three phenotypes are illustrated in the accompanying photograph. Heterozygous females have transverse black stripes reminiscent of the tabby markings of cats, and for this reason the gene is named *'tabby'* (symbol *Ta*). Homozygous females and hemizygous males have a number of abnormalities resembling in every way those produced by the autosomal recessive gene *crinkled*[5,6]. Though the different modes of inheritance prove the two genes to be at different loci, a direct test of allelism is being made.

The remarkably exact and detailed reproduction of the same complex syndrome by two different genes is of more general interest than the fact of sex-linkage. It argues forcibly in favour of the 'principle of unity of gene action'[7], and it provides a laboratory parallel to the situation frequently met with in human genetics, where the same clinical condition behaves as if sex-linked in some pedigrees and autosomal in others[1].

The evidence that *tabby* is sex-linked is briefly as follows. The gene arose by spontaneous mutation and was first observed in a single male which had the 'pseudo-crinkled' phenotype. Mated to normal females, this male produced twenty-nine daughters all with the 'tabby' phenotype, and thirty-eight sons, all normal. These tabby females mated to unrelated normal males produced thirteen tabby and nine normal daughters, seven pseudo-crinkled and eleven normal sons. A tabby female mated to the original pseudo-crinkled male produced four pseudo-crinkled and four tabby daughters, two pseudo-crinkled and six normal sons.

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Production of Congenital Defects in the Offspring of Pregnant Mice Treated with Compound *F*

IT has previously been shown[1] that cortisone, injected into pregnant mice, will produce cleft palates and other congenital malformations in the resulting offspring. The incidence of cleft palate was found to vary with the genetic constitution of the treated mice, the dosage of cortisone used, and the gestational stage at which treatment was begun.

In view of the evidence that compound *F* may be the substance produced by the adrenal cortex in response to stimulation by adrenocorticotrophic hormone[2], it seemed desirable to know whether this compound would have teratogenic effects similar to those of cortisone when injected into pregnant mice. Accordingly, pregnant females of stocks previously shown to be susceptible to the teratogenic effects of cortisone[1] were injected at susceptible stages of gestation with compound *F* (17-hydroxycorticosterone acetate, Merck). Cleft palate (without cleft lip) has not been observed in any offspring of untreated females of these stocks.

Five pregnant females, each injected intramuscularly with four daily doses of 2·5 mgm. of compound *F* starting on the tenth or eleventh day, gave birth to a total of 25 offspring. Of these offspring, five were partially eaten before the state of the palate could be determined. Of the twenty whose heads were examined, nineteen (95 per cent) had median, post-alveolar clefts of the palate. Nine of these were born alive. No external abnormalities were seen by macroscopic examination in any offspring of treated females.

It thus appears that compound *F* and cortisone have similar effects on the development of the embryonic mouse palate.

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Luminescent Responses in *Chætopterus* and the Effects of Eserine

ANIMAL luminescence comprises two broad categories, namely, those in which light is produced by the animal, and those in which it is due to symbiotic bacteria; but in either event it may be under the control of the animal. The emission of light would then be a significant indication of the progress of underlying physiological events, and by quantitative recording should provide information about the physiology of luminescent effectors. Considerations of this kind have prompted a study of the physiology of light production in the polychæte *Chætopterus variopedatus*. This is a well-known luminescent worm about 10 cm. long that discharges a luminescent secretion from definite circumscribed glands in certain regions of the body[1]. The glandular cells responsible are elongated eosinophilic elements packed with a paraplasmic mass of secretory granules, and when animals are excited and killed simultaneously by dropping into a fixative, the photogenic cells are caught in the act of secreting, and have the appearance of squeezing forth their contents[2].

Although weak, the luminescent response of *Chætopterus* has been recorded with the use of a photomultiplier cell, galvanometer or oscilloscope, and camera. Preliminary studies have shown that the luminescent response in *Chætopterus* is under nervous control[3]. With electrical stimulation it has been found that a single impulse evokes a bright response, and, furthermore, that the intensity of response can be increased by raising the frequency or increasing the number of impulses. The results obtained have many analogies with neuro-muscular functioning, and can be interpreted as due to the activity of contractile units effecting evacuation of the photogenic glands.

Chemical transmitters have been implicated in the responses not only of muscles, but also of glands and chromatophores, and it seemed of value to investigate this problem in connexion with the luminescent