drogenase and the eye color of the adult. In vivo, $ry^2$ is lethal in larvae reared at high temperatures (30°C), but, if reared at lower temperatures, larvae with $ry^2$ survive. Connected with this is our finding that cells isolated from *ma-l* embryos exhibited the same growth rate and optimum temperature as those from embryos of wild-type *D. melanogaster*, while cells from $ry^2$ exhibited a lower optimum temperature (25°C) and grew more slowly, with a mean generation time of 42 hours in the log phase.

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## Mycotoxins: Aflatoxin Isolated from Penicillium puberulum

Abstract. Penicillium puberulum *Bainer was found growing on a sample of moldy peanuts. It also grows on shredded wheat, potatoes, and laboratory culture media such as wort, potato dextrose, and Sabouraud agars, and synthesizes aflatoxin on these substrates. Thin-layer chromatograms of the chloroform-soluble toxin produced by the mold when grown on shredded wheat show fluorescent bands with* $R_F$ *values identical with those of the fractions* $B_1$, $B_2$, $G_1$, *and* $G_2$ *of the toxin produced by* Aspergillus flavus. *This extract produces typical bile duct proliferation type of liver damage in 2- to 3-day-old Peking white ducklings.*

Initially aflatoxin was isolated by extraction from toxic peanut meal contaminated with *Aspergillus flavus* Link ex Fries, as described by Austwick, and identified by the strain number V-3734/10 (*1*), in our nomenclature, M-3. For some time, the toxin was considered to be a unique product of these particular strains of *A. flavus*. Now there is evidence (*2*) that certain other molds produce aflatoxin, such as *A. parasiticus* (Austwick, strain number IMI 15, 957 ii). The *A. parasiticus* is a subculture of the original strain isolated and described by Speare (*2*). We now report that *Penicillium puberulum* Bainer (Hodges, strain number M-56) produces aflatoxins. This strain of *Penicillium* was isolated from a sample of rejected moldy peanuts.

Mold mycelia were found on the inner surface of the cotyledons of many nuts. Samples of the different mycelia were transferred with a sterile needle to wort agar medium in a petri dish, and after 2 to 3 days of incubation at room temperature, mold colonies were transferred to Czapek agar slants. One of the mold cultures was initially recognized as a *Penicillium* species (*3*) and was later identified as *P. puberulum* Bainer (*4*).

This strain of *P. puberulum* has been cultured on sterilized potato plugs, moist shredded wheat, Sabouraud's agar slants, and potato dextrose agar slants. Chloroform extracts of all these cultures exhibited a strong fluorescence when excited by 365 mμ of light. A substrate of shredded wheat was chosen for toxin production after a preliminary evaluation of the relative intensity of fluorescence of the extracts from the several cultures.

The chloroform-soluble extract (M-56) was a light tan amorphous powder, (*5*). Analytical tests were performed on this preparation with ascending thin-layer chromatography on silica gel G-HR (distributed by Brinkmann Instrument Co.). The chromatograms were developed with a mixture of chloroform and methanol (95 : 5) in sealed, lined tanks. The chromatographed M-56 extract exhibited four characteristic blue and green fluorescent spots with $R_F$'s identical to known $B_1$, $B_2$, $G_1$, and $G_2$ aflatoxins. Thin-layer quantitative analysis of the extract, with pure aflatoxins $B_1$ and $G_1$ as standards, indicated that the extract is composed of 15 percent each of aflatoxins $B_1$ and $G_1$ and 1 percent each of aflatoxins $B_2$ and $G_2$ (by weight).

A propylene glycol solution of the M-56 extract, when tested by our qualitative screening test in 2- to 3-day-old Peking white ducklings, produced the typical bile duct proliferation type of liver damage that is characteristic of the aflatoxin effect. The oral acute toxicity of the extract in similar ducklings was measured, ten birds being used for each dose, and may be expressed in milligrams per kilogram with 95 percent confidence limits (*6*) as follows: The 50-percent lethal dose, $LD_{50} = 2.30$ (2.12 to 2.48); slope = 1.14 (0.05 to 1.39).

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## Reduction of Cardiac Stores of Norepinephrine in Experimental Heart Failure

Abstract. *Constriction of the ascending aorta in the guinea pig resulted in ventricular hypertrophy and congestive heart failure. In animals with heart failure which were killed 5 to 40 days after constriction, the norepinephrine stores in both ventricles were strikingly reduced; the extent of reduction was related to the severity of the constriction.*

Marked reductions of the norepinephrine concentration in the atrial appendage have recently been found in some patients with chronic congestive heart failure (*1*). This finding has led to the suggestion that there may be depletion of the norepinephrine stores during heart failure which could interfere with sympathetic nervous transmission. In view of the important positive inotropic effects on the heart of activity of the sympathetic nervous system (*2*), such interference with adrenergic function could possibly further impair cardiac performance in heart failure. To study this problem in a controlled experimental situation, we have produced heart failure in guinea pigs by graded aortic constriction and have examined its effects on the stores of norepinephrine in each ventricle and in the kidney.