these viruses are related to one another. Similar data have also been obtained by comparing the gs proteins of woolly monkey, gibbon, and RD-114 type C viruses (*16*). Although RD-114 satisfies the morphological and immunological criteria for classification as a type C virus (*13*), it is not yet possible to assign its species of origin. Additional isolates from other primates, including man, will be necessary to establish the generality of a primate type C virus group based on the antigenic properties of the reverse transcriptase.

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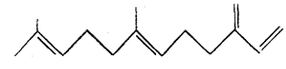
Aphid Alarm Pheromone: Isolation, Identification, Synthesis

Abstract. *A broadly interspecific aphid alarm pheromone was isolated from several economically important species of aphids and identified as* trans-*β-farnesene.*

Aphids are repelled by the odor of droplets released from their cornicles (*1*). When an aphid is attacked by an insect predator, these droplets are produced causing nearby aphids to escape or drop from their feeding sites on plants (*2*). Such a repellant substance is therefore defined as an alarm pheromone (*3*).

Aphids were homogenized in a Ten Broeck tissue homogenizer in a mixture of diethyl ether and methanol (3 : 1, by volume), the homogenate was filtered, and the filtrate was dried over anhydrous sodium sulfate. After evaporation of the solvent in vacuo, the residue was dissolved in hexane and chromatographed on a Florisil column. Only the hexane eluate (hydrocarbon fraction) showed biological activity. The hydrocarbons were further separated by column chromatography over Florisil impregnated with silver nitrate, and eluted with increasing concentrations of diethyl ether in hexane. Biological activity was recovered in the fraction eluted with 6 percent ether in hexane. Comparison of the chromatographic mobility of the active material with that of multiply unsaturated hydrocarbon standards indicated the presence of a compound containing three to four olefinic double bonds. The active fraction displayed two prominent peaks by gas-liquid chromatographic analysis on Carbowax 20-M. Biological activity was recovered from the gas chromatograph by trapping the effluent in capillaries chilled in Dry Ice. One peak contained the alarm pheromone, which was then examined by combined gas chromatography–mass spectrometry (*4*). The pheromone had a molecular ion of 204, which, on the basis of an entirely hydrocarbon composition, was equivalent to the molecular formula $C_{15}H_{24}$ with four degrees of unsaturation, and which, in turn, suggested a farnesene-type hydrocarbon. *trans,trans-α*-Farnesene has been identified from Dufour's gland in ants (*5*). To investigate the possibility of a similar compound in aphids, the more readily prepared *trans-β*-farnesene was synthesized by heating 10 g of nerolidol in 100 ml of dimethyl sulfoxide in a glass-lined reaction bomb at 150°C for 20 hours (*6*). The reaction yielded 74 percent of hydrocarbon products, from which *trans-β*-farnesene was isolated by preparative gas chromatography. The structure was authenticated by comparison of its infrared, ultraviolet, and nuclear magnetic resonance spectra with reported values (*7*).



trans-β-Farnesene

The retention times on apolar (SE-30, XE-60) and polar phases (Carbowax 20-M) in gas-chromatographic analysis were identical to those for the aphid alarm pheromone.

The mass spectra of synthetic *trans-β*-farnesene and the aphid alarm pheromone were identical not only in the intensities of all the ions, but also with respect to the shapes and intensities of the metastable ions, especially those at *m/e* (mass to charge) 24.3, 37.1, 63.8, 89.0, and 127.0.

Further proof of identity was obtained by treating portions of aphid pheromone and the synthetic *trans-β*-farnesene with one molar equivalent of *m*-chloroperoxybenzoic acid in dichloromethane solution. Both reacted to give an epoxide with identical retention times on polar and apolar phases in gas chromatography. The mass spectra reveal a monoepoxide (parent ion *m/e* 220) in which the intensity of all the ions, and especially the metastable ions at *m/e* 63.1, 63.9, 77.2, and 88.8, are identical in shape and intensity.

Finally, biological assay of the synthetic *trans-β*-farnesene against ten species of aphids indicated complete biological activity (*8*).

trans-β-Farnesene was identified as the alarm pheromone by the above

chemical and spectroscopic techniques in the rose, pea, greenbug, and cotton aphids (*9*). The effectiveness of *trans*-β-farnesene against six other aphid species (*10*) indicates that this compound is perhaps the most interspecifically active pheromone to be discovered.

Since exposure to the alarm pheromone causes aphids to escape rapidly from their feeding sites, especially by dropping from the host plant, an interesting and novel method of insect control may be realized.

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9. Rose aphid, *Macrosiphum rosae* (L.); pea aphid, *Acyrthosiphon pisum* (Harris); greenbug aphid, *Schizaphis graminum* (Rondani); and cotton aphid, *Aphis gossypii* Glover.
10. Potato aphid, *Macrosiphum euphorbiae* (Thomas); English grain aphid, *Macrosiphum avenae* (Fabr); foxglove aphid, *Acyrthosiphon solani* (Koltenbach); green peach aphid, *Myzus persicae* (Sulzer); corn leaf aphid, *Rhopalosiphium maidis* (Fitch); and oat birdcherry aphid, *Rhopalosiphum padi* (L.).

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Relation between REM Sleep and Intracranial Self-Stimulation

Abstract. *Depriving rats of rapid eye movement (REM) sleep was shown to lower their thresholds and raise their response rates for rewarding brain stimulation. Conversely, allowing rats to self-stimulate while they were being deprived of this sleep form reduced the amount of REM rebound during recovery from deprivation. These results demonstrate a reciprocal relation between rewarding brain stimulation and REM sleep.*

Although traditionally sleep has been thought of as a state of relative quiescence, at least one type of sleep, rapid eye movement (REM) sleep, is a state of extreme activation of the central nervous system (*1*). Dement and others (*2*, *3*) have shown that there is a lowering of threshold of many neural and behavioral activities following REM deprivation (*3*). Their data suggest a generalized neural hyperexcitability after REM deprivation, which in turn is hypothesized to be reflected in heightened behavioral activity.

In the first experiment, we chose to look at the effect of REM deprivation on positively reinforcing electrical brain stimulation, also called intracranial self-stimulation (ICSS) (*4*). This is of particular interest in that ICSS measures activity of the central nervous system (CNS) through behavioral methods. In addition, some of the "drive" behaviors that Dement and his associates (*3*) have elicited as a result of REM deprivation have also been elicited from hypothalamic regions that are ICSS areas (*5*). Our specific prediction in the first experiment was that thresholds for ICSS would be lowered following REM deprivation. In other terms, we predicted that subjects would be more sensitive to ICSS following REM deprivation.

To test this hypothesis, 12 male albino Holtzman rats weighing approximately 250 grams at the start of the study were used as subjects. Two pairs of insulated stainless steel electrodes were implanted in each rat with the use of a stereotaxic device while the animal was under sodium pentobarbital and chloral hydrate anesthesia. One pair of electrodes was aimed at the medial forebrain bundle, a structure yielding strong self-stimulation effects. A second pair recorded activity from the dorsal hippocampus. Two cortical screws were implanted to record from the frontal and occipital cortex. After completion of the experiment, animals were killed by perfusion. Histological sections stained according to the Luxol fast blue and cresyl violet method were made and confirmed the electrode placements.

Electrical brain stimulation consisted of biphasic rectangular waves. Each pulse pair consisted of two 0.2-msec waves, opposite in polarity and separated by an interval of 0.2 msec. Pulse pairs were delivered at a frequency of 100 per second, and train duration was held constant at 0.25 sec. Current was varied according to the demands of the experiment and was continuously monitored on an oscilliscope.

Subjects were taught to self-stimulate; ICSS was delivered when the subject pressed a lever in an experimental chamber. The range of current intensities that would support responding on a continuous reinforcement schedule was explored, and rate-intensity functions (*6*) were collected for 10 days. Then, ascending rate-intensity functions were collected during 1-hour sessions conducted at the same time during each of 4 consecutive days (baseline data). Immediately after the 1-hour session of the 4th day, six subjects (REM deprivation group) were deprived of REM by the platform method (*7*), and six subjects were placed on a larger platform (13 cm) and used as a yoked control group (*8*). The REM deprivation and yoked control subjects were on their respective platforms approximately 23 hours. After this period, all subjects were placed in a self-stimulation test chamber, and ascending rate-intensity functions were again obtained. The cycle of 23 hours on the platform followed by 1 hour of ICSS testing was continued for 4 days. After the 4 days of REM deprivation, subjects were returned to their home cages, and rate-intensity functions were again ascertained each day for an additional 4 days (recovery period). Following a 2-week rest period, four subjects that originally were in the REM deprivation group were placed on the large platform and used as a nonyoked control group (*8*).

In general, both control groups showed a lowering of response rates and a rise in ICSS threshold, while the REM deprivation group showed the opposite, increased response rates and lowered ICSS threshold. In all respects, these control animals were treated identically to animals that were on the REM deprivation platform, and both yoked and nonyoked controls fell off