effective in the first experiment, and an unusually large number of $S_4S_4$ plants should be obtained in the second experiment. Both of these experiments have been tried, and the results have been negative. It seems, therefore, that the major effect of these associations between slow-growing and fast-growing pollen tubes is upon the compatibility-incompatibility reactions which form the second important feature in the biology of pollen-tube growth.

---

## *A NEW GENE AFFECTING BEHAVIOR AND SKELETON IN THE HOUSE MOUSE*

BY L. C. DUNN

DEPARTMENT OF ZOÖLOGY, COLUMBIA UNIVERSITY

Communicated March 9, 1934

A new recessive mutant character has recently appeared in the house mouse which is unusual in combining effects similar to those associated with two previously known genes. The new mutation is known as shaker-short (symbol $s^t$). It is recognizable at birth by the shortened tail which varies from no bony tail at all (one case) to three-fourths of the length of the normal tail and usually ends in a slender filament which contains no vertebrae and falls off soon after birth. It resembles closely the brachyury mutant described by Dobrovolskaia-Zavadskaia and Kobozieff (1927). Brachyury, however, acts as a dominant and is lethal when homozygous.

Beginning at about five days of age, the new mutant type shows severe disturbances in equilibration. Erratic circus movements, more chaotic than those seen in the waltzing mouse, appear a little later, and it shows an ataxia of the head, accompanied by tremors, more extreme than the somewhat similar condition in the shaker mutations previously described by Dobrovolskaia-Zavadskaia (1928), and by Lord and Gates (1929). As adults the mutants show marked lack of coördination. Most of them show a tendency to circle but seldom consistently in one direction. Generally they appear too weak and erratic for active waltzing. All short-tail mutants which have lived to three weeks of age have developed this extreme choreic behavior. All such have also proved to be completely deaf, this being another point of resemblance to the waltzers and the two different shaker varieties. The fertility of eight adult mutants (four males and four females) has been tested by matings with normal mice. All have been completely sterile.

In addition to these peculiarities many of the mutants have at birth a marked lesion near the median point of the parieto-occipital suture. The

lesion appears either as a bleb filled with blood or lymph which later dries to a small scab, or it may consist of such a scab at birth. This may provide a valuable clue to the central nervous disturbances which appear later.

The new type acts as a simple recessive to normal. Since the homozygotes are sterile, the only data are those from matings of heterozygotes. To date such matings have produced 120 offspring of which 85 were normal and 35 were short-tailed at birth, the monohybrid expectation being 90:30. Only litters from known heterozygotes are included and since the production of one mutant offspring is required to establish the heterozygosity of the parents, it is probable that there have been omitted several litters, consisting of normals only, from heterozygous parents which have not yet produced a mutant. For this reason the segregation ratio cannot be used for judging the prenatal viability of the mutant. 29 litters borne by heterozygotes mated *inter se* have given an average of 4.1 young per litter; 35 litters from the same heterozygotes mated to homozygous normal sibs have given 4.9 young per litter, so there may be a little higher mortality among the mutants before birth.

The new mutation appeared in a stock unrelated to any of the mutant types which it resembles. The stock originated from a pair of hairless mice caught wild in London in 1924 and bred by Mr. H. C. Brooke who sent several specimens to Professor F. A. E. Crew of Edinburgh University in 1925. Professor Crew was kind enough to give me, in 1927, a male and two females, heterozygous for the hairless gene (as well as for piebald, indicating that outcrossing had occurred) and these were inbred for the hairless character, with the exception of one outcross to the Bagg albino stock ten generations before the mutant appeared. Because of the striking nature of the new variant and the antecedent inbreeding we may assume that the new complex of characters arose by mutation within the last few generations.

This is the fourth recorded mutation in mice affecting equilibration and nervous coördination in similar ways. The previous three, viz., waltzer, shaker 1 (Lord and Gates), shaker 2 (Dobrovolskaia-Zavadskaia) have all been shown to be due to mutations at different loci. The new form, judging by its extremely abnormal behavior and the tail defect associated with it, is probably also due to a separate mutation. It is being tested for allelomorphism with the other mutants, but since the homozygotes are sterile these tests will require some time. In spite of its close resemblance to the brachyury mutant, the fact that it is entirely recessive, not lethal and also choreic indicates that it is probably not a recurrence of this mutation.

The new mutation probably affects the development of both the vertebral column and the brain. A similar effect on the axial skeleton, brought about by the dominant brachyury mutation, has been shown by Chesley

(1932) to involve abnormalities in the early mesoderm and neural tube. One step in the determination of the extreme defects in the nervous system of the homozygous form is the failure of the notochord to differentiate (Chesley[1]). Genes which affect such fundamental tissues are of prime importance in attempting to relate the gene to the processes of differentiation, and the new material appears to be especially valuable for this purpose. Since previous attempts to disclose the structural defects responsible for the waltzer and shaker conditions have not been successful, an investigation of the early development of the brain in the new form should be of unusual interest.

[1] Dissertation (unpublished).

Dobrovolskaia-Zavadskaia, N., *Archives Biologie*, **38**, 457–501 (1928).

Dobrovolskaia-Zavadskaia, N., and Kobozieff, N., *C. R. Soc. Biol.*, **97**, 114–118 (1927).

Lord, E. M., and Gates, W. H., *Am. Nat.*, **63**, 435–442 (1929).

Chesley, Paul, *Proc. Soc. Exp. Biol. Med.*, **29**, 437–438 (1932).

---

## *PROJECTIVE DIFFERENTIATION OF SPINORS*

BY J. W. GIVENS, JR.

PRINCETON UNIVERSITY

Communicated March 10, 1934

In the paper by Veblen and Taub on "Projective Differentiation of Spinors" in this volume of these PROCEEDINGS, **20**, 85–92(1934), it is shown that $\gamma_{,\lambda} = \gamma f_\lambda$ where[1]

$$f_\lambda = \frac{1}{2} \text{ Trace } \left( \gamma^{-1} \frac{\partial \gamma}{\partial x^\lambda} \right)$$

is a necessary condition for the reality of $\Gamma^\alpha_{\beta\lambda}$. It is then stated that: "Hence the conditions (2.1), (2.2), (2.3) and (2.4) are self consistent and imply that $A_{\beta\lambda\mu}$ is real and anti-symmetric in $\beta$ and $\mu$ but otherwise arbitrary." Since (2.1) states that $\gamma_{,\lambda} = 0$ this conclusion does not follow without further argument, but fortunately the missing step is not hard to supply.

Writing the fifth equation of paragraph seven in the form

$$K^*_\lambda = \gamma^{-1} K_\lambda \gamma + \gamma^{-1} \frac{\partial \gamma}{\partial x^\lambda} - \frac{1}{2} \text{ Trace } \left( \gamma^{-1} \frac{\partial \gamma}{\partial x^\lambda} \right) .1$$

and taking the trace gives

$$\text{Trace } \left( \gamma^{-1} \frac{\partial \gamma}{\partial x^\lambda} \right) = 0.$$