delay and the same abnormal phyllotaxy. However, its embryonic structures made no progress in the two years in spite of the apical position on the shoot system. As it was necessary to remove all the large expanding buds, this GA_3 formation might be compared with the small basal 'sleeping' buds on every shoot. The latter were, however, much better prepared, as they produced regularly decussate leaves if the shoot portion about them was cut off early in spring. These effects were even more accentuated when 1 per cent GA_3 lanoline paste was applied below the shoot tips on May 7, 1963. The leaves remained healthy until the end of October, and the length of the shoots was almost the same as usual; their axis was, however, 10–15 mm in diameter (for example, the lowest internode 9 mm, the highest one 15 mm in diameter) and all the buds were only 1·5–3 mm long. The xylem width below the apical node was from two to three times larger than in the corresponding control shoots with axis diameter of 5·5 mm at the top and with floral buds 10–15 mm long.

It seems probable that GA_3 lowers the correlative inhibition of the leaves, yet this particular activity of this growth-substance appears to be noxious at the time when this inhibition is necessary for the formation of the bud scales. There is only a certain number of well-developed bud scales which enable the initiation of leaves and flowers, respectively, inside the winter buds, as has been experimentally established[6]. Further, the lack of leaf or flower primordia in the buds results in a conspicuous thickening of the shoot axis, especially at its upper region, which is also promoted by the activity of the root system. Of course, the effect of the same GA_3 lanoline paste was the opposite if applied to the lilac buds when they were beginning to expand, and afterwards they showed the usual effect of this so-called 'shooting hormone'[7].

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MICROBIOLOGY

A Natural Reservoir of Penicillin-resistant Strains of *Staphylococcus aureus*

The story of the increasing incidence of antibiotic-resistant *Staphylococci* in such man-made selective environments as the hospital ward has been well documented. Similar processes are infrequently encountered in the soil or in other natural situations, and it is rare that antibiotic production can be shown to have ecological significance in a natural environment[1]. In the course of investigations into the microbial flora of the hedgehog, a naturally occurring biocenose which appears to be selective for penicillin-resistant *Staphylococci*, has been uncovered.

Hedgehogs in New Zealand are heavily infected with the dermatophyte *Trichophyton mentagrophytes* var. *erinacei*, strains of which have been recovered from 44·7 per cent of 114 animals[2]. The infection is found in animals of all ages and involves large areas of skin.

Nasal and skin swabs from 35 animals of the same series have been inoculated on to media selective for *Staphylococci*. Coagulase positive *Staphylococci* were isolated from the nostrils of 40 per cent, and the ventral skin surface and paws of 63 per cent and 71 per cent, respectively. It was noticeable that areas of skin carrying the dermatophyte yielded large numbers of coagulase-positive *Staphylococci* in almost pure culture. This colonization of mycotic lesions by pathogenic *Staphylococci* is well known and is frequently observed to occur in tinea pedis. The isolates were phage-typed and their antibiotic sensitivities were investigated.

Approximately 90 per cent of the isolates could be typed using human staphylophages although not more than 50 per cent could be classified into the human groups. Although a wide variety of phage-patterns was recorded, it was found that 79 per cent of nasal strains and 92 per cent of skin isolates were resistant to penicillin, but were sensitive to streptomycin and to the tetracyclines. Penicillin resistance was not associated with any particular phage pattern but was found equally distributed through the staphylococcal types.

Dermatophytes are known to produce antibiotics and penicillin has been isolated from *T. mentagrophytes*[3]. The hedgehog variant of this species *in vitro* can be shown to produce considerable quantities of a penicillin-like substance, inhibitory to sensitive *Staphylococci* and neutralized by penicillinase. The type strain of the variant *IMI.* 101.051 and 3 other isolates were grown in Sabouraud broth shake cultures for 5 days. The filtrates of all produced zones of inhibition equivalent to 1·5 units/ml. of penicillin in plates seeded with the Oxford strain of *Staphylococcus aureus*. Addition of penicillinase to the filtrates destroyed their inhibitory powers.

It appears probable that the chronic mycotic infection of hedgehog skin provides an environment in which penicillin-resistant *Staphylococci* have a selective advantage over sensitive strains and that the hedgehog surface is one in which the natural production of an antibiotic is affecting the structure of the biocenose. The ecological relationships of the microbial community living on hedgehog skin are being further investigated.

We thank Mrs. Jean Markham for phage-typing the isolates.

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VETERINARY SCIENCE

Use of Lactic Dehydrogenase for Detection of Avian Leukosis

In the United States, the loss of birds from the avian leukosis complex is of the order of 1,000,000 dollars per week[1]. The loss to the poultry industry in the United Kingdom is estimated to be about £7,500,000 annually[2]. Investigation and control of the disease are impeded by lack of a rapid, dependable diagnostic test. The resistance-inducing factor (RIF) technique[3] offers a valuable research tool but is too time-consuming and complicated for practical flock testing.

A simple, accurate test for the presence of leukosis and, in particular, for virus-shedding chickens, would be of inestimable value. Leukosis-free birds could be identified for use as foundation stock and elimination of the leukosis virus from egg embryo and chick embryo cell vaccines would be greatly simplified.

Previous investigators have reported increased plasma lactic dehydrogenase (PLDH)-levels in animals with tumour growth[4,5]. Variation in PLDH-levels due to breed and age of chickens has been reported[6], but the presence or absence of clinical leukosis was not noted. This communication reports our observations on increased PLDH-levels from chickens with clinical leukosis.

PLDH determinations were made according to the method of Berger and Broida, using reagents obtained from Sigma Chemical Co.[7]. Plasma samples were obtained using 0·1 ml. of a 50 mg/ml. solution of sterile heparin