crassa ad basin truncatam, apice hyalina, 2-Vel 3-furcata.

Synnemata subulate, black, erect, scattered, 1–2.5 mm long, 80–250 μ at the base, 30–60 μ at the apex (figure 1A). Conidiophore threads brown, 3–5 μ thick along most of their lengths, bearing conidiogenous cell at the apex. Conidiogenous cells monotratic, integrated, terminal (figure 1B). Conidia arise as blown out ends from the pores at the apex of the conidiophores (figure 1C) obclavate to fusiform, pale-brown, snooth, 5–15 septate, 70–160 μ long, up to 12–23 μ broad, 4–8 μ thick at the truncate base with a hyaline apex bi or trifurcate (figure 1D).

On dead twigs of *Ficus religiosa* Linn collected from Gaumukh, Mt. Abu, August 1984.

Specimen deposited with C.M.I., Kew.

Herb. IMI 289328 and Botany Department, University of Jodhpur. Coll. No. JUML 955.

The authors are grateful to Dr P. M. Kirk for his help in identification of the fungus. Thanks are also due to Rev Fr K. M. Mathews for the Latin translation. Financial assistance from CSIR, New Delhi is gratefully acknowledged.

21 April 1986; Revised 11 July 1986

EFFECT OF AFLATOXIN B_1 ON MITOTIC INDEX

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AFLATOXIN B_1 (AF-B_1) is one of the most common and widely prevalent mycotoxins. It is a very strong carcinogen, liver being the most preferred target of attack[1]. As the carcinogenic property is a manifestation of the genetic damage and impairment in the mitosis-regulating system, it was considered essential to study the effect of this toxin on mitotic-index.

Two test-systems were selected for this purpose: (i) meristematic cells of onion (*Allium cepa*) root-tip, and (ii) bone-marrow cells of young weaning guinea pigs. For the first system, the bulbs (40–50 g wt) were grown for 48 hr over various concentrations of the AF-B_1 aqueous solution. Thoroughly washed tips were fixed in aceto-alcohol, and aceto-carmine stained squash preparations were made. Total number of cells in a particular field, and among them those undergoing mitosis were counted for calculating the mitotic-index. For the second system, the guinea-pigs were orally administered with aqueous solution of AF-B_1 daily for 16 weeks. At the end of this period, flame-dried giemsa-stained preparations of bone-marrow cells were made. The total number of mitotically dividing cells out of the erythroblasts seen in a certain focus was recorded. Randomness in both the experiments was achieved by standard procedure[2].

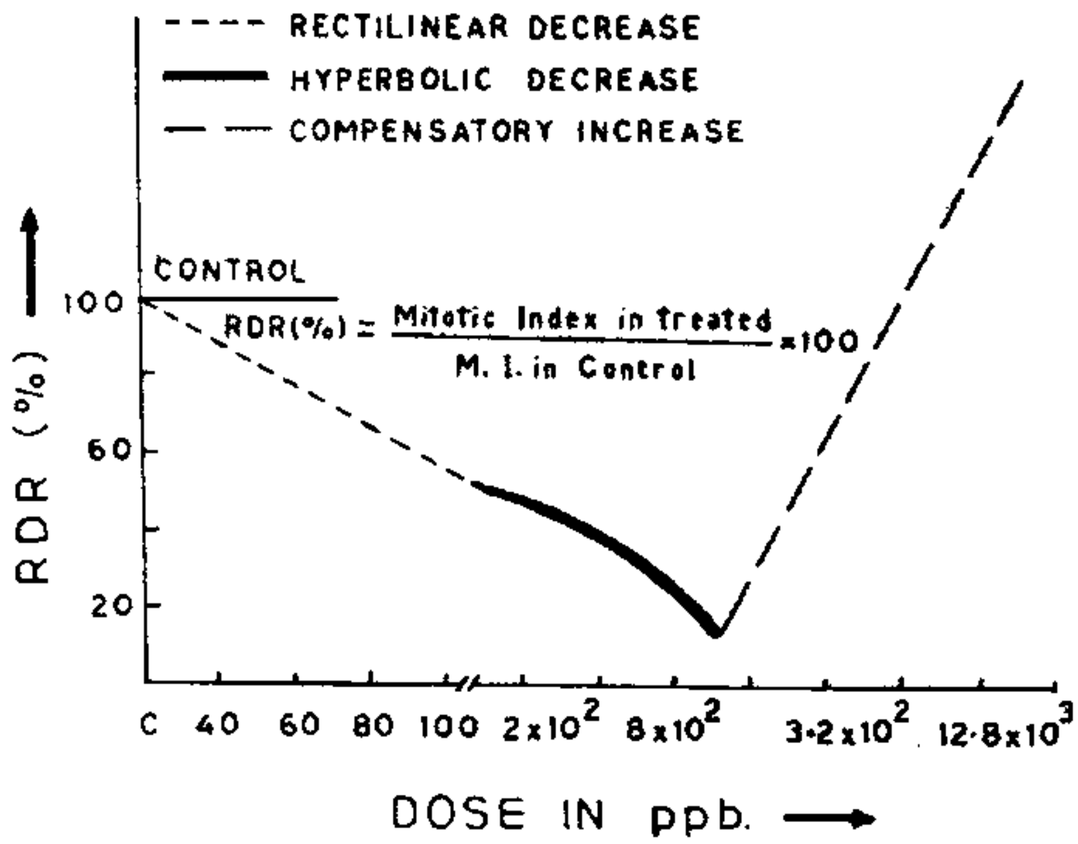

Figure 1. A diagrammatic representation of dose-rate dependence of mitotic-index after aflatoxin treatment.

A series of doses of the AF-B_1, ranging from 10^1 to 12.8×10^3 ppb were used in both the systems. The mitotic-index was expressed in terms of the relative division rate (RDR) which was, in fact, a percentile ratio of mitotic indices in treated organisms with respect to those in the corresponding control variants.

The results show a gradual decline in RDR value in the concentration range of 10^1 to 10^2 ppb, and this decrease was almost rectilinear in nature. In the concentration range of 1×10^2 to 8×10^2 ppb, although a further fall was recorded the nature of the curve was hyperbolic, and a stage was reached when the mitosis almost ceased. However, a sudden increase in RDR was noted at about 10^3 ppb concentration which continued to rise steeply surpassing even the control level (figure 1). It is thus evident that the lower concentrations of AF-B_1 are mito-inhibitory and the higher ones are mito-promotary.

The basic nature of this toxin, as in almost all the xenobiots, is perhaps to disturb the internal mileu of the sensitive cells in such a manner that their biosynthetic pathways get disturbed. Consequently, many cells fail to complete their division cycle and may even get damaged or destroyed[3]. Mitoinhibitory effect noted at lower concentrations is a manifestation of it. As an optimum and physiological viable size is needed for any organ/tissue to work[4], many of the cells that escaped destruction (resistant cells) started dividing more rapidly to compensate the loss[5]. It seems that this defensive mechanism "clicked on" at concentration of 100 ppb. With gradual increase in concentration of AF-B_1, the intensity of compensatory division also increased and hence the nature of the curve was hyperbolic up to 800 ppb concentration. At very high concentrations, this compensatory division

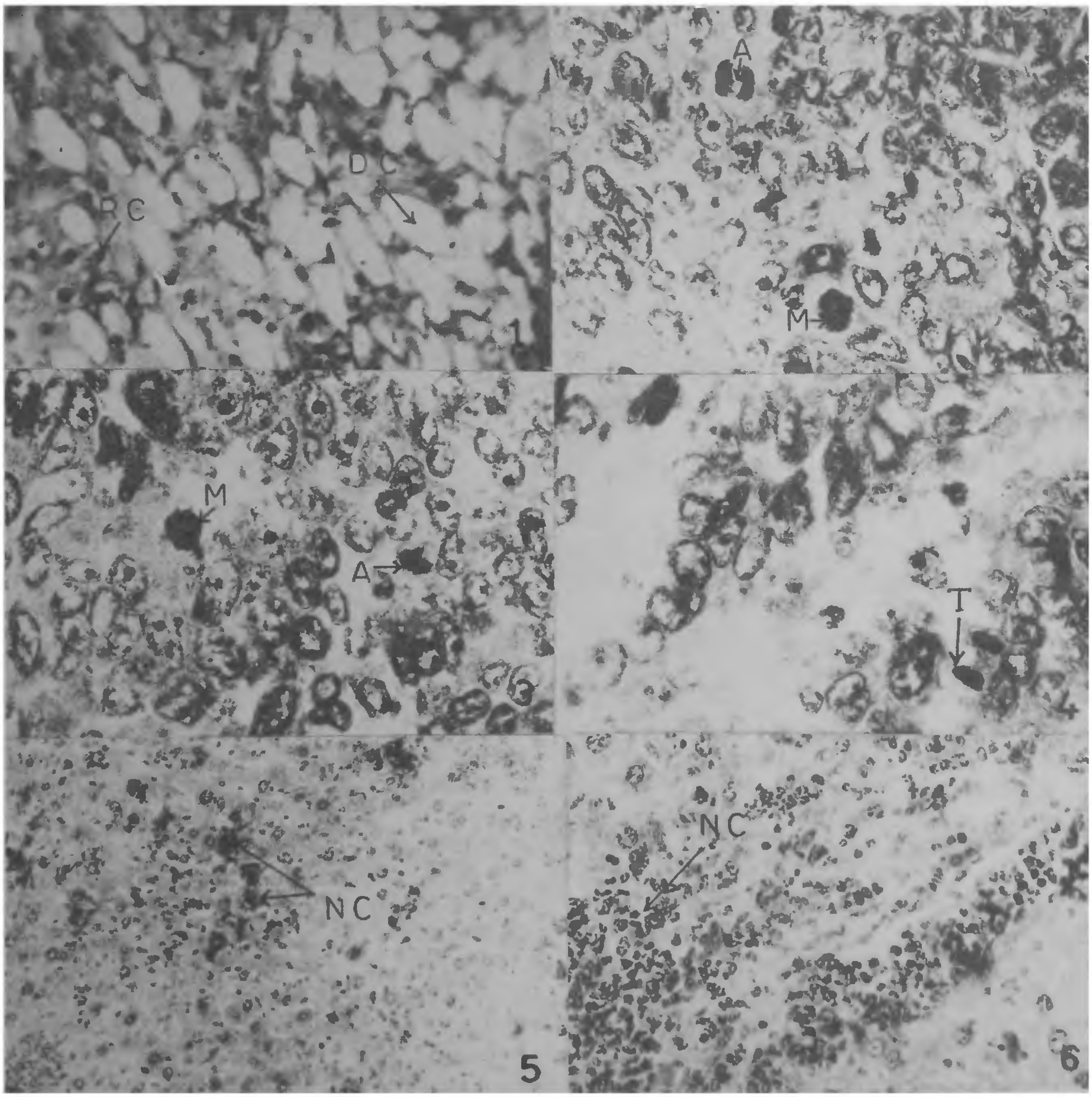

Figure 2. Damages in liver cells and their regeneration resulting in hepatoma. **1.** Degenerating cells (DC), along with resistant cells (RC) (×160); **2–4.** Mitosis in resistant cells (M-metaphase, A-anaphase, T-telophase) (×400); **5.** Dividing neoplastic cells (NC) in comparatively lesser number (×80); **6.** Abundance of neoplastic cells (NC) causing hepatoma (×160).

possibly becomes unbriddled of all the possible control mechanisms resulting in neoplasia. Mutation in mitosis controlling genes also cannot be ruled out. By virtue of such toxin–tissue interaction, the same toxin can act both as mitoinhibitory (at lower doses) and carcinogenic (at higher doses). This is further supported by the fact that in separate experiments we recorded the damage of liver cells in guinea pigs which was followed by appearance of mitotic configurations in the remaining hepatic tissue (neoplastic hepatoblasts, figure 2).

7 May 1986

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ARROWROOT (*MARANTA ARUNDINACEA* L) IS A NEW COLLATERAL HOST FOR KATTE DISEASE OF CARDAMOM (*ELETTARIA CARDAMOMUM* L) MATON

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MOSAIC or marble disease of cardamom (*Elettaria cardamomum* (L) Maton) is a threat for cardamom cultivation. The loss in yield due to the disease depends upon the number of years that the plants have been infected. Naidu[1] reported that the yield reduction may vary from 38–68% in one to two years. In nature, the virus is known to be transmitted through an aphid vector (*Pentalonia nigronervosa* f. *caladii* Vander Goot)[2]. Besides cardamom, the virus has been found to infect the other wild host plants viz *Amomum cannecarpam* and *A. involucratum*[3]; *A. microstephanum*[4], *Alpinia nutan*[5] and *Curcuma neilgherrensis*[6] which might serve as a source of inoculum in the plantations.

During the survey of katte disease of cardamom around Sringeri, the authors observed about 2–3% arrowroot (*Maranta arundinacea*) plants showing typical symptoms of katte virus of cardamom. On the infected plants the chlorotic flecks noticed on the leaves measured 2–8 mm in length and 2–3 mm in diameter. The mosaic symptoms were pronounced on the youngest leaves. Further, mosaic and mottling symptoms were observed on the leaf sheath and young pseudostem.

An attempt has been made to prove the pathogenicity by transmitting the virus reciprocally between arrowroot and cardamom. The aphid vector *P. nigronervosa* f. *caladii* was used for transmission of the virus. The pure virus free aphid culture was maintained on healthy cardamom plants. Such aphids were allowed to feed on inoculated arrowroot leaf for 24 hr. The viruliferous aphids were transmitted to 50 days old healthy cardamom seedlings and were allowed to feed for 24 hr. Observations were recorded at regular interval for the expression of early symptoms on young leaves. The experiment was repeated six times to confirm the transmissibility of the virus and the percentage transmission was recorded up to 100%. Back transmission of the virus from cardamom to healthy arrowroot plants was also successful.

Based on the similarity in symptoms and transmissibility through the aphid vector (*P. nigronervosa* f. *caladii*) the virus has been found to be identical to katte disease of cardamom (*E. cardamom*). Hence arrowroot (*M. arundinacea*) is a new record of a collateral host to katte virus in nature and serves as a source of inoculum for spread of katte virus of cardamom in an established plantation.

26 May 1986; Revised 27 June 1986

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