that neutralize both LLV and FLC[14], and exhibit lymphoid cells that prevent and even cure Friend disease (unpublished results). The need for such awareness is eloquently illustrated by the present results.

Recent studies[17,18] have shown that plant lectins concanavalin A (Con A) and phytohemagglutinin may interact with FLC, abolishing or reducing its infectivity, and that a proportion of mice surviving inoculation with FLC pretreated with Con A (Con A-FLC) acquire a high level of resistance to subsequent challenge with FLC. These findings have been interpreted as indicating that FLC after inactivation with Con A retains its immunogenicity and may therefore be an efficient means of immunization.

We have tested the alternative possibility that the immunizing activity of Con A-FLC might be due to residual LLV infectivity. On day – 60, inbred female BALB/c mice aged 8–10 weeks were i.p. inoculated with 0.2 ml of a plasma preparation of FLC which had been preincubated for 1 h at room temperature with an equal volume of Con A (Miles Laboratories, Kankakee, Illinois, USA) in phosphate-buffered saline (PBS) at a final concentration of 30 μg/ml. The virus, NB-tropic anemia-inducing and LDH virus-free, had been prepared as described[19] and diluted to contain $10^{2.5}$ mean infective doses per 0.1 ml. On day 0, whereas none of 15 control mice inoculated with FLC preincubated in PBS alone survived, out of 40 mice injected with Con A-FLC 24 were alive and 18 had no palpable splenomegaly.

At this time the 18 Con A-FLC-injected mice with no appreciable splenomegaly were ear-marked and individually bled (0.3 ml of blood) by retroorbital puncture. They were then i.v. challenged with $10^{2.5}$ infective doses of FLC on the same day and their spleens were weighed on day + 21. The individual blood samples obtained from the protected mice were immediately diluted 1:10 in PBS, clarified at low speed and separately tested for LLV in $S^+ L^-$ cells of the D-245 line by focus formation[20], and in BALB/c mice by a 3-week spleen weight assay[14], and by the ability to protect against FLC injected 21 days later. 8 mice were positive for LLV by one or more of the assays used, with maximum sensitivity exhibited by the protection test which was positive in all 8 mice, followed by the $S^+ L^-$ test (5 mice) and by the spleen weight assay (4 mice). As shown by the table, all LLV-positive mice proved resistant to FLC, whereas none of the LLV-negative animals showed significant levels of resistance, as judged from the weight of their spleens 3 weeks after FLC challenge. In a similar experiment, the resistance to FLC conferred by Con A-FLC could be passaged serially with plasma 4 times (and then the experiment was interrupted) provided that a 3-week-interval was allowed between plasma passage and FLC challenge, thus confirming the infective nature of the resistance-inducing agent.

These results corfirm the FLC-inactivating action of Con A and the resistance of Con A-FLC-treated mice to FLC challenge[17,18]. More importantly, by showing 100% correlation between resistance to FLC and presence of LLV in the blood of Con A-FLC-injected mice at the time of challenge, they clearly demonstrate that the immunizing activity of Con A-FLC is due to residual LLV infectivity. The mechanism by which Con A interacts with FLC is not known[18], but to explain the present findings it is not necessary to postulate a selectivity of Con A for different FLC components. Due to the higher titer of LLV in FLC preparations as compared to the other component(s), any inactivating treatment of FLC, unless complete, is bound to leave traces of this virus unaltered. In turn a proportion of recipients of partially inactivated FLC preparations develop an LLV infection that immunizes against FLC. In contrast, the recipients that do not become infected with LLV remain non-immunized, as shown by the absolute lack of resistance to FLC exhibited in the present results by LLV-negative mice.

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A new type of neurosecretion in the cerebral ganglion of a sipunculid

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Summary. A lipid neurosecretion is described in the giant cells of the cerebral ganglion in Sipunculus nudus (Sipunculida).

Metalnikoff[1] described 4 types of neurons in the cerebral ganglion of Sipunculus nudus:

I: Small unipolar cells, diameter about 5 μm, concentrated in aggregates. II: Small pear-shaped cells resembling typical neurons, diameter about 18–20 μm, scattered through most parts of the ganglion. III: Giant cells located in the posterior part of the ganglion. Same shape as type II; length of the cell body 40–60 μm, very large axon. IV: Bipolar cells, usually spindle-shaped, grouped in the anterior part of the ganglion on the boundary line between cerebral ganglion proper and cerebral organ.

According to earlier research, types II, III and IV are neurosecretory cells[2–4]. Gabe[4] observed that neurosecretory material (NSM) of the small pear-shaped cells (type II of Metalnikoff) stains with eosin, azocarmin, iron hematoxylin and PAS, NSM of giant cells (type III of Metalnikoff) with chrome hematoxylin, paraldehyde fuchsin (PF) and PAS, NSM of bipolar cells (type IV of Metalnikoff) with chrome hematoxylin and PF. On the other hand, Åkesson[5] could not find any signs of neurosecretory activity in the small pear-shaped cells (type II). This apparent disagreement led us to reinvestigate the neurosecretory system of Sipunculus nudus with histological and histochemical techniques.

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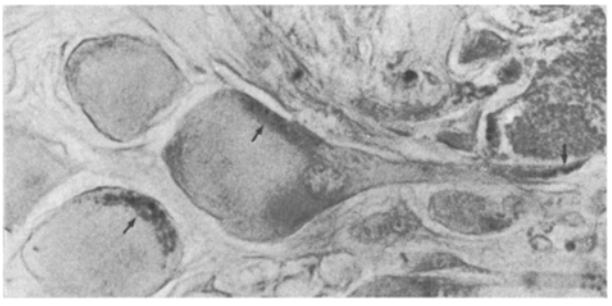
Materials and methods. Sipunculids (Sipunculus nudus) were obtained from the Stazione Zoologica of Naples. The segments containing the cerebral ganglion were fixed in Bouin's fluid or in 10% formaldehyde. Sections were stained with chrome hematoxylin-phloxine, PF, dihydroxy-dinaphthyl-disulfide, alkaline tetrazolium, PAS, Sudan black B. Formaldehyde fixed sections were observed under the fluorescence microscope (wavelength 3650 Å).



Giant cells of cerebral ganglion of Sipunculus nudus. Bouin; Sudan black B (SBB). × 400. Note SBB-reactive secretion product in pericarya and axon (arrow).

Results and discussion. The results of our histological and histochemical investigations of the Sipunculus cerebral ganglion do not confirm earlier descriptions of this organ. Neurosecretory activity was detected in giant cells (type III) and bipolar cells (type IV) only, whereas the small pear-shaped cells (type II) never showed any traces of neurosecretory material. Our results agree with Gabe's findings that the NSM of bipolar cells (type IV) is definitely of proteinaceous nature. Neurosecretory granules in cytoplasm and axons of giant cells (type III) did not react with PAS, which is contrary to Gabe's description of these cells as containing a glucide component. In our experiments, NSM of type III cells stained deeply with Sudan black B (figure); with fluorescence microscopy neither dissolved carotenoid pigments nor lipofuscins were detected in these lipid neurosecretory granules. The cerebral ganglion of Sipunculus nudus therefore contains 2 different types of neurosecretion: one type, designated as peptide neurosecretion, is found in bipolar cells. A second type, detected exclusively in giant cells, is characterized by secretory granules containing no peptide material but instead large amounts of lipids. The chemical nature of these lipids remains to be elucidated.

Pentagastrine, histamine et acide ascorbique de la muqueuse gastrique chez le rat

Pentagastrin, histamin and ascorbic acid in rat's gastric mucosa

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Summary. Pentagastrin and histamine, 2 potent gastric acid stimulants, lower ascorbic acid in the rat's gastric mucosa. They give the same result as nerve stimulation by desoxyglucose. Explanation of ascorbic acid function in gastric physiology needs further work.

L'acide ascorbique (AA) a récemment été localisé dans les cellules bordantes gastriques[1] et la teneur en AA de la muqueuse diminue au cours de la genèse de l'ulcère de contrainte chez le rat alors que le pH de l'estomac s'abaisse[2]. De plus l'administration de désoxyglucose (DG), qui provoque une sécrétion gastrique acide par intervention du vague, conduit au même résultat[1]. Nous avons cherché à savoir dans ce travail si 2 autres agents stimulants de la sécrétion acide, la pentagastrine (PG) et l'histamine (HT) provoquaient les mêmes modifications et si l'on pouvait établir une liaison entre les phénomènes observés.

Matériel et méthodes. L'expérimentation porte sur des rats mâles Wistar pesant suivant les essais successifs 180–230 g. Ces animaux, arrivés 8 jours avant l'essai, sont nourris avec un régime équilibré en éléments nutritifs et vitamines, et regroupés 6–8 par cage dans une animalerie thermostatée à 24 °C, éclairée artificiellement 12 h sur 24. Ils sont soumis la veille de l'expérimentation à une diète hydrique de 14 h. Les injections de pentagastrine (Peptavlon®, ICI) et d'histamine (Histamine Chlorhydrate, Merck) sont réalisées par voie sous-cutanée sur des rats préalablement tirés au sort entre le groupe témoin et le groupe traité. A la fin de l'essai les animaux sont assommés et les estomacs prélevés. Le pH est déterminé au moyen d'un papier indicateur au dixième d'unité appliqué sur la muqueuse étalée et un fragment de celle-ci (environ 200 mg) est prélevé par grattage, puis homogénéisé dans un liquide déprotéinisant. Le dosage de l'AA est effectué par la méthode au dichlorophénol indophénol[3] sur le surnageant après une double centrifugation à 3000 × g dans les 3 h qui suivent le prélèvement.

Résultats et discussion. L'administration de PG (1,5 mg/kg) provoque un abaissement significatif de la teneur en AA de la muqueuse (tableau), ainsi que des pH gastriques (Test U, Mann et Whitney), il existe de plus une corrélation statistiquement significative entre pH et teneur en AA ($r = 0{,}53$; $y = 79x + 65$). Pour une dose plus faible (0,5 mg/kg) et un délai plus important (60 min) la variation en AA n'atteint pas le seuil de significativité; la corrélation n'est plus retrouvée.

L'administration d'HT (100 mg/kg, 30 min) produit également un abaissement de l'AA de la muqueuse. Avec une dose plus faible (30 mg/kg, 60 min) ou un temps plus long (100 mg/kg, 60 min) les diminutions en AA, n'atteignent pas le seuil de significativité. Avec les conditions expérimentales retenues, nous ne mettons pas en évidence de corrélation entre AA et pH gastrique.

La PG produit une diminution de l'AA de la muqueuse aux doses généralement utilisées pour son action sur la sécrétion gastrique acide[4]. De plus cette variation est liée

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