Prof. J. H. Biggart has very kindly further confirmed the nature of the brain lesions by sectioning.

While our work on this subject is not yet completed, we think it advisable to direct attention to these observations in view of their possible practical significance. It is possible under war-time conditions that potatoes and dried potato products may frequently be regarded as cereal substitutes in chick-rearing rations, which are also likely to contain cod-liver oil. Incorporation of cod-liver oil with cereal products is well known to lead to oxidative destruction of vitamins E, and where the proportion of cereal is low or stale meals are used, nutritional encephalomalacia may be encountered. It is clear that, under war-time conditions, obscure types of leg-weakness other than rickets or curled-toe paralysis (riboflavin deficiency) may be due to nutritional encephalomalacia and should be checked by macroscopic and, where possible, microscopic examination of the cerebellum.

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p-Cresol and Œstrone in Urine

THE presence of *p*-cresol in human urine, as well as in the urine of horses, cows and other animals, has been known for a long time. It occurs mainly as a salt of *p*-tolylsulphuric acid. According to Siegfried and Zimmermann[1], the average concentration of *p*-cresol in normal human urine is of the order of 18 mgm. per litre, and in a man with an adrenal tumour a value of 25 mgm. per litre has been reported[2]. In the urine of pregnant mares, Marshall has found that the concentration is 60 mgm. per litre[3].

It is significant that these increases in *p*-cresol are accompanied by corresponding increases in the concentrations of œstrogenic hormones, mainly œstrone, present in the urine, which have been reported as 0·016 mgm., 0·3 mgm. and 10 mgm. per litre respectively in the three cases. The urine of stallions provides an even richer source of œstrone, the concentration reported being of the order of 17 mgm. per litre[4]. We have confirmed this figure for total œstrogen, and in addition have found that the concentration of *p*-cresol in this instance reaches the remarkably high value of 550 mgm. per litre. Approximately 25 gm. of *p*-cresol (benzoate, m.p. 72°; aryloxyacetic acid derivative, m.p. 134°) were isolated from the strong phenolic fraction from 10 gallons of the mixed acid-hydrolysed urine from two stallions. It is also known that the œstrone content of human urine increases progressively during pregnancy, and Falsia[5] has reported that there is a corresponding increase in the *p*-cresol content.

The origin of the phenol and cresols in urine is usually attributed to tyrosine, and in support of this view it has been claimed that the concentration of phenols in urine increases with an increase in protein intake. On the other hand, Fricke[6] has pointed out that the phenolic constituents of urine are most abundant in Herbivora, and has claimed that the quantity in human urine is increased on a vegetable diet. These considerations seem to indicate that tyrosine and intestinal putrefaction may not be the sole source of phenols in urine, and the above correlation between the elimination of œstrone and *p*-cresol suggests the possibility that not only the former but also the latter may be derived from the male hormone or some related steroid. On this basis the *p*-cresol could arise from ring *A* of the steroid molecule, in which the hydroxyl and methyl group are correctly placed. This possible connexion between the natural sex hormone and simple phenols, coupled with the known œstrogenic activity of many phenolic compounds of comparatively simple structure, recalls the suggestion, originally due to Dodds[7], that the true œstrogenic agents may be relatively simple compounds resulting from the breakdown of the cyclopentanopolyhydrophenanthrene system.

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A Search for Endemic Areas of Trichinosis in Great Britain

ALTHOUGH the life-history of *Trichinella spiralis* has been known for nearly a century, we are still very ignorant of the epidemiology of trichinosis in man—a gap in our knowledge which remained unsuspected until Hall[1] discovered the frequent occurrence of sub-clinical human infestation in the United States. A repetition of Hall's work more recently carried out in Great Britain by Van Someren[2] and later by Miss M. Young[3] has revealed that a similarly high incidence of infection is to be found here. Interest in the epidemiology of the infection in Great Britain has been still further stimulated by the outbreaks of clinical trichinosis which occurred during the winter of 1940–41 at Wolverhampton, Penrith and Harpenden[4,5] accentuating the urgency for the discovery of the origin of infection.

In the past, it has been generally supposed that outbreaks of trichinosis in Great Britain are traceable to the importation of infected pork, which occasionally finds its way through the meat inspection; but this now appears to be an inadequate explanation, and it seems more than likely that reservoirs of infection exist among animals in our own country.

Although Leiper[6] was able to demonstrate infection in rats at centres where trichinosis had been diagnosed in man, the general incidence of rat infection throughout Great Britain appears to be very low. The attempted detection of hitherto unrecognized endemic areas through the examination of rat carcases appeared therefore to be an unpromising line of research, and it was thought that the examination of the carcases of rat-eating animals might be more likely to produce a result, as their skeletal muscles would present evidence of trichinosed flesh having been eaten at any time during their whole lives. Some 636 stoats, 78 weasels and 2 polecats were secured for this purpose, through the medium of the pest officers in various counties, and were subjected to a thorough examination. In this process