a trail pheromone laid down by the scouts might function as the principal orientation cue which leads to the battleground.

The following findings support this hypothesis. While a raid organized by scout ants was in full progress (that is, once the ants had become initially aroused, presumably by the jerking behavior of the recruiting ants), I drew artificial trails intersecting the natural trail at 90° to it. The raiding ants did not respond to artificial trails laid with extracts from poison glands or Dufour's glands, but a significant number of workers followed the trail laid with hindgut extract. The territorial raids of *M. mimicus* thus seem to be organized by an alarm-recruitment system. Behavior observations suggest that the ants are alerted by a jerking motor display performed by the recruiting scouts. Experimental results support the hypothesis that they are subsequently guided to the combat area by a hindgut pheromone trail laid by the scout ants. Some ant species employ an elaborate alarm recruitment system in defense against particular interspecific enemies (*3*). I demonstrate here that a similar system can also be used in intraspecific territorial defense (*4*).

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References and Notes

1. The distinctions between ritualized and escalated aggression and between tournaments and displays within the category of ritualized aggression have been examined by J. Maynard Smith and G. R. Price [*Nature (London)* **246**, 15 (1973)] and J. Maynard Smith [*J. Theoret. Biol.* **47**, 209 (1975)].
2. For a review of the general biology of honeypot ants, see E. O. Wilson, *The Insect Societies* (Harvard Univ. Press, Cambridge, Mass., 1971).
3. E. O. Wilson, *Science* **190**, 798 (1975).
4. A fuller account of the pheromone assay and other aspects of the alarm-recruitment response is in preparation.
5. Supported by grant BMS-75-06447 from the National Science Foundation. I thank Dr. R. Snelling for species identifications and T. Hölldobler for the photographs.

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Hippocampal Activity and Scopolamine

The report by Teitelbaum *et al.* (*1*) does not account for previous data which seem to invalidate their conclusion that behavior-related hippocampal rhythmical slow activity is abolished by injections of scopolamine. In a previous report (*2*) it was shown that hippocampal rhythmical slow activity persists during locomotion after a dose of scopolamine HBr of 10 mg/kg intraperitoneally. Teitelbaum *et al.* used a dose of 10 mg of scopolamine HCl per kilogram. The slight difference in the amount of base given by these two dosages is probably unimportant. The effects of atropinic drugs on hippocampal activity were clarified in another report (*3*) which concluded, in part, that "Preservation of reasonably clear RSA [rhythmical slow activity] during gross movements following atropinization could be demonstrated *only* at sites where the RSA, in the absence of any drug, had a large amplitude (preferably 1 mv or more) and little admixture of fast wave activity. This point requires emphasis since there have been reports, based on recordings lacking these features, that atropinic drugs essentially abolish RSA during behavior." Apparently I did not emphasize the point sufficiently, even though it was illustrated by a figure [figure 4 in (*3*)]. The tracings shown by Teitelbaum *et al.* contain considerable fast activity and have an amplitude of about 0.5 mv. Such tracings may continue to exhibit rhythmical slow activity after injection of atropinic drugs (*2*) but usually they do not.

There are two further points. First, despite the assertion by Teitelbaum *et al.* (*1*, p. 1115), I have not claimed a relation between atropine-sensitive rhythmical slow activity and "behavioral arousal." As I point out (*3*), hippocampal activity of this type can occur during surgical anesthesia, and its behavioral significance is largely unknown. Second, hippocampal rhythmical slow activity is present not only during the initiation of voluntary movement but also during its long-continued performance, for example during running in a wheel or walking on a treadmill (*4*).

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References

1. H. Teitelbaum, J. F. Lee, J. N. Johannessen, *Science* **188**, 1114 (1975).
2. C. H. Vanderwolf, B. H. Bland, I. Q. Whishaw, in *Efferent Organization and the Integration of Behavior*, J. D. Maser, Ed. (Academic Press, New York, 1973), p. 229.
3. C. H. Vanderwolf, *J. Comp. Physiol. Psychol.* **88**, 300 (1975).
4. B. H. Bland and C. H. Vanderwolf, *Brain Res.* **43**, 67 (1972); I. Q. Whishaw, *Can. J. Psychol.* **26**, 219 (1972); _____ and C. H. Vanderwolf, *Behav. Biol.* **8**, 461 (1973).

19 June 1975

While our conclusions (*1*) were not based on scopolamine data alone, our findings with regard to the effects of this drug on hippocampal theta patterns are in agreement with those of Bennett (*2*) and Friedman and Wikler (*3*).

We were able to mimic the movement-related theta response with physostigmine and we showed that the same dose of scopolamine that blocked physostigmine-induced theta also blocked theta produced by treadmill running without affecting the rate or pattern of movement. Both the physostigmine response and the behaviorally induced hippocampal theta response were abolished with lesions of the medial septal nucleus.

The negative findings of Vanderwolf (*4*) with regard to the effects of muscarinic blocking agents were discussed in detail in our report (*1*). Apparently, Vanderwolf has seen this effect in his laboratory (as indicated above). Because our signal was a mere 500 μv in amplitude and contained "considerable" [we disagree, see figure 1 in (*1*)] high-frequency activity, he has arbitrarily decided to ignore such data. We are in no position to make such a decision—nor is he.

Winson (*5*) and Fox and Ranck (*6*) have provided evidence for the existence of at least two (possibly three) theta-generating layers in the dorsal hippocampus of the rat, one located high in CA_1 (the location of our recording electrodes), another located more ventrally in the dentate gyrus, and possibly a third in the supra-pyramidal layer of CA_3. With their staggered bipolar electrode configuration, Vanderwolf *et al.* (*4, 7*) are recording differentially between two neurochemically distinct movement-related theta generators that are synchronous but phase reversed [see Winston (*5*) for a description of phase relationships between theta generators in hippocampus]. In recording such bioelectric activity from freely moving animals, movement artifacts and electrical noise are reduced with employment of differential amplifiers that have common mode rejection characteristics. According to Giles (*8*), the output voltage

$$E_{out} = (R_f/R_s)(E_{in_2} - E_{in_1})$$

where R_f = resistance in the feedback circuit and R_s = resistance of source; E_{in_2} is the peak potential at one electrode (for example, dorsal theta generator) and E_{in_1} is the peak potential at the other source (for example, ventral generator). Since E_{in_1} and E_{in_2} are synchronous, but phase reversed, E_{in_1} is a negative value that is multiplied by a

negative. In such a situation E_{in_1} and E_{in_2} would summate so that $E_{out} = (R_f/R_s)(E_{in_1} + E_{in_2})$.

Instead of recording a slow wave of 500 μv generated from one source, the potentials from the frequency-coupled sources would add, giving Vanderwolf's gargantuan (1000 μv) rhythmic slow pattern that cannot be blocked by any one specific neurotransmitter antagonist or blocking agent (*4*, *7*).

Of particular relevance are the recent findings of Bird and Aghajanian (*9*) showing that microiontophoretic application of scopolamine and quinuclidinyl benzilate (QNB) blocked the excitation of hippocampal pyramidal cells by acetylcholine in the rat. We have shown that the dorsal movement-related theta generator is muscarinic (*1*), and Winson (*5*) provides evidence that the ventral theta generator may be nicotinic.

We suggest that Vanderwolf consider changing his electrode configuration so that he can record from one neurochemically distinct system at a time.

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References

1. H. Teitelbaum, J. F. Lee, J. N. Johannessen, *Science* **188**, 1114 (1975).
2. T. L. Bennett, *Behav. Biol.* **9**, 541 (1973).
3. M. J. Friedman and A. Wikler, *Psychopharmacologia* **17**, 345 (1970).
4. C. H. Vanderwolf, *J. Comp. Physiol. Psychol.* **88**, 300 (1975).
5. J. Winson, *Brain Res.* **103**, 57 (1976).
6. S. E. Fox and J. B. Ranck, Jr., *Exp. Neurol.* **49**, 299 (1975).
7. T. E. Robinson, B. A. Pappas, C. H. Vanderwolf, *Neurosci. Abstr.* **1**, 333 (1975).
8. J. H. Giles, *Semiconductor Linear Integrated Circuits Applications Handbook* (Fairchild, Mountain View, Calif., 1967), pp. 19–20.
9. S. J. Bird and G. K. Aghajanian, *Neurosci. Abstr.* **1**, 387 (1975).

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Immunosurveillance of Naturally Occurring Feline Leukemia

Essex *et al.* (*1*) have advanced epidemiological data "supporting the concept of immunosurveillance in an outbred species of mammal," the cat.

That their results "clearly indicate that a low FOCMA [feline oncornavirus-associated cell membrane antigen] antibody titer in the presence of FeLV [feline leukemia virus] positivity is a risk indicator for leukemia, while high antibody titers are associated with resistance to leukemia development" seems established by their data. I also agree that the data are consistent with the immunological surveillance concept; however, the data are also consistent with alternative concepts which, in my opinion, need more emphasis.

The prospective study does not, of course, rigorously rule out the possibility that the tumor was the cause rather than the result of low antibody titers since it is impossible to know how long before overt disease the pathological process may have started. Perhaps more likely, however, is the hypothesis that the relation between antibody level and neoplasia need not be etiological, regardless of which came first; rather, both may be the result of an underlying immunological abnormality. The scenario could be something like the following: Some cats, for unknown reasons, do not respond to virus with normal levels of antibody to virus. Leukocytes of the antibody-producing mechanism are therefore stimulated by the feedback controls that regulate antibody production. This high level of stimulation eventually results in neoplasia in the stimulated leukocytes (*2*). The mechanism would, in principle, be analogous to the production of ovarian tumors by excess gonadotrophin in the rodent ovary transplanted into the portal circulation of an ovariectomized recipient (*3*).

The above possibility makes it extremely difficult, in my opinion, to draw conclusions concerning the immunological surveillance concept from studies of tumors derived from cells of the immune mechanism itself.

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References

1. M. Essex, A. Sliski, S. M. Cotter, R. M. Jakowski, W. D. Hardy, Jr., *Science* **190**, 790 (1975).
2. R. S. Schwartz, *N. Engl. J. Med.* **293**, 181 (1975).
3. G. R. Biskind and M. S. Biskind, *Am. J. Clin. Pathol.* **19**, 501 (1949).

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