cyted by leucocytes, is followed by characteristic degenerative changes and death of the latter. On the other hand, doses of up to 500 mgm. of lipid in liquid paraffin inoculated intraperitoneally in guinea pigs produced no evidence of general systemic toxic effects.

There are obvious similarities between this toxic lipid of *C. ovis* and the surface lipoid component ('cord factor') of the virulent tubercle bacillus recently reported by Bloch and others[1], which, in addition to having toxic properties, appears to be closely concerned with the mechanism of the immunological response to the tubercle bacillus[2]. In contrast with the reported toxicity of 'cord factor', which produces death with pulmonary hæmorrhages in mice two to three weeks after intraperitoneal injection, *C. ovis* lipid has a local toxic action which appears within twenty-four hours after intradermal injection, leucocytes are especially vulnerable and no delayed systemic toxic effects have been observed in guinea pigs.

A detailed account of this work, including chemical and immunological studies at present in progress, will be published elsewhere.

H. R. Carne
Nancy Wickham
Joan C. Kater
Department of Veterinary Pathology
and Bacteriology,
University of Sydney.

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Occurrence of New *Salmonella* Types from South Africa

Upon bacteriological examination of day-old chicks imported by air from the Union of South Africa and found dead on arrival at Elizabethville, the following types of *Salmonella* were detected by culturing the bone marrow of fourteen of them on the usual media : 2 strains of *S. saint paul* (4,5,12 : *e,h* : 1,2) ; 1 strain of *S. vejle* (3,10 : *e,h* : 1,2) ; 1 strain of *Salmonella* unidentified, probably *S. newport* (6,8 : *e,h* : 1,2) rough.

Salmonella saint paul and *S. vejle* seem never to have been isolated in South Africa[1–3]. A few strains of *S. newport* have been isolated by Henning[2], one from a human being and another from a duck.

In the Belgian Congo, *S. saint paul* has only been isolated from human stools (7 strains)[4]. *S. vejle* has been isolated only once from a native child's stools[4]. *S. newport* is well known and has been isolated several times from human material and from horse and goat[5–7].

In East Africa, between 1948 and 1953, Mackey[8] isolated in Dar es Salaam 3 strains of *S. saint paul*, all from human origin ; none of *S. vejle* and 11 strains of *S. newport* (9 from humans and 2 from lizards).

We might thus consider that *S. saint paul* and *S. vejle* are new for South Africa.

This is also their first isolation from animals in the Belgian Congo, and the fact that they were found in imported chickens is one more hint on the complex epizootology of salmonellosis in animals.

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J. Deom
Veterinary Laboratory,
Elisabethville,
Belgian Congo.
March 23.

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Influence of Nutritional State on the Heat Tolerance of Cattle

On completion of an experiment in which two differently fed groups of cattle were used for studying the effect of low nutrition on seasonal changes in the hair coat, opportunity was also taken to make observations on the heat tolerance of the animals. The results of these latter observations are reported here.

On April 20, 1955, eight poll shorthorn heifers aged 6–8 months and weighing an average of 351 lb. were randomly divided into two groups of four animals each. One group, kept on clean straw bedding, was fed liberally, calf meal (18 per cent crude protein) being supplied at the rate of 2 lb. per animal per day together with as much lucerne hay as the animals would consume. On this ration, individuals of the group increased to an average of 672 lb. weight by February 28, 1956. The feed of the other group was restricted as necessary to maintain constant weight of the individuals over the same period. They were bedded on sawdust and received meal at the rate of 4 oz. per animal per day, one-quarter the amount of lucerne hay supplied to the well-fed group and less straw than sufficient to satisfy appetite. Both groups received vitamin supplement in the form of a commercial preparation of fish liver oil.

On February 28, 1956, immediately following their usual morning feed, the eight heifers were subjected to a heat tolerance test in a psychometric room. The duration of the test was three hours unless an animal's rectal temperature reached 107° F., in which case the individual was withdrawn. The room temperature during exposure was 105° F. dry bulb and 92° F. wet bulb. Rectal temperatures were taken at the end of each hour, and each animal's heat tolerance coefficient was calculated from its rectal temperature at the end of the test, using Rhoad's[1] formula :

$$\text{Heat tol. coeff.} = 100 - 10\,(\text{rect. temp.} - 101)$$

The mean heat tolerance coefficient of the fat, well-fed group was 43 ; that of the thin, poorly fed group 55. The difference of 12 between the group means is highly significant ($P = 0{\cdot}01$) and reflects the greater heat tolerance of the thin animals. This was also evidenced by their relative freedom from drooling, their calmer breathing and less anxious state at the end of the test.

Following this test, the poorly fed group was placed on the liberal ration, whereas no feed at all was allowed the well-nourished group. These feeding conditions obtained for the post-test period of February 28, for all of February 29 and for the morning of