

es derived from the *au* and ten of the normal Texas stock, namely, the central portion of each visible abdominal sternite and the distal portion of the membrane and mounted membranous wings which is free of veins. On the sternites, the setae were counted in a single area 0.01 mm² in the middle of the sternite, with the aid of a graticule in a 15× ocular in combination with a 10× objective. In the membranous wings the setae within five square millimeters of each wing selected at random, were recorded with a 15× ocular and 44× objective.

In the sternites of the normal beetles the mean setal number in a sample of 50 specimens varied from 9.4 ± 0.2 to 10.7 ± 0.4 setae. In *au* these values varied from 23.7 ± 2.1 to 30.6 ± 1.4 setae, or approximately two to three times as many as in the normal beetles. In the membranous wings the mean number of setae in the sample of 50 measurements in each strain was 10.84 for the normal and 11.10 ± 1.66 for the mutant. The difference in the means is not statistically significant ($P > .4$).

The micrographs obtained with the scanning electron microscope are shown in Fig. 1. At the top, on the left, is a view of the head and the prothorax of the normal beetle, and on the right that of the *au* mutant. Clearly, the number of pits and associated bristles are greatly increased in these two parts of the body. The cervical bristles on the anterior margin of the prothorax are also greatly increased in number.

The micrographs of the abdominal sternites (in the middle of Fig. 1) show to what extent the number of pits and bristles is increased in the mutant. Finally, on the bottom of the figure are two micrographs which contrast the compound eye of the normal and the mutant. In the normal beetle (left) there are only single bristles between the ommatidia, while in the mutant (right) interommatidial bristles are often doubled.

The cytogenetic basis of most mutants other than those from *Drosophila*

by about 33 extra hairs. The increase in hair number was less marked in males.

Cytological examination and genetic data revealed that the increase in hair number resulted from a duplication. The duplication essentially doubled the number of extra hairs on the wing (4). The autosomal recessive "hairy," which increases the numbers of hairs on the wings and other parts of the body, interacted with *Hw* to increase the number of hairs on the wings even further (5).

In *Tribolium* the cytogenetic basis of the aureate mutation has not been investigated because, even with the most powerful compound microscope, the chromosomes are too small to detect chromosomal aberrations such as duplications or deletions. As techniques are developed in conjunction with the scanning electron microscope, it should be possible to examine cytological material and determine whether chromosomal aberrations (such as duplications in *Drosophila*) are responsible for the modification of the phenotype of *Tribolium*.

Be that as it may, our data indicate that the aureate mutation in its effect appears to be unique so far, not only for the genus *Tribolium* but for the order Coleoptera (6). Furthermore, because the scanning electron microscope gives micrographs of high resolving power even at high magnifications, it has been possible to obtain a detailed record of the phenotype of normal and mutant to a degree not previously attainable.

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Chromosome Studies on Marshall Islanders Exposed to Fallout Radiation

Abstract. *Cytogenetic studies of blood lymphocytes of Marshall Islanders, 10 years after their exposure to radiation from fallout in 1954, show chromosome-type aberrations in 23 of 43 exposed persons. Half the aberrations are of the exchange type. An unexpectedly large number of acentric fragments, but no exchange-type aberrations, appear in a few unexposed people on the same island.*

Chromosome aberrations in blood lymphocytes have been demonstrated in several population groups exposed to ionizing radiation, including patients during and after radiotherapy for ankylosing spondylitis (1) or malignant tumors (2), persons exposed during diagnostic procedures (3), and others exposed in the course of their work (4). Similar findings have been reported from individuals involved in radiation accidents (5) and in survivors of the atomic bombings of Hiroshima and Nagasaki in 1945 (6-8). One of the more interesting and possibly more significant points in all these studies was the observation that chromosome aberrations can persist in circulating lymphocytes for many years after the exposure. It seemed of interest to determine whether residual damage of this type also occurs in Marshall Islanders

cation of the method of Moorhead *et al.* (11); they were harvested at 48 and

The total numbers of all types of chromosome aberrations in the two groups

in the low-exposure group was that among the highly exposed. W

incidence of acentric fragments in a control population of Japanese studied by the same authors was 0.11 percent—20 times less than our finding among the eight unexposed Marshall Islanders. In a controlled cytogenetic study of sampled survivors of the atomic bombings of Hiroshima and Nagasaki, Bloom *et al.* have found exchange-type chromosome aberrations in 33 persons—35 percent of 94 survivors examined 20 years after exposure (8); the incidence of aberrations was 0.6 percent in the exposed group—less than half our finding among the Marshall Islanders. Bloom *et al.* found only a single acentric fragment in 8847 cells from the 94 controls, an incidence of 0.01 percent; for the 33 aberration-positive individuals, the dose ranged from 237 to 891 rads, and for none of the

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haploid cells that are capable of vegetative growth.

Recently it has been observed that newly formed zygotes may follow an alternate course of development (1): rather than differentiating into mature zygotes, some may divide mitotically to give rise to diploid cells that remain vegetative. I now describe a selective method for obtaining diploid strains, and some of the characteristics that distinguish them from haploid strains.

My method for recovering diploid strains is based upon the inability of mutant auxotrophic haploid cells to grow under conditions that allow prototrophic cells to proliferate rapidly. These conditions are met by plating a mating mixture of two different auxotrophs on a minimal agar medium lacking the required growth factors.

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