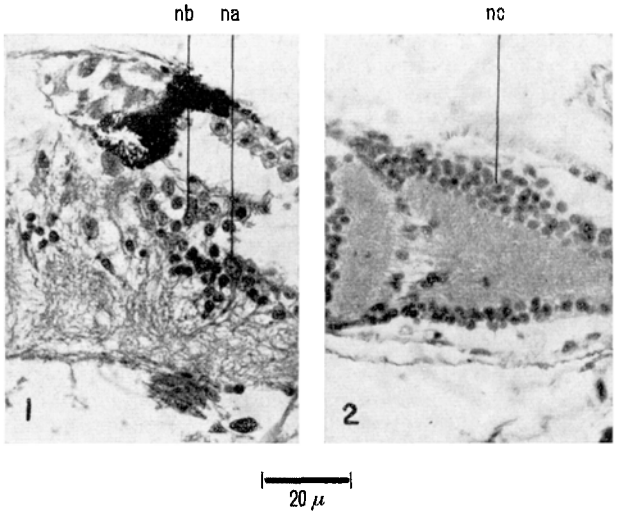

Fig. 1. Horizontal section of the supraoesophageal ganglion of *Artemia* showing large neurosecretory cells (na) and small cells (nb), Gömöri.

Fig. 2. Horizontal section of the eye-stalk in the region of the X-organ of *Artemia* showing the third type of neurosecretory cells (nc), Gömöri.

Release of granules appears to take place both through the cell membrane and by axon transport. In the middle of the brain ventrally, there is a deeply staining region receiving axons from the large cells described above. This is probably a storage organ like the Y-organ of the eye stalk of decapod crustacea.

The study of neurosecretion in *Artemia* would seem to offer a new and profitable approach to many problems of its physiology and ecology. Since this race of *Artemia* of Sambhar lake is parthenogenetic, the relation of their neurosecretory activities to their reproduction raises problems of unusual interest. Work on these lines is in progress.

Zusammenfassung. Mit der Gömöri-Technik wurden bei *Artemia* drei Typen neurosekretorischer Zellen nachgewiesen. Im Gehirn sind grosse, ovale Zellen mit vakuolisiertem Cytoplasma vorhanden, von denen einige grosse Axone besitzen. Der 2. Typus ist kleiner und weist keine zum Augenstiel führenden Fortsätze auf. Der 3. Typus liegt als traubenförmige Gruppen von Zellen im X-Organ des Augenstiels. Wahrscheinlich ist ventral am Gehirn ein Depotorgan für das Sekret des 1. Typus vorhanden. Die Beziehung dieser Zellen zur parthenogenetischen Vermehrung der Art wird untersucht.

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Immunoelectrophoretic Characteristics of Plasma from Rats with Adjuvant Arthritis

Adjuvant arthritis in rats is an experimental syndrome which can be partially or completely inhibited by treatment with various anti-inflammatory agents[1]. There are currently no reports in the literature pertaining to alterations in plasma immunoelectrophoretic patterns of arthritic rats, nor of the influence of anti-inflammatory agents on these patterns. In this communication, the immunoelectrophoretic characteristics of plasma from both treated and non-treated adjuvant-injected rats are reported.

A total of 27 male albino rats, derived from the Wistar strain, were used in this experiment. Group I (9 rats) received an intradermal injection in the mid-tail region of 0.8 mg heat-killed *Mycobacterium butyricum* in 0.1 ml mineral oil; Group II (9 rats) received the same but were injected daily with 10 mg/kg hydrocortisone acetate (Hydro-Adreson, Organon) subcutaneously, starting on the day of adjuvant injection; Group III (9 rats) received 0.1 ml of mineral oil only. 21 days after adjuvant injection, all animals were anaesthetized with ether, and blood was obtained by heart puncture with heparinized syringes. Plasmas were separated by centrifugation and stored at −20°C until use.

Antisera were obtained by immunization of rabbits with a 1:1 suspension of serum from normal rats and complete Freund's adjuvant. The immunization schedule involved intramuscular injection of 0.4 ml of the suspension the first week and 0.6 ml suspension for each of the succeeding 3 weeks. Rabbits were bled 10 days after the last injection and the separated antisera were stored at −20°C until use.

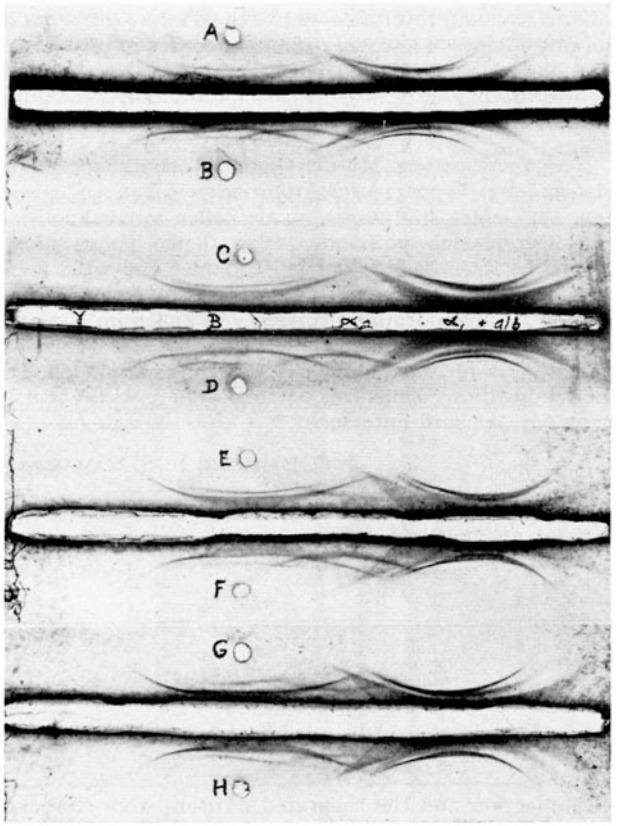
A micro-immunoelectrophoretic procedure, using microscope slides and an apparatus designed by Wieme[2], was used. A constant current of 30 mA per slide at 10°C was employed, and the duration of electrophoresis was 45 min. After electrophoresis, antisera grooves were filled and the slides were allowed to set for 36 h in a moist chamber at room temperature. Following the washing and drying procedures, the slides were stained with Ponceau S.

During the second and third weeks after adjuvant injection, 7 out of 9 animals in Group I developed mild to very severe arthritis. The symptoms included swelling of the joints of the legs, nodular lesions on the external ears, and extensive necrotic areas on the tail. Animals which were treated with hydrocortisone had no joint swelling or gross manifestations of arthritis, the ear lesions were absent, and the tail lesions were less severe than those of the untreated animals.

Typical immunoelectrophoretic patterns of plasmas from animals of the three groups are shown in the Figure. Patterns from animals of the vehicle-control group had a minimum of one rather broad band for albumin, two α-1 glycoproteins, a prominent α-2 protein, one rather long and dense β-globulin line near the antiserum groove and another shorter and finer one nearer the electrophoretic axis, and a very faint γ-globulin precipitin line. Patterns

[1] B. B. Newbould, Brit. J. Pharmacol. Chemother. *21*, 127 (1963).
[2] R. J. Wieme, Clin. chim. Acta *4*, 317 (1959).

from untreated rats of the arthritic group either lacked the α-2 precipitin line completely (animals with the most severe arthritis), or exhibited a very greatly reduced line in this area. The patterns from animals of this group also showed an increase in the β-globulin line nearest the electrophoretic axis. Adjuvant-injected rats which were treated with hydrocortisone had patterns which were essentially the same as those from control animals.



Immunoelectrophoretic patterns of plasmas from untreated, treated, and control rats. Plasmas A, C, and E are from rats of the untreated group, graded 4+, 2+, and negative, respectively, according to severity of arthritis prior to sacrifice. Plasma G is from a rat treated with hydrocortisone. Plasmas B, D, F, and H are from four different vehicle-control animals.

BLUMENKRANTZ et al.[3] reported that rats with adjuvant arthritis have increased urinary excretion and decreased serum concentration of glycoproteins. The results of the present experiment are of interest, in this regard, in that the complete lack of, or great reduction in, the α-2 protein was observed only with plasmas from rats of the arthritic group. Hydrocortisone administration, in addition to preventing development of gross manifestations of arthritis, apparently also prevented the loss of this protein component.

LOWE[4] recently reported that starch-gel electropherograms of sera from rats, between the 16th and 20th days after adjuvant injection, showed the appearance and subsequent disappearance of a protein migrating near the slow α-2 globulin. In view of this, immunoelectrophoretic analyses of plasma from rats during the early course of the arthritic syndrome are currently in progress.

Zusammenfassung. Das Plasma-protein unbehandelter und mit Hydrocortison behandelter Ratten wurde 21 Tage nach Adjuvantinjektion immunelektrophoretisch untersucht. Arthritische Ratten zeigten eine Zunahme von β-Globulinen bei Abwesenheit oder Abnahme von α-Globulinen. Ein plasma-immunelektrophoretischer Unterschied zwischen mit Hydrocortison behandelten Tieren und Kontrolltieren wurde nicht gefunden.

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with the technical assistance of J. COOLEN

Institute of Pathology, Free University of Amsterdam, (Netherlands), March 5, 1965.

[3] H. BLUMENKRANTZ, J. A. HOUSSAY, A. B. HOUSSAY, and M. E. B. RENDO, Acta physiol. latin-am. *13*, 313 (1963).
[4] J. S. LOWE, Biochem. Pharmac. *13*, 633 (1964).

Sur l'apparition de glucose-6-phosphatase dans le pancréas du rat blanc au cours du diabète alloxanique

Il a été signalé[1-4] que dans le pancréas du rat blanc normal, on ne trouve pas de glucose-6-phosphatase histochimiquement décelable. Or, beaucoup d'auteurs[5-7] attribuent un rôle important à cette enzyme dans la sécrétion de l'insuline et éventuellement dans la pathogénie du diabète. Il est vrai que cette enzyme est hautement spécifique pour les cellules B des ilôts de Langerhans de divers animaux: lapin, souris, cobaye, chien, etc. C'est pourquoi l'étude de cette enzyme au cours du diabète mérite d'être entreprise dans le pancréas d'espèces où normalement elle est absente comme c'est le cas du rat blanc.

Méthode. Trente rats blancs mâles, de la race «Wistar», pesant chacun de 150 à 200 g après jeûne de 24 h, ont été rendus diabétiques. Le diabète était provoqué par une seule piqûre intrapéritonéale d'alloxane «Roche», à rai-

[1] J. VERNE et P. PETKOV, Annls. Endocr. *22*, 965 (1961).
[2] J. VERNE, R. WEGMANN, P. PETKOV et S. GUHA, Annls. Histochim. *6*, 33 (1961).
[3] P. PETKOV, Thèse (en bulgare) (Sofia 1962), p. 134.
[4] W. GEPTS et D. TOUSSAINT, Annls. Endocr. *24*, 688 (1963).
[5] S. S. LAZARUS, Proc. Soc. exp. Biol. Med. *102*, 303 (1959).
[6] S. S. LAZARUS, Proc. Soc. exp. Biol. Med. *101*, 819 (1959).
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