(*a*) Exchange reaction between 'Lipiodol *UF*' and iodine-131. 0·1 ml. of 0·1 N potassium iodide, 0·1 ml. of 0·01 N sodium nitrite, 1 ml. potassium iodine-131 (10 mc.), 2 ml. carbon tetrachloride and 0·15 ml. 0·02 N hydrochloric acid are pipetted into a glass-stoppered flask and intensively stirred for 1 h. The organic phase containing the iodine-131 released is separated and washed three times with 1·5 ml. water. The iodine-131 solution in carbon tetrachloride together with a mixture of 2 ml. of carbon tetrachloride and 1 ml. of 'Lipiodol *UF*' is heated to 50°–60° in a tapered flask under reflux. The solution is then extracted twice with 2 ml. of 0·001 N $Na_2S_2O_3$, twice with 2 ml. 0·001 N potassium carbonate and twice with 5 ml. water. The carbon tetrachloride solution is evaporated *in vacuo*, dissolved in ethanol, filtered if necessary, and evaporated again. A clear oil is obtained: radioactivity 9 mc. iodine-131 (yield 90 per cent). The product is chromatographically identical with the starting material and does not contain any free iodine-131.

(*b*) Exchange reaction between 'Lipiodol *UF*' and potassium iodine-131, 1 ml. 'Lipiodol *UF*', 35 ml. of acetone and 2 ml. of an aqueous solution of potassium iodine-131 and 0·5 ml. 0·001 N potassium iodide carrier are mixed and heated under reflux for 2 h. The solvents are evaporated, 5 ml. petrol ether and 2 ml. water added and the layers separated. The petrol ether solution containing 87 per cent of the initial radioactivity is evaporated to give a colourless oil.

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BIOLOGY

Resistance to Warfarin in the Common Rat

A case of apparent resistance of the common rat to warfarin occurred in 1958 on a farm in West Scotland[1], and further evidence of resistance from the same farm was given in 1963 (ref. 2). No other report on this matter is known to me.

In 1962 some difficulties arose in controlling rats (*Rattus norvegicus*) on a couple of farms in Jutland, Denmark, and some rats caught on the farms during the winter of 1962–63 were consequently tested for warfarin resistance in the laboratory. After being fed for two or three weeks on the same diet as brown rats from the rat farm of the Laboratory, they were compared with these rats in four tests, each consisting of four rats from Jutland (*J*-rats) and four rats from the rat farm (*RI*-rats). The two groups were identical in regard to sex and rather uniform in regard to weight. They were placed singly in metal cages and supplied with water from a bottle with tube at the top of the cage.

Table 1

Dose of warfarin	No. of rats *J*-rats	No. of rats *RI*-rats	No. of rats surviving *J*-rats	No. of rats surviving *RI*-rats	Average No. of days from first portion eaten to death *J*-rats	Average No. of days from first portion eaten to death *RI*-rats
Test 1 0·025% 1 ml. for 5 days	4	4	2	1	10 (2 *r*)	4·7 (3 *r*)
Test 2 0·045% 1 g for 5 days	4	4	3	0	11 (1 *r*)	6·5 (4 *r*)
Test 3 0·049% 1 g for 5 days	4	4	3	0	6 (1 *r*)	6·5 (4 *r*)
Test 4 0·024% 1 g for 5 days	4	4	4	0	—	7·3 (4 *r*)
Total	16	16	12	1		

(-*r*) = (-rats). All the dead rats had pronounced internal hæmorrhages.

In test 1 each rat had 1 ml. of a warfarin solution dripped on a piece of white bread on each of five successive days.

In tests 2–4 each rat had 1 g of a warfarin preparation mixed up in white bread on each of five successive days. When five portions had been eaten, the rats were fed with ordinary rye bread.

In test 4 the *J*-rats were survivors from tests 1, 2 and 3.

As two groups of *J*-rats, each consisting of seven rats, were received during October–November 1963, two supplementary tests were carried out.

In test 5 the seven *J*-rats from the first group were fed a bait containing 0·05 per cent warfarin. On the first day each rat was offered 30 g of the bait as the only food and more was added the following days, the rat always having a surplus of bait to eat. After 51 days the test was broken off and the amount of bait eaten was measured. One rat died in seven days with internal hæmorrhages, the other six remained sound, eating on an average 630 g bait each in the period (504–887 g).

In test 6 four *J*-rats from the second group were fed a bait containing 0·025 per cent warfarin + 0·025 per cent sulphaquinoxaline (an antibacterial agent supposed to eliminate the bacteria in the intestinal tract of the rat, and in this way reducing the amount of vitamin K, the antidote to warfarin). These rats were compared with the three remaining *J*-rats being fed on an ordinary 0·025 per cent warfarin bait.

One of the four *J*-rats died after 11 days with no sign of warfarin poisoning (inflammation of the lungs and enteritis), the rest remaining sound for 30 days, when the test was broken off. In this period each rat had eaten on an average 410 g (358–460 g).

The surviving *J*-rats from tests 1–4 plus later received rats were placed in an empty rat pen at the laboratory in order to let them breed.

Besides showing an astonishing resistance to warfarin in the *J*-rats, the test may indicate that the vitamin K-producing bacteria in the intestinal tract are not primarily responsible for this resistance.

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Retino-hypothalamic Connexions in Cetacea

The existence of retino-hypothalamic neural connexions in mammalian forms has long been controversial. The more recent investigations of Blümcke[1] in the cat and guinea pig, of Frey[2] in the guinea pig and of Knoche[3,4] in man, dog and rabbit strongly suggest that a bundle of fibres leaves the optic pathway at a chiasmatic level and passes dorsalward into the area of the paraventricular nucleus of the adjacent hypothalamus and also into the posterior lobe of the hypophysis. Altman[5], using the Nauta–Gygax staining technique to demonstrate fibre degeneration following optic nerve section in the cat, observed fibres of retinal origin above the optic chiasm and in the lateral hypothalamus immediately adjacent to the optic tract. Giolli[6], on the other hand, using