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TWITTERING—A VOICE MUTATION IN *MICROTUS*

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It has been impressively demonstrated that behaviour, like structure, is genetically determined. It must, therefore, be affected by mutation. The isolation ("Kaspar-Hauser") experiments of ornithologists in particular have demonstrated that a great number of vocal performances in animals are genetically determined, but little is known about mutations affecting them. As rare mutations are most often observed in large laboratory stocks, rodents may be suitable subjects, provided that the breeder pays attention to the vocalisations of his animals (which is seldom the case).

So-called "singing" in mice (*Mus musculus*) has been observed on several occasions and was studied by Dice (1932). "The musical mouse produced a song which was bird-like in quality, but weak in volume. . . . Generally the song consisted of a series of pleasing musical chirps and twitters. . . . Some of the F_1 offspring had a faint song, which might better be called a 'chitter'. The chitter may be described as a faint rapid trill, quite musical in character." This was quoted by Grüneberg (1952), who himself continues: "Neither in the F_2 , nor in the F_3 nor in repeated backcrosses of daughters, grand-daughters, etc. to the original male were animals produced whose vocal performances equalled that of the original male. Thus, while its singing habit was not inherited, the weak 'chitter' in its descendants was probably determined genetically, though the mode of its inheritance could not be ascertained."

The present author has discovered a similar phenomenon in the Continental European Vole. The effect is genetically determined and appears to be the first well-established demonstration of a mutation directly affecting a behavioural character.

1. ORIGIN AND DESCRIPTION

A study of the mode of inheritance of several coat colour mutations found in nature has been carried out on the author's laboratory population of voles for several years (Frank and Zimmermann, 1957). These included recessive (*s*) and dominant (*W*) spotting. After inbred lines had been established following crossbreeding individuals with these two mutations, two females (related cousins) with deviant vocalisation appeared among the descendants in 1957.

Whereas the normal threatening call of *Microtus arvalis* is a monosyllabic, loud and shrill squeak (something like "dyeeep"), the deviant animals

produced a delicate, clear, and melodious twitter ("tee-tee-tee-tee-tee-tee"). This twitter closely resembles the vocalisations of shrews, for example the "greeting call" of *Crocidura* (Frank, 1953), and the vocalisations of some birds, such as certain *Limicolæ*. Thus, the vole vocalisation described is quite a new call, differing in intonation, pitch, and rhythm from the normal threatening call of the species. The deviant vocalisation is unmistakable from the very beginning: it is already recognisable as soon as the threat vocalisation begins to make its appearance in the sucklings. The striking difference between the calls of normal and twittering voles is clearly visible on sonagrams (Plate I) kindly prepared by Dr G. Thielcke (Max-Planck-Institut für Verhaltensphysiologie, Möggingen, Germany).

However, the descendants of the two original females included animals which produced a "broken twitter" and some which were entirely voiceless, in addition to the predominant "clear twitters". The voiceless animals could not be provoked into uttering any sounds by any of the means which normally elicit them. Further breeding has shown that these voiceless animals and the broken twitters also are homozygous for the twittering mutation.

The fact that the new mutation has a general effect upon vocalisation and thus may produce all gradations from complete dumbness to clear twittering was also demonstrated by some homozygotes whose vocalisation altered during the course of their lives. In some cases, originally voiceless animals twittered at a later stage, while clear twitterers sometimes changed to become broken twitterers or even completely voiceless.

The production of vocalisations is a very complex process. It stands to reason therefore that a mutation affecting the voice will not be manifested in such a uniform and distinct manner as, for instance, coat colour mutations. For this same reason, it has so far proved impossible to establish whether this abnormality in vocalisation is a general one caused by morphological changes in the larynx or specifically based upon nervous processes, though the latter explanation would seem to be the more likely.

This mutation of the voice has no effect upon the general behaviour of the voles. Normal and twittering animals lived together without any disturbance of their social communication. This is not surprising, since the auditory sense plays a minor role in communication between small mammals as compared to the senses of smell and of touch.

2. MODE OF INHERITANCE

The two original females were paired to males from separate wild-coloured lines, in which aberrations in vocalisation had never been observed. The twittering mutation proved to be recessive. In order to confirm the full accuracy of this result, many control lines were bred in the following years by re-crossing the descendants of the original females to normal lines. The results amply corroborated previous evidence.

However, the degree of manifestation of the mutation (column 6 of table 1) always remained under the expected values for recessive inheritance. Some sources of error must be noted in this connection, especially the crude method of scoring the animals by ear instead of employing objective appliances. Some of the animals classified as "normals" in this way turned out to be recessive homozygotes in the course of further breeding. In

Plate 1

Sonograms of the calls of *A.* normal vole *B.* twittering mutant, both recorded at a speed of 19 cm./sec. and reproduced at a speed of 9.5 cm./sec., carried out by G. Thielcke.

addition the loss of recessive homozygotes caused by higher mortality of the sucklings must be taken into account. Nevertheless, the distinct appearance of normally-voiced young in the litters of homozygote recessive pairs and the gradual disappearance (but never complete disappearance) of such exceptions during further breeding indicates that the twittering mutation exhibits incomplete penetration. This agrees with the observations made by Dice on *Mus musculus*, as quoted above.

The twittering mutation is not sex-linked, neither is it coupled to either of the two spotting mutations. However, the mutation was originally associated with a striking reduction in vitality, which expressed itself in the retardation of the speed and extent of bodily growth and in a higher mortality of suckling homozygotes as compared to heterozygotes. But consistent breeding with the strongest descendants and permanent cross-breeding

TABLE 1

Cross	Total offspring	Normal	Voiceless	Broken twittering	Clear twittering	Percentage manifestation (columns 3-5)
$tw/tw \times +/+$	504	504	...	...	...	0
$+ / tw \times + / tw$	533	434	24	23	52	18.6
$+ / tw \times tw/tw$	1152	661	77	107	307	42.6
$tw/tw \times tw/tw$	765	117	52	193	403	84.7
(early generations)						
$tw/tw \times tw/tw$	1067	99	8	142	818	90.7
(later generations)						

with robust animals of normal lines gradually improved the vitality of the homozygotes up to a normal or supernormal level within about ten generations. Thus, the striking reduction in vitality was apparently not attributable to the *tw* gene itself but to other, linked genes, and this disadvantageous coupling was corrected by recombination.

Similar associations of mutations with reduction in vitality are common and may be significant in ensuring genetic stabilisation of such characteristics as are themselves insufficiently controlled by natural selection. The latter is surely the case with vocal performances (at least in animals such as small mammals, in which vocalisation does not play a major part in social communication) so that the stabilisation of the specific vocal performance may be possible (or may in any case be facilitated) only when genetic coupling to another factor diminishing vitality is present.

3. SUMMARY

1. Two individuals in a laboratory population of *Microtus arvalis* (Rodentia) proved to be homozygous for a mutation affecting voice.
2. The normal threatening sound is a monosyllabic, loud, shrill squeak.
3. Homozygous mutants generally produce a clear or, less often, a broken twitter but they are sometimes voiceless.
4. The voice of the mutants may change with age.

5. The *tw* (twittering) mutation is recessive and of limited penetrance. It does not affect any other aspect of behaviour but it was originally associated with a striking reduction in viability.
6. Stocks are available on request.

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TWO RACES OF COD AT FAROE

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THE known transferrin types in the blood sera of cod (*Gadus morhua* L.) are controlled by five co-dominant allelomorphic genes which occur in different proportions in the races of cod tested at Norway (Møller, 1966) and at Greenland (Jamieson, 1967). Recent tests on transferrins in cod at Faroe suggest that there is some genetic isolation between the cod on the Faroe Plateau and those on the Faroe Bank. The Plateau is a shelf surrounding the islands, extending to the 200 metres isobath. The Bank lies about 150 kilometres south-west of the islands; its shallowest water is about 100 metres deep and its margin is also 200 metres deep. A channel 830 metres deep at its shallowest part separates the Bank from the Plateau. The area of the Plateau is about five times that of the Bank.

In a review of the statistics and biology of cod at Faroe, Jones (1966) discussed earlier, non-genetic evidence for this isolation. The results of tagging experiments over the past fifty years include rare examples of cod migrating between the Plateau and the Bank, and even fewer examples of cod migrating between Faroe and other regions. A study of otolith types also indicates that immigration from other regions occurs only rarely. In comparison with Plateau cod, the Bank fish appear lighter in colour, grow faster and have a higher mean number of vertebrae. These combined observations indicated the presence of distinct cod stocks at Faroe Plateau and Faroe Bank, but provided no direct demonstration of any genetic diversity.

The genotypic distributions of cod samples tested at both Faroe Plateau and Faroe Bank, on cruises of R.V. *Ernest Holt* in May 1966 and again in March 1967, are given in table 1; all of the five known *Tf* alleles in cod were found in fish from both areas. Estimates of the cod *Tf* allele frequencies are given in table 2, and the final column compares the frequencies on the Plateau and the Bank. Arrows show the order of the five relationships, which remained consistent for both cruises.