virus described in 1953 by Stanley, Dorman and Ponsford[4,5] in Australia. Oily coats and yellow foci in the liver were characteristic features. The neurotropic properties of their virus were brought out by serial passage. The cerebral behaviour of our virus has not been fully studied. It seems probable that the Australian, British and American viruses are interrelated and are widely distributed in mouse colonies as latent invaders. Direct comparison of the three agents is clearly indicated.

JOHN B. NELSON

The Rockefeller Institute,
New York 21.

GEORGE S. TARNOWSKI

The Sloan-Kettering Institute
for Cancer Research,
Walker Laboratory,
Rye, New York.

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Griseofulvin and Colchicine: Lack of Carcinogenic Action

DURING the investigation of the toxicology of griseofulvin, it was shown[1] that the antibiotic when given parenterally is a mitotic poison, in some respects similar to colchicine. Clinical investigation[2,3] showed that this did not represent an acute toxic hazard in clinical use. Nevertheless, interference with mitosis seems to us to imply a greater possibility of carcinogenic action on long-term administration than is the case with compounds devoid of this property. Colchicine has not yet been studied adequately from this point of view, and it was decided to include a group treated with this drug in a study of the long-term effects of griseofulvin.

Four groups of albino, Wistar rats were used. All were newly weaned at the commencement of the experiment. Group I received colchicine, 0·5 mgm./kgm., twice weekly for 42 weeks. The colchicine was initially given subcutaneously in saline, but because of local necrosis the vehicle was changed to arachis oil after four weeks. Group II received saline and arachis oil as controls to Group I. Group III received a finely ball-milled suspension of griseofulvin as a 10 per cent w/v dispersion in 0·5 per cent aqueous mixture of 'Dispersol *LN*' and 'Dispersol *OG*', at a level of 200 mgm./kgm., twice weekly by intraperitoneal injection for 93 weeks. Group IV received the suspending agents alone as a control to Group III. Each group comprised 10 male and 10 female animals.

Colchicine proved toxic in the initial stages, but griseofulvin was well tolerated until the seventy-fifth week of the experiment when mortality began to mount. At the end of 52 weeks dosing, eight animals treated with colchicine, but only two treated with griseofulvin, had died. At the end of 78 weeks experiment, nine animals of each group treated with the drugs had died. Five animals given colchicine and seven given griseofulvin survived until the experiment was terminated at the end of the ninety-third week.

All animals were examined after death for the presence of tumours and, unless postmortem autolysis was advanced, blocks of all the major organs were fixed for histological examination. The following tumours were encountered: Group I: colchicine treatment; one fibroadenoma of mammary gland; Group II: control to colchicine, four adenomata of thyroid; one leiomyosarcoma of the vagina and one sarcoma at injection site; Group III: griseofulvin treatment, one adenocarcinoma of cervix uteri; Group IV: control to griseofulvin, one interstitial cell tumour of the testis.

In the animals treated with either griseofulvin or colchicine, the nuclei of the liver and stomach mucosa of some appeared rather more variable in size than is normally the case. Other pathological changes were found equally in control and treated groups.

Although the numbers in these experiments are relatively small, it is clear that neither griseofulvin nor colchicine is a potent carcinogen, and indeed these experiments give no evidence of carcinogenic action of any sort of either compound.

G. E. PAGET
SHIRLEY J. ALCOCK

Imperial Chemical Industries, Ltd.,
Pharmaceuticals Division,
Alderley Park, Macclesfield,
Cheshire.

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The Hedgehog as a Source of Human Ringworm

THE dermatophyte *Trichophyton mentagrophytes* has been isolated from a variety of animals, but although Georg[1] lists 18 wild and domestic mammals living in different geographical areas which are known to be spontaneous carriers of this pathogen, the hedgehog does not appear to have been included in any survey.

During March 1960 an adult female patient attended the mycological diagnostic clinic attached to this Department, complaining of ringworm, which she stated to have been contracted from a pet hedgehog. On examination, several raised itchy lesions were found, from which a pathogenic fungus was isolated. This was readily assigned to the genus *Trichophyton*, but less readily placed in a definite species. During the same month a similar isolate was obtained from a lesion on the wrist of an adult male, who had recently returned from a country holiday.

A series of 26 living and dead hedgehogs, collected in this area, were examined for the presence of a pathogenic fungus. Isolates of *Trichophyton*, similar in macroscopic and microscopic characters, were recovered from 7 (27 per cent) of the animals. Positive cultures were obtained from the quills of 4 and from the hair on the ventral body wall, the head or the ears of a further three. A hedgehog isolate (*H*1) was sent to Dr. Lucille Georg, Communicable Diseases Centre, Georgia, United States, who kindly identified it as *Trichophyton mentagrophytes*.

Mycelial fragments taken from the *H*1 strain were applied to an area of scarified skin on a guinea pig