lated to produce *S. richteri* hydrocarbons when in contact with the host. It is also evident that cuticular components are in a continuous state of flux.

If the host hydrocarbons are acquired, then this multiple-host myrmecophile should be able to change its hydrocarbon pattern to match that of the host species. To test this hypothesis, we collected beetles from *S. richteri* colonies, isolated them for 2 weeks, and then introduced them into laboratory colonies of *S. invicta*. After 5 days, the beetles were removed and analyzed for cuticular hydrocarbons as described. The data (Fig. 2, A and B) show that the *M. excavaticollis* taken from *S. richteri* colonies acquired the cuticular hydrocarbons of its new host, *S. invicta*. The same phenomenon occurred when previously isolated beetles were introduced into *S. geminata* and *S. xyloni* colonies. Although the switching of hydrocarbon patterns from one host to another weakens the likelihood that they are synthesized by the beetle, we also found that freshly killed isolated beetles had acquired *S. invicta* hydrocarbons within 2 days after exposure to the ant colony. These data eliminate biosynthesis as a possibility and support a passive mechanism of hydrocarbon acquisition. When initially introduced into a host colony, the *M. excavaticollis* were immediately attacked. The response of the beetles was to play dead and wait for the attacks to cease, or they moved to an area less accessible to the ants. Within 2 hours after introduction into a host colony, the beetles' cuticle contained 15 percent of host hydrocarbons. The accumulation of hydrocarbon continued up to 4 days until the beetles' cuticle contained about 50 percent host hydrocarbons. Beetles surviving this long were generally no longer attacked.

The beetles can acquire the host cuticular hydrocarbons by ant-beetle contact, grooming behavior, regurgitation of ant postpharyngeal gland contents (which contain large amounts of species-specific hydrocarbons), and by ingestion. However, the overall mechanism used for integration of *M. excavaticollis* into its host colonies involves the initial passive defensive behavior that must enable it to survive long enough to acquire the species odor of its host as well as the environmental part of the host's colony odor.

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Lesion-Induced Sprouting in the Rat Dentate Gyrus Is Inhibited by Repeated Ethanol Administration

Abstract. *The effect of ethanol on hippocampal axonal sprouting was studied with a histochemical technique for identifying acetylcholinesterase. Unilateral lesion of the entorhinal cortex in adult rats produced an increase in the density of acetylcholinesterase staining in the outer molecular layer and a concomitant increase in the width of the pale-staining commissural-associational zone of the dentate gyrus. Other rats were given ethanol (11.3 ± 0.45 grams per kilogram) for 2 weeks before and 9 days after receiving the lesion. Ethanol abolished the expansion of the commissural-associational zone. The effect of ethanol on sprouting axons suggests that it may inhibit recovery of function after brain injury.*

Each year about 297,000 persons are hospitalized for initial stroke (*1*) and another 422,000 for head injury (*2*). Ethanol is a significant factor associated with central nervous system dysfunction and recovery (*3–8*). Since about 9 percent of Americans are heavy drinkers (*9*), there is a compelling need to understand the effects of ethanol on recovery from injury to the central nervous system.

The behavioral deficits that follow brain injury often show some recovery with time. Reactive synaptogenesis (axon sprouting) has been proposed as one mechanism underlying such recovery (*10*). The dentate gyrus is ideal for determining the effects of ethanol on lesion-induced sprouting. The precision with which afferents of the dentate gyrus are organized into laminar terminal fields has permitted accurate documentation of the changes that follow brain lesions. After infliction of an entorhinal cortical lesion (*11, 12*), most of the remaining afferents of the dentate gyrus sprout into the deafferented area of the molecular layer. Some of these afferents have been shown to form functional connections (*13, 14*).

Alterations in the cholinergic innervation of the molecular layer can easily be monitored by acetylcholinesterase (AChE) histochemistry. Within 4 to 6 days after infliction of an entorhinal lesion, a marked intensification of AChE staining is observed throughout the outer molecular layer. Biochemical studies have demonstrated increased activity of AChE and choline acetyltransferase following such lesions (*12, 15*). In addition, the pale-staining commissural-associational (CA) zone in the inner molecular layer widens and exhibits a concomitant decrease in the density of AChE staining. The expansion of the CA zone corresponds closely to the extent of commissural sprouting (*16*). A variety of studies recently provided evidence that long-term exposure to ethanol damages the hippocampal formation (*3, 17*). We now report that heavy consumption of ethanol inhibits the typical post-lesion sprouting response of the CA zone in the rat dentate gyrus.

Adult Sprague-Dawley rats of both sexes were maintained on a 12-hour light-dark cycle, with the period of light beginning at 700 hours. Nine days after unilateral lesion of the entorhinal cortex, the presence of axonal sprouting was determined by analyzing alterations in the laminated pattern of AChE staining in the molecular layer of the dentate gyrus. The ethanol-fed lesioned group

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(N = 10) consumed an average daily dosage of 11.3 ± 0.45 g of ethanol per kilogram as 35 percent of the total caloric intake in a liquid diet (*18*). The pair-fed lesioned control rats (N = 7) were individually matched with rats in the first group and pair-fed the same liquid diet, except that ethanol was replaced by an isocaloric amount of maltose and dextrin. The lesioned control group (N = 6) received a unilateral entorhinal cortex lesion. The normal control group (N = 6) received neither ethanol nor lesion. Both of the latter control groups were maintained on standard laboratory feed. The ethanol-fed lesioned rats were given ethanol for 2 weeks, lesioned, and continued on ethanol. At 9 days after infliction of the lesion, the brains were processed for histochemical identification of AChE (*18*). For each animal, the mean difference between the widths of the molecular layer, the outer and supragranular bands of AChE staining, and the CA zone were determined on both sides of sections from the dorsal hippocampal formation (*18*). There were no significant differences in the lesions, which included all the medial and lateral entorhinal cortices except at the most ventral levels.

Characteristic signs of sprouting were seen in the pair-fed lesioned control and lesioned control rats, which exhibited a marked expansion on the lesioned side of the CA zone compared to the side contralateral to the lesion. Moreover, the differences between the ipsilateral and contralateral sides of the CA zone were significantly larger in these two groups than in the normal control rats (Fig. 1 and Table 1). The borders of this pale-staining band were more distinct on the lesioned side than on the nonlesioned side, and were more distinct than those in normal control rats. This, coupled with an apparent retraction of AChE-positive fibers, caused the region to appear almost devoid of AChE staining.

In ethanol-fed lesioned rats the width of the CA zone on the lesioned side did not differ from the width of the CA zone on the nonlesioned side. In most cases these rats also exhibited smaller increases in the intensity of AChE staining in the outer zone than the other lesioned rats (Fig. 1). These findings indicate that ethanol abolished the lesion-induced expansion of the CA zone and reduced the other changes associated with lesion-induced sprouting. It will be important to examine the possible differential effects of ethanol during the intervals before and after lesion infliction.

Lesions of the entorhinal cortex normally induce expansion of the CA zone and a decrease in the density of AChE staining in the region. Moreover, increased AChE staining is observed in the outer molecular layer. These changes correspond to axonal sprouting of the CA fibers, axonal sprouting of the cholinergic fibers, and alterations in the spatial relation between CA and cholinergic fibers (*12, 19*). We observed similar results in our lesioned control and pair-fed lesioned control groups. However, the ethanol-fed lesioned group failed to exhibit the widening of the CA zone and the decrease in AChE staining density

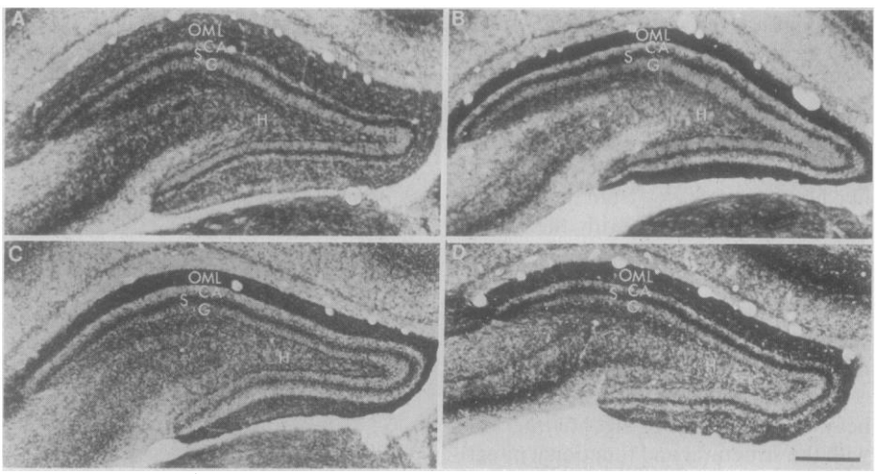

Fig. 1. Acetylcholinesterase staining in the dentate gyrus. (A) Normal control rat. (B) Lesioned control rat 9 days after receiving a unilateral lesion of the entorhinal cortex. (C) Pair-fed lesioned control rat 9 days after receiving the lesion. (D) Ethanol-fed lesioned rat 9 days after the lesion. Abbreviations: *CA*, commissural-associational zone; *G*, granule cell layer; *H*, hilus; *OML*, outer molecular layer; and *S*, supragranular layer. Scale bar, 500 μm.

Table 1. Effect of entorhinal cortex lesions and ethanol on the molecular layer of the rat dentate gyrus. The width of the molecular layer is equal to the sum of the widths of the outer zone, CA zone, and supragranular zone for each hippocampal section. Note that the differences between the ipsilateral and contralateral CA zone in the ethanol-fed lesioned group do not differ significantly from the differences in normal control rats ($.1 > P > .05$, Student's t-test), but do differ significantly from the differences in pair-fed lesioned rats ($P < .05$). Thus, ethanol abolished the width increase in the CA zone accompanying lesion-induced sprouting. Values are mean micrometers ± standard errors.

Group	Width, lesioned side	Width, nonlesioned side	Difference
	Molecular layer		
Ethanol-fed lesioned	160.1 ± 1.7	198.6 ± 1.8	−38.5 ± 1.5*†‡
Pair-fed lesioned	147.2 ± 1.9	185.6 ± 1.9	−38.4 ± 1.3*†
Lesioned control	148.1 ± 2.6	201.2 ± 2.4	−53.1 ± 2.6*
Normal control	196.7 ± 2.2	201.9 ± 1.9	−5.2 ± 2.1
	Outer zone		
Ethanol-fed lesioned	98.1 ± 1.6	139.8 ± 1.8	−41.7 ± 1.3*†‡
Pair-fed lesioned	88.0 ± 1.7	135.0 ± 2.3	−47.0 ± 1.5*†
Lesioned control	82.8 ± 2.2	141.0 ± 2.2	−58.2 ± 2.3*
Normal control	135.8 ± 2.2	140.1 ± 1.7	−4.3 ± 1.8
	CA zone		
Ethanol-fed lesioned	33.7 ± 0.57	33.8 ± 0.62	−0.13 ± 0.76
Pair-fed lesioned	37.4 ± 0.64	28.4 ± 0.72	+9.0 ± 0.95*†
Lesioned control	41.6 ± 0.89	33.5 ± 0.84	+8.0 ± 1.1*
Normal control	34.2 ± 0.79	33.6 ± 0.80	+0.63 ± 1.0
	Supragranular zone		
Ethanol-fed lesioned	26.9 ± 0.56	24.2 ± 0.46	+2.8 ± 0.67*†‡
Pair-fed lesioned	21.6 ± 0.55	19.8 ± 0.45	+1.8 ± 0.68*†
Lesioned control	22.8 ± 0.70	24.7 ± 0.61	−1.9 ± 0.79*
Normal control	25.1 ± 0.57	26.9 ± 0.63	−1.9 ± 0.78

*Effects of lesion: mean differences highly significant ($P < .05$) for ethanol-fed lesioned, pair-fed lesioned, and lesioned control rats compared to normal control rats (Newman-Keuls test). †Effects of liquid diet: mean differences highly significant ($P < .05$) for ethanol-fed lesioned and pair-fed lesioned rats compared to lesioned control rats. ‡Effects of ethanol: mean differences not significant ($.5 > P > .2$) for ethanol-fed lesioned rats compared to pair-fed lesioned rats.

observed in other lesioned groups (Fig. 1). Thus, ethanol abolished two parameters of lesion-induced axonal sprouting in the dentate gyrus. This could reflect inhibition of sprouting in the CA or septal cholinergic neuron systems to various degrees. Alternatively, sprouting of CA fibers may have occurred without being observable because the AChE-positive fibers did not retract from the CA zone. Indeed, the outer (AChE-positive) zone on the side of the lesion was larger in the ethanol-fed lesioned rats than in the pair-fed lesioned and lesioned control groups. However, the failure of the AChE-positive fibers to withdraw from the CA zone would be inconsistent with the critical afferent theory (*16*) of sprouting axons. Further experiments are required to determine which of these systems is most sensitive to the inhibitory effects of ethanol on axonal sprouting.

There is little doubt that prolonged heavy consumption of alcohol interferes with the structural and functional integrity of the brain. In rats, long-term exposure to ethanol results in loss of dendritic spines from neurons in the hippocampus and dentate gyrus (*3*). Moreover, ethanol exacerbates the destructive effects of cerebrospinal trauma in cats (*20*). Increased cerebral atrophy in alcoholic patients has been identified by computerized axial tomography (*4, 5, 21*), pneumoencephalography (*6*), measurement of brainstem-evoked potentials (*22*), and autopsy (*7*). We have now shown significant inhibition of axonal sprouting with daily exposure of rats to ethanol for 2 weeks before and 9 days after an entorhinal lesion. High levels of ethanol may exert a toxic effect on sprouting axons.

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18. Rats in the ethanol-fed lesioned group were given unlimited access to ethanol in a liquid diet (Bio-Serv). This diet, which was the sole source of food and water, was imbibed through calibrated drinking tubes. The individually housed rats were fed the ethanol-containing diet for 2 weeks. After receiving unilateral electrolytic lesions (*14*), the rats were allowed to recover while receiving the liquid diet. Body weight dropped slightly in the ethanol-fed and pair-fed lesioned groups when the liquid diet was started, but was not significantly different on the day of the lesion. The rats were killed 9 days after surgery and their brains were processed for AChE histochemistry by a modification of the method of F. A. Geneser-Jensen and T. W. Blackstad [*Z. Zellforsch.* **114**, 460 (1971)]. For each animal the section used for analysis was taken at the point along the septotemporal axis of the dorsal hippocampal formation where the dorsal and ventral limbs of the dentate gyrus first become continuous. Each section was projected and a MOP-AMO-3 digitizer (Zeiss) was used to measure the widths of each zone in the molecular layer at 15 randomly selected points along the dorsal limb of the dentate gyrus. The measurements for each group were pooled and the mean width of each layer was computed (Table 1). The minimum number of samples required per section (*15*) was determined by the technique of progressive means (M. A. Williams, *Quantitative Methods in Biology* (Elsevier/North-Holland, Amsterdam, 1977).
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Synchronous Neural Afterdischarges in Rat Hippocampal Slices Without Active Chemical Synapses

Abstract. *Extracellular field potential and intracellular recordings from neurons in rat hippocampus show that, even with synaptic transmission blocked, antidromic electrical stimuli can trigger afterdischarges of up to 9 seconds duration; during these discharges action potentials of a single neuron were synchronized with extracellularly recorded population spikes. Apparently mechanisms other than recurrent chemical synapses can synchronize and recruit epileptiform events. Measurements of transmembrane potential indicate that transient extracellular electrical fields (ephaptic interactions) contribute to the observed synchrony; electrotonic coupling and changes in the concentration of extracellular ions may also contribute.*

During seizures, bursts of action potentials and membrane depolarizations are abnormally synchronized across large populations of cortical neurons. The electrical properties of individual neurons that appear epileptic have been investigated (*1, 2*), but the mechanisms that synchronize epileptiform discharges and cause their local spread are not known. Seizures can spread from one area of the brain to a remote region through projection pathways involving chemical synapses (*3*). Some investigators (*1, 2, 4–6*) have suggested that chemical synapses also locally synchronize and propagate epileptiform activity, especially by recurrent excitation. However, other local synchronizing mechanisms may include electrotonic synapses (*5, 7–9*), electrical field effects (ephaptic interactions) (*10*), and changes in concentration of extracellular K^+ and Ca^{2+} (*1, 11, 12*). We investigated the possibility that synchronized epileptiform events can occur when chemical synaptic transmission is inoperative.

Extracellular and intracellular recordings of CA1 pyramidal cells were made by conventional techniques (*13*). Transverse slices (450 μm thick) of freshly

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