chromosome breaking agent was unable to induce chromosomal aberrations in this plant species. We know, however, that many chemicals which damage animal chromosomes, may not induce chromosomal abnormalities in plants as was found in connection with the cytogenetic studies of cyclamates[11–14], and different plant species may react differently to the same chemical as was revealed by a study of RILEY and NEUROTH[15] on the effect of LSD on plant chromosomes.

Zusammenfassung. 7,12-Dimethylbenz(a)anthracen (DMBA) in verschiedenen Konzentrationen anstelle von Auxin, Kinetin und Kokosmilch im Nährmedium induziert die Bildung von Wurzeln und beblätterten Sprossen auf den Kalli von *Haworthia variegata*. Dieses Ergebnis gleicht demjenigen im vollständigen Kontrollmedium ohne DMBA, mit dem Unterschied, dass unter dem Einfluss von DMBA weniger Sprosse und eine grössere Anzahl von Wurzeln gebildet werden. Cytologische Untersuchungen an Kallus- und Wurzelzellen aus dem DMBA-Medium zeigten eine normale Konstitution des Chromosomensatzes ohne chromosomale Aberrationen.

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Induced Morphogenetic Variations in *Aspergillus oryzae* by 8-Azaguanine

One of the most potent modifiers of genetic transcription in metazoan systems is 8-azaguanine, which is known to replace guanine in messenger RNA (m-RNA)[1]. The knowledge that such replacement can be environmentally programmed, led the authors to investigate the impact of 8-azaguanine on diverse strains of *A. oryzae*, cultured in defined nutrient milieu. This communication reports the new[2] morphological variations induced in *A. oryzae* by this aza-base.

Materials and methods. The diverse cultures of *A. oryzae* figuring in this investigation are from KAKKAR's personal stock, and these strains were originally isolated from the soil and rhizosphere microflora of wheat fields of Allahabad and suburbs in 1960–1961. During their entire period of retention under cultural conditions, these strains have retained their rigid structural pattern and general morphology, as described by RAPER and FENNELL[2] without any apparent discernible abnormality.

The basal medium of the present investigation was the modified Czapek's medium containing: Sucrose, 10 g; KH_2PO_4, 1 g; $NaNO_3$ 3.0 g; KCl, 0.5 g; $MgSO_4$, $7H_2O$, 0.5 g; $FeSO_4$, 0.01 g; and pyrex thrice distilled water to make 1 l. 8-azaguanine was added to the basal medium in the concentration of 85 mg/l. The reinforced medium was apportioned in pyrex (white label) 150 ml Erlenmeyer culture flasks, each flask containing 25 ml of the culture solution. The flasks containing the culture solution were subjected to fractional sterilization in Arnold's steamer, by steaming them for 30 min each day for 3 consecutive days. The pH of the culture fluid was 6.5.

The inoculum was prepared from the conidia of diverse strains of *A. oryzae*, which were washed by centrifugation and finally suspended in double distilled water. Inoculation was performed by pipetting 1 ml. of the standardized spore suspensions (approx. 25,000 spores) into the flasks containing the culture solution. After inoculation, the

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[2] K. B. RAPER and D. I. FENNELL, *The Aspergilli* (The Williams and Wilkins Company, Baltimore, Maryland 1965), p. 370.

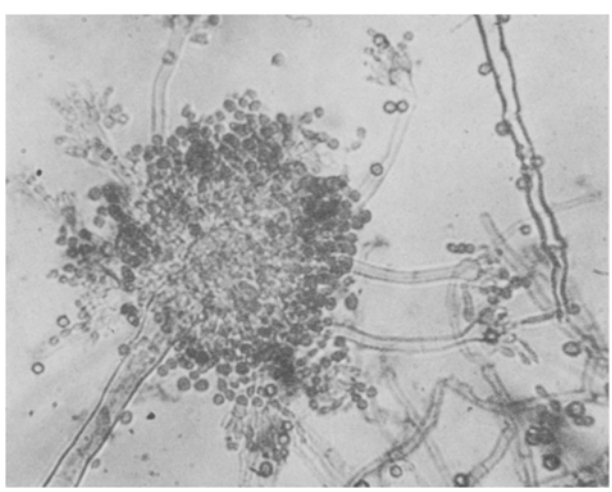

Fig. 1. An abnormal head of *A. oryzae* with 6 secondary septate stalks, terminating into fertile miniature vesicles bearing chains of globose or sub-globose conidia. ×800.

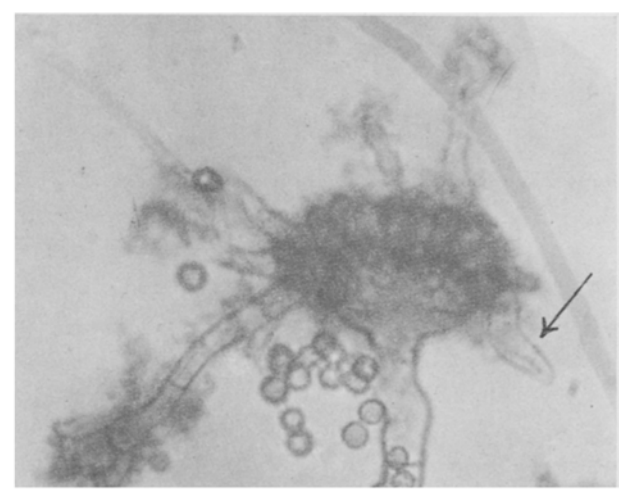

Fig. 2. An abnormal head of *A. oryzae* with secondary stalks in various stages of development. ×800.

culture flasks were incubated at a fixed temperature of 25° ± 1°C for 25 days. The controls, which were devoid of 8-azaguanine, were simultaneously maintained in respect of each isolate.

Results and discussion. The results of a representative experiment are presented in the Table, and the morphogenetic variations of interest are depicted in Figures 1 and 2.

After 15 days of growth on the medium reinforced with 8-azaguanine, the colour of the strains was completely altered from the normal Rainette green (Ridgway Pl. XXXIX) to Sayal brown (Ridgway Pl. XXIX) on the upper side, and Pale Chalcedony yellow (Ridgway Pl. XVII) on the lower side[3].

The other induced morphological variations are enumerated below: Proliferation of the sterigmata into secondary stalks (only in heads bearing biseriate sterigmata); stalks septate, 6 to 14 in number, terminating into miniature fertile heads with uniseriate sterigmata, bearing chains of globose or sub-globose conidia in basipetal succession. Stalks varying in length from 65 to 150 µm, and breadth 5 to 9 µm. Secondary miniature vesicles 5 to 8 µm long and 6 to 8 µm broad; Conidia 5 to 7.5 µm in diameter.

These morphogenetic variations have been repeatedly observed in diverse strains of *A. oryzae*, as we repeated our experiments to confirm the observations. The frequency of the abnormal heads obtained in diverse strains of *A. oryzae* is depicted in the Table. The controls, which were devoid of 8-azaguanine, remained unaffected and no variations were detected in them. The diverse strains produced 52% to 73% abnormal heads under the impact of 8-azaguanine. Similar aberrant development of the phialids was also induced in the present strains of *A. oryzae* by 6-azauracil (80 mg/l) and 2-amino-pyrimidine (110 mg/l). The earlier reports of proliferation in *Aspergilli* are those of THIELKE[4], MILLER and ANDERSON[5] and TURIAN[6].

During repeated transfers from 8-azaguanine-reinforced medium to 8-azaguanine-reinforced medium, the strains persisted in producing a high percentage of abnormal heads, but on transfer to the medium devoid of 8-azaguanine, the strains reverted to normal within 72 h, and the abnormal traits quickly disappeared. This clearly proves that the morphogenetic variations encountered in *A. oryzae* strains were in response to the programmed environmental signal of 8-azaguanine.

In view of the foregoing observations the authors are tempted to hazard the explanation that the synthesis of abnormal m-RNA's, following the incorporation of 8-azaguanine, invariably leads to the production of altered but stable catalytic proteins with novel potentialities, which may be responsible for inducing the recognizable morphogenetic variations in *A. oryzae*[7].

Résumé. Proliférations de l'appareil sporifère d'*Aspergillus oryzae* traité à l'azaguanine.

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Frequency of proliferating biseriate abnormal heads produced after 15 days of growth of *A. oryzae* cultures on complete modified Czapek's medium to which 85 mg/l of 8-azaguanine had been added, as compared to controls to which no 8-azaguanine had been added

Aspergillus oryzae cultures		
No.	Without 8-azaguanine (%)	With 8-azaguanine (%)
136	0.0	63.0
151	0.0	66.0
159	0.0	52.0
168	0.0	73.0
190	0.0	69.0

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[7] The authors are deeply indebted to Dr. O. ISLER of F. Hoffmann-La Roche, Basel (Switzerland) for his generous gift of 8-azaguanine and 6-azauracil. They are also grateful to the University Grants Commission, at New Delhi, which financed this research programme by grant-in-aid No. F-8/5-63 (G), to R.K.K.

DNA Repair Synthesis after X-Irradiation in *E. coli*

Ionizing radiation disrupts DNA, and the killing effects of such radiation on cells are to a large extent, the result of damage to DNA[1]. Although the precise nature of this damage is unknown, X-rays are known to produce discontinuities in the sugar-phosphate backbone of the molecule[2]. The damage is probably not limited to the production of hydrolyzed phosphadiester bonds but likely involves the ribose moiety[3] and the bases as well[4]. Evidence for enzymatic repair of radiation-induced damage to DNA has been found in many systems and has resulted largely from the study of bacterial mutants having altered radiation sensitivity[1], and from measurement of changes in the molecular weight of DNA following irradiation of living cells.

In *E. coli*, the time course of strand rejoining, presumably the final step in the repair process, is similar in UV- and X-ray induced damage, being essentially complete in 1 h[2,5]. The early steps in the latter case are completely unknown. A serious difficulty in the search for early enzymatic steps in X-ray repair is the lack of a well defined lesion in the DNA molecule, such as pyrimidine dimers following UV, of which the reversal or removal could be followed.

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