Mechanism of Brain Growth by Environmental Stimulation

Cummins *et al.* (*1*) have proposed an appealing explanation for the effects of environmental stimulation on brain development. Their hypothesis that "the development of some neurons can be described as environment-dependent" [(*1*), p. 692] is consistent with what is known from environmental complexity and other types of developmental studies (*2*). We feel, however, that arguing "that the basic mechanism is that of nonspecific activation of the cortex during arousal" [(*1*), p. 694] is not justified by the available data.

If arousal can be defined on the basis of "altered levels of motor activity" (*3*), administration of amphetamine, which increases motor activity, should elicit changes in brain weight regardless of the environment. Bennett *et al.* (*4*) showed that this is not the case. Arousal theory cannot explain why rats forced to perform complex acrobatic skills do not have larger brains than passive controls (*5*). These animals were aroused when hanging upside down while eating. Additional evidence against the arousal hypothesis comes from the study of "observer rats" living in individual wire mesh cages located inside the enriched environment (*6*). These observer animals were aroused four times a day by changing the location of their cages, at which times they showed typical orienting response toward and interaction with the grouped rats. They did not show the typical anatomical or behavioral changes elicited by enrichment.

The observer rats were not deprived of sensory stimulation; they could see, hear, and smell the enriched environment, but their cortical weights were identical to those of impoverished controls. Thus, we also disagree with the statement that "sensory deprivation suffered by the isolated animals is likely to be the critical factor" [(*1*), p. 694] in determining the enrichment-impoverishment differences.

We thus believe that a nonspecific cortical stimulation (arousal) by itself does not induce brain growth. It seems instead that a more specific mechanism must be invoked. We have proposed that processing a wide variety of stimuli coupled with motor feedback is necessary (*5*). This is equivalent to restating that learning (acquisition, memory storage, or both), if complex enough, will trigger a trophic response.

Perhaps Walsh and Cummins agree with us in a way. When the arousal hypothesis was first published, they said, "Despite the emphasis on arousal in this paper, we did not intend to suggest that this is the only mechanism involved in the complexity-isolation changes. Other mechanisms, such as learning, are almost certainly involved, since an altered arousal state must have as its consequence an altered level of sensory awareness and probability of engram storage" [(*3*), p. 995]. But in *Science* (*1*), they mention arousal as the basic mechanism without mentioning any other mechanism or offering any additional proof for their theory.

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15 September 1978

Ferchmin and Eterović's (*1*) evidence against our concept of brain development through nonspecific activation of the cortex during arousal (*2*) fails for the following reasons.

1) Walsh and Cummins (*3*) emphasized that the "state of arousal can be estimated in so many different ways" (*3*, p. 989). To define arousal on the basis of "altered motor activity" takes their definition out of context. While altered motor activity may well be an expression of arousal, it is not the cause of it. There are many causal bases for activity and, in fact, Rosenzweig *et al.* (*4*) have shown that motor activity per se will not induce cerebral development.

2) The study of "observer rats" (*5*) is difficult to interpret since social stimulation alone is ineffective in producing changes in the brain (*6*). The study of "acrobatic rats" (*7*) argues more convincingly against the learning hypothesis than against the arousal hypothesis. Although Ferchmin and Eterović state that these animals were aroused when hanging upside down while eating, they provided no evidence for this (*7*). The learning of this skill was developed gradually with training proceeding through six stages, and "after some training this whole procedure was performed quickly and efficiently" (*7*, p. 457).

3) In the amphetamine study by Bennett *et al.* (*8*), the comparison was between isolated saline-injected and isolated amphetamine-injected animals. Eterović and Ferchmin (*9*) have shown that stimulation derived from an injection procedure alone is sufficient to raise the cerebral weights of isolates to that of enriched animals. Such stimulation could account for the lack of effect in (*8*) by causing the brain weights of both injection groups to move toward their developmental ceilings, thereby attenuating the difference induced by amphetamine.

Ferchmin and Eterović's suggestion that "amphetamines . . . should elicit changes in brain weight regardless of the environment" misses the point of our theoretical model, namely the existence of a developmental ceiling for brain development. Because of this ceiling, the ability of any stimulation procedure to effect change is intimately linked with the total amount of stimulation the animal receives.

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24 May 1979

0036-8075/79/0803-0522$00.50/0

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Science **205** (4405), 522.
DOI: 10.1126/science.451619