soap water traps. The reason for this difference was probably due to the fact that the surface tension of pure water is too high to kill or to trap the attracted insects. Close examination of the water pheromone trap, shows a large amount of scales on the water, indicating that trapped insects can easily escape. We measured the surface tension of the detergent solutions used and found a significant correlation between the insect numbers caught and water surface tension. If the surface tension of the detergent water in our trap is higher than 71.2017 dyn/cm (0.05 g/800 ml) no moths are caught. The best results are obtained when the solution has a surface tension from 70.8427 dyn/cm to 69.6460 dyn/cm (0.5 g/800 ml to 1.5g/800 ml). In the second experiment, we trapped more male moths because the amount of female pheromone used was double that of the first experiment. So the percentage of females in the table was lower than that in the figure. In conclusion, it is evident that gravid females are attracted by water, probably because they need it for egg maturation. Thus low humidity is a limiting factor for the life of insects pests in a storage houses. The result may have some evolutionary or practical importance in the study of storage pests[16].

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Role of Friend-associated lymphatic leukemia virus in immunization against Friend leukemia complex[1]

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Summary. Mice inoculated with Friend leukemia complex (FLC) pretreated with concanavalin A are resistant to FLC challenge only when they have become infected with the FLC-associated lymphatic leukemia virus (LLV). In interpreting states of resistance to FLC induced by various immunizing procedures, the possibility that immunity is sustained by an unrecognized LLV infection should always be considered.

The strains of Friend leukemia complex (FLC) used in different laboratories may present dissimilarities, of which some (ability to infect and exert pathological effects in various mouse lines) are due to the passage history of the strains[2], and others (concerning the characteristics of the disease produced, such as production of splenic foci, induction of anemia or policytemia, tendency to regress) have less certain explanations and are attributed to variations in the relative proportions of the viruses forming the complex, to the presence of additional viral entities or to other factors[3–5]. Despite such dissimilarities, all FLC stocks must be assumed to contain a lymphatic leukemia virus (LLV) which is regarded as an indispensable helper of the FLC component responsable for the rapidly evolving hepatosplenomegaly with erythroleukemia characterizing Friend disease[6]. This assumption is not only justified by the nature of the interactions that are believed to occur between the FLC components, but finds experimental support in the fact that, whenever it has been looked for, LLV has invariably been detected[7–12]. LLV is in great excess over the other component(s) of the complex[7] and single infection of adult mice with LLV results in a chronic viremia with scanty signs (slight and transient hyperplasia of the spleen[7,13,14]) that can easily remain unrecognized. Thus some of the symptoms that are currently attributed to the entire FLC may in fact be due to LLV alone, and the presence of LLV should be constantly born in mind when the results of experiments with FLC are interpreted. Nevertheless, the multiviral nature of FLC has received adequate consideration in certain areas of research, signally in the genetics of host susceptibility[2], but not in others. For istance, the role of LLV in the immunodepressive properties of FLC has only recently received recognition[15]. A field that requires particular awareness of the complexity of FLC is the study of immune responses and of ways of inducing immunity to FLC, because mice infected with LLV alone become strongly resistant in a few days to FLC and to FLC-transformed cells[7,11,16], develope circulating antibodies

LLV infection and resistance to FLC in mice injected with Con A-treated FLC

Treatment	Presence of LLV in blood at day 0	Spleen weight at day +21
Con A-FLC at day –60, FLC at day 0	yes (8)	198 ± 57**
	no (10)	2121 ± 318
Con A at day –60, FLC at day 0	no (12)	2324 ± 513

*Number of mice in the group. **Mean ± SD.

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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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