Open Peer Commentary

Commentaries submitted by the qualified professional readership of this journal will be considered for publication in a later issue as Continuing Commentary on this article. The following are among those who will provide Continuing Commentary in a forthcoming issue: N. J. Mackintosh and H. Ursin.

by **David B. Adams**

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Motivational systems: fear or defense? pain or recuperation?

The new and provocative model presented by Bolles & Fanselow does not attempt to identify the neural substrates of their proposed fear and pain motivational systems, but there are many points of consonance with my own analysis of a "defense motivational system," which was based upon data from brain research and presented recently in this journal (Adams 1979a). Specifically, their Figure 2 is consonant with my Figure 1. The following comparisons may be made. Their "expectancy of a US" corresponds in part to my "sensory filters for motivating stimuli," pathways that are shown converging upon the midbrain in my Figure 1: a forebrain pathway, a pretectal visual pathway, an auditory pathway, and a pain/tactile pathway from the hindbrain. Their "fear" corresponds to activation of the "defense motivational mechanism" located in the midbrain central gray. Their "species-specific defense reactions" correspond to motor patterns organized by motor patterning mechanisms such as those of striking, lunge-and-bite attack, upright and sideways postures, squeal and hiss, freezing, and fleeing, shown in my Figure 1. The inhibition of pain shown in their figure is not illustrated in my analysis, but is consonant with data from Mayer and Liebeskind (1974) that stimulation of the midbrain central gray produces analgesia. Finally, the inhibition of other motivational systems shown in their figure is consistent with data suggesting that the defense motivational mechanism directly inhibits the offense motivational mechanism (Mink and Adams, in preparation).

The analysis by B & F does not illustrate releasing and directing stimuli for specific motor patterns (their SSDRs) of defense (their fear), although I think that their analysis would be strengthened by considering them. By distinguishing separate releasing and directing stimuli that act directly upon motor patterns, as opposed to motivating stimuli that act upon the motivational mechanisms, they could explain why specific behaviors are, in their words, "flexible" and "adaptive to the possibilities afforded by environmental supports." And, by being more explicit about motivating stimuli, they could explain why, in their words, "different motivational systems can evidently be aroused in different ways."

My own analysis of the defense motivational system differs from theirs in my emphasis upon motivating stimuli for defense other than conditioned stimuli. Other motivating stimuli such as sudden noise or visual movement, dorsal tactile stimulation, certain olfactory stimuli, and stimuli that evoke neophobia may be more important under natural conditions. Because of the peculiar way in which they are raised in very restricted and homogeneous environments with no opportunity to escape into a nest box or burrow, laboratory animals may have very high sensory thresholds for most of these stimuli (Clark and Galef 1977; Adams 1979b), and therefore pain and stimuli conditioned to pain have been overemphasized in laboratory studies. I am intrigued by B & F's suggestion that only stimuli conditioned to pain, and not pain itself, can serve as a motivating stimulus for defense (their "fear"), but I am not completely convinced by the evidence they present.

I question their use of the term "species-specific defense reactions." By comparing rats and cats, they emphasize taxonomic differences across orders, not species, while there are very few good data on cross-species comparisons of behaviors in rodents. In particular, there are good data on species-specific differences in copulatory patterns in muroid rodents (Dewsbury 1975). I have recently reviewed the motor patterns of defense in muroid rodents and found that most of them are remarkably similar across many species (Adams, submitted for publication). Only three classes of motor patterns related to agonistic behavior typically vary from species to species of muroid rodent: motor patterns of scent-marking, threat, and alarm signals, and only the last two are motor patterns of defense.

Most cases of "species-specific" motor patterns – or, for that matter, species-specific differences in any aspect of a motivational system – probably arise as premating isolation mechanisms (Dobzhansky 1937) that protect the female against sterile matings with males of other similar species. For example, the motor patterns of copulation and of scent-marking, as mentioned above, can vary more between related species than between genera, which points to an active force for divergence such as those of pre-mating isolation mechanisms (Dewsbury 1975; Adams, submitted for publication). Other aspects of the neural substrate of motivational systems of agonistic behavior are more likely to be similar, not only across species, but even across the entire class of mammals (Adams 1979a; Adams 1980).

Indiscriminate use of the term "species-specific" reinforces an unfortunate tradition in comparative psychology that overemphasizes the genetic aspect of individual and species differences [cf. Eibl-Eibesfeldt: "Human Ethology" *BBS* 2(1) 1979]. As mentioned above, in the process, critical ontogenetic factors are often overlooked, and the role of releasing and directing stimuli, both conditioned and unconditioned, are not sufficiently recognized.

by **Mortimer H. Appley**

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Stress and arousal in pain perception

Rejecting Hobart Mowrer's now forty-year-old concept of fear as the conditioned form of pain, Bolles & Fanselow offer a provocative and interesting new interpretation of fear and pain as independent motivational states. According to their perceptual-defensive-recuperative (PDR) model, fear may arise independently of experienced pain (e.g., as in phobias of height or snakes, where the pain was/is never experienced) but in any case is associated with the *anticipation* of pain and has the effect of inhibiting pain and pain-induced recuperative behaviors. This is necessary, they point out, because such behaviors are or would be incompatible with the effective defensive behaviors that should occur in the presence of fear cues if the organism is to protect itself properly. The mechanism by which fear effects the inhibition of pain is an endogenous analgesic brain peptide (endorphin) production which blocks pain following fear and the effects of which can themselves be inhibited by administration of a narcotic antagonist, naloxone.

The PDR model of fear and pain is at best, as its authors note, "a rough heuristic." Its differentiation of perceptual-learning (P), defensive (D) and recuperative (R) phases of traumatic situations is interesting, but sketchy at best, especially as regards the last of these. B & F would have greatly strengthened their arguments had they attempted to relate their theory/model to the considerable research and theoretical literatures on arousal, anticipation, anxiety, and stress. What, for example, is the relation between the defensive (D) and recuperative (R) phases described here and the stages of resistance and exhaustion in Hans Selye's General Adaptation Syndrome (see Cofer and Appley 1964, pp. 442ff. for discussion of systemic and psychological stress)? Or of these stages to those in Mowrer's (1960) discussion of hope and hoplessness, Lazarus's (1966) description of coping, Appley's treat-