Table 1. GROWTH OF RICE EMBRYO (VAR. BHASAMANIK) KEPT UNDER WATER AND IN AIR, 144 HR. OLD AT 25° C. IN DARKNESS

	Under water			In air		
	Mesocotyl (mm.)	Coleoptile (mm.)	Root (mm.)	Mesocotyl (mm.)	Coleoptile (mm.)	Root (mm.)
Embryo with full endosperm	nil	34·5	0·1	45·8	7·0	58·4
Embryo with ¾ endosperm	nil	35·0	0·4	43·8	7·2	70·2
Embryo with ½ endosperm	nil	51·3	1·5	41·3	7·4	61·3
Embryo with ¼ endosperm	nil	18·2	nil	20·0	6·0	37·1

It is evident from Table 1 that the coleoptile of submerged embryos and the root growth of air-grown embryos have been accelerated by the elimination of a portion of endosperm. When more than half the endosperm was removed, no such effect of acceleration was observed. Under water, the growth of root and second leaf is almost completely suppressed, the hollow coleoptile elongates by its tapering end and it never breaks open. In complete darkness the germination of rice seed under water is significantly inferior to that in light (even so little as one or two minutes exposure in every twenty-four hours). In darkness, suppression of the mesocotyl growth of the submerged embryo is another interesting contrast to the much elongated mesocotyl of air-grown embryos.

Germination of oat seedlings under constant illumination results in the suppression of growth of the mesocotyl. The reasons for this are not well understood. Van Overbeek[3] has stated: "The growth of the mesocotyl is quite dependent on the amount of auxin left over by the coleoptile. If this amount is large, a large growth will result; if the amount is small, then little mesocotyl growth will result". Galston and Baker[4] have shown that the suppression of growth of pea epicotyl is due to the deactivation of auxin by light in the presence of riboflavin. Yamada[1] has suggested that, under water, deactivation of auxin is less and hence the length of the coleoptile is greater; but there is no reason for the suppression of the rice mesocotyl, which should not occur if the deactivation of auxin is decreased. Further, growth of the coleoptile is found to be still higher when half the endosperm is eliminated and the coleoptile presumably supplied with less auxin either in the active state or in the form of a precursor. The reasons for the acceleration of growth by elimination of endosperm fractions are not clear.

It may be presumed that the amount of auxin or some unknown factor present in the rice endosperm is more than optimal for the early growth of the embryo; hence elimination of a fraction of endosperm makes the level of auxin or the unknown factor optimal for embryo growth, until such elimination reaches a limiting value. Compared with the control (embryo with full endosperm and without added auxin), it has also been found that, on addition of indolylacetic acid to the growing embryo with three-quarters endosperm, acceleration effects on root growth were not observed. Coleoptile tip also plays an important part in the production of auxin in the embryo, for Das[5] has shown that excised embryo of rye can produce its own auxin without the intervention of endosperm. It appears that the whole problem is complex and more work is required on the nature of transport of auxin from the endosperm of the embryo, the quantity produced by the coleoptile itself and lastly how the growth of different parts of the embryo are regulated under different conditions.

Further work is in progress and the results will be published shortly. We thank Dr. I. Banerjee, head of the Department of Botany, University of Calcutta, for facilities for this investigation.

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A Mosaic Disease of Leguminous Plants caused by a Strain of Tobacco Mosaic Virus

WHILE collecting viruses for use in investigating the inhibition of virus infectivity by extracts of *Theobroma cacao* L., one was found causing a systemic mosaic disease in cow-pea (*Vigna unguiculata* (L.) Walp.) and Bengal bean (*Mucuna aterrima* Holland). The behaviour and properties of the virus suggest that it is related to tobacco mosaic virus, although we have been unable to find any previous report of strains of this virus infecting leguminous plants systemically.

The leaves of infected cow-pea and Bengal bean show light and dark green mottling which varies in severity, and they are somewhat distorted. The disease is now prevalent in these two crops at Moor Plantation; but there is no reason to think that it is a recent introduction. Mosaic symptoms have been recorded commonly in cow-pea and Bengal bean since 1941, not only at Moor Plantation but also at several farms of the Department of Agriculture in the Western and Northern Regions of Nigeria.

The virus is easily transmitted by inoculating sap from diseased plants to cow-pea, Bengal bean, *Nicotiana tabacum* L., *Nicotiana glutinosa* L. and several varieties of French bean (*Phaseolus vulgaris* L.). The only symptoms in *N. glutinosa* are necrotic local lesions; in *N. tabacum* var. Virginia Hybrid, chlorotic primary lesions are followed by a systemic mosaic and distortion of the leaves. The virus from cow-pea causes a systemic mosaic on the fourteen varieties of French bean tested, in contrast to our stock strain of tobacco mosaic virus, which produces discrete local lesions on some varieties but no systemic symptoms in any.

Colourless preparations of the cow-pea mosaic virus were readily made from the sap of infected tobacco plants by repeated precipitation with ammonium sulphate, and solutions of the purified virus showed the phenomenon of anisotropy of flow strongly. Mr. F. C. Bawden, of Rothamsted Experimental Station, found that a purified preparation was precipitated specifically with an antiserum prepared against his stock strain of tobacco mosaic virus, and with the electron microscope Mr. H. L. Nixon found that it contained rod-shaped particles of varying lengths, indistinguishable from the particles of strains of tobacco mosaic virus he had previously examined. In the Rothamsted glasshouses in December, the

virus produced a systemic mosaic disease in *P. vulgaris* var. Prince.

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Normal Mouth and Intestinal Flora of the Ferret (*Mustela furo* L.)

Although the ferret is widely used as an experimental animal, particularly in the study of virus diseases, very little work has been done on the commensal bacteria of the animal itself. Some work on its pathological conditions has been published by Hughes[1], Momberg-Jorgensen[2], Symmers and Thomson[3], Symmers, Thomson and Iland[4], and Skulski and Symmers[5]; but none has been found concerning the bacterial flora which can commonly be isolated from the mouth and intestine of the healthy ferret. The present investigation was made to study this flora, in a colony of tuberculosis-free ferrets, maintained in first-class conditions, which has its own unit of breeding gills, and is augmented at intervals by fresh stock purchased from breeders in the surrounding area.

One hundred and eighteen ferrets were examined as soon as possible after joining the colony. Swabs were taken from the mouth and throat and from the rectum. These were cultured on horse-blood agar and McConkey's medium, and incubated aerobically and anaerobically at 37° C. for a minimum period of 72 hr. The animals were fasted for 16 hr. before being swabbed orally.

As it was not practicable to kill healthy ferrets, the internal organs were not examined. Therefore, no results are available for comparison with those of Schweinburg and Sylvester[6] in other laboratory animals.

The flora isolated from the ferrets was usually mixed, comprising at least two bacterial species; but these were not always the same from the mouth and the rectum. The commonest organism was *Bacterium coli*, which was isolated from 108 of the 118 ferrets (91·5 per cent). Most strains were lactose fermenters but nine were atypical varieties, The other bacteria isolated were strains of *Proteus vulgaris*, *Staphylococcus albus*, alpha- and beta-hæmolytic streptococci, *Micrococcus catarrhalis*, diphtheroids chiefly *Corynebacterium xerosis* types, *Pseudomonas pyocyanea* and Gram-positive spore-bearing bacilli. These bacteria were not further identified nor was any selected for individual study.

The observations are grouped in Table 1 to show the site of origin of each bacterial species and the number of times it was isolated, and in Table 2 to show the number of ferrets from which one or more bacterial species were isolated.

From these observations it would appear that the flora of the alimentary tract of the ferret is similar to that of other animals. The relatively high proportion of strains of *Proteus* which was isolated from 74 of 118 ferrets (62·7 per cent) might be significant, although a comparable proportion was reported by Goret *et al.*[7], who found it in the fæces of 68·1 per cent of dogs which showed no signs of intestinal infection. The absence of anaerobes such as the *Clostridia* commonly present in the fæces of many animals is also noteworthy.

Table 1

Bacterium	Number of ferrets from which it was isolated			
	Oral swab	Rectal swab	Both	Total
Bact. coli, typical	19	27	53	99
Bact. coli, atypical	5	3	0	8
P. vulgaris	12	48	14	74
Staph. albus	43	7	5	55
Streptococcus				
alpha-hæmolytic	25	6	0	31
beta-hæmolytic	3	7	0	10
M. catarrhalis	21	0	0	21
Diphtheroids	14	0	0	14
Ps. pyocyanea	17	1	1	19
Spore-bearing bacilli	3	3	0	6

Table 2

Number of bacterial species isolated per ferret	1	2	3	4	5	6	7	8
Number of ferrets	9	35	36	29	8	1	0	0

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Effect of Chloretone on Tunicate Embryos

It has been known for many years that amphibian embryos, allowed to develop under narcosis before muscular movements start, show the normal reflex and muscular activity when removed from the anæsthetic[1]. Amphibian tadpoles can be narcotized only for short periods, as they need to feed.

The tadpoles of *Ciona intestinalis* do not suffer this disadvantage, as they do not feed before metamorphosis. I have found that embryos of *Ciona* placed in 0·1 per cent chloretone in sea water five hours before hatching do not show the muscular twitching seen in controls and show no hatching movements at all. In the majority of cases the egg membranes eventually disintegrate, but in some they remain intact until metamorphosis is well advanced. The narcotized tadpoles metamorphose at approximately the same time as free-swimming controls. A period of free swimming has previously been thought necessary for metamorphosis[2] in the non-viviparous ascidians; but the observation reported here does not support this.

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