were significant differences, however, in the relative percentage composition of the neutral lipids (Table 1).

In the infective larvae, free fatty acids were the principal component of the neutral lipid fraction and the amounts of triglyceride were relatively small, while in the parasitic adult triglycerides formed a much larger percentage of the total neutral lipids.

The difference in the lipid composition between the free-living larvae and the parasitic adult can be related to their different modes of life.

Respiratory studies have shown that the free-living infective larvae of *N. brasiliensis* and *S. ratti* do not metabolize exogenous substrates, and they therefore must rely entirely on their endogenous food stores (ref. 2 and my unpublished results). These infective larvae contain negligible amounts of glycogen, but they do have large stores of lipid which decrease with age (ref. 3 and my unpublished results). Thus if the larval lipids are being extensively catabolized this could well be associated with high levels of free fatty acids. High levels of free fatty acid are found in tissues which are very active metabolically such as insect fat bodies[4] and mollusc digestive glands (my unpublished results).

Table 1. LIPID COMPOSITION OF THE FREE-LIVING INFECTIVE LARVAE AND PARASITIC ADULTS OF *Strongyloides ratti* AND *Nippostrongylus brasiliensis*

| | *Strongyloides ratti* | | *Nippostrongylus brasiliensis* | |
|---|---|---|---|---|
| | Larvae | Adults | Larvae | Adults |
| Total lipid as % of dry weight | 25 | 10 | 15–20* | 11·9† |
| Lipid components as % of total lipid | | | | |
| Triglycerides | 12 | 32 | 19·5 | 55 |
| Free fatty acids | 67 | 40 | 59 | 20 |
| Cholesterol | 1·2 | 3 | 3 | 2·2 |
| Mono and diglycerides | < 1 | < 1 | < 1 | < 1 |
| Phospholipids | 18 | 20 | 16 | 20 |

Each reading is the average of five replicates, mean coefficient of variation 7 per cent.
* Data from Wilson[3].
† Data from Roberts and Fairbairn[5].

In the adults, on the other hand, lipid catabolism may be much less important[5] and the chief function of lipid at this stage may be as a food reserve for the gametes.

The change from free fatty acids to triglycerides could also be related to the change from the free-living to a parasitic mode of life. When the larvae become parasitic there is a big increase in the ambient temperature. In general, it is found that animals which live at higher temperatures have lipids with a higher melting point than animals which live at lower temperatures[6]. Thus, if the same considerations apply, when the infective larvae become parasitic a change must take place in the composition of their lipids. The melting point of the lipids could be raised by increasing the degree of saturation and/or increasing the chain length of the fatty acids. Also the lipids synthesized after infection are probably different in composition from those synthesized before infection. The presence in the free-living larvae of relatively large amounts of free fatty acids might in some way be related to this change, in that free fatty acids may be more readily altered when in the free form than when esterified as triglycerides.

I thank Dr D. L. Lee (Houghton Poultry Research Station) for providing the *Nippostrongylus* material. This work was carried out during the tenure of a Medical Research Council scholarship.

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Received April 8; revised May 1, 1968.

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## Removal of Plasminogen and Factor XIII from Fibrinogen

ALLINGTON[1] described a method in which a fibrin gel is ultimately formed by dilution of a urea solution of fibrin monomer. Some investigators may prefer to have the original gel structure formed consequent to the action of thrombin on fibrinogen, so it should be noted that a simple charcoal adsorption procedure is highly effective in the removal of plasminogen from a variety of human and animal fibrinogens, including commercially available ones[2–6].

Although the matter was not emphasized in previous publications, it is also known that the adsorption removes Factor XIII along with the plasminogen. For example, clots were made containing 3 mg/ml. human fibrinogen (AB Kabi, Grade *L*), 2 units of thrombin, $5 \times 10^{-3}$ M cysteine, and $5 \times 10^{-4}$ M $CaCl_2$. After 1 h, an equal volume of 60 per cent urea was added. Clots made with the original fibrinogen gave no sign of dissolution in over 24 h, while those formed from charcoal-adsorbed fibrinogen dissolved completely in about 2 h.

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Received February 1; revised April 29, 1968.

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## Single Unit Activity evoked by Thermal Stimulation of the Cervical Spinal Cord in the Guinea-pig

EVIDENCE has been accumulating for the existence in warm-blooded animals of temperature sensors in the spinal cord[1–3], as well as the well known cutaneous thermal receptors and the thermosensitive structures in the anterior hypothalamus. In the guinea-pig these thermosensitive structures seem to be confined to the cervical and upper thoracic part of the spinal cord[3]. Heating this restricted area, using radio-frequency, results in the complete suppression of shivering induced by external cooling. The thermosensitive area receives, through a vascular connexion[4], heat generated in the brown interscapular adipose tissue. This tissue is found in most mammals during the neonatal period and after cold adaptation. Shivering is suppressed in these animals when they are exposed to a cool environment as long as the brown adipose tissue produces heat sufficient to maintain the cervical spinal cord at a constant temperature[3,5]. More recently[6] radio-frequency heating has been carried out after partial or total sectioning of the spinal cord segment at $C_5$, and it was shown that suppression of shivering is possible in muscle groups supplied from segments above the lesion, as long as a small medio-ventral area of white matter is still intact. On the other hand, to impede the suppression of shivering in the rostral muscle groups only this medio-ventral area needs to be destroyed at $C_5$. These results suggest that this medio-ventral area of the spinal cord contains afferent fibres which serve to connect the spinal thermosensors with some intracranial centres. To further elucidate the function of these postulated thermal sensors, we have carried out electrophysiological studies in young guinea-pigs (4–10 weeks old), anaesthetized with 'Nembutal' (40 mg/kg).