Separate analysis of diastole-triggered EVR's for the REG, however, yielded a statistically significant difference between the prestimulus and poststimulus segments (Fig. 2). Although 87.5 percent of the waves were classified accurately [$F(7, 8) = 3.92$, $P < 0.05$], only 68 percent of the stimulus and prestimulus waves were classified accurately during diastolic phase for blood volume responses of the arm. Stimulation during systole was not significantly discriminated from the prestimulus wave with any analysis.

To our knowledge, this is the first report of transient, rapid changes in a measure of the cerebral vasculature from conscious human subjects. This rapid EVR was evident only when stimulation was synchronized with the diastolic phase of the ophthalmic artery. Although the response was observed in the periphery, it did not occur reliably. The latency of the EVR defies the time course related to previously described vascular changes in response to altered metabolic activity. Thus, this response may be a neurogenically mediated vascular event in preparation for altered metabolic demand.

Enhancement of the EVR during diastole is consistent with previous reports with the ERP (*16*). Auditory stimulation synchronized with diastolic phase resulted in a 10 to 20 percent augmentation of the attentional (N1) component of the ERP. Our results suggest that rapid changes in blood volume occur according to a similar schedule and are of similar proportion (*19*). The temporal similarities between response of the cerebral vasculature and the electrical response of the brain suggest common generating mechanisms. For instance, decreased baroreceptor activity (as during diastole) is associated with increased cerebral blood flow and release from neuronal inhibiton (*13*). Our results suggest that stimulation during diastole can evoke rapid changes in cerebral vascular events and augment the ERP. The EVR may prove to be a neurally mediated phenomenon that not only yields clinically relevant information regarding the integrity of vasomotor systems but also may provide new information of brain metabolism related to detecting and processing external information in real time.

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16. A major difference among these methods is that only relative measurements are obtained with the REG, although it has the advantage of providing dynamic rather than static indices of volume. Since rapid changes in volume were of interest, the relativity of the measurement was not a problem.
17. Photoplethysmography used a reflective transducer, which combines an infrared light-emitting diode and a silicon phototransistor.
18. Tone presentation as well as data collection epochs were initiated when the amplitude-calibrated ophthalmic photoplethysmograph reached 75 percent of its maximum amplitude in either the "ascending" systolic phase or the "descending" diastolic phase. Analysis of simultaneously synchronized data collection sequences began approximately 270 msec after the R wave of the electrocardiogram, and the diastolically synchronized sequence began approximately 480 msec after the R wave.
19. It is not possible to separate the contribution of blood volume and blood flow with this procedure. Even though these two measures are related, it would be of interest to dissociate them. Our data indicate that external stimulation is related to perturbation in the cerebral vasculature. The differential contribution of volume and flow of capillaries, arterioles, venules, and major veins is not known. S. A. Radvan-Ziemnowiez [*Methods in Psychophysiology* (Williams & Wilkins, Baltimore, 1967), pp. 129–157] suggested that the REG represents blood flow contributed by the internal carotid artery. Studies of localization and of patients with known pathologies are needed to clarify this important point.
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Predator-Induced Defense in a Marine Bryozoan

Abstract. *Laboratory experiments showed that predation by both trophically specialized and generalized nudibranch species triggers rapid induction of defensive spines in the bryozoan* Membranipora membranacea. *Spines effectively control the pattern and extent of intracolony mortality caused by nudibranch predation. Previously found only in plants, rotifers, and cladocerans, consumer-induced defenses may be widespread among clone-forming or colonial taxa exposed to nonfatal encounters with predators.*

Most plants (*1*) and some asexually reproducing invertebrates (*2*) respond to attack by consumers with production and mobilization of inducible defenses. Inducible defenses are produced only in response to stimuli from consumers and consequently are as temporally and spatially intermittent as the stimuli triggering them. Among animals, the capacity to respond to predation with induced defenses has been demonstrated only by asexually reproducing groups such as cladocerans and rotifers (*2*). Attacks by internal parasites also trigger induced immunological defensive responses in most metazoan hosts (*3*). Attacks on plants, metazoans (by parasites), and rotifers share the common feature that the prey are only partially damaged and can therefore respond with an appropriately timed defense. Although individual units may be killed, predation on rotifers and cladocerans is usually nonlethal to the genome (the appropriate evolutionary unit), which is represented by a spatially discontinuous collection of ramets (*4*). Partial predation on any taxon may favor the evolution of induced defenses if initial damage is a reliable predictor of additional tissue loss and if sufficient time and resources are available for the prey to mobilize an effective defense. I now describe a novel morphological defense elaborated by a cheilostome bryozoan in response to attack by slowly feeding, spatially and temporally intermittent molluscan predators.

Bryozoans, like other colonial inverte-

brates, can be well endowed with chemical defenses (*5*), but neither structural nor consumer-induced defenses have been shown. Because most colonies are large relative to individual predators and are constructed of numerous replicated units, they are only rarely killed in a single attack (*6*). *Membranipora membranacea* is a circumglobal cheilostome bryozoan that grows epiphytically on kelp. The large encrusting colonies are attacked by several kinds of predators but most commonly by *Doridella steinbergae*, a nudibranch that is trophically specialized on *Membranipora* (*7*). Yoshioka (*8*) suggested that spines present on colonies of *Membranipora membranacea* are predator-induced and, further, that two closely related spined species—*M. villosa* and *M. serrilamella*—might be conspecific morphs of the unspined *M. membranacea* (*8*). Induced morphological defense in *Membranipora* should be an effective strategy for surviving attack from a slowly feeding, intermittent predator. Production of spines is probably costly (*8*), resulting in slower growth. The price of slowed growth is high in a space-limited system, where final size is ultimately limited by abutting conspecifics (*9*).

In controlled laboratory experiments (*10*), spines were induced on *M. membranacea* within 2 days by exposing colonies to direct predation by two species of nudibranch (Table 1 and Fig. 1, a and b). Within a single zooid, flexible chitinous spines grow at two locations: corner spines from existing buds at the corner of each zooecium, and membranous spines directly out of the frontal membrane (Fig. 1, b and c). Colonies in treatments exposed to the nudibranchs *Doridella steinbergae*, a *Membranipora* specialist, and *Onchidoris muricata*, a bryozoan generalist, produced spines, whereas control colonies did not ($P < 0.001$, χ^2); there was no statistical difference between the number of spined colonies produced in the *Doridella* or *Onchidoris* treatments ($P > 0.05$, χ^2). In the induction experiment, only colonies subject to direct, mechanical damage by nudibranchs produced spines. For example, four out of 32 colonies surviving the *Doridella* treatment failed to produce spines; these colonies did not appear to be attacked by *Doridella*. This suggests that neither waterborne cues from nudibranchs nor chemical signals from injured colonies are sufficient to trigger induction (*11*). The absence of spines on control colonies shows that general mechanical damage alone cannot trigger induction, because even control colonies suffered some mechanical damage (killed polypides and broken zooecia) during removal from kelp substrate during the transplantation procedure (*10*).

Table 1. *Membranipora membranacea* spine induction experiment. Colonies were subjected to three treatments: predation by the nudibranchs *Doridella steinbergae* and *Onchidoris muricata* and a control treatment with no predation.

Colony type	*Doridella* ($N = 40$)	*Onchidoris* ($N = 30$)	Control ($N = 40$)
Spined	28	12	0
Unspined	4	2	29
Dead	8	16	11

Spines were induced on existing and developing zooids around the entire perimeter of an attacked colony, thereby fortifying the edges of colonies. The consistent formation of regular bands of spined zooids on most colonies producing spines suggests that information about a nudibranch attack can be translocated to a site remote from the attack and can trigger a programmed response of peripheral spine development. Although the possibility that peripheral bands form in response to peripheral foraging patterns of nudibranchs cannot be excluded, the regularity of these bands on most colonies with spines suggests translocation. In laboratory cultures, spine growth on colonies was immediately terminated (within a day) when nudibranchs were removed. Existing spines were permanent; thus the presence of spined zooids on colonies is an indicator of previous nudibranch attack. I have found colonies in nature that are completely armored with spines; this may indicate that colonies can continue producing spines when nudibranchs pose a constant threat.

Spines can effectively control intracolony feeding patterns of the nudibranchs and slow nudibranch feeding rates. On 67 percent ($N = 126$) of partially spined *Membranipora* colonies censused from field populations, nudibranch damage was restricted to the central, unspined portions of the colony; spined marginal zooids were largely undamaged (Fig. 1d). The defended marginal zooids can usually regenerate new tissue to replace damaged central zooids. Feeding rates of nudibranchs on spined colonies were significantly lower than on unspined colonies ($P < 0.05$, Mann-Whitney U test). Unspined colonies were consumed by individual nudibranchs at a median rate of 44 zooids per day [mean = 58.3, standard deviation (S.D.) = 41.7; $N = 17$]; spined colonies were consumed at a median rate of eight zooids per day (mean = 19.4, S.D. = 20.1; $N = 5$). Spines therefore effectively control the pattern and magnitude of intracolony mortality due to nudibranch predation.

These results have important implications for the study of coevolution and paleoecology as well as predator-prey interactions involving clonal or colonial prey. The reciprocal adaptation demonstrated in the interaction of *Membranipora membranacea* and its primary predator, *Doridella steinbergae*, represents one of the strongest in a limited number of examples (*12*) of coevolution in the sea. Important adaptive features maintaining the specificity and reciprocal nature of the interaction are (i) temporal synchronization of the seasonal appear-

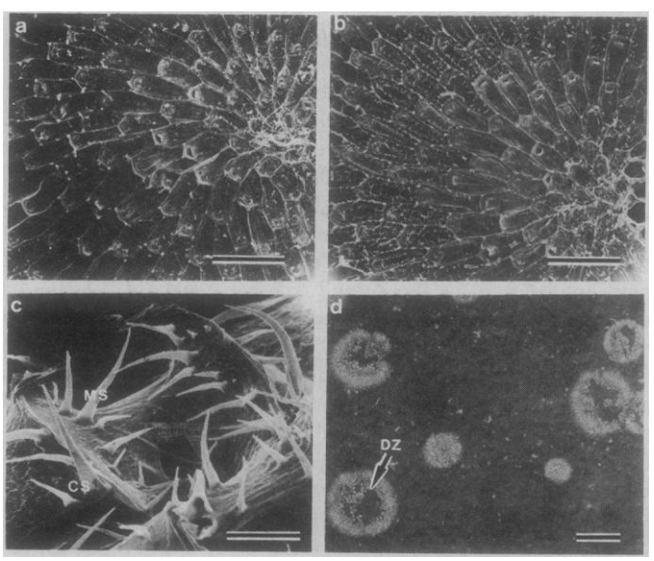

Fig. 1. Induced spines on *Membranipora membranacea*: results of a controlled laboratory experiment. (a) Scanning electron microscope (SEM) photograph of a colony grown without nudibranchs (control treatment); scale bar, 1000 μm; (b) SEM photograph of a colony grown with *Doridella steinbergae*; scale bar, 1000 μm; (c) SEM photograph of a magnified, spined zooid of *Membranipora membranacea*; *CS*, corner spine; *MS*, membrane spine; scale bar, 100 μm. (d) Pattern of intracolony mortality on a spined colony due to nudibranch predation. The peripheral, defended margin of the colony is not killed; *DZ*, dead zooids; scale bar, 10 mm.

ance of predator and prey, both of which are sub-annual species (presumably an adaptation of *Doridella* to exploit *Membranipora*); (ii) requirement of *Doridella* larvae for contact with *Membranipora* to trigger metamorphosis to the adult form (*7*); (iii) cryptic coloration of *Doridella* with respect to *Membranipora*; and (iv) a fast-acting, induced defense by *Membranipora* in response to predation by *Doridella* and the other, more generalized nudibranch predator.

Little is known about the chemical or physical arsenal available to most colonial organisms in the evolutionary arms race with their predators or about the patterns of spatial and temporal deployment of constitutive and induced defenses. As shown with *Membranipora*, induced or spatially variable patterns of colony defense may determine intermittent foraging patterns displayed by many predators on bryozoans (*13*) and other colonial prey.

Bryozoans are well represented in the fossil record (*14*). On the basis of information extracted from present-day interactions, paleontologists make inferences about events structuring interactions in "paleo-assemblages." However, predation has been a notoriously difficult process for paleontologists to quantify (*15*). If other species of recent and fossil anascan bryozoans respond to predators with the production of spines, then the existence of fossil bryozoans with bands of spined zooids would allow us to make inferences about the prevalence and incidence of predation in paleoseas.

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11. There is evidence that airborne substances released by injured trees can be detected by conspecifics and trigger an induced defense in the receiver [D. F. Rhoades, *Am. Chem. Soc. Symp. Ser.* **208**, 55 (1983); I. T. Baldwin and J. C. Schultz, *Science* **221**, 277 (1983)]. Some clonal marine animals appear to emit warning pheromones upon attack by nudibranchs; these signals are detected by spatially distant individuals which respond with a defensive posture [L. G. Harris and N. R. Howe, *Biol. Bull.* **157**, 138 (1975)].
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Decreased Neuronal Inhibition in Vitro After Long-Term Administration of Ethanol

Abstract. *The pathophysiology of brain dysfunction was studied with an animal model of chronic alcoholism. Rats were fed a liquid diet with or without ethanol for 20 weeks and then the diet without ethanol for three more weeks. Hippocampal slices were prepared and intracellular recordings were obtained from dentate granule and CA1 cells. Significant depression of orthodromically elicited inhibitory postsynaptic potentials and postspike afterhyperpolarizations was observed in neurons from ethanol-exposed animals. No differences were observed in other active or passive membrane characteristics. These results suggest that a loss of neuronal inhibition could contribute to brain dysfunction in chronic alcoholism.*

Generalized learning deficits in rats and mice (*1*), loss of dendritic spines (*2*), decreased dendritic branching (*2, 3*), and cell death (*4*) have been reported in the hippocampus after three or more months of ethanol administration and several weeks of withdrawal on liquid diets. However, physiological changes in neurons have been little studied in animals with ethanol-induced brain damage. Augmentation of paired-pulse facilitation (*1, 5*) and changes in the distribution of synaptic current in CA1 cells (*6*) were measured extracellularly in the rat hip-

Table 1. Effects of long-term exposure to ethanol on intracellularly measured physiological parameters of granule cells. The amplitude and duration of the AHP were normalized to the number of spikes. Statistical analysis was done with a two-tailed *t*-test for all parameters except input resistance, subthreshold IPSP, and AHP amplitude and duration, for which a two-tailed Mann-Whitney *U* test was used because standard deviations for the two groups were significantly different by analysis of variance ($\alpha = 0.05$). N.S., not significant.

Measure	Control group		Ethanol group		*P*
	Mean ± standard error	*N*	Mean ± standard error	*N*	
Resting potential (mV)	55.65 ± 1.22	59	54.48 ± 1.23	49	N.S.
Action potential (mV)	73.70 ± 1.33	62	73.49 ± 1.54	53	N.S.
EPSP (subthreshold) (mV)	5.22 ± 0.49	32	4.87 ± 0.62	27	N.S.
IPSP					
Subthreshold (mV)	0.60 ± 0.13	34	0.39 ± 0.09	31	< 0.05
Maximum (mV)	2.25 ± 0.19	54	1.51 ± 0.22	46	< 0.025
Maximum (second)	0.580 ± 0.064	54	0.290 ± 0.046	46	< 0.01
Rheobase current (nA)	0.090 ± 0.009	11	0.040 ± 0.010	9	N.S.
AHP (mV per spike)	0.84 ± 0.28	19	0.25 ± 0.05	17	< 0.015
AHP (second per spike)	0.210 ± 0.055	19	0.08 ± 0.27	17	< 0.02
Input resistance (megohms)	66.64 ± 9.71	22	51.84 ± 4.61	22	N.S.
Time constant (msec)	21.86 ± 1.32	22	20.17 ± 1.01	22	N.S.
Rectification	0.240 ± 0.023	27	0.220 ± 0.023	27	N.S.