Chelone midas and the snakes Thamnophis sir-talis and Natrix sipedon. Papez considered this nucleus to be similar to nucleus Z in the alligator or described by C. C. Wiber and F. C. Corebu nucleus to be similar to nucleus Z in the aligator as described by C. C. Huber and E. C. Crosby [J. Comp. Neurol. 40, 97 (1926)]. The third and fourth cranial nerve nuclei are located below the floor of the cerebral aqueduct

- 10. ventral to the central gray in the caudal mid-brain. The third nerve nucleus is split by the dorsal and ventral division. The sixth cranial nerve nucleus is located just below the central gray at the level of attachment of the fifth crania and fourth cranial nerve nuclei accumulate a much greater amount of HRP than the neurons of the sixth cranial nerve nucleus, as iudged from the intensity of the reaction in the third and fourth nerve nuclei and the relative paucity of
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- We have repeated these experiments with four reptiles (Cordylus cordylus, common cape gir-dled lizard; Gerrhonotus coeruleus coeruleus, San Francisco alligator lizard: Gerrhosaurus val*idus*, Smith's plated rock lizard; and *Eumeces laticeps*, broad-head skink). Orthograde translaticeps, port of HRP to the terminal regions of the retino-fugal axons occurred in all specimens. In addi-tion, a nucleus was identified which was labeled by retrograde transport of HRP following intraocular injection in these animals. The location of the nucleus varies in the four species, bein located, for example, in the diencephalon adja cent to the optic tract in *Cordylus cordylus* and located in the caudal mesencephalic tegmentum
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- critical review of the manuscript. Supported by NIH grant NS12152.

23 July 1976

Aspergillus oryzae (NRRL Strain 1988): A Clarification

El-Hag and Morse (1) chose to ignore two opinions regarding the identity of their aflatoxin-producing "variant of Aspergillus orvzae NRRL 1988," and reported incorrectly the two opinions they acknowledged (2). The culture, as eventually sent to the Food and Drug Administration by Morse and to NRRC by El-Hag, proved to be a strain of Aspergillus parasiticus contaminated with a yeast. The subculture sent to me also was heavily infested with culture mites. This information was made available to El-Hag well in advance of publication.

We had supplied El-Hag with two freeze-dried preparations of A. oryzae NRRL 1988 (lyophilized on two different dates) prior to his first report of aflatoxin production on millet by this strain (3). Immediately following this report, I opened one preparation from each of the two dates and compared the subculture with the stock culture in our collection. All three were identical and were A. oryzae as it is known to me. Subcultures from each source were tested for production of aflatoxin on wheat, corn, millet, and rice. Later, when El-Hag stated that the aflatoxin production on millet was an "artifact" (4) and changed the substrate, the stock culture was also tested on cowpeas and soybeans. No aflatoxin was produced on any of these substrates by any of the three subcultures. El-Hag was informed of these results.

I believe it most unlikely that the culture distributed by El-Hag arose as a variant of NRRL 1988, a strain of A. oryzae that has remained unchanged in our collection through 30 years of maintenance by periodic transfer. A more likely explanation, in my opinion, would be that A. oryzae NRRL 1988 has been replaced by their A. parasiticus through mite infestation. This opinion of its origin was transmitted to El-Hag. [A complete summary of our contacts with their investigations will be sent to any interested person (or persons) on request.]

The variability in aflatoxin production reported by El-Hag and Morse, and attributed by them to instability of the culture, probably reflects varying degrees of contamination of their fermentations. I am not surprised that the culture they returned to me produces aflatoxins B_1 , B_2 , G_1 , and G_2 . Aspergillus parasiticus is known for this ability.

In my opinion, manufacturers of industrial enzymes from A. oryzae and users of their products need not be disturbed. Strain NRRL 1988 and other strains of A. oryzae used in the food and enzyme industries have failed repeatedly to produce aflatoxin when tested at this center on corn, wheat, rice, millet, cowpeas, sovbeans, and a liquid medium. Our results corroborate the extensive Japanese work (5-8) demonstrating that none of hundreds of industrial strains of A. oryzae produce this mycotoxin.

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28 June 1976; revised 13 September 1976

We have read the comments by Fennell and think it is interesting to compare them with a text by the same author (1).

The Aspergilli are characterized by great diversity and variability as they are isolated from nature, and these differences may be interpreted as having emerged by processes of variation and mutation similar to those that occur in the laboratory.

The term variant is applied to strains arising through gradual change from normal members of identifiable species. The characters of a variant are generally not stable but subject to continued change and further variation.

This statement appears to be at variance with the position taken by Fennell.

When we first encountered rat mortality, followed by isolation of aflatoxins, we were anxious to identify the cause. We sent samples of the culture to two collaborators and to the Northern Regional Research Center and the Food and Drug Administration. The two collaborators gave the identification described in our report. Prepublication copies of the report were sent to collaborators and to the Northern Regional Research Center. Collaborators acquiesced to the report as written; but phone calls from Fennell produced the reversal described in Fennell's comment.

All in all it has been an episode that we would like to have closed. In our view the question remains open.

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4 October 1976