

enced density function are used, the properties of the bottom water found by Amos and Gerard are not those of the water ~ 1000 m above.

Amos and Gerard also incorrectly state that Geochemical Ocean Sections Study (GEOSECS) station 28 (39°N, 43°59'W) shows a steplike increase in light-scattering near the bottom. The profiles of θ , S , σ_t , and light-scattering at this station are reproduced in Fig. 1. In fact, the bottom layer has a lower level of light-scattering than the water mass immediately above. A turbidity current cannot explain the profiles found at GEOSECS station 28. In contrast, a density current of Denmark Straits Norwegian Sea overflow characteristics is seen intruding beneath water of North Atlantic Deep Water characteristics.

Similar anomalous layers, all at $\theta \sim 1.81^\circ\text{C}$, with Denmark Straits overflow characteristics, have been identified at many stations throughout the western North Atlantic (5). The anomalous water found at the bottom at station Lynch 47-186 (1) and GEOSECS station 28 (6) as a density current is also seen as an intrusive feature away from the basin margins in deeper water.

Is it not possible that the high turbidity found by Amos and Gerard is due to resuspension by high mesoscale currents in the area? They found that the current at 1.5 m above bottom averaged 23 cm/sec. Schmitz reports distributions of mean eddy kinetic energy along 55°W (7). At 40°30'N, 55°W (2° east of station Lynch 47-186), a mean eddy kinetic energy of 80 cm² sec⁻² was found at a depth of 4000 m. Velocities as high as 50 cm/sec were recorded (8).

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We acknowledge that Armi's explanation (1) of the anomalous conditions is a valid and perhaps less speculative alternative than our own (2). We concur with Armi that the nonconservative nature of in situ temperature and density (σ_t) in the deep ocean make these properties unsuitable for water mass tracing. It was our intention to show that properties at the bottom at station Lynch 47-186 (including the dissolved oxygen content) are similar to those at the bottom higher up on the continental slope [figure 2c in (2)]; we stated that these properties trend toward those of Denmark Strait water. The use of a potential density profile instead of σ_t in our figure 2b would have been preferable, and we did not intend to imply that the water column was unstable. We were in error in quoting the adiabatic gradient as being 0.096°C per kilometer (figure 2b); it should be 0.13°C per kilometer.

In defense of our hypothesis, we would point out that downslope advection does occur in several parts of the world's oceans and obviously did occur at this location during the 1929 earthquake (3). Our suggestion that turbidity current activity is responsible for the anomalous conditions at the bottom at station Lynch 47-186 is based upon the extraordinary amount of suspended material found there. The amount (~5000 $\mu\text{g/liter}$) exceeds by two orders of magnitude that found at Geochemical Ocean Sections Study (GEOSECS) station 28 [figure 1 in (1)] and, to our knowledge, is one of the highest concentrations of suspended material yet found in the deep ocean. We pointed out (2) that the addition of such quantities of material will increase the density of the sediment-water suspension by an amount comparable to the density effects contributed by the temperature-salinity changes in the near-bottom water column.

Large Doses of Ecdysterone May Inhibit Mosquito Behavior Nonspecifically

Beach (1) has reported that biting behavior in the unfed mosquito *Anopheles freeborni* is inhibited by the injection of ecdysterone, an insect hormone that is produced by the ovaries during oogenesis (2). This suggested role of ecdysterone as a behavioral inhibitor warrants a closer examination.

Central to Beach's hypothesis is the report (2) that in *Aedes aegypti*, ecdysteroids reach a peak of approximately 275 pg per female at 18 hours after a blood

The source that we used for GEOSECS station 28 nephelometer data was figure 2 in (4). Compared to Armi's figure 1, Broecker and Bainbridge's data from the same original station profile look considerably different. A drop in light-scattering is shown in figure 2b in (4) but, compared to the increase in light-scattering immediately above, this decrease is small. Broecker and Bainbridge may not have presented their data referenced to a zero ordinate on the arbitrary scale of units used by the GEOSECS nephelometer. This would enhance the apparent increase in light-scattering that we refer to.

More recent measurements of near-bottom ocean turbidity were taken as part of the High-Energy Benthic Boundary Layer Experiment (HEBBLE) program in an area of the Nova Scotia continental rise about 500 km west of our reported stations (5). Results from two cruises (6) in 1979 indicate a very strong bottom nepheloid layer in depths of about 5000 m, with particulate concentrations about twice the values reported in (2). These high concentrations are associated with strong but variable bottom contour currents.

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meal. By 30 hours after a blood meal the concentration returns to its low pre-blood meal level. Since this assay was performed on whole body extracts, it is not known how much, or indeed if any, hormone occurs outside the ovary. In *Ae. aegypti*, there is no humoral inhibition of either biting or host-seeking when ecdysteroid concentrations have peaked; the first indications of host-seeking inhibition are at 30 hours after a blood meal, with the greatest inhibition occurring be-

tween 36 and 72 hours after a meal (3). Since the endogenous concentration of ecdysteroids is low at the time when host-seeking behavior is inhibited, it is unlikely that ecdysterone is the humoral inhibitor in *Ae. aegypti*.

Although ecdysterone has not been demonstrated in *An. freeborni*, Beach injected this species with up to 5 μg of the hormone, which is 18,000 times the physiological concentration in *Ae. aegypti*. Biting was inhibited at doses of 1 to 5 μg , but not at 0.5 μg . Therefore, it is unlikely that a physiological dose would have inhibited biting either. Ecdysterone administered within physiological ranges does have definite effects on *Ae. aegypti* and other insects (4), and one should question why Beach observed biting inhibition only when ecdysterone was applied in such large amounts.

Are these massive doses of a hormone specifically inhibiting biting behavior in *An. freeborni*, or are they acting nonspecifically to suppress behavior in general? I have found that 1 μg of ecdysterone injected into *Ae. aegypti* does not inhibit biting behavior; however, both host-seeking and oviposition behaviors are blocked (5). Mosquitoes generally do not seek a host when gravid but do readily oviposit, and it would not be biologically plausible to propose that ecdysterone naturally inhibits both behavioral sequences. Instead, these mosquitoes might have been unable to respond to any stimuli because they were "drugged."

Conclusions regarding the effects of hormones on behavior should be drawn with caution, especially when doses many times the physiological concentration are required and when the behavior being evaluated is the absence of a measurable response.

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Klowden states that ecdysterone does not inhibit host-seeking in *Aedes aegypti* because the titer of the hormone returns to pre-blood meal levels prior to the disappearance of the behavior. This interpretation assumes that elevated ecdysterone titers in *Ae. aegypti* directly affect that part of the nervous system governing host-seeking behavior. However, ecdysterone could mediate the change from host-seeking to non-host-seeking behavior in other ways. For example, high ecdysterone titers may elicit a second factor that inhibits biting behavior. Elaboration of a hormone and expression of the behavioral state it effects are not necessarily contemporaneous.

Although I must inject pharmacological rather than physiological levels of ecdysterone to inhibit biting behavior, I disagree with Klowden's contention that these doses "drugged" my test animals. Twelve hours after injection with 1 μg of ecdysterone, 3- to 5-day-old *Anopheles freeborni* males seek out and mate with females of the same extent as untreated males of the same age (1). Such mating activity would not be observed in ecdysterone-injected males if the hormone were acting as a nonspecific inhibitor of behavior. The experimental designs in the reports cited by Klowden as evidence that physiological levels of ecdysterone "have definite effects . . ." differed from my own. In the first report (2), the effect of ecdysterone on tsetse fly Malpighian tubules was studied in vitro rather than in vivo. Would the same amount of exogenous ecdysterone work in vivo, or would much higher doses be required to compensate for the degradation of the hormone within the intact fly (3)? The second study (4) utilized two ecdysterone injections separated by a fixed interval of time, rather than a single injection. The length of this interval was critical (5), and it may be that the number and timing of injections determine

whether physiological levels of exogenous ecdysterone will elicit specific physiological changes in mosquitoes. Injection of pharmacological rather than physiological hormone doses in experiments on ecdysterone-related changes in mosquitoes is the rule rather than the exception (6-9).

Klowden also reports that 1 μg of ecdysterone injected into *Ae. aegypti* blocks host-seeking and oviposition but not biting behavior. However, he does not mention how biting behavior was measured in this experiment. Is he using an assay similar to mine or one involving a different experimental design? The results of this experiment seem to conflict with his contention that 1 μg of ecdysterone nonspecifically suppresses behavior in general.

I disagree with the comment on the biological implausibility of post-blood meal ecdysterone inhibiting oviposition and biting behavior. Expression of either behavior prior to egg maturation would only jeopardize the chances of producing viable progeny. Therefore, why not a mechanism that suppresses both behaviors during oogenesis? Finally, nowhere do I state that ecdysterone inhibits *only* biting behavior. Ecdysterone also delays oviposition in *An. freeborni* (10) and my feeling is that the hormone may act to inhibit any extraneous activity during oogenesis.

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