is lightly smeared with a non-oily analgesic jelly prior to insertion.

A programme touching on several aspects of ruminant physiology and nutrition has been built up. The detailed results of these researches will be published from time to time over the next two years.

Acknowledgment is made of the financial support of the Research Committee of the University of New Zealand and of the co-operation of Mr. W. M. Webster, head of the Veterinary Department, Massey Agricultural College, who was responsible for many helpful suggestions and the surgery which he will report on elsewhere.

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Bovine Leptospirosis in Kenya

In Africa evidence for the existence of leptospirosis in ruminants has been found in the Belgian Congo[1], Algeria[2] and Tunisia[3]. We now record disease in cattle, sheep and goats in Kenya caused by leptospiræ.

During May 1956, deaths occurred in all three species on ten farms on the Uaso Nyiro River in the Nanyuki District. Jaundice, red discoloration of milk, photosensitization-like lesions of unpigmented areas of skin, particularly that covering the udder and teats, and darkening of urine were the main symptoms noted in the acute syndrome in cattle. Subacute cases were also seen, with death after a long period of ill-defined sickness with loss of condition. Native and cross-bred sheep and native goats were also affected, but merino sheep on adjoining parts of the farms did not become infected. Disease in the smaller ruminants was usually hyperacute. At post-mortem the main findings were nephritis with inconstant jaundice.

The first laboratory confirmation was in necropsy material from a cow during the natural outbreak, by the observation of leptospiræ in the formalinized urine sample using dark-ground microscopy and of masses of leptospiræ within kidney tubules in histological sections stained by the Warthin–Starry method. Similar findings have since been made in specimens from goats and further cattle. A series of animals was inoculated with fresh kidney material from a clinical case in a goat. The acute disease was reproduced in a pregnant ewe and chronic infections established in sheep, goats, yearling steers and pigs. In pigs, massive urinary excretion of leptospiræ has occurred without clinically detectable illness.

The guinea pig has proved to be relatively insusceptible to the leptospiræ concerned, but infections with death at 9–11 days after intraperitoneal inoculation have been regularly reproduced in golden hamsters.

Leptospiræ have been isolated in pure culture in Fletcher's and Korthof's media from hamster tissues and blood, during passage in this species. Isolations have also been made, seven days after inoculation, from the heart blood of chicks given infective material by the intraperitoneal route at two days of age.

Chronic kidney lesions have been found at the abattoir in apparently healthy steers from affected farms. Histologically, the lesions resemble those described by Hadlow and Stoenner[4] in chronic *L. pomona* infection. Leptospiræ have been isolated from some of these and seen in others by dark-ground microscopy.

The species of leptospira involved is being determined.

It is hoped to publish this work more fully elsewhere.

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Normal Resistance-level of *Anopheles funestus* Giles to Insecticides

The tolerance to insecticides of normal and resistant strains of *A. gambiae* Giles has been measured by a number of workers[1–3], but so far no figures for *A. funestus*, the second main malaria vector of Africa, seem to have been published. This communication records the results of tests carried out on this species in an area of Sokoto Province, Northern Nigeria, about ten miles west of the sprayed zones of the Western Sokoto Malaria Control Pilot Project. The test for susceptibility described by Busvine[4] was applied to wild-caught females in the blood-fed and gravid states. Results are summarized in Table 1.

These results have been evaluated by the method of Litchfield and Wilcoxon[5], and values for the median lethal concentrations of DDT and gamma-BHC, with confidence limits for 95 per cent probability, are respectively 1·32 per cent (1·39 and 1·24 per cent) and 0·0033 per cent (0·0040 and 0·0027 per cent). In the case of dieldrin the data are significantly heterogeneous as shown by the χ^2 test; the median lethal concentration apparently lies

Table 1

Toxicant	Concentration (per cent)	No. of mosquitoes exposed	Mortality (per cent)
DDT	0·75	33	97
	0·50	69	90
	0·33	90	54
	0·25	47	28
Control	—	27	0
Gamma BHC	0·01	33	90
	0·007	70	71
	0·004	51	60
	0·002	34	29
	0·001	17	0
Control	—	31	0
Dieldrin	0·1	74	81
	0·075	52	35
	0·050	56	27
	0·033	44	14
	0·025	55	4
Control	—	29	0