DAVIS and REBERT[10] continued the electrophysiological studies on the antennal chemoreceptors of *A. aegypti* initiated by LACHER, using both his microscopic setup (Leitz Laborlux with Ultropak Illuminator, X700) and his classification of the antennal sensilla. It became apparent during these subsequent studies that further clarification of the nomenclature used to identify the antennal sensilla was needed.

Positive identification of single antennal sensilla was achieved by the following method. Immediately after recording the electrophysiological responses to chemical stimulation, the sensillum was marked by enlarging the hole made by the recording electrode, and its location on the antenna was mapped. When the electrophysiological experiments on a given mosquito were finished and any other sensilla were similarily marked and mapped, the antennae were prepared for examination with a scanning electron microscope (SEM). One of the 39 sensilla examined in this manner is shown in the Figure.

A scanning electron micrograph of a sensillum basiconicum (A3) on segment 9 of the antenna of a female *A. aegypti* mosquito, showing the hole left after the removal of the recording electrode.

The results of the correlation of the electrophysiological responses to chemical stimulation with the morphology of individual chemoreceptor sensilla are as follows. The electrophysiological responses of an 'A4' sensillum[5] can only be obtained from one morphological type sensillum – the sensillum basiconicum – referred to by others as an A3. The electrophysiological responses from the sensillum referred to by LACHER[11] and DAVIS and REBERT[10] as an 'A3' can also be obtained from one of the A2 type sensilla – the A2-M. SEM examination of these two sensilla revealed that both are sensilla trichodea and are morphologically indistinguishable from one another. Thus, with a lack of separable physiological and morphological features, it would seem that the 'A3' and the A2-M are in fact the same sensillum type. No other sensilla were observed on the 17 antennae examined with the SEM that do not fit into the categories described earlier, i.e., a long sensillum trichodeum (A1), 3 types of shorter sensilla trichodea (A2 family), and sensilla basiconica (A3).

This reclassification of the morphological types of antennal chemoreceptors of *A. aegypti* is now in agreement with the similar identification of these receptors by other investigators. It should be noted that the electrophysiological responses of the various chemoreceptor sensilla to chemical stimulation previously reported by LACHER[5,11] and DAVIS and REBERT[10] are not in question here, only their identification of the proper sensilla from which the responses were obtained. This paper is intended to correct that error[12].

Résumé. Deux corrections sont faites dans la classification des chémorécépteurs antennaires de la femelle du moustique *Aedes aegypti*. Le sensillum «A4» est classi comme sensillum basiconicum (A3), et l'«A3» de LACHER[11], et DAVIS et REBERT[10] comme sensillum trichodeum (type A2). Les résultats électrophysiologiques antérieurs ne sont pas contestés.

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Peripheral Neuropathy Associated with Inhalation of Methyl-*n*-Butyl Ketone

Methyl-*n*-butyl ketone (MnBK) is a solvent used extensively in vinyl and acrylic coatings and adhesives. There was recently an outbreak of peripheral neuritis in a Columbus, Ohio, concern using ink to print fabric. MnBK was suspected to be responsible for this outbreak and the following experiments were devised to investigate the effects of inhalation of MnBK by rats.

Nine rats were exposed to 200 ppm MnBK (by volume) for 6 weeks, 5 days per week, 8 h per day. 8 other rats were exposed to a mixture of 200 ppm MnBK and 2000 ppm methyl-ethyl-ketone (MEK), for a similar period; 3 rats died during this experiment. 4 rats were used as controls. The rats exposed to MnBK alone, presented with a muscular weakness of all limbs which lasted a few hours after the experiment each night. The animals exposed to the MnBK-MEK mixture were similarly weak and the recovery time took at least 24 h.

The rats were killed with ether and perfused with cold 10% formalin, buffered with 0.1 *M* sodium phosphate, pH 7.2. The sciatic nerves of all the rats were removed and examined. The following histological methods were used to examine the nerves, hematoxylin-eosin, Holmes

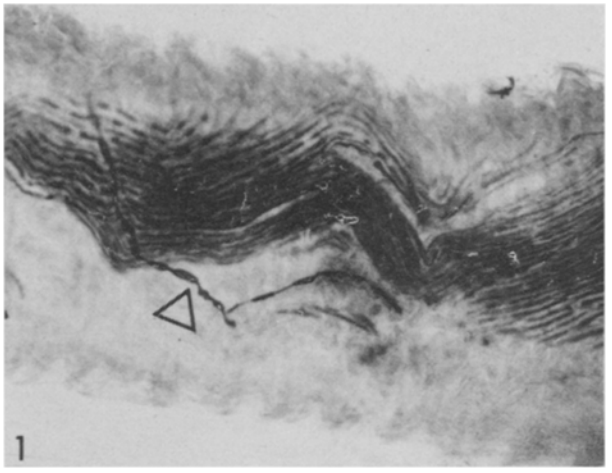

Fig. 1. Axonal beading in the sciatic nerve (Holmes silver). Low magnification.

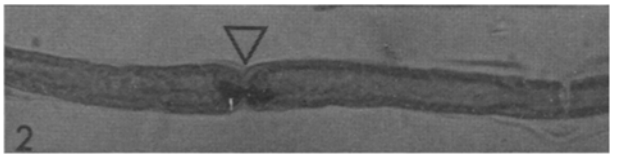

Fig. 2. Perinodal myelin breakdown (arrow). Marchi. Glycerin dissection.

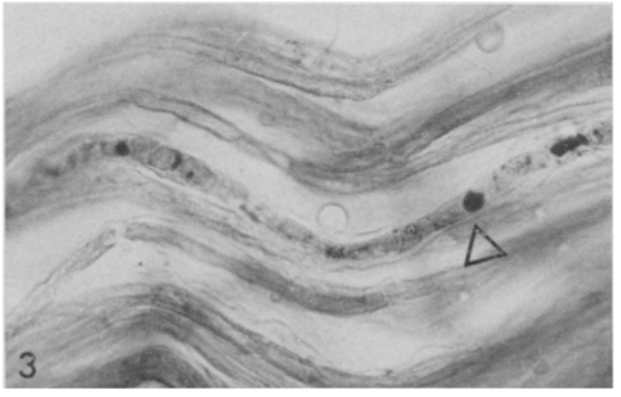

Fig. 3. Paranodal myelin breakdown (arrow). Marchi. Glycerin dissection.

silver, Nauta-Gyggax, luxol-fast-blue, PAS, oil-red-O. The Marchi preparations were dissected in glycerin. The tissues were examined with a polarizing microscope.

The histological examination of the sciatic nerves showed that in all the rats, except the controls, there was axonal hypertrophy, beading and degeneration (Holmes silver, Nauta-Gyggax), associated with widespread perinodal and segmental breakdown of myelin (Marchi, ORo, Polarizing scope) (Figures 1–3). This picture is interpreted as representing early changes of a neuropathy, which is primarily axonal with secondary myelin breakdown. The cause of the neuropathy is MnBK inhalation and the possible role, if any, of MEK in the second group of rats is now being investigated.

Résumé. Dix-sept rats sont exposés pendant 6 semaines 5 jours par semaine, 8 h par jour, à une atmosphère contenant soit du méthyl-*n*-butyl kétone seul soit un mélange de MnBK et de méthyl-éthyl kétone. Tous les rats présentèrent une faiblesse musculaire généralisée après l'inhalation qui dura de quelques h à 24 h avec récupération motrice totale. En dépit de cette apparence normale, l'examen histologique révéla une hypertrophie, un ballonement en grains de chapelet, et une dégénérescence des axones, associée à une démyélination secondaire, habituellement située dans la région des nodes de Ranvier. La toxicité du MnBK est prouvée, celle du MEK est encore à l'étude.

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Molecular Coding of Maze Learning; Demonstration by Bioassay

Since 1965 an increasing number of publications have reported detection of behavior-inducing substances in the brain of trained animals by means of behavioral bioassays[1–5] and two of the active substances have been isolated and identified[6,7]. Although evidence has been produced for the specificity of the method[8–11], it still remains controversial. The experiments reported in this paper bring further support to the validity and specificity of the behavioral bioassay.

Experiment I. The first series of 3 experiments, done at Baylor College of Medicine, used a maze (Figure 1, bottom) consisting of a white plastic outer shell into which a balsa-wood system of partitions could be inserted. Swiss albino mice (male, 25 g), water deprived 48 h prior to the first training session, were trained to run the maze (up to a maximum of 5 min) until they reached the water cup and drank for a few sec. With 1 daily training session, the

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