results indicate that 1/2 is the equilibrium value, and that the violation of stable age distribution does not itself alter the value from that predicted under Fisher's theory.

We have discussed both these models in terms of varying ability to survive; however, the results should also hold for other fitness components which vary over time (such as competition for territories; do early spring-born males procure the best territories?). The basic difference between these two models is that the life history changes are predictable in the cyclical model and unpredictable in the perturbation model. Therefore, in the latter case the sex ratio response can only occur after the life history change. Both models predict that selection will favour a shift in offspring sex ratio towards the sex with improved reproductive success relative to the other sex.

Our theory requires that animals or plants are able to alter the sex ratio among their offspring, and that they can do it at the appropriate time (so that there must exist appropriate cues). We indicate here some case histories of particular interest.

Haplodiploidy (fertilised eggs produce females, unfertilised eggs males) occurs among Arthropods and gives the female much control over the sex ratio of the brood[11,12]. There is evidence for temporal shifts of sex ratio in some parasitic Hymenoptera[12,13]. Photoperiod (a seasonal cue) affects progeny sex ratio in the parasitoid wasp *Pteromalus puparum*. Under LD10 : 14, two to three times as many female progeny are produced than at LD14 : 10, although fertility is unaffected[14]. In another parasitic wasp, *Camporetes perdisticus*, the greatest percentage of females is produced under LD12 : 12 (ref. 15). Among mites of the genus *Macrocheles,* offspring sex ratio varies inversely with the adult sex ratio[16,17]. Such a response is predicted under the perturbation model.

Life history expectations vary during the population cycle of multi-voltine rodents and these changes are different for males and females[18-22]. Juvenile sex ratios have been found to change with the population cycle in several species[21-23]; however, this is difficult to interpret because of potential bias. The effect could be due to changes in behaviour, mortality after weaning, or rates of maturation. In one case, litter sex ratios of field populations of the bank vole (*Clethrionomys glariolus*) were examined by bringing pregnant females into the laboratory. The sex ratio of resulting litters was found to vary significantly with the population cycle[24].

There are several phenomena consistent with the prediction of the perturbation model that a scarcity of one sex leads to its overproduction in the next generation. It has been demonstrated experimentally in the guppy (*Lebistes reticulatus*) that offspring sex ratio varies inversely with adult sex ratio[25]. In the plants *Silene dioca*[26] and *Rumex acetosa*[27], the sex ratio of seedlings varies inversely with density of pollen on the stigma. The physiological mechanism is speculated to be slow pollen tube growth of Y-bearing pollen. When pollen densities are low (a male

perturbation?) the Y-bearing pollen has an improved chance of fertilising ova. In a small population, the effect of random sampling on the adult sex ratio would have the same influence as a perturbation, therefore favouring a facultative response.

Another line of evidence for the perturbation model is the effect of delayed fertilisation on sex ratio. In many organisms, a delay in mating or fertilisation causes a dramatic increase in the proportion of males in the progeny. This is exactly to be expected if the mating delay reflects an abnormal scarcity of males. Hertwig[28] showed in the anuran *Rana exculenta* that the sex ratio of late fertilisation eggs gave a much greater proportion of males. Kuschakewitch[29] demonstrated that the result was not due to differential mortality (according to Huxley[30] and James[31]). Delayed fertilisation leads to a predominance of males in the trout (*Salmo iridens*)[32], *Drosophila melanogaster*[33], several species of mealy bugs (*Pseudococcus*)[31], a butterfly (*Talaeproia tubulosa*)[34], and three species of copepod (*Tisbe*)[35].

In the mealy bug, mating 0, 6, 8 and 10 weeks after emergence resulted in 102, 181, 327 and 991 males per 100 females, respectively. Sex ratio was ascertained at the prepupal stage; however, James[31] showed that the changes in sex ratio were not due to differential mortality between conception and the prepupal stage, and therefore must be due to prezygotic mechanisms.

Certainly, more detailed life history information is necessary to test the models conclusively; however, the examples mentioned are suggestive. Experiments are now being carried out in an attempt to select for faculative sex ratios in *Drosophila melanogaster*.

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1. Fisher, R. A. *The Genetical Theory of Natural Selection* (Oxford University Press, 1929).
2. Shaw, R. F. & Mohler, J. D. *Am. Nat.* **87**, 337–342 (1953).
3. Kolman, W. *Am. Nat.* **94**, 373–377 (1960).
4. Bodmer, W. F. & Edwards, A. W. F. *Ann. hum. Gen.* **24**, 239–244 (1960).
5. MacArthur, R. H in *Theoretical and Mathematical Biology* (eds Waterman, T. & Morowitz, H.) 388–397 (Blaisdell, Lexington, 1965).
6. Leigh, E. *Am. Nat.* **104**, 205–210 (1970).
7. Charnov, E. L. *Evolution* **29**, 366–368 (1975).
8. Leigh, E. G., Charnov, E. L. & Warner, R. R. *Proc. natn. Acad. Sci. U.S.A.* **73**, 3565–3660 (1976).
9. Latham, R. M. J. *Windl. Mant. Bull., Ottawa* **11**, 139–149 (1947).
10. Hamilton, J. B. *Recent Prog. Horm. Res.* **3**, 257–322 (1948).
11. Flanders, S. E. *Insectes Soc.* **3**, 325–334 (1956).
12. Clausen C. P. J. *N.Y. ent. Soc.* **47**, 1–9 (1939).
13. Flanders, S. E. *Q. Rev. Biol.* **21**, 135–143 (1946).
14. Bouletrean, M. *Ent. Exp. Appl.* **19**, 197–204 (1976).
15. Hoelscher, C. E. & Vinson, S. B. *Ann. ent. Soc. Am.* **64**, 1373–1376 (1971).
16. Filipponi, A., Mosna, B. & Petrelli, G. *Riv. Parasitt.* **32**, 193–218 (1972).
17. Filipponi, A. & Petrelli, G. *Riv. Parasitt.* **36**, 295–308 (1975).
18. Evans, F. C. *J. Mammal.* **30**, 351–363 (1949).
19. Petrusewicz, K. *Acta theriol.* **4**, 103–137 (1960).
20. Krebs, C. J., Gaines, M., Keller, B., Meyers, J. & Tamarin, R. *Science* **179**, 35–41 (1973).
21. Kalela, O. *Ann. Zool. Fenn.* **8**, 452–455 (1971).
22. Canham, R. P. *Can. J. Zool.* **48**, 809–811 (1970).
23. Fordham, R. A. *Ecology* **52**, 138–146 (1971).
24. Nanmov, S. P., Gibet, L. A. & Shatlova, S. P. *Zh. obshch. Biol.* **30**, 673–680 (1967).
25. Geodakian, V. A., Kosobutsky, V. I. & Bileva, D. S. *Genetika* **9**, 154–163 (1967).
26. Correns, C. *Hanb. Vererbursw.* **2**, 1–138 (1928).
27. Rychlewsk, J. & Kazimierez, Z. *Acta biol. cracov.* **18**, 101–114 (1975).
28. Hertwig, R. *Biol. Zbl.* **32**, 1–146 (1912).
29. Kuschakewitsch, S. *Festschrift R. Hertwig* **2**, 61–224 (1910).
30. Huxley, J. S. *J. Genet.* **10**, 265–276 (1920).
31. James, H. C. *Proc. R. ent. Soc. Lond.* **12**, 92–98 (1937).
32. Mrsic, W. *Arch. Mikr. Anat.* **98**, 129–209 (1923).
33. Hannah, A. *Z. Ord. Abst. Vererbl.* **86**, 574–599 (1955).
34. Seiler, J. *Retz, Arch. Zellforsch.* **15**, 249–268 (1920).
35. Volkmann-Rocco, B. *Biol. Bull.* **142**, 520–529 (1972).