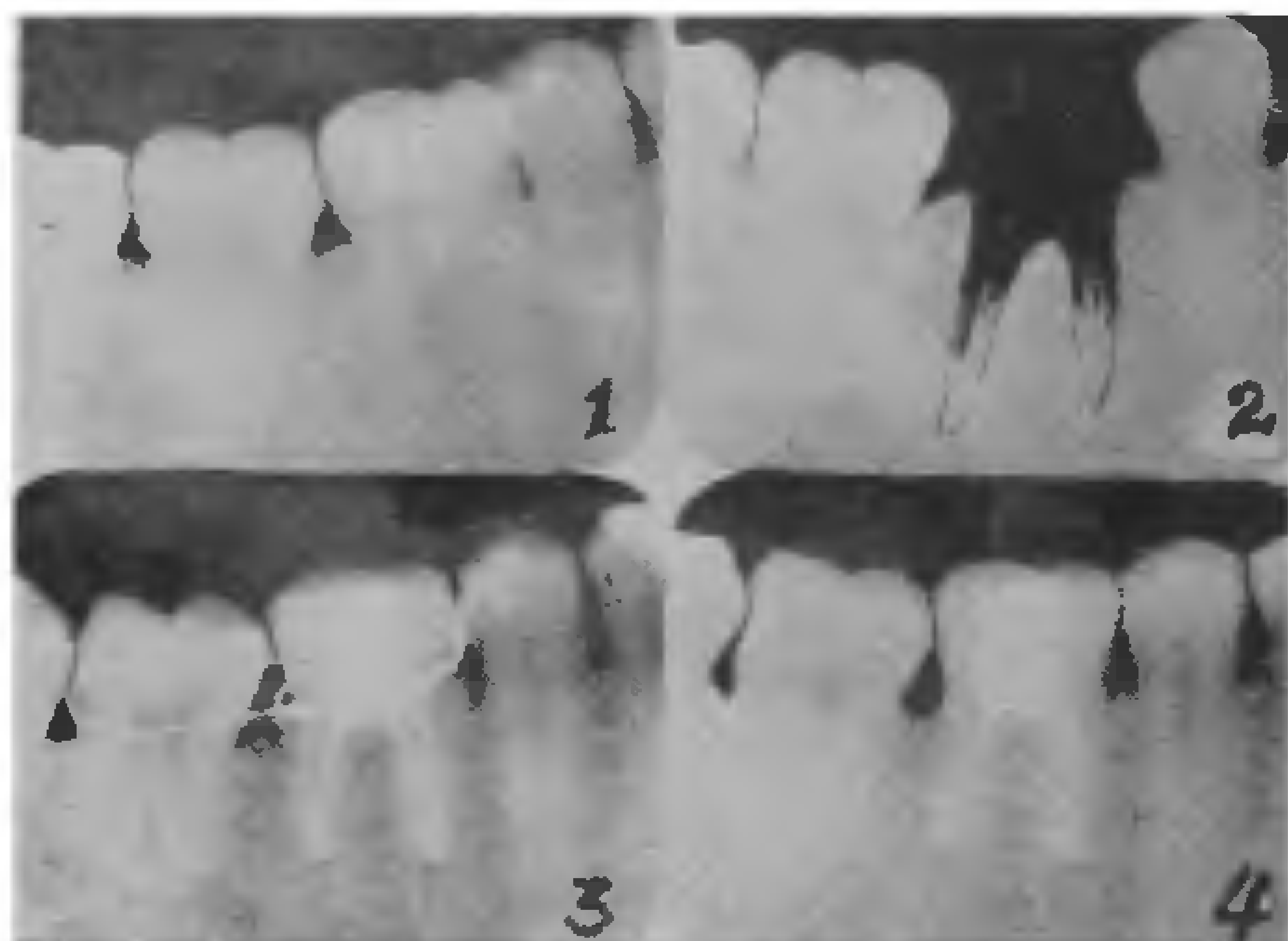
reference to the adult molars and to check the viability of the periodontal ligaments when excised, though a few attempts have been made in relation to the replantation of permanent anterior teeth[1-5].

Root canals of carefully extracted molars of ten adult humans were treated *in vitro* (Figs. 1 and 2) with ready-made gutta percha points and zinc oxide-eugenol cement. Zinc-free silver amalgam fillings were provided at the apical region, following the removal of 2–3 mm of the apical portion of the roots. Crowns of the teeth were restored by permanent fillings and the occlusal surfaces of the teeth were ground to prevent contact with the opposing teeth. Effective reinsertion in the socket was made with wiring with neighbouring teeth (Figs. 3 and 4), for a period of 6–8 weeks.



FIGS. 1–4. Successive stages of replantation procedure of human molar.

Though the exact biological processes involved in the successful replantation of intentionally or accidentally displaced teeth are not known, the procedure is gaining importance owing to its aesthetic value especially in teeth with calcified root canals where radiographic evidence of apical pathology is not exhibited. Extensive root resorption has been reported[6] in the replantation studies of the anterior teeth of children where the ligaments and the supporting tissue were suggested to be in embryonic stage. Present study confirms the periodontal membrane and the exposed adult teeth portions being viable at least for a period of 20–30 min., as in, 90% of the analysed samples no resorption occurred for a period of 4–5 years. In spite of the indication of the post-operative regeneration of the periodontal ligament being similar to the periosteum of the bone grafts[6], the successful replantation of the adult molars strongly favours experimental furtherance in terms of physiological stability or loss, and histology of regenerating supportive tissue, over extended periods.

Thanks are due to Prof. B. P. Rajan, Professor of Operative Dentistry, Dental Wing, Madras Medical College, for his interest in the work.

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1. Grossman, L. I. and Chacker, F., *Trans. Fourth Internat. Conf. Endodont.*, 1968, 127.
2. Deeb, E., *Ibid.*, 1968, 147.
3. Flaragen, V. D. and Myers, H. I., *Oral Surg. Med. Path.*, 1958, 11, 1179.
4. Rajan, B. P., *Internat. Coll. Dent. News Letter*, 1973, 10.
5. Parameswaran, A. and Ramesh, R., *Dental Dialogue*, 1975, 2 (3), 25.
6. Monsour, F. N. T., *Aust. Dent. J.*, 1970, 15, 361.

IDENTITY OF THE PATHOGEN CAUSING ERGOT OF PEARL MILLET IN INDIA

ERGOT contamination of pearl millet (*Pennisetum typhoideum*) is a serious problem in India, since consumption of ergoty pearl millet leads to disease in humans characterized by nausea, vomiting, giddiness and prolonged sleepiness[1].

The pathogen causing ergot of pearl millet in India was identified by Shinde and Bhide[2] as *Claviceps microcephala*. Loveless[3] designated the pathogen causing ergot of pearl millet in Rhodesia as *Claviceps fusiformis*. An attempt was, therefore, made to establish the correct identity of the pathogen of pearl millet in India based on morphological and chemotaxonomic criteria.

A number of samples of ergot of pearl millet was collected from various parts of India and examined. The sclerotia were obpyriform or irregularly shaped up to 0·7 mm long and up to 0·4 mm wide, generally slightly curved with maximum width at the base and usually narrowing towards the tip. The conidia were hyaline, fusiform to broadly falcate, and 28·0–40·0 μ wide. Comparison of the Indian material with the type material of *Claviceps fusiformis* established that the two were identical.

A study of the alkaloids in the honeydew stage, after extraction and quantitation by the method described earlier[4] for sclerotial stage, showed that 100 gm of the material contained about 5 mg of the alkaloid.. The alkaloid consisted of agroclavine, elymoclavine, chanoclavine, setoclavine and penniclavine. The alkaloid profile was found to be similar to that seen in the sclerotial stage. However, quantitatively in the sclerotial stage the alkaloid content was found to be ten fold higher than that in the honeydew stage. The pathogen which causes ergot of pearl millet produces

the clavine group of alkaloids both in sclerotial as well as the honeydew stages, whereas, the pathogen *C. purpurea* responsible for ergot of rye produces the ergotoxine-ergotamine group of alkaloids. Thus based on morphological and chemotaxonomic characters it is clear the pathogen that leads to ergot of pearl millet in India is *Claviceps fusiformis* Loveless.

Grateful thanks are due to Dr. S. G. Srikantia and Dr. P. G. Tulpule of National Institute of Nutrition, Hyderabad, for helpful criticisms. Thanks are due to the Director, Commonwealth Mycological Institute, London, for lending the type material (IMI-121140).

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1. Krishnamachari, K. A. V. R. and Bhat, R. V. *Ind. J. Med. Res.*, 1976, 64, 1624.
2. Shinde, P. A. and Bhide, V. P., *Curr. Sci.*, 1958, 27, 499.
3. Loveless, A. R., *Trans. Brit. Mycol. Soc.*, 1967, 50, 15.
4. Bhat, R. V., Roy, D. N. and Tulpule, P. G., *Toxicol. Appl. Pharmacol.*, 1976, 36, 11.

CHLORAMPHENICOL RESISTANCE IN GENUS *BACILLUS*

CHLORAMPHENICOL resistant bacteria such as *Escherichia coli*[1, 2, 3]; *Pseudomonas aeruginosa*[1]; *Pseudomonas fluorescens*[4]; *Proteus mirabilis*[1] and *Staphylococcus aureus*[5,6] have been reported and the mechanism of the resistance has been studied. S. Osawa isolated a number of chloramphenicol resistant mutants from *Bacillus subtilis ATCC* 6633 which are resistant to 5 μg of chloraamphenicol[7]. However, there has been no report on naturally occurring strain of *Bacillus* which is resistant to chloramphenicol. The author has isolated several chloramphenicol-resistant strains of *Bacillus* from soil. One of the isolates, showing similar bacteriological properties as *Bacillus megaterium*, was used throughout the experiment (Strain 7). It was gram positive spore forming bacilli, giving typical large, round, opaque whitish colony on nutrient agar, produces acid only from mannitol, sucrose, glucose, arabinose and xylose. Lactose was variable. Isolates were identified following the scheme of *Bergey's Manual of Determinative Bacteriology*, 7th Ed. and *A Guide Book to the Identification of Genera of Bacteria* (V.B.D. Skerman, 1967). *Bacillus megaterium* strain 7, could grow in the presence of 10 μg/ml and was trained to grow by subculture in increasing concentrations of chloamphenicol (50 μg/ml chloramphenicol). *Bacillus megaterium KM* and *Bacillus cereus* T, used as standard, could not grow at the concentration of 10 μg/ml. In order to measure chloraamphenicol activity, the cells of chloramphenicol-sensitive and resistant strains of *Bacillus megaterium* were incubated in the nutrient broth containing 250 and 500 μg/ml of chloramphenicol for 0, 8 and 20 h at 37° C with shaking. [Nutrient broth consisted of meat extract (Kyokuto Seiyaku Co., Tokyo) 10·0 g.; polypeptone (Daigo Eiyo Kahaku Co., Osaka) 10·0 g.; sodium chloride 5·0 g; deionized water 1000 ml pH 7·3]. The amount of chloramphenicol in the medium was bio-assayed using the cup-plate method. Only a weak inactivation was observed when the resistant strain was used. To increase the sensitivity of the test, the cells of either the resistant strain (*Bacillus megaterium* strain 7) or the sensitive strain (*Bacillus megaterium KM* obtained from the collection of this laboratory) was incubated with ^{14}C– chloraamphenicol in nutrient broth and the degradation products of ^{14}C-chloramphenicol in the supernatant were analyzed by chromatography and autoradiography. Degradation products of ^{14}C-chloramphenicol were detected only when the resistant strain was used (uninduced condition). The amounts of degradation products were markedly increased when the cells of the resistant strain, which were pregrown in the presence of 10 μg/ml of chloramphenicol, were incubated for 20 h with ^{14}C-chloramphenicol (induced condition). No degradation products, however, were detected when the cells of *Bacillus megaterium KM* were incubated with ^{14}C-chloramphenicol under similar uninduced or induced conditions. Degradation of ^{14}C-chloramphenicol was also demonstrated when the cell-free extract of *Bacillus megaterium* strain 7 was incubated with ^{14}C chloramphenicol, acetyl-CoA and Mg^{++} ion, while no degradation was observed with the cell-free extract of *Bacillus megaterium KM*. Thus, chloramphenicol decomposing enzyme in *Bacillus megaterium* strain 7 may partly be responsible for the chloramphenicol resistance of this organism. Further work on the study of mechanism of the resistance is in progress.

Author wishes to express his sincere thanks to Dr. K. Izaki, Tohoku University, Japan, for his kind guidance and to the Japanese National Commission for UNESCO, ICRO and also to the Japanese Government for awarding the fellowship and providing with necessary facilities for pursuing the research work.

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