

Sexual selection in guppies called into question

SIR—Breden and Stoner¹ attempted to test an important prediction from recent sexual-selection models^{2,3}. Their results may be consistent with the hypothesis that selection by predators on colour patterns of male guppies has led to a correlated evolutionary response in female mating preferences, but, as Endler⁴ points out, alternative explanations are not excluded. Here, I discuss some of the points made by Breden⁵ in his reply to Endler's criticisms, and raise some additional issues.

The existence of genetic differences in female mating preferences, suggested by Breden and Stoner's data, is further supported by my study⁶ of two different guppy populations. Females from the brighter (more orange) population appear to prefer bright males but females from the more drab population have no apparent preference, rather than a preference for drab males as suggested by Breden and Stoner's data. Both of these studies are in contrast with the results of a field introduction of a high-predation guppy population to a low-predation locality by Endler⁷. The change in male colour patterns after the introduction suggests that females in the original high-predation population prefer bright males, although the strength of the preference could have been less than in low-predation populations. The differences between these studies suggest that female preferences can differ in degree as well as in direction.

Breden and Stoner argue that their data on female behaviour provide evidence for mating preferences. Endler suggests that the females could respond to the models as though they are predators; other possibilities are that females respond to the models as potential sources of food or recognize the dull model as a female rather than as a male. Breden states that females perform 'move-toward' behaviour⁸ directed at the models, indicating that the responses are sexual, but it is not clear if my criteria⁸ would accurately distinguish sexual responses from other forms of directed swimming when females and males or models are separated by partitions. Comparisons of the responses of female guppies to other females, males, models, predators and other stimuli in partitioned and open aquaria are needed to determine whether the technique gives clear-cut evidence of female choice.

Even if Breden and Stoner's data do reflect differences in female mating preferences, their results could arise through processes other than correlated change in female preference in response to direct selection on male colour patterns. Any change in mating preferences could lead to a corresponding change in colour patterns, leading to a similar prediction of correlated variation in female preferences and male colour patterns across popu-

lations. The possibility that selection on females could act through risk of predation while close to a bright male should not be rejected without testing; other direct effects are possible. Selection against hybrids between different locally adapted populations (outbreeding depression) could favour females that prefer males from their own population. This could include the adaptation of colour patterns to local predation regimes, as in Breden and Stoner's model, but local adaptation unrelated to predation could also favour females that prefer local males. Direct selection unrelated to sexual selection could lead to differences in the visual system of females that could affect mate choice. Differences in the visual environment⁴ between streams could lead to biases in female choice. Female preferences might also diverge in separate populations as the result of random events during independent processes of sexual selection^{2,3}.

Breden and Stoner's argument that differences in female preferences have resulted specifically from differences in predation regime depends upon a comparison of only two high-predation and two low-predation populations, not a sufficient sample to warrant such a strong generalization. Differences in female preferences resulting from any of the processes described above could produce the observed results by chance. A substantial amount of colour pattern variation among populations appears to be unrelated to differences in predation; our unpublished data suggest this may be especially true of the particular populations studied by Breden and Stoner. It will be necessary to compare preferences in many more populations with known predation level and known colour pattern distribution, and to validate the experimental techniques, before we will know if the conclusions of Breden and Stoner are correct.

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Spot the mistake

SIR—There must be *something* wrong with Voorhees and Poggio's image analysis (*Nature* **333**, 364; 1988); their picture of a 'leopard' is, in fact, of a cheetah.

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Cystic fibrosis and chloride-secreting diarrhoea

SIR—In commenting on Rotter and Diamond's article¹ on principles by which recessive alleles are maintained at high frequency, Pritchard proposed² that the cystic fibrosis (CF) allele is increased in frequency because of increased fertility of men carrying a single allele. The frequency of the CF gene (1/20 in Caucasians) is too high to be sustained by mutations alone (see ref. 3). Conclusions about the maintenance and increase on the CF gene must take into account data on the biochemical basis of this disease.

Since the finding³ that CF sweat ducts have a decreased chloride permeability, much effort has led us closer to identifying the basic defect in CF. Two recent reports^{4,5} show that the chloride channels of the apical membrane in airway epithelial cells of CF patients does not open, even though they are known to be present, when stimulated by the catalytic portion of the cyclic AMP-dependent protein kinase. This suggests that, in CF, the apical Cl⁻ channels (or regulatory units) cannot be phosphorylated or do not respond. Although these results are obtained from airway cells, the same defect is probably present in other epithelial cells of affected organs such as pancreas or intestine.

Among the diseases that could produce a selective pressure are diarrhoeal diseases mediated by toxin-secreting bacteria. Two of these toxins, *Escherichia coli* LT (heat-labile) and cholera toxin, mediate their diarrhoeal effect through an increased level of cyclic AMP followed by a Cl⁻ secretion, particularly in the crypt cells⁷. This secretion could be mediated by the apical Cl⁻ channels defective in CF. Heterozygotes for the CF gene probably express 50 per cent defective cyclic AMP-dependent Cl⁻ channels as are the defective proteins in other recessive diseases. CF heterozygotes should thus have fewer Cl⁻ channels that could be opened and suffer less fluid losses on these types of diarrhoeas. This could have given CF heterozygotes a selective advantage when diarrhoeas caused many deaths in Europe and explain the high frequency of the CF gene found today.

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