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Brain Mechanisms for Offense, Defense and Submission

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Abstract: A preliminary attempt is made to analyze the intraspecific aggressive behavior of mammals in terms of specific neural circuitry. The results of stimulation, lesion, and recording studies of aggressive behavior in cats and rats are reviewed and analyzed in terms of three hypothetical motivational systems: offense, defense, and submission. A critical distinction, derived from ethological theory, is made between motivating stimuli that simultaneously activate functional groupings of motor patterning mechanisms, and releasing and directing stimuli that are necessary for the activation of discrete motor patterning mechanisms. It is suggested that motivating stimuli activate pathways that converge upon sets of homogeneous neurons, called motivational mechanisms, whose activity determines the motivational state of the animal.

A defense motivational mechanism is hypothesized to be located in the midbrain central gray. In addition to tactile, auditory, and visual inputs from the paleospinothalamic tract, lateral lemniscus, and (perhaps) from the pretectum, it may receive inputs from a major forebrain pathway whose functional significance is not yet understood.

A submission motivational mechanism is also thought to be located in the central gray. In addition to inputs for defense, it is thought to receive a necessary input from a "consociate (social familiarity cue) modulator" located in the ventromedial hypothalamus, which can switch behavior from defense to submission. The location of the hypothetical offense motivational mechanism is not known, although the pathways by which it is activated are traced in some detail.

Brain mechanisms of aggression in primitive mammals and in primates are apparently similar to those in rats and cats.

This paper represents a preliminary attempt to analyze the intraspecific behavior of mammals in terms of specific neural circuitry. Despite the large number of studies and reviews on the brain mechanisms of intraspecific aggression in cats and rats, there is still no agreement on the general outline of the neural circuitry involved. Hopefully, this paper will help to frame a working model that can stimulate and help in the design of future experiments.

My own involvement in this problem began with a study in which I recorded from individual neurons during affective defense behavior in the cat (Adams 1968). Some of the results of that study were encouraging; in the midbrain central gray neurons were found that fired if and only if the animal showed affective defense. Those results were expected, since other investigators such as Hunsperger (1956) and Skultety (1963) had already established that the central gray plays a critical role in affective defense. Other results were discouraging; despite the fact that electrical stimulation of the hypothalamic ventromedial nucleus produced affective defense, the neurons of that nucleus were not active during affective defense elicited in a semi-natural situation. Puzzled, I came to the conclusion that our behavioral control was not adequate to the demands of our neurophysiological techniques.

For the past ten years I have followed a strategy of developing refined behavioral understanding that might be applied to neurophysiological experiments. I have concentrated on the behavior of muroid rodents, which are the best-studied of all mammals in terms of behavior, and which are amenable to neurophysiological techniques. I felt that a more thorough behavioral analysis was needed in order to provide the kind of functional information needed to evaluate the neurophysiological data.

In particular, I am greatly indebted to the techniques and terminology of ethology as it has been developed by Lorenz (1970), Tinbergen (1951), Leyhausen (1956), and Eibl-Eibesfeldt (1970). [See also Eibl-Eibesfeldt: "Human Ethology" BBS 2(1) 1979.] From them I have taken a critical distinction between two aspects of stimuli: *motivating stimuli*, which bias the organism towards the performance of a number of functionally related motor patterns such as those of sexual behavior, defense, and so forth; and *releasing and directing stimuli*, which trigger or orient the performance of one particular motor pattern without regard to its functional or temporal relationship to other motor patterns. Whereas the time course of the action of a motivating stimulus is usually on the order of minutes or more, the time course of a releasing stimulus may be on the order of milliseconds. In muroid rodents, and to some extent in cats, the motivating stimuli for social behavior are often olfactory, while releasing and directing stimuli are most often visual or tactile. It should be noted that this distinction is *not* a classification of stimuli, but a distinction between two ways in which stimulus information is processed by the nervous system. One and the same physical stimulus may function simultaneously as a releasing, directing, and motivating stimulus. For example, the vibrissal perception by one rat of the presence and vibration of the vibrissae of a second rat may release and direct a motor pattern of upright posture and, at the same time serve as a motivating stimulus to the "consociate modulator" that ensures that the rat will not lunge

and bite the nose of the opponent (Kanki & Adams 1978). By "consociate," I mean a familiar individual of the same or a different species. The "modulator" (discussed further in the section on Submission) is a hypothetical mechanism for processing social cues of this sort.

Because the present analysis refers to specific, albeit hypothetical sets of potentially identifiable neurons, the terminology for these neurons does not correspond exactly to earlier ethological terminology, which was not intended to be so specific. I have continued to use the term "motivation," however. Although this terminology is not always welcome among neurophysiologists (for an exception. see Bindra 1969), an ethological analysis such as the one used here would be incomplete without referring to the concept represented by motivation, and the attendant scientific tradition seems to warrant using the word itself. In the present terminology, a critical role is played by *motivational mechanisms*, which are hypothetical sets of homogeneous neurons whose activity is held to be responsible for the motivational state of the animal. These are the neurons that are activated in the presence of *motivating stimuli*, and which are in turn responsible for the activation of a number of related *motor patterning mechanisms*. Motor patterning mechanisms coordinate the production of the *motor patterns* of the animal that are its observed or measurable behavior, including not only postures and acts, but also vocalizations, autonomic effects, and hormonal and pheromonal secretion. Motor patterning mechanisms are activated by simultaneous inputs from a motivational mechanism and from sensory filters responsive to *releasing* and *directing stimuli* specific to that motor pattern. I have coined the term *motivational system* to refer to the entire complex of motivating stimuli, releasing and directing stimuli, the neural mechanisms that alter all these stimuli, motor patterning mechanisms, and the particular motivational mechanisms through which they are all related.

A detailed survey of intraspecific aggressive behavior in muroid rodents has led me to propose that there are three motivational systems involved: offense, defense, and submission (Adams, submitted for publication). It has become quite common in the last few years to make a distinction between offense and the other motivational systems. The distinction has been made for the cat on the basis of behavioral observation (Leyhausen 1956), and for the rat on the basis of pharmacological (Miczek 1974), behavioral (Blanchard et al. 1975; Adams 1976), and neurological data (Adams 1971). Offense is shown, for example, by the resident male rat; its motor patterns include approach, an offensive sideways posture, piloerection, and a bite-and-kick attack. Other behaviors are shown by an intruder male rat; its motor patterns include fleeing, freezing, a full submissive posture, squealing, and ultrasound vocalization.

A further distinction is also necessary between defense and submission, and this distinction has not been made in most of the literature. Defense is the behavior shown by wild animals or laboratory animals with forebrain lesions, and it may be quite damaging. It includes the lunge-and-bite attack directed at the face or protruding parts of the body of the opponent. It also includes various other defense motor patterns, including squealing, upright posture, fleeing, freezing, and various types of warning noises and vocalizations. Submission is the

behavior usually observed in laboratory animals under attack by conspecifics. It consists of a less damaging response. including many of the same motor patterns of defense, such as squealing, upright posture, fleeing, and freezing, but also including specific *submissive* behaviors such as the full submissive posture (lying on the back) and ultrasound vocalizations that have been shown to inhibit conspecific offense (Lehman & Adams 1977).

A further set of evidence for the distinctions among offense, defense, and submission derives from the different effects of hormones on muroid rodent social behavior. Offense is dependent upon gonadal hormones; in most species males show offense more than females, and the effect may be manipulated by castration or administration of testosterone (shown by many investigators). Maternal aggression is enhanced by prolactin (Wise & Pryor 1977), and an analysis of the postures of lactating rats in our laboratory indicates that maternal aggression includes both offense and defense. The effect of the prolactin upon defense is probably due to the suppression of the hypothetical "consociate modulator" and release of defense from its inhibitory influence. Corticosteroids, which enhance submission (Moyer & Leshner 1976), probably exert their effects by facilitation of the hypothesized consociate modulator.

The differences between offense, defense, and submission may be understood on another level, in terms of their communicative function and their evolutionary histories. Defense, it may be assumed, evolved under the pressure of attack by predators. As such, its primary purpose is not communication, at least not in relatively small and weakly-armed animals such as the muroid rodents. Instead, freezing enables the animal to avoid detection or to avoid releasing a predatory attack, fleeing brings the animal to the safety of a burrow or tree, and attack is a "last-resort" behavior that depends for its effectiveness upon infliction of pain or damage. In a predator such as the cat, defense may have more of a communicative function, since the cat is a relatively well-armed animal, and other carnivores (such as the dog) may respond to threat by desisting from an attack. Offense and submission, on the other hand, are primarily systems of communication. Offense, in the cat or rat, consists of threat and an attack, which is ritualized to such an extent that it does not usually produce serious damage. For example, the bite-and-kick attack of the rat inflicts superficial wounds on the flank of the opponent, a part of the body least vulnerable to serious damage. In species for which the land is partitioned into territories or domains (Brown 1975), such threat and ritualized attack enables an individual territory holder to maintain an arrangement of "loyal opposition" with its neighbor, in which each animal is dominant on its own territory but knows and respects the others. Since the neighbors do not kill each other, they remain familiar opponents and do not have to reestablish their relationship, often with potentially damaging combats. Submission may be seen as the opposite, complementary side of this communicative system. Use of the upright posture and the full submissive posture, which protect the flank from the bite-and-kick attack, and use of ultrasound, which inhibits offense, enables an animal to acknowledge the momentary dominance of its opponent without receiving a wound. Another aspect of ritualization is the exaggeration of threat postures and (in the cat) vocalizations which enable the opponent to

escape before fighting occurs (Eibl-Eibesfeldt 1970). As a result, in both offense and defense, motor patterns tend to occur in a graded hierarchy from low-intensity threat to high-intensity attack or escape. The complex evolution of threat among muroid rodent species has been reviewed elsewhere (Adams, submitted for publication).

In the following pages the data on the brain mechanisms of intraspecific aggression, obtained primarily from the cat and rat, will be considered in terms of the three proposed motivational systems: defense, submission, and offense. The neural organization of the motivational mechanism, sensory filters for motivating stimuli, sensory filters for releasing and directing stimuli, and motor patterning mechanisms will be considered in that order. An extension of the discussion to other mammals, in particular, primitive mammals (the opossum) and primates, will be made at the conclusion of the other analyses.

Defense: Motivational mechanism

There are neurons in the midbrain central gray and adjacent tegmentum that meet the criteria for the hypothetical motivational mechanism for defense in the rat. A lesion that totally destroys this region produces a syndrome in rats that includes the loss of all motor defense patterns in response to all motivating stimuli that have been tested. Defensive upright posture and boxing, escape, and vocalization are all abolished in response to footshock, and freezing is abolished in the open-field test situation (Edwards & Adams 1974). Vocalization is abolished in response to restraint and dorsal tactile stimulation (Chaurand et at 1972), and biting and escape as well as vocalization are abolished in response to these motivating stimuli (observation of animals described in Edwards & Adams 1974). Escape in response to footshock (Halpern 1968; Liebman et al. 1970) or loud noise (Lyon 1964) is also abolished by central gray lesions in the rat.

Electrical stimulation of the central gray and adjacent midbrain in the rat produces escape behavior, biting, and vocalization (Wolfle et al 1971; Waldbillig 1975), although it has not been reported to produce the upright posture. Neurons in this region are specifically active during shock-elicited defense (Pond et al. 1977).

Data from the cat are similar to those from the rat. In the cat, lesions of the midbrain central gray abolish defense motor patterns including vocalization, threat postures, attack, and defense in response to various types of motivating stimuli (Kelly et al. 1946; Hunsperger 1956; Skultety 1963). Electrical stimulation (Hunsperger 1956; Skultety 1963; Adams 1968) and chemical stimulation (Baxter 1968) of this region in the cat produce escape and "affective defense," which includes defense postures, vocalization, striking, and biting. There are neurons in this region in the cat that are active if and only if the animal is engaged in affective defense (Adams 1968).

There are even data from the distantly-related chicken that suggest that the same brain region may contain a defense motivational mechanism (DeLanerolle & Andrew 1974). Midbrain central gray lesions in the chicken abolish or depress defense vocalizations, defensive pecking, and freezing in a novel environment (Andrew & DeLanerolle 1974),

while electrical stimulation of the region produces escape and defense vocalization (Andrew 1973).

There are two instances of contradictory data from the cat that require discussion. Lesions of the midbrain central gray fail to abolish the aggressive behavior of the "thalamic cat" (Carli et al. 1963), and such lesions only temporarily interrupt the aggressive behavior following lesions of the ventromedial hypothalamus (Glusman 1974). There are two possible explanations consistent with the hypothetical role of the central gray as the defense motivational mechanism. First, it is possible that the observed behavior was offense rather than defense. Second, it is possible, at least in the latter case, that the lesions did not destroy the entire defense motivational mechanism but left some tissue remaining that became supersensitive to remaining inputs.

There is some evidence that following extensive hypothalamic lesions the behavior that is seen may be offense rather than defense. In the rat, lesions of the posterior portion of the ventromedial nucleus lead to offense rather than defense (Olivier 1977), while lesions of the anterior part of the nucleus lead to defense. I have noticed offense behaviors following similar lesions that destroyed both lateral and medial hypothalamus at the level of the ventromedial nucleus (in animals studied by Adams 1971). These rats displayed offensive sideways posture and bite-and-kick attacks when tested in a shock box - an effect I have never seen in normal animals. Upon examination of the histology it was observed that the offense had been exhibited only by animals with sparing of the posterior hypothalamus.

In order to abolish defense, lesions of the midbrain central gray must be complete. The entire rostral-caudal extent of the central gray must be destroyed, as well as part of the tegmentum adjacent to the lateral borders of the central gray. Partial lesions cause *increased* defense rather than diminishing it (Edwards & Adams 1974). This suggests that there may be some inhibitory processes in the circuitry of the defense system within the central gray which can be released by partial lesions. Although the midbrain central gray is often considered to be separate from the tegmentum that lies lateral to it, this separation may be artificial and due simply to the intrusion of tectofugal and posterior commissure fibers, which swing down and surround the central gray region. Both anatomical (Mehler 1969) and physiological (Ruth & Rosenfeld 1977) data suggest that the separation is artificial, and that the central gray and adjacent tegmentum on its lateral borders should be considered a single functional anatomical unit. The entire rostral-caudal extent of the central gray is also critical for defense. Lesions that destroy only the rostral end, only the caudal end, or both rostral and caudal ends, leaving the central zone intact, do not produce complete deficits (see Figure 6 in Edwards & Adams 1974). Similar data have been reported in the cat (Skultety 1963): lesions that destroyed more than 80% of the central gray abolished defense; lesions that destroyed between 50% and 80% blocked defense for several weeks only; and lesions with less than 50% destruction did not reduce defense.

Consistent with the notion of the role of the midbrain central gray as the defense motivational mechanism, this area appears to be the major locus of the effects of morphine on defense Jacquet & Lajtha 1973; Yaksh et al. 1976; Dostrovsky & Deakin 1977).

The defense motivational system is illustrated in Figure 1. At the center of the system are two hypothetical pools of neurons in the midbrain central gray, one of which corresponds to the defense motivational mechanism, as described above. It is shown as receiving its input from a second set of neurons in the central gray, which receive the motivating inputs for both defense and submission. This distinction between two pools of neurons in the central gray is hypothesized in order to account for the ability of an animal to switch from defense to submission, as explained in the following section on the submission motivational system. Motivating inputs converge upon the central gray, as shown, and outputs diverge from the central gray to activate the various motor patterning mechanisms of defense.

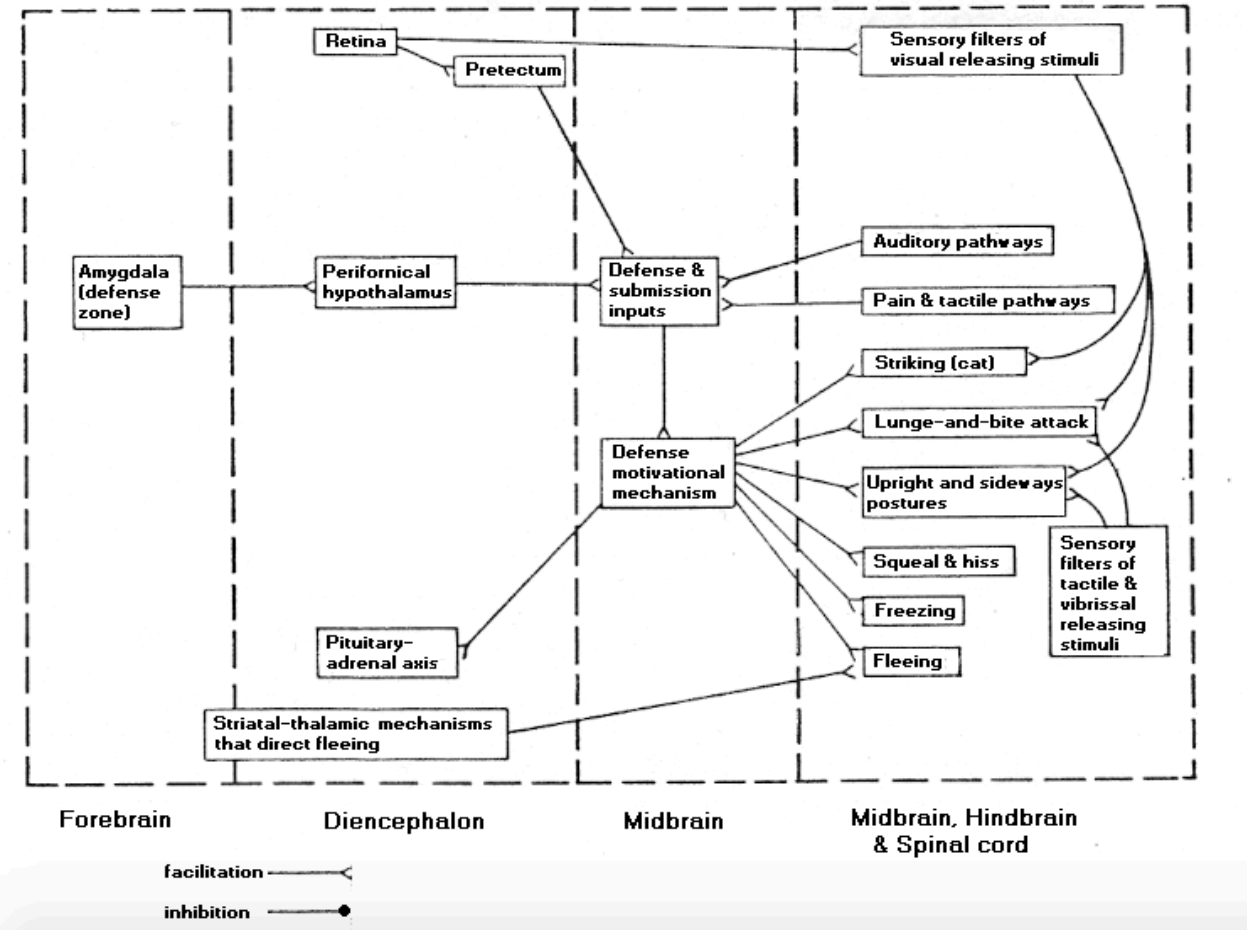

Figure 1. Neural circuity for defense. Motivating stimuli activate the "defense zone" of the amygdala (neophobia? defense pheromones?), pretectum (moving visual stimuli), auditory

pathways (sudden noise), and pain and tactile pathways. These motivating influences converge on input neurons of the midbrain central gray, which, in turn, activate neurons of a motivational mechanism for defense. The defense motivational mechanism activates motor patterning mechanisms for striking (in the cat), lunge-and-bite attack, upright and sideways postures, squealing and hissing, freezing, and fleeing. In most cases these mechanisms do not produce motor patterns unless they simultaneously receive appropriate visual or tactile releasing inputs, or, in the case of fleeing, releasing inputs from striatal-thalamic guidance mechanisms. Also illustrated is a motor patterning output to the pituitary-adrenal axis.

Defense: Motivating stimuli

The following types of motivating stimuli have been identified as activating the defense motivational system in wild muroid rodents: pain, sudden noise, sudden visual movement, dorsal tactile stimulation, restraint, certain olfactory stimuli (including defense pheromones), and the stimuli that evoke neophobia (Adams, submitted for publication). Experimental analyses have been hampered by the fact that some stimuli are not as effective in laboratory animals as in wild animals, especially sudden noise, sudden visual movement, and neophobia. This may be due to genetic differences in favor of docile animals created by selection pressure in the laboratory, as well as to artificial laboratory rearing practices which frustrate the normal ontogenetic development of the neural mechanisms that process these stimuli (Clark & Galef 1977).

Many of the motivating stimuli for defense do not require forebrain mechanisms. In the rat, startle and freezing may be elicited by loud sounds, and noxious tactile stimulation can produce jumping, vocalization, biting, struggling, urination, and defecation in chronic preparations from which the entire forebrain has been removed (Lovick 1972; Woods 1964). In cats, defense can be obtained in response to pain, dorsal tactile stimuli, and restraint after complete removal or transection of the forebrain (Woodworth & Sherrington 1904; Bazett & Penfield 1922; Keller 1932; Magoun et al. 1937; Kelly et al. 1946; Bard & Macht 1958).

Pain as a motivating defense stimulus was considered by classical neurologists to reach the midbrain tegmentum and central gray by way of the ventrolateral columns of the spinal cord (Woodworth & Sherrington 1904) and the paleospinothalamic tract in the brainstem (Mehler 1969). More recently, however, the mechanisms responsible for the sensory filtering of pain have turned out to be more complex than previously thought, and they may involve projections to the central gray from the dorsal columns and medial lemniscus as well (Liebeskind & Mayer 1971).

Auditory stimuli that motivate defense may reach the central gray directly by way of the lateral lemniscus without being relayed through the inferior colliculus. Lesions that destroy the inferior colliculus do not abolish an escape response to noise in the rat (Lyon 1964), while lesions that destroy the ventral portion of the central gray do abolish the response. Central gray neurons in the rat are responsive to click stimuli, as has been shown by

evoked potential (Hara et al. 1961) and single-unit recording techniques (Adams 1968). Central gray neurons in the rat that fire maximally during shock-elicited fighting are also facilitated by auditory stimuli (Pond et al. 1977); handclaps also produced upright defensive posture and boxing in the absence of shock stimulation in these animals.

Visual stimuli that activate defense may reach the central gray by way of the pretectum. This is suggested by the results of Schneider (1969), who found that undercutting the pretectum abolishes the freezing response of the hamster to overhead visual movement, while undercutting the superior colliculus, if anything, enhances the freezing response. Support for this also comes from Schaefer's (1970) findings that electrical stimulation of the pretectum produces flight in rabbits. This neural system may be homologous to the one that has been systematically studied in the pretectum of the toad, by which visual stimuli of a certain large size, movement velocity, and contrast produce escape behavior (Ewert 1970; Ingle 1976). The anatomy of the system in mammals is not clear, however; while there are projections from the central gray to the pretectum in the cat (Hamilton & Skultety 1970), reciprocal connections have not been reported (Berrnan 1977).

The neural pathways for visual, auditory, tactile and painful motivating stimuli for defense are shown converging upon the central gray in Figure 1. Since the output pathways from the defense motivational mechanism to motor patterning mechanisms for defense are primarily descending ones, this illustrates why defense is not abolished after removal of the forebrain.

There is at least one major forebrain pathway that activates defense. Lesions of this pathway do not abolish defense, presumably because other motivating stimuli activate pathways that ascend to the defense motivational mechanism through the hindbrain and midbrain. Electrical stimulation of this pathway elicits coordinated defense patterns in the cat (Hess & Brugger 1943; Hunsperger 1956; and many other authors) and rat (Panksepp 1971). The pathway extends from the amygdala to the lateral preoptic area by way of ventral efferent fibers (Milton & Zbrozyna 1963) to the perifornical hypothalamus and then back to the midbrain central gray. The directionality of this pathway has been established by combined stimulation and lesion experiments (Fernandez de Molina & Hunsperger 1962; Hunsperger 1956). Thus, stimulation effects from the amygdala depend upon an intact hypothalamus and central gray, but not vice versa, and stimulation effects from the hypothalamus depend upon an intact midbrain central gray.

Details of the forebrain pathway activating defense have been revealed in experiments using brain stimulation. To some extent defense patterns are a function of the releasing and directing stimuli present during stimulation; thus if no attackable object is present, the stimulated cat may flee, but if a stuffed cat is present, then striking or biting may occur (Brown et al. 1969a). There may also be differences in motor patterns as a function of the locus of stimulation. Affective defense from the perifornical hypothalamus is obtained from a core region surrounded by an area from which fleeing responses are obtained (Hunsperger 1956). Similarly, in the amygdala there are two adjacent regions for affective

defense and for fleeing (Ursin & Kaada 1960). As will be discussed later, this may reflect an interaction between defense and submission systems.

The functional significance of the forebrain defense pathway is not known. Bilateral destruction of the perifornical region of the hypotha1amus in the cat does not impair affective defense against an attacking dog (Hunsperger 1956). There are other types of motivating stimuli for defense, however, that have not been tested in animals with forebrain lesions. In particular, pheromones and stimuli associated with neophobia might be expected to involve forebrain pathways. And while defense, as will be noted later, is inhibited by familiar animal odors, it may be facilitated by unfamiliar ones.

One particular surgical effect may be related to destruction of input pathways to the defense-motivational mechanism. Lesions of the far lateral midbrain at the level of its junction with the diencephalon in cats produce a syndrome in which defense behavior is greatly depressed. The cat fails to respond defensively to another attacking cat or dog; it has a high threshold for defense to painful tactile stimulation (Sprague et al. 1961) and does not show the hyperdefensiveness normally seen after lesions of the ventromedial hypothalamus (Kaelber et al. 1965; Glusman et al. 1961), The effect is apparently not due to interruption of medial lemniscal afferents to the thalamus and cortex, since lesions of the thalamic relay nuclei do not produce such an effect (Glusman et al. 1961). Instead, it may be due to interruption of afferents to the central gray from ascending tactile and pain pathways (Sprague et al. 1961) and from visual afferents as well, since lesions that abolish visually-motivated defense always involve undercutting the connections from the pretectum and superior colliculus to the central gray (Sprague et al l961).

Defense: Motor patterning mechanisms

There are a large number of motor patterning mechanisms activated by defense motivational mechanisms. In muroid rodents these mechanisms organize sideways or quadrupedal defense postures, defensive upright posture, freezing crouch, escape leaps and fleeing locomotion, lunge-and-bite attack, hissing, squeal or chit vocalization, urination, defecation, release of defense pheromones, activation of the adrenal medulla and the pituitary-adrenal axis and various warning or threat signals, including piloerection, teeth-chattering, tail-rattling, tail-raising, hind-foot thumping, and forefoot pattering (Adams; submitted for publication). In the cat many analogous motor patterns are activated during defense, including sideways and upright postures, escape leaps and fleeing locomotion, lunge-and-bite attack, hissing, screaming, urination, defecation, endocrine activation, and piloerection (Leyhausen 1956). In addition, striking is shown by cats during defense.

The defense patterns do not all occur at the same time but are organized in a graded hierarchical series corresponding to threat at low intensities and attack or escape at high intensities. This is apparently organized within the brain in terms of the strength of neural connections from the defense motivational mechanism to the motor patterning mechanisms. As shown in the classical work on brain stimulation of defense in cats (Hess & Brugger 1943), low-intensity brain stimulation of appropriate loci produces piloerection,

pupil dilation, and low-intensity vocalization. Intermediate intensities of stimulation produce higher intensities of the foregoing motor patterns along with postural effects such as sideways postures and arching of the back. High-intensity stimulation produces attack or escape, provided that the appropriate releasing stimuli are present. Apparently the motor patterning mechanisms of piloerection, vocalization, and other threat patterns have low thresholds for activation by the defense motivational mechanism, while motor patterning mechanisms of attack and fleeing have higher thresholds. Threshold differences are also indicated by the fact that the threat patterns have shorter latencies for activation by brain stimulation than do the attack and fleeing patterns.

Since most of the motor patterns for defense have been obtained after removal of the forebrain in rats and cats, it would appear, as noted earlier, that the motor patterning mechanisms lie at midbrain or hindbrain levels. The one exception, it may be assumed, is pituitary-adrenal activation, which accompanies defense; this would be expected to involve ascending pathways from the midbrain to the hypothalamus and thence to the anterior pituitary. Some evidence for such a pathway may be found in the work of Giuliani et al. (1961), who found that midbrain sections abolish the pituitary-adrenal response to ether anesthesia, abdominal surgery, electric shock, anoxia, and certain neurotransmitters. As one would expect, electrical stimulation of the region just lateral to the central gray activates ACTH secretion in the chronic cat (Slusher & Hyde 1966).

The efferent fibers from the midbrain central gray projecting to various caudal neural structures that presumably function as motor patterning mechanisms may correspond to those fibers that leave the central gray in a radial stream pattern throughout the lateral extent of the midbrain. These fibers have been called Weisschedel's radiations (Nauta 1958). One set of such fibers has been traced and related to the function of hissing and screaming vocalization in the cat. Fibers of this pathway leave the midbrain central gray laterally and swing posteriorly and ventrally to travel in the ventral pons beneath the medial lemniscus (Magoun et al. 1937; Kanai & Wang 1962; Berntson 1972); their ultimate destination, and the location of the motor patterning mechanisms for hissing and screaming, have not been determined, however.

The motor patterning mechanisms for freezing and fleeing may include the so-called "locomotor region" of the midbrain. This is a region in the midbrain tegmentum beneath the inferior colliculus, where electrical stimulation produces running in the cat, and where single-pulse stimulation produces monosynaptic excitation on reticulospinal neurons thought to provide the "throttle" for locomotion in the cat (Orlovsky 1970). This region, often called the cuneiform nucleus, receives a major input from the central gray (Hamilton & Skultety 1970). Excitation of these neurons would presumably cause fleeing, and inhibition would cause freezing.

Defense: Releasing and directing stimuli

The releasing and directing stimuli for the defensive upright posture have been studied in detail in the rat. Vibrissal or facial tactile stimuli are necessary to release the behavior in naive animals (Thor & Ghiselli 1975), although visual stimuli may suffice to release it in experienced animals (Kanki & Adams 1978). Vibrissal stimuli are necessary for directing the behavior; this is shown by the fact that an experienced animal will continue to show upright posture in response to visual releasing stimuli following removal of the vibrissae, but the posture is not properly oriented with respect to the opponent (Kanki & Adams 1978).

The neural mechanisms of vibrissal and visual-releasing stimuli for the defensive upright posture are known in some detail for the rat. Vibrissal releasing stimuli require only subcortical mechanisms, since they remain effective after bilateral destruction of projections to the thalamic relay nucleus for tactile sensation (Kanki & Adams 1978). Presumably, therefore, the main pathway goes more or less directly from the trigeminal complex to the motor patterning mechanisms of the upright posture, as illustrated in Figure 1.

There is an alternative set of pathways by which visual stimuli can release the upright posture in experienced animals This pathway includes neocortical structures; visual releasing stimuli are unaffected by lesions of the superior colliculus but are rendered ineffective following lesions of the visual cortex (Adams & Severini 1977). The cortical circuitry involved in visually-released boxing includes the ventrobasal thalamic relay nucleus for tactile sensation, since visual stimuli can no longer release the behavior following bilateral destruction of this nucleus (Kanki & Adams 1978). Apparently the visually-released boxing, which is a learned behavior, is elaborated by a cortical system that combines its visual input with the thalamocortical projections of the tactile system.

Although the releasing and directing stimuli of biting in cats during interspecific attack have been studied in some detail (Flynn 1972), the role of these stimuli in the biting in cats or rats during intraspecific defense has not been systematically studied. One may assume, as in the case of the upright posture, that they involve the trigeminal complex, since vibrissal and facial tactile inputs appear to be very important.

The motor pattern of fleeing probably requires releasing stimuli that are processed by a complex forebrain circuitry concerned with a "cognitive map" of a flight path or escape route. Behavioral evidence for this assumption includes observations that animals flee more readily if placed into a familiar place (Metzgar 1967), and that they do not attempt to flee if they know that there is no escape from the test chamber (Blanchard et al. 1976). According to a review of his many studies on this question, Thompson (1978) lists the following forebrain structures as critical for escape: the caudate nucleus and putamen, globus pallidus, entopeduncular nucleus, subthalamus, and ventromedial thalamus (all of which are connected by the lateral forebrain bundle), and the anterior thalamus. Reduction or abolition of escape following midline thalamic lesions has also been noted in both the cat

(Mitchell & Kaelber 1966) and the rat (Bohus & deWied 1967). We have found in our laboratory that the midline thalamic lesions that abolish escape behavior in response to shock do not affect upright posture and boxing response to the shock This suggests that the effects of these lesions are specific to the motor patterning mechanism of escape - that is, its releasing and directing stimuli - and do not affect the motivating stimuli for defense in general.

There are no data to indicate that specific releasing or directing stimuli are necessary for activating the motor patterning mechanism for hissing or squealing.

Submission

Submission differs from defense on a number of levels. In terms of its motor patterns, submission includes the full submissive posture and ultrasound (in rats), while defense includes a lunge-and-bite attack and striking (in cats). Other motor patterns are similar for defense and submission. In terms of motivating stimuli, submission is shown against an animal's consociates (i.e. familiar conspecifics or other animals with whom it has lived), and defense is shown against other animals. In terms of life history determinants, defense is shown primarily by wild animals, and submission is shown primarily by tame or laboratory animals. One may assume that the process of taming wild animals consists, in part, in shifting the dominant behavior from defense to submission. Functionally, defense is often damaging, while submission is not. The defensive animal is a dangerous one, likely to inflict a bite on the face of its opponent (or the hand of an experimenter), while the submissive animal is a vulnerable one, likely to show a full submissive posture that might inhibit conspecific offense but would not be expected to be effective against a predator. Phylogenetically, defense probably evolved first to deal with predators, and the submission system evolved later to modify defense behavior when the animal was confronted with a conspecific whose offense behavior could be inhibited by particular submissive postures. Finally, as will be reviewed below, defense and submission depend upon different, although parallel, neural substrates. In particular, lesions of the amygdala may enhance submission, while lesions of the septum and ventromedial hypothalamus enhance defense.

Most of the motor patterns and motivating inputs of defense and submission are similar. This suggests that their respective motivational mechanisms consist of sets of homogeneous neurons with very similar neural connections, and that they may have the same locus, submission having evolved as a subset of defense during the course of phylogeny. As in the case of defense, the motor patterns of submission appear to be organized in a graded hierarchical series depending upon intensity of activation of the submission motivational mechanism and differential thresholds of activation of the various motor patterning mechanisms. Since most of the motor patterning mechanisms of the two systems may be shared, their differential thresholds would be expected to be the same for both systems.

I suggest that there is a mechanism that switches the behavior of an animal from defense to submission in the presence of a consociate, or familiar individual. Such a mechanism may be called a "consociate modulator." In a previous publication (Adams 1977) I suggested

that it should be called a "conspecific defense modulator," but upon further reflection I think that the term should be "consociate modulator" because there are old experiments that show that behavior towards individuals is modified by familiarity, even if they are not members of the same species; for example, cats can be made to live peacefully with rats rather than treating them as prey (Kuo 1930). And I have dropped the term "defense," because such a mechanism may ultimately turn out to modulate other behaviors besides defense, such as feeding and sex.

The consociate modulator, according to the present formulation, is a set of homogeneous neurons (similar to those of motivational mechanisms) that is activated by stimuli associated specifically with consociate animals, and that, in turn, facilitates the submission motivational mechanism and inhibits the defense motivational mechanism. For rats, the stimuli that activate the consociate modulator are probably olfactory; in cats the cues may come from other sensory modalities as well.

A number of lines of evidence, none of them compelling when considered alone, have suggested to me that the ventromedial nucleus of the hypothalamus may be the site of the hypothetical consociate modulator. These lines of evidence include: a reanalysis of the results of single-neuron recording from the ventromedial hypothalamus during affective defense in the cat (Adams 1968); consideration of data on the neuronal circuitry and the inputs of the ventromedial hypothalamus; an attempt to explain why fleeing and affective defense are obtained from different (although neighboring) zones with electrical stimulation of the forebrain; and speculations on the potential interactions of defense and submission in the forebrain, which might explain why forebrain lesions can release defense.

A reanalysis of single-unit recording results from the ventromedial nucleus of the cat during affective defense suggests that its neurons may be active during submission rather than during defense. In my original study I was quite surprised to find that neurons of this nucleus were not active during affective defense, despite the general belief expressed in the literature that cells in this region should be involved in aggressive behaviors (Adams 1968). One neuron recorded in the capsule of the ventromedial nucleus acted in a way that I could not interpret at the time. It fired at high rates if and only if the animal cowered or tried to escape from its opponent. This neuron was not reported in the publication, however, because I could not record from it during affective defense, which was, after all, the purpose of the study. In what at the time seemed an unfortunate occurrence, the cat refused to show affective defense during testing of the neuron, and instead simply cowered and tried to flee. This event reflected a bias I had built into the design of the experiment: it focused on defense and not submission. Those cats (about 50%) in the colony who showed cowering and flight (i.e. submission) were not used for the experiment; only those who showed consistent affective defense were implanted with the recording electrodes.

The neural connections of the ventromedial nucleus of the hypothalamus are consistent with what one would expect in a consociate modulator. Neurons of the ventromedial nucleus receive convergent inputs from the amygdala, septum, and midbrain tegmentum, including the midbrain central gray (Dreifuss & Murphy 1968; Tsubokawa & Sutin 1963),

as well as anatomically-demonstrated inputs from the anterior hypothalamus (Chi 1970). The amygdala, in particular, is a brain structure that one would expect to be involved in processing the olfactory stimuli that make possible the recognition of consociates (Gloor 1978). The septum may be involved in discriminating conspecific opponents in terms of their vibrissal "jitter" rates, since both septal lesions and abolition of vibrissal jitter in the opponent lead to biting by rats in response to shock (Kanki & Adams 1978). Inputs from the amygdala and septum not only facilitate ventromedial neurons, but they also produce a complex excitatory-inhibitory effect thought to reflect complex feedback and feed-forward inhibition (Murphy 1972; Ono & Oomura 1975), such as one might expect from an "and-gate" logical device. There is also a major projection from the ventromedial nucleus to the midbrain central gray (Saper et al. 1976; Morrell et al. 1978); this corresponds to what one would expect if a ventromedial consociate modulator were the main input to a submission motivational mechanism in the central gray.

The outlines of a hypothetical neural circuitry controlling submission are shown in Figure 2. Afferent pathways from the amygdala, septum, and anterior hypothalamus, activated by stimuli from familiar consociates, converge upon a consociate modulator located in the ventromedial hypothalamus, which then sends projections activating the submission motivational mechanism in the midbrain central gray. At some point there is a logical and-gate that operates only when *both* inputs to the consociate modulator and motivating stimuli for defense and submission are simultaneously activated. The exact place where these inputs converge is not known; perhaps it is at the level of the ventromedial hypothalamus, or perhaps, as shown in the figure, it is at the level of the central gray. If the convergence occurs at the ventromedial nucleus, then there would have to be projections from the central gray to the ventromedial hypothalamus. There are some data indicating central gray projections to the ventromedial hypothalamus on anatomical (Szentagothai et al. 1962) and physiological (Beyer et al. 1962; Tsubokawa & Sutin 1963) grounds, but other anatomical studies have instead found that the central gray projections go only as far as the posterior hypothalamus (Chi 1970; Hamilton & Skultety 1970).

If one assumes that there are separate, parallel pathways for submission and defense in the forebrain, then it is possible to explain many peculiarities in the results of electrical stimulation. In some loci it is possible to obtain the full range of defense patterns by electrical stimulation; these include freezing, fleeing, and lunge-and-bite attack. In other loci it is possible to obtain freezing and fleeing, but without any accompanying affective defense or lunge-and-bite attack. Stimulation at the former loci, we may propose, activates the forebrain pathways that convey motivating cues for defense and submission. Stimulation at the latter loci, however, may activate the consociate modulator, which facilitates submission but not defense. In the cat, fleeing without affective defense may be elicited from a so-called "flight" zone of the amygdala (Ursin & Kaada 1960), and from areas that surround the perifornical region of the hypothalamus where defense may be stimulated; these areas include the anterior hypothalamus, lateral hypothalamus, and ventromedial hypothalamus, and certain areas of the central gray of the midbrain (Hunsperger 1956; Adams 1968). In the rat, fleeing without lunge-and-bite attack may be

elicited from comparable regions, including the anterior hypothalamus (Woodworth 1971), lateral hypothalamus (Panksepp 1971), ventromedial hypothalamus (Woodworth 1971), and midbrain central gray (Waldbillig 1975). In the rat, in which boxing may be considered as a submissive response, dopaminergic stimulation of the corticomedial amygdala (Rodgers et al. 1976) and cholinergic stimulation of the basolateral amygdala (Rodgers & Brown 1976) can increase boxing in response to footshock without facilitating the lunge-and-bite response of defense.

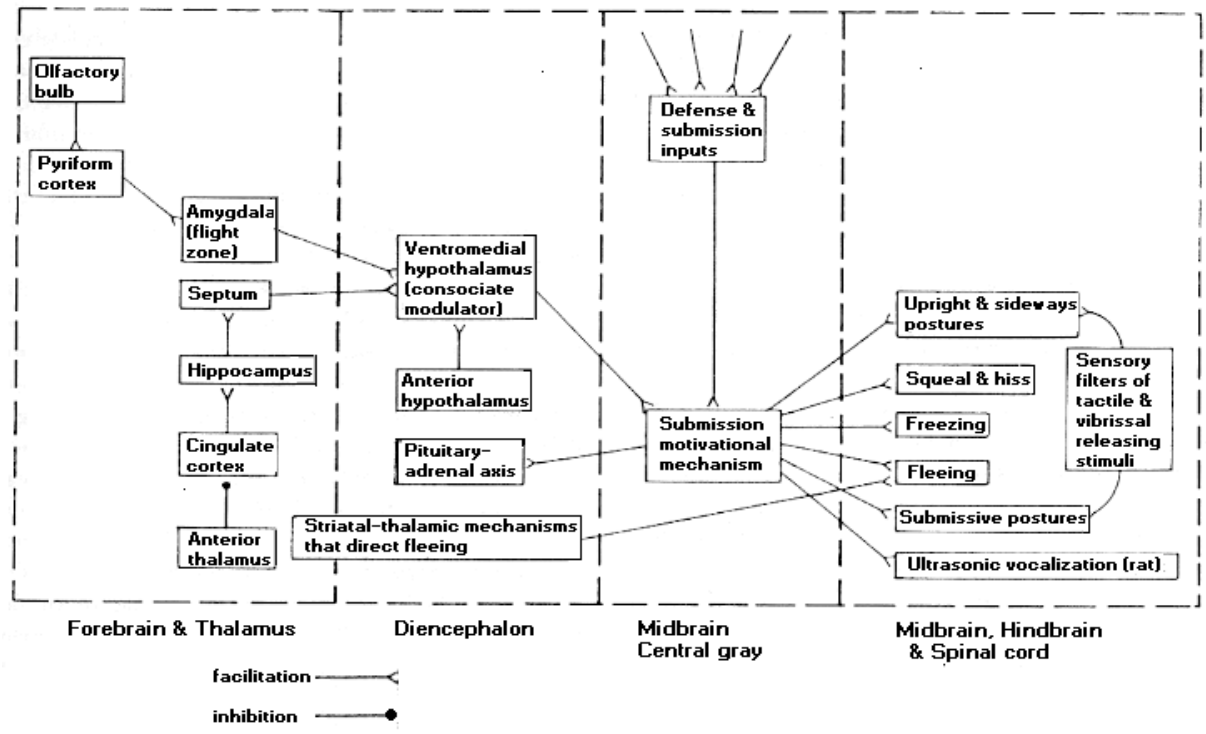

Figure 2. Neural circuity for submission. Most of the inputs and outputs of the submission motivational mechanism, located in the midbrain central gray, are similar to those for defense, as shown in Figure 1. Motor patterning mechanisms differ in several respects, including ultrasound and submissive postures, and not including striking (in the cat) and the lunge-and-bite attack. The most significant difference consists of a necessary input from a "consociate modulator" (see text) located in the ventromedial hypothalamus, which receives motivating inputs from the fleeing control zone of the amygdala as well as from a circuit including the anterior thalamus-cingulate cortex-hippocampus-septum. These motivating inputs reflect familiar consociate stimuli. Another major input to the ventromedial hypothalamus comes from the anterior hypothalamus, but its functional significance is not known.

There is a report, from work on the rat, that if electrical stimulation is confined within the ventromedial hypothalamus, the effect is suppression of defense without fleeing (Veening 1975). This might be predicted in case there was no stimulation of the perifornical system, which conveys motivating stimuli for defense and submission. In cats, on the other hand, stimulation of the ventromedial nucleus may produce either defense or fleeing (submission), perhaps by stimulation of the dendrites of perifornical neurons that extend into the ventromedial nucleus (Millhouse 1969).

The existence of separate parallel pathways for submission and defense is also indicated by the results of experiments in which two points are stimulated simultaneously, or in which both electrical and chemical stimulation are used. Whereas electrical stimulation in the hypothalamus of the cat produced defense (called "attack" by the author), chemical stimulation with carbachol from the same electrode produced submission (called "fear" by the author); when the chemical-stimulation effect was blocked, the electrical-stimulation effect remained intact (Baxter 1967). In another experiment it was shown that simultaneous stimulation of two fleeing points or two affective defense points in the cat summated in their effects, whereas simultaneous stimulation of a fleeing point and a defense point canceled each other's effects (Brown et al. 1969b). Further arguments in support of separate systems of submission ("flight") and defense may be found in Kaada (1967).

The effects of forebrain lesions on defense and submission may be explained if one assumes that the parallel pathways of defense and submission in the forebrain have reciprocal inhibitory interactions. Two such inhibitory connections have been illustrated in Figure 3, one from the amygdala to the ventromedial hypothalamus, and a second from the ventromedial hypothalamus to the defense mechanism in the midbrain central gray. According to this model, lesions that disrupt the forebrain pathways of submission should release the defense from inhibition, making the animal more likely to show a lunge-and-bite or striking attack. This is the reason, it is suggested, why lesions of the following structures increase defense behavior: ventromedial hypothalamus in the rat (Anand & Brobeck 1951) and cat (Wheatley 1944); septum in the rat (Brady & Nauta 1953) and cat (Spiegel et al. 1940): anterior hypothalamus in the rat (Maire & Patton 1954) and cat (Fulton & Ingraham 1929), and secondary olfactory structures in the rat (Cain 1974) Most of these effects have been confirmed by many authors, although it should be noted that the effects of septal lesions are transitory and not always observed (Slotnick et al 1973; Sodetz et al 1967). Many authors have concluded that denervation supersensitivity must be involved, because the resulting defense behavior is quite strong. This may be true, but on the other hand it may reflect a subjective judgment on the part of observers who have not seen the defense behavior of wild animals. Although the lesion-induced defense is excessive in comparison to that of laboratory rats and house cats, it may appear normal if compared to that of wild-trapped rats and feral cats.

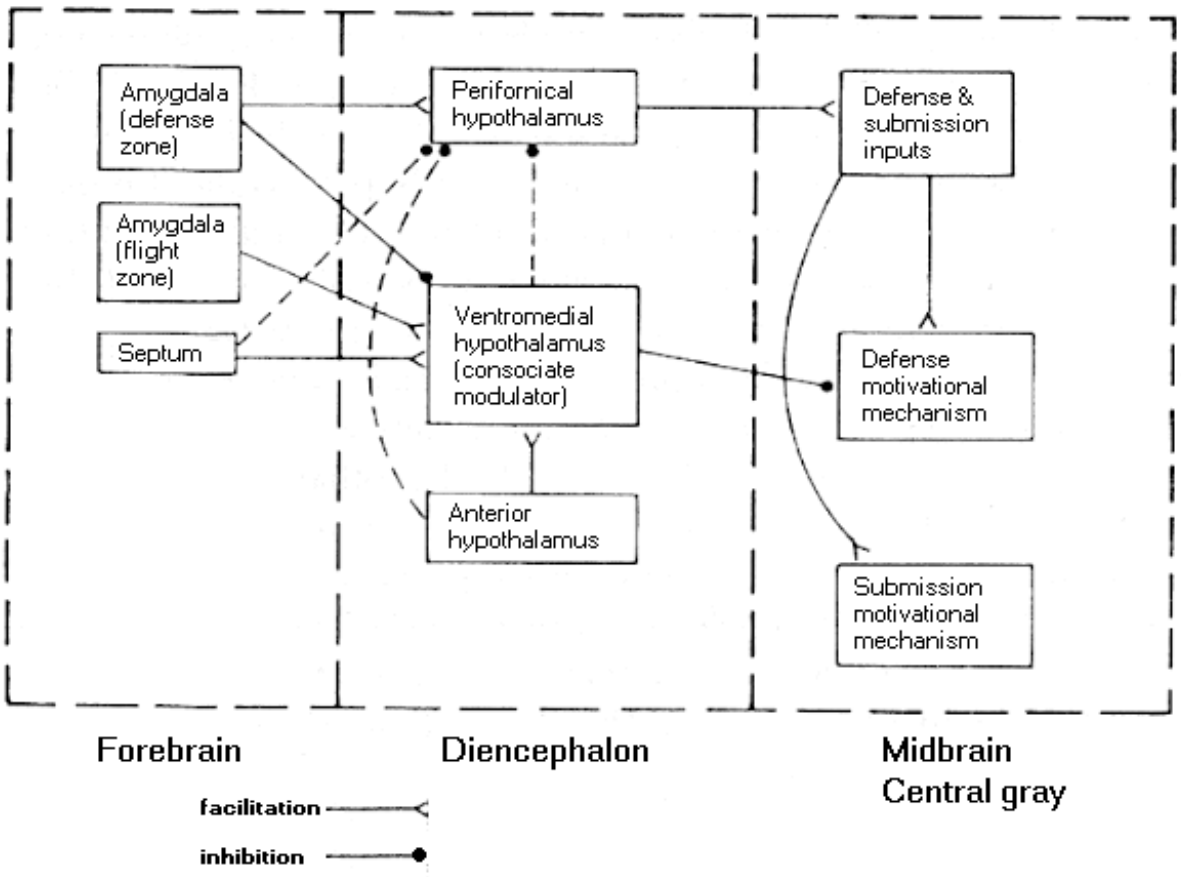

Figure 3. Interactions between defense and submission. Pathways facilitating defense are shown above, and those facilitating submission are below. The principle interactions are mediated by two inhibitory pathways, shown as solid lines, from the defense zone of the amygdala to the consociate modulator of the ventromedial hypothalamus, and from the ventromedial hypothalamus to the defense motivational mechanism in the midbrain central gray. Other possible inhibitory pathways, shown as dotted lines, include one from the ventromedial hypothalamus to the perifornical portion of the forebrain defense pathway, one from the septum to the forebrain defense pathway, and one from the anterior hypothalamus to the forebrain defense pathway.

Lesions that destroy the amygdala would be expected to have the opposite effect, decreasing defense behaviors. The effect should be complicated, however, according to the model. Lesions would: 1) destroy inhibitory projections to the ventromedial hypothalamus, thus releasing its inhibition of defense; 2) destroy afferents of the forebrain pathways that provide motivating inputs for both defense and submission; and 3) destroy a part of the afferent system to the ventromedial nucleus that responds to consociate stimuli. Of these three effects, the first might be expected to dominate, since both the second and third

destroy only *part* of the relevant inputs to their target nuclei. Destruction of the remaining afferents to those nuclei might reverse the effect, however. For this reason, it may be possible to explain why lesions of the amygdala have a pronounced taming effect in the rat (Woods 1956) and cat (Schreiner & Kling 1953), but the taming effect is reversed following additional lesions of the septum in the rat (King & Meyer 1958). According to the present analysis, the taming effect should depend upon the ventromedial nucleus, which may explain why lesions of the ventromedial nucleus reverse the taming effect produced by amygdaloid lesions (Kling & Hutt 1958). If the inhibitory projections to the ventromedial nucleus are M-cholinergic, this may explain why M-cholinergic blockade in the ventromedial nucleus suppresses affective defense produced by chemical stimulation in the midbrain central gray of the cat (Romaniuk & Golebiewski 1977).

Stimulation of structures that project to the ventromedial hypothalamus should facilitate submission and suppress defense according to the model. Considerable published data support this prediction. In rats, electrical stimulation of the septum suppresses defense (lunge-and-bite attack) previously enhanced by ventromedial hypothalamic lesions (Brayley & Albert 1977) and inhibits affective attack in cats (Siegel & Skog 1970). Similar stimulation facilitates submission (fleeing) in response to footshock or electrical stimulation of the central gray and adjacent tegmentum in rats (Gardner & Malmo 1969) and facilitates hissing and escape from hypothalamic stimulation in cats (Siegel & Skog 1970). In cats, chemical stimulation of the amygdala suppresses defense behavior previously enhanced by chemical stimulation of the hypothalamus (Decsi & Nagy 1974); chemical stimulation of the hippocampus, which provides the main input to the septum, can also suppress defense that has been enhanced by chemical stimulation of the hypothalamus (Nagy & Decsi 1974), while the opposite effect, as one would predict, is produced by lesions of the hippocampus, especially if combined with neocortical damage (Rothfield & Harman 1954). There is a parallel between the latter finding and that of Yutzey et al (1964) that neocortical damage prolongs the hyper-defensiveness of rats produced by septal damage.

There are two thalamocortical circuits that appear to modulate the balance between submission and defense. In one circuit, involving projections from the medial dorsal thalamus to the fronto-orbital cortex, there is apparently an inhibitory projection, since the former appears to inhibit defense and facilitate submission, while the latter has the opposite effects. Thus, stimulation of the medial dorsal thalamus produces submission ("fear") in cats (Roberts 1962), and lesions of the medial dorsal thalamus increase affective defense in cats (Schreiner et al 1952), while lesions of the fronto-orbital cortex increase boxing but not biting during shock-elicited fighting in rats (Kolb & Nonneman 1974); the latter would appear to represent increased submissive behavior. In another circuit, involving projections from the anterior thalamus to the cingulate cortex (and thence to the hippocampus), there is again a reciprocal relationship. since the former appears to inhibit submission and facilitate defense, while the latter appears to facilitate submission and inhibit defense. Thus, lesions of the anterior thalamus increase submission and suppress defense in the cat (Schreiner et al 1952), while stimulation of the cingulate cortex and its

fiber tract, the cingulum, produces submission (called "fear" by the authors), and lesioning them increases defense behavior in the cat (Koridze & Oniani 1972).

There may be other points in the forebrain where there are inhibitory interactions between the defense and submission systems. These have been indicated by dotted connections in Figure 3. From their work with knife cuts and injections of blocking agents in the rat, Albert & Richmond (1977) came to the conclusion that the septum and olfactory structures inhibit the defense pathways in the lateral hypothalamus. This conclusion is further warranted by the finding by Brayley & Albert (1977) that septal stimulation suppresses defense even after ventromedial hypothalamic lesions. It is also possible that there are inhibitory projections from the ventromedial hypothalamus, not only to the defense motivational mechanism in the midbrain, but also to the defense pathway in the lateral hypothalamus. This could explain why knife cuts between the medial and lateral hypothalamus increase defense behavior in the rat (Paxinos & Bindra 1972; Sclafani 1971).

Offense

The offense motivational mechanism. **The neural basis of offense has not been investigated in the cat (Flynn 1976), and only occasionally in the rat; therefore, there are few data available on the question of the neuroanatomical locus of the hypothetical offense motivational mechanism. It is probably not located in the forebrain, since, as noted earlier from studies by Olivier (1977) and Adams (1971), all of the motor patterns of offense have been displayed in coordinated fashion by the rat following destruction of the hypothalamus. It probably lies in the midbrain in a location that receives projections from the lateral hypothalamus, since lesions of the latter abolish offense (Adams 1971), while electrical stimulation produces offense (Panksepp 1971; Woodworth 1971; Koolhaas 1978). There is one report on a midbrain lesion that abolishes offense in rats; the locus was the ventral raphe nucleus (Kostowski & Valzelli 1974).**

Motivating stimuli for offense. **In muroid rodents there are three primary types of facilitative motivating stimuli for offense (Adams, submitted for publication). The first is effective in males only and consists of pheromonal stimuli that depend upon testosterone in the opponent. The filter for these stimuli is presumably activated by testosterone and, for that reason, is not normally present in females. The second is effective in both males and females and consists of stimuli, principally olfactory, that identify the opponent as an unfamiliar conspecific. The third is present in both males and females, and consists of a complex stimulus situation that elicits competitive fighting. This type of fighting is elicited in animals who have been deprived of food or water and are given a limited supply such that they must compete with an opponent for acquisition (Zook & Adams 1975). [See also Toates: "Homeostasis and Drinking" BBS 2(1) 1979.]**

Laboratory rats and mice differ from wild muroid rodents and other laboratory species such as gerbils and hamsters in depending primarily upon the motivating stimulus of testosterone-dependent pheromones. For that reason, laboratory rat and mouse females show little offense except in a competitive fighting situation, whereas among wild rats and

mice, gerbils, hamsters, and so forth, females are often as likely as males to attack unfamiliar conspecifics. Also, laboratory rats and mice generally do not show intermale fighting after destruction of the olfactory bulbs, while other species continue to show fighting with unfamiliar conspecifics. The reason for these differences is not known. It may depend upon a selection against offense in the breeding of laboratory mice and rats, which has reduced the effectiveness of olfactory filters for motivating stimuli tuned to unfamiliar conspecific odors. It may also depend, in part, upon the relatively homogeneous diet and bacterial flora in laboratory colonies, which may reduce the variability in odors among laboratory animals, making all the animals in the colony relatively "familiar."

The sensory filters for the motivating stimuli of offense tuned to unfamiliar conspecific odors are apparently located in the amygdala, while those tuned to testosterone-dependent odors are apparently located in more medial structures such as the septum or preoptic area. This conclusion is based upon apparently contradictory data concerning the effects of amygdaloid lesions upon offense

Lesions of the amygdala disrupt offense in some types of muroid rodents but not others. They disrupt offense in those animals that do not depend upon testosterone-dependent pheromones as motivating stimuli for offense: in male hamsters (Bunnell et al. 1970; Shipley & Kolb 1977), and in wild rats (Galef 1970). They do not disrupt offense in those animals that depend primarily upon testosterone-dependent pheromones as motivating stimuli: male laboratory rats (Busch & Barfield 1974; Bunnell 1966). The role of the amygdala as a sensory filter for unfamiliar conspecific stimuli that activate offense is complementary to its role as a sensory filter for consociate stimuli activating the hypothalamic switching circuit for defense and submission. The critical input to the amygdala for offense may come from the vomeronasal organ and the accessory olfactory bulb, since these structures project to the corticomedial amygdala, where lesions abolish offense in the hamster (Lehman et al. 1978). The output pathway may be the stria terminalis through the medial preoptic area to the lateral hypothalamus; this might explain why lesions of the medial preoptic area, but not of the anterior hypothalamus, disrupt offense in female hamsters (Hammond & Rowe 1976).

In muroid rodents the sensory filters for the offense stimuli that are dependent upon testosterone and tuned to testosterone-dependent pheromones may be located in the septum or preoptic hypothalamus. In one study, implantation of testosterone in the septum reinstated intermale fighting (offense) in laboratory mice after it had been abolished by castration (Owen et al, 1974). A more recent study, employing a similar experimental design in the laboratory rat, found that implantations in the preoptic area were more effective (Bermond 1978).

There are other experimental data indicating that the septum modulates offensive behaviors in various muroid rodents. In the mouse (Slotnick & McMullen 1972) and rat (Lau & Miczek 1977; Blanchard et al. 1977), septal lesions reduce offense behavior. In the hamster, septal lesions increase offense (Sodetz & Bunnell 1970; Johnson et al. 1972), and septal stimulation inhibits it (Potegal et al. 1978). The functional significance of these effects

is not known. By analogy with its role in submission and defense, one might expect the septum to process information indicating that the opponent was a conspecific, which might be expected to facilitate offense. Another possibility is that septal activation reflects familiarity or lack of familiarity with the test environment. The septum receives a major input from the hippocampus, which has been implicated in the process of spatial recognition (Q'Keefe & Nadel 1978). This may be important for offense, since offense is more readily exhibited by an animal when it knows that it is in a familiar "home" environment or territory (Adams 1976).

The motivating stimuli for competitive fighting are probably processed in the amygdala and hypothalamus. Miczek et al. (1974) have found that lesions of the cortical amygdala, periamygdaloid cortex, and stria terminalis abolish fighting in rats during food competition. These data are supplemented by findings that amygdaloid lesions in the rat reduce "incentive motivation" (Gaston 1978) and reduce the "frustration effect" of non-reward in a feeding situation (Henke 1973). There are reports that ventromedial hypothalamic lesions (Grossman 1972) and knife cuts between the ventromedial and anterior hypothalamus (Grossman & Grossman 1970) abolish competitive fighting in female rats. A thesis in our laboratory (Severini 1973) found that anterior hypothalamic lesions abolished the competitive fighting, but that ventromedial hypothalamic lesions did not. These contradictory findings indicate that further research is needed on the role of the hypothalamus in competitive fighting.

The neural mechanisms of offense are outlined in Figure 4. The motivating stimuli for offense are shown as activating a number of forebrain nuclei, with unfamiliar conspecific stimuli activating the amygdala and septum, male odors activating the olfactory tubercle (in males), and the stimuli of competitive fighting activating the amygdala. These influences are shown as converging upon a pathway that runs in the lateral hypothalamus from the preoptic hypothalamus caudally and into the midbrain to an offense motivational mechanism.

Motor patterning mechanisms and releasing and directing stimuli for offense. **The motor patterning mechanisms for offense are similar in the rat and cat. In the rat and other muroid rodents they consist of approach locomotion, offensive sideways posture, offensive upright posture, and the bite-and-kick attack (Lehman & Adams 1977). In the cat they consist of approach, a straight-backed sideways posture, and a biting attack (Leyhausen 1956). In both cat and rat, offense is accompanied by piloerection. Both the sideways and upright offense postures in the rat are probably coordinated by the same motor patterning mechanisms as the corresponding postures of offense (Lehman & Adams 1977). The neural mechanisms for the offense motor patterns of the cat and rat have not been specifically studied. However, if it is true that the patterning mechanism for offensive upright posture is the same as that for defensive upright posture, then the same releasing and directing stimuli would be involved. As mentioned earlier, these have been studied in our laboratory (Kanki & Adams 1978). The releasing and directing stimuli for the other offense motor patterns have not been specifically studied. It would appear from casual observations that**

most of the motor patterns require visual releasing and directing stimuli, since they can occur at distances from the opponent that are beyond the reach of tactile communication.

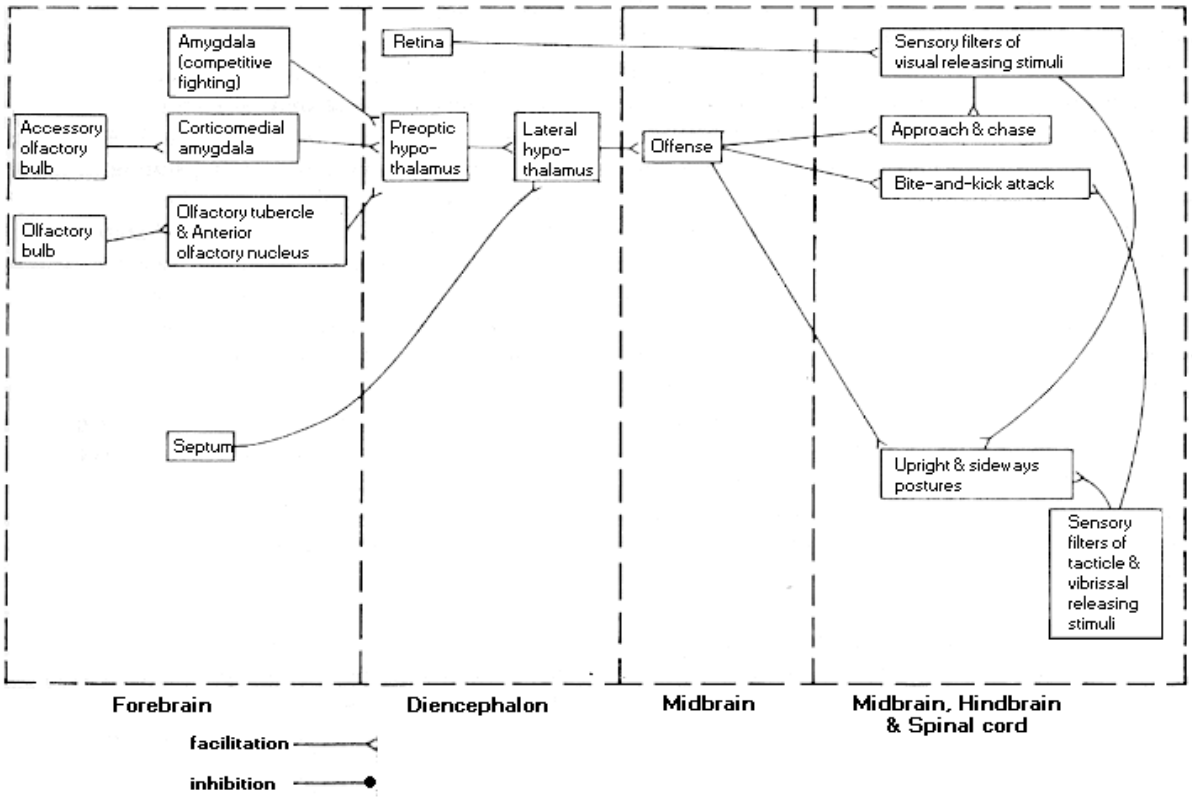

Figure 4. Neural circuitry for offense. Various types of motivating stimuli activate the amygdala (competitive fighting stimuli), corticomedial amygdala (unfamiliar conspecific stimuli), olfactory tubercle or anterior olfactory nucleus (testosterone-dependent pheromones, effective only in males), and septum (familiar territory?). These motivating influences are conveyed to a midbrain motivational mechanism for offense by way of the medial forebrain bundle in the lateral hypothalamus. The offense motivational mechanism activates motor patterning mechanisms for approach and chase, bite-and-kick attack, and upright and sideways postures. These mechanisms do not produce motor patterns unless they simultaneously recieve appropriate visual or tactile releasing stimuli.

Data from primitive mammals and from primates

Although most available data are from studies on cats and rats, it is useful from the standpoint of an evolutionary analysis to examine data from the opossum and from primates as well. The opossum can tell us something of the general evolution of brain mechanisms of aggression in mammals as a whole, since it is similar to primitive mammals who evolved tens of millions of years ago. The primates can tell us something of the evolution of brain mechanisms for aggression in humans. In general, as will be noted, there are more points of similarity than difference among all these animals. The interpretation of data from the opossum and from primates is hampered by lack of an ethological analysis that could distinguish motivational systems of offense, defense and submission.

Midbrain mechanisms have been studied in the rhesus macaque. A study, completed over 40 years ago by Magoun et al. (1937) showed clearly that the midbrain pathways for defensive vocalizations and facial expressions are the same in the monkey as in the cat. Stimulation of the central gray has also been carried out in freely-moving macaques in a social situation, in which case it elicits a full range of defensive (and offensive?) motor patterns including chasing, jumping, biting, and fighting (Delgado 1963). Although midbrain studies, as such, have not been performed in opossums, it was the tentative conclusion of Bergquist (1970) that the main output pathway from the hypothalamus concerned with defense in the opossum is the periventricular fiber system to the midbrain central gray, just as it is in the cat.

There is a major forebrain pathway for defense in the opossum and in primates that may be activated by electrical stimulation of the lateral hypothalamus in preoptic and perifornical regions. This has been demonstrated in the opossum (Roberts et al. 1967; Bergquist 1970), the rhesus macaque (Robinson et al. 1969; Alexander & Perachio 1973), and the squirrel monkey (Renfrew 1969). The anatomical sites for stimulation of defense are similar to those in the cat and the rat, and the elicited behaviors appear to be homologous to biting defense in the rat and affective defense in the cat.

As in the cat and rat, there are two separate but adjacent zones of the hypothalamus and amygdala of the rhesus monkey from which defense (including attack) and submission (including only escape) can be obtained by electrical stimulation (Perachio & Alexander 1975). This suggests that there may be a consociate modulator in primates as well, and that it may switch the animal from defense to submission.

The taming effects of amygdala lesions, mentioned earlier with regard to the cat and rat, were originally described in primates (Kluver & Bucy 1939). The effect has been carefully studied in primate group social interactions (Rosvold et al. 1954). It has also been reported for the opossum (Hara & Myers 1973).

Septal lesions, which sometimes increase defense in cats and rats, have not increased defense and submission in primates and opossums. Defense reactions to tactile stimulation in opossums remain unchanged after septal lesions, while defense behavior due to visual

and auditory stimulation is temporarily decreased (Hara & Myers 1973). Emotional behavior of squirrel monkeys (Buddington et at 1967) and macaques (Votaw 1960) is not changed by septal lesions. As noted earlier, the effects of such lesions in cats and muroid rodents are also variable from species to species and experiment to experiment.

Thalamocortical lesions in primates yield effects similar to such lesions in the cat and the rat. Lesions of the cingulum decrease defense (Glees et al. 1950), just as they do in the cat (Koridze & Oniani 1972). Lesions of the orbital frontal cortex of rhesus monkeys increase submission (called "aversive reactions" by the authors) and decrease defense (called "aggressive reactions") in response to a doll or a snake (Butter et al. 1970).

One report on primates is difficult to compare with data from the cat and rat: lesions of the globus pa1lidus abolish the penile display of squirrel monkeys, which has been interpreted as an "aggressive" motor pattern (MacLean 1978). One would presume that this would correspond to offense in the cat or rat, although the relevant comparative ethological analysis has not yet been undertaken. There are no comparable data on pallidal or other striatal lesions abolishing offense in other mammals, although there are suggestive data in the finding that dopamine metabolism in the mouse changes as a function of fighting (Hutchins et al. 1975).

Discussion

There are many parallels in the neural circuitry of offense, defense, and submission. These may be seen in the composite diagram of the neural circuits presented in Figure 5. All three involve forebrain pathways that process motivating stimuli in the septum and amygdala convey these influences to the hypothalamus, and then relay them caudally to motivational mechanisms in the midbrain. The motivational mechanisms with known locations are in the central gray of the midbrain, and the other motivational mechanism (offense) may he there as well. From the central gray, descending projections fan out to motor patterning mechanisms; although their exact locations are not known, most are presumed to lie in the midbrain and hindbrain, since most motor patterns can be obtained in decerebrate animals. Releasing and directing stimuli may require forebrain pathways, however.

Offense, defense, and submission may be differentiated by appropriate brain lesions, despite their many parallels. Offense and defense may be differentiated by hypothalamic lesions that, if very extensive, can abolish offense while enhancing defense (Adams 1971). Defense and submission can be separated by lesions of the medial hypothalamus or amygdala; the former enhance defense and abolish submission, while the latter have an opposite effect.

Whereas previous classifications of aggressive behavior have been based upon logical distinctions made by the observer, the present classification is based ultimately upon the neural circuitry involved. This leads to differences in the conclusions reached. In particular, the present conclusions may be compared to three previous classifications based upon

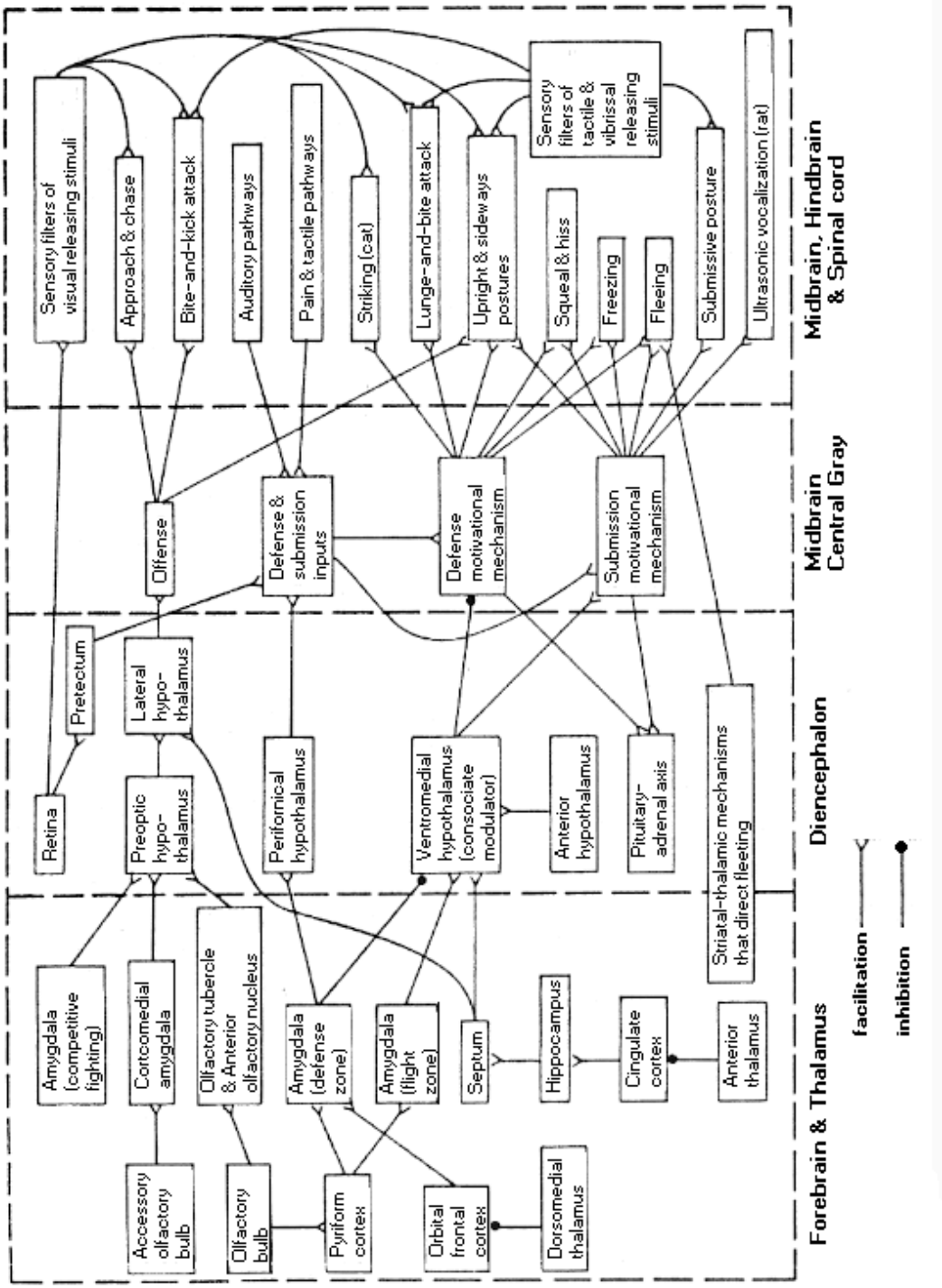

Figure 5:Composite diagram of offense, defense, and submission neural circuitry. This figure is composed from an overlay of Figures 1-4; detailed description may be found in the earlier figure captions.

stimulus situations eliciting aggression (Moyer 1968), response patterns of aggression (Flynn 1976), and a functiona1 classification of aggression (Wilson 1975).

Offense, according to the present analysis, consists of behavior under the control of an offense motivational system. In cats and rats this system is activated by several types of motivating stimuli processed by the amygdala and, perhaps, the preoptic hypothalamus: olfactory stimuli that characterize male conspecifics (in males only); olfactory and other stimuli that characterize the opponent as unfamiliar; and/stimuli associated with competition for food or water when the animal is food- or water-deprived. The motor patterns of offense include approach and chase, bite-and-kick attack, sideways postures, piloerection, and (in the rat) an offensive upright posture. In the classification system of Moyer (1968) offense subsumes inter-male aggression, territorial defense, that portion of irritable aggression elicited by competitive interactions, and that portion of maternal aggression that involves such motor patterns as bite-and-kick or sideways postures. In the classification system of Flynn (1976) it corresponds to offensive threat, and (perhaps) to certain quiet attacks that might take place in intraspecific encounters. In the classification system of Wilson (1975) it corresponds to territorial aggression and dominance aggression.

Defense and submission, according to the present analysis, consist of behaviors under the control of defense and submission motivational systems. In cats and rats the motivating stimuli of these two systems are the same, involving a forebrain pathway through the "defense zone" of the amygdala and perifornical hypothalamus, which may convey olfactory motivating stimuli, and ascending pathways, which convey pain, dorsal tactile stimulation, sudden noise, or visual movement. There is considerable overlap in the output motor patterns, including upright and sideways postures, squealing and hissing, freezing, and fleeing. There are also differences, however, with lunge-and-bite attack and striking (in the cat) shown only during defense, and submissive postures and ultrasound (in the rat) shown only during submission. The animal normally shows defense against unknown opponents, but if its consociate modulator is activated by stimuli of a known conspecific or other life-long associate, then the behavioral control is shifted from the defense to the submission motivational system.

Defense and submission have not been differentiated by previous authors. In the classification of Moyer (1968) they subsume the categories of fear-induced aggression, that portion of irritable aggression elicited by pain, and a part of maternal aggression that involve a lunge-and-bite attack. In the classification of Flynn (1976), defensive threat and attack are corresponding categories. And in the classification of Wilson (1975) they include the category of antipredatory aggression.

Certain categories of aggression in previous classifications are not included here. Predatory aggression and quiet attack on another species are not included because they are not intraspecific aggression, and because they appear to be under the control of a different motivational system. Moyer (1968) includes a category of instrumental aggression, for which I do not know of any examples in the ethological or neuroscience literature. Wilson (1975) includes categories of sexual aggression, parental disciplinary aggression, weaning

aggression, and moralistic aggression, which have not been extensively studied, or in some cases observed, in the species under consideration here.

Because the present classification is not based solely upon a set of logical distinctions and categories, such as response or stimulus categories, its conclusions may seem at times to be surprising, illogical, or, at least, not parsimonious. In particular, it is surprising that there should be so much overlap between the two motivational systems of defense and submission. In fact, in an earlier paper (Lehman & Adams 1977) I suggested that defense and submission should logically involve separate and non-overlapping patterns. Data from brain lesions and stimulation require a different conclusion, however. Freezing, fleeing, and upright postures, along with a number of other motor patterns, are exhibited by animals both before and after lesions of the medial hypothalamus that shift the animal from submission to defense. Only the lunge-and-bite attack appears for the first time after such lesions. Although we may wish to assume that the neural mechanisms of behavior are ultimately "logical," the logic may not be immediately obvious and may require a functional and evolutionary analysis that goes beyond the scope of the present review.

Although the emphasis has been placed here upon genetically-determined, "hard-wired" neural circuitry of social behavior, there is no doubt that important aspects of the functioning of this circuitry, and perhaps part of the circuitry itself, change as a function of the animal's experience. Some of these changes are due to the actions of hormones. Others are more properly considered as examples of learning and memory. I have argued elsewhere in detail (Adams, in press) that in muroid rodents there are seven points at which learning plays an important role in the motivational systems of offense, defense, and submission. 1) Previously-neutral stimuli may become conditional motivating stimuli for defense and submission if they are paired with unconditional motivating stimuli. 2) The directing stimuli for the routes followed during approach and escape locomotion in a familiar place may be learned by experience. 3) The stimuli, largely olfactory, by which an animal determines if its opponent is a consociate, are partly learned and may, in some cases, be considered as a type of imprinting. 4) The motivating stimuli of neophobia are of necessity dependent upon learning. 5) The ability of sudden movement and noise to serve as motivating stimuli for defense depends upon early experiential factors (Clark & Galef 1977). 6) The ability of restraint and dorsal tactile stimuli to serve as motivating stimuli may be lessened by experience with handling. 7) Visual stimuli can be conditioned to serve as releasing stimuli for the upright posture in the rat (Kanki & Adams 1978).

In keeping with the purpose of this paper, which is to help focus and direct research questions concerning brain mechanisms of aggression, the remainder of the discussion will concentrate on the types of experiments that might be performed to test and improve this hypothetical model.

One way in which the present model is undoubtedly oversimplified is its unidirectionality of neural projections -that is, information flow. As shown, information is received from stimuli, processed, and conveyed to motivational mechanisms that activate motor patterning mechanisms that organize motor patterns. No reverse flow is shown. Attentional

mechanisms that might alter perceptual systems are not included. In brain-stimulation experiments concerning the interspecific attack of a cat upon a rat, it has been shown that perceptual systems are modified by the activation of motivational systems (Flynn et al. 1971). Although the disruption of intraspecific aggression following brain lesions has often been attributed to attentional losses (e.g. Sprague et al. 1961), a definitive study of the question would require brain stimulation experiments similar to those done on interspecific attack. It seems likely, from the fact that most neuro-anatomical connections are reciprocal, and from the findings on interspecific attack, that such reverse connections will be found when the appropriate experiments are done for offense, defense, and submission.

The location, and indeed the existence, of the hypothesized offense motivational mechanism needs to be determined. Probably the most efficient experimental strategy would involve reliable elicitation of offense by electrical stimulation of the lateral hypothalamus or by isolation-induced offense in the rat, and the systematic placement of midbrain lesions in order to find a location at which all the motor patterns for offense are abolished. Then lesions of that region would be tried on animals who show all the various types of offense - that is, inter-male fighting, offense by females (in the hamster and gerbil), and competitive fighting - to make sure that the lesions abolish all these types of offense.

The least known aspect of the neural mechanism for aggression consists of the locus and functioning of the motor patterning mechanisms. The locus of these mechanisms could be determined by tracing the projections from the various motivational mechanisms using Nauta degeneration or autoradiographic histological procedures. Discrete lesions in areas to which these fibers project would be expected to eliminate specific motor patterns of the various motivational systems while leaving others intact. The time is coming when it should be possible to link our knowledge of sensory processes and motivational systems on the afferent side of the brain with the growing knowledge of motor systems such as those of locomotion (Shik & Orlovsky 1976; Orlovsky & Shik 1976).

Releasing and directing stimuli for the motor patterning mechanisms have also received too little attention. The method of research by which we have investigated the releasing and directing stimuli for the upright posture (Kanki & Adams 1978) could be used to investigate similar stimuli for other motor patterns as well.

A major question remains that of the functional significance of the forebrain pathway for defense. Does it reflect motivating stimuli of defense pheromones, neophobia, unfamiliar conspecific opponents, or still other motivating stimuli? This question could be answered by lesion studies on behavioral preparations in which defense could be elicited reliably in response to the various motivating stimuli listed above. Since these stimuli are not usually effective in laboratory rodents raised under standard conditions, either wild animals or laboratory rodents raised under special conditions should be used. Raising animals with nest boxes (Clark & Galef 1977) might provide suitable experimental subjects.

Following successful lesion experiments on defense motivating stimuli, it should be feasible to employ single-neuron recording to investigate the neural basis of aggression. The

principle challenge is to develop behavioral preparations that respond with defense, submission, and offense to various motivating stimuli in rapid succession while fitted with chronic recording microelectrodes (see Adams 1968; Pond et al 1977). In this way it should be possible to determine the precise functional neural circuitry of the septum, amygdala, hypothalamus, and midbrain central gray.

Ideally, by means of experiments that combine behavioral techniques, chronic microelectrodes and chronic stimulating electrodes, it should be possible to classify neurons on the basis of both behavioral and physiological characteristics; let us suppose, for example, that neurons in the ventromedial hypothalamus that fired maximally during submission were also found to be those that respond to central gray stimulation. Providing that the physiological characteristics were not abolished by certain anesthetics (and this could be tested by giving anesthetics to a functioning chronic preparation), it would then be possible to conduct acute neurophysiological experiments on neurons with known behavioral functions, and thereby to extend our knowledge far beyond its present confines.

The study of the brain mechanisms of aggression could gain a great deal from new developments in the understanding of basic mechanisms of olfaction. Although it appears from behavioral research that the olfactory qualities of familiar versus unfamiliar stimuli, and qualities of pheromones based on the presence or absence of gonadal hormones in the opponent, are critical in motivating stimuli for social behavior, the neural mechanisms for these processes are not known at all.

An evolutionary perspective would be strengthened by data on sequences of motor patterns related to aggression in opossums and primates obtained in experimental paradigms such as we have used on the rat (Lehman & Adams 1977). Such data might help determine whether these species have motivational systems of offense, defense, and submission homologous to those of cats and rats. At the present time several of us are conducting such experiments in stumptail macaques, and, hopefully, other species will be studied in other laboratories as well.

Finally, there is a possibility that there is a brain mechanism, not yet studied to any extent, by which the organism chooses among possible motor patterns that might be performed at any given instant. To some extent the activation of a motor patterning mechanism is presumably due to activation of motivational mechanisms and the presence of the requisite releasing and directing stimuli. It may be possible for more than one motor patterning mechanism to be active simultaneously. Thus, for example, vocalization, locomotion, and piloerection may accompany a number of postures in the rat and cat. But, beyond this there appear to be situations in which there is a sharp discontinuity among the activations of related motor patterning mechanisms such as those of freezing, fleeing, and lunge-and-bite attack during defense in muroid rodents. It is possible that there may be a "master switch" in the brain for such motor patterns that ensure that only one is activated at a time. Where would one look for such a "master switch?" I would suggest looking in old parts of the cerebellum that appear to receive projections from and send projections to most or all of the motor initiation centers of the brain (Nieuwenhuys 1967).

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Comments by D. J. Albert
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The consociate modulator. **Adams has developed a thought provoking and novel model. Its wide scope allows commentary from numerous points of view, but I will confine mine to the proposed consociate modulator in order to deal with it in detail.**

The consociate modulator is viewed as being localized in the ventromedial nucleus of the hypothalamus. A concern that this immediately raises is whether the localization can be made precisely in this way. This is important inasmuch as further developments of the model attribute behavioral effects to afferents and efferents of this nucleus; It does not appear that existing evidence warrants this degree of localization since many of the results that implicate the medial hypothalamus in the control of aggressive behavior damage substantial tissue outside the ventromedial nucleus. Further, in a recent attempt to delimit the region involved in the modulation of intermale attack, mouse killing, and reactivity to an experimenter, the critical area was fairly diffuse in the rostromedial hypothalamus while in the posterior hypothalamus it appeared to be centered in the region between the ventromedial nucleus and the fornix (Albert and Wong 1978a).

With respect to the proposed connections of the consociate modulator, the circuit drawn connecting the cingulate cortex, hippocampus, and septum with the ventromedial nucleus seems quite shaky. Three experiments report no effect of cingulate cortex stimulation, either in terms of reactivity to the experimenter or of mouse killing (Brayley and Albert 1977, 1977a; Albert, Brayley, and Milner 1978). There does not appear to be substantial evidence for hippocampal efferents through the septum playing much of a direct role in the modulation of aggression. The septum itself, which contains these hippocampal efferents, is a relatively ineffective site for inducing increased aggression using lesions. The more effective site in this general area is ventral to the lateral septum along the medial edge of the rostral limb of the anterior commissure (Albert and Brayley 1979; Albert and Richmond 1975; Albert and Wong 1978b). Finally, there is little evidence that the lateral septum or the region ventral to the anterior septum modulates aggression by way of input to the ventromedial nucleus since the reactivity to the experimenter induced by medial hypothalamic lesions is suppressed by stimulation in the region of the septum (Brayley and Albert 1977).

Functionally, the consociate modulator is initially proposed to broadly regulate the tendency to behave defensively or submissively to a "familiar individual of the same or different species." However, the detailed development of the model is concerned primarily with intraspecific aggression. Existing evidence itself suggests that intraspecific aggression is modulated by the medial hypothalamus in the same way as an attack on a mouse (Albert and Wong 19788) Defensiveness, as manifested in increased reactivity to an experimenter, is modulated independently of the tendency to attack a mouse or another rat (Albert and Brayley 1979; Albert and Wong 1978a; Eclancher and Karli 1971; Panksepp 1971a). Because it appears that the medial hypothalamus modulates a broad spectrum of

interspecific and intraspecific aggressive behavior. Adams's suggestion that the medial hypothalamus regulates defense-submission seems too limited. An alternative conceptualization is that the medial hypothalamus is one part of a neural system modulating the tendency to emit an attack and the tendency to act defensively. The neural control of these two dimensions of behavior appears to overlap in the medial hypothalamus, but whether there is an integrated control of these two dimensions of behavior is not clear (Albert and Wong 1978a, 1978b).

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Comments by R. J. Andrew
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Avian data on aggression. **In the course of his article Adams effectively re-interprets some data of mine and DeLanerolle (Andrew 1973; Andrew and Delanerolle 1974) He argues that lesions in the central gray of the chick midbrain "depress defense vocalizations, defensive pecking and freezing in a novel environment." In fact, what we described was rather different and (we felt) rather more interesting. Our lesions destroyed the intercollicular area, whose mammalian homologue is probably the subcollicular area. Both could indeed be regarded as including lateral extensions of the midbrain central gray, but surprisingly, lesions (in work I have not yet published), confined to the midline central gray of the chick did not produce the characteristic effects such as muting, which follow from intercollicular lesions. It is interesting therefore that Adams notes that "part of the tegmentum adjacent to the lateral borders of the central gray" must be destroyed in order to produce the full syndrome that he observes in the rat following complete lesions of the central gray. In the chick (but not of course necessarily in mammals) it is enough to destroy structures on the lateral borders of the central gray to produce what may be a comparable syndrome.**

However, it is the behavioural changes resulting from intercollicular lesions in the chick that I would like to discuss more fully. One initial interest was in the muting that results. All calls are lost, and not simply the peeps given by cold, frightened, or frustrated chicks; these latter might perhaps be termed defense calls, in that they are often given when fleeing and may precede or follow (but very rarely accompany) defensive pecking.

We then discovered, to our surprise, that all of the phases of behaviour that are normally accompanied by calling had also disappeared. It is necessary to use a word like "phase" because, for example, feeding was normal, except that the extended session of excited feeding (normally accompanied by twitter calls, which intact chicks give on finding a food source) was absent. Instead, the first bout was exactly the same as subsequent ones. Equally, small beads and similar objects that normally are attractive to chicks and evoke repeated pecking with twitters were ignored. It is important to note that such pecking differs from defensive pecking both in its form and in the stimuli that release it. Defensive pecking is typically delivered from a crouch with outspread wings, after sustained fixation in which the animal remains still; often it has backed away from the stimulus just before such fixation. Such a peck can be evoked by large stimuli as well as small and, if so, is aimed at the centre of the large stimulus rather than at some surface marking. A very similar difference holds for the pecking at large objects that is facilitated by testosterone; here the hormone only affects pecks at large stimuli (Andrew and Clifton, unpublished), We suspect that the same lower-level mechanism may be involved in both testosterone-facilitated pecking (which might be "offense" in Adams's terminology) and defensive pecking.

Intercollicular lesions do, as part of their global effects, also affect behaviour evoked by frightening stimuli (eg fleeing, hiding, and visual scanning in a novel environment are almost or quite abolished). However, we maintained (and still believe) that this is only one aspect of a more basic change that we characterised as a loss of the ability to respond to visual (and probably other) stimuli as if they were conspicuous or highly valent; one crucial aspect of such response is the appearance of "emotional" behaviour.

More extensive speculation on this hypothesis will be found in Andrew (1975); it would be out of place here, where indeed it could be argued that findings in chicks, which apparently disagree with hypotheses derived from mammalian data, may reasonably be ignored.

I would like to suggest, nevertheless, that some deductions may be drawn that are pertinent to the present discussion. Firstly, I would argue that ethologists have been much to blame in the rigidity with which they have clung to the categories of behaviour that were adopted in the infancy of the discipline. This rigidity has often resulted in the choice of a limited range of experimental situations, which of necessity confirm the categories. Thus (to take a real example) if a male fish is tested by the presentation of a sexually receptive female, or a territorial rival, or a place to lay eggs, it is not surprising that analysis of the types of behaviour shown should suggest that the components of behaviour can be classified as sexual, aggressive, or parental.

The same sort of constraint may well affect the study of defensive, submissive, and offensive behaviour. In the cat emotional responses, which are often listed as being the crucial indices distinguishing "affective" attack (which would be classified as defensive by Adams) from confident attack, probably also occur in a much wider range of situations. These deserve formal investigation. Frustration is one obvious example: in many primates the same set of calls is given in such situations (and in a number of others) as occur in social interactions involving defense (Andrew 1962).

There may be, in addition, a more difficult problem to solve, if we are properly to disentangle the causal mechanisms of affective behaviour. Learning may lead to the use in adults of stabilised patterns of behaviour as effective responses in social interaction. Defense and submission are obvious candidates. The overlap between the "motivational systems" for defense and submission noted by Adams as surprising might, on this argument, reflect the fact that these are not the best ways of categorising behaviour at (say) the midbrain level, but they do correspond to crystallised patterns used by the whole animal in very clear and specific situations.

I believe that a very important role of studies like Adams's is to make us dissatisfied with present causal models of behaviour. Clearly Adams feels this too and has taken some valuable and concrete steps in this direction. I am arguing only that we should push the process much further.

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Comments by Ronald Baenninger
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Limits of neurophysiological approaches to aggression. **As a student of aggressive behavior but a nonspecialist in the research area addressed by David Adams, I am pleased to comment on his provocative and scholarly article in the general terms appropriate to an interested outsider.**

What I find mildly disturbing is that the doctrine of neural centers appears to have been resurrected as an unspoken theme throughout Adams's article. For example, we are told that "The sensory filters for the motivating stimuli of offense tuned to unfamiliar conspecific odors are apparently located in the amygdala. ..." This suggests that there is a one-to-one correspondence of behavior (or function) and anatomical locus, a point of view which I understood to be outdated, partly because logically there is a "proving the null hypothesis" quality about it. If neurons of structure A are active during a particular behavior X, this by no means proves that structure A is the locus of behavior X, since cells of structures B, C, F. and Z may also be active but unrecorded by the investigator. If structure A is stimulated electrically or chemically, behavior X may occur, but similar logical problems prohibit us from concluding that A is the seat of X. If removal of A is followed by cessation of behavior X, there are the worst kinds of logical problems, reminiscent of the case of a psychologist who removed all the legs from a cockroach trained to jump on a verbal command. The conclusion (when the now legless cockroach fails to jump) that cockroaches have their auditory apparatus in their legs is similar to the type of conclusion frequently drawn in physiological psychology from lesion/ablation studies. Because the body of literature so ably integrated by Adams is based largely on recording,

stimulation, and lesioning studies, a great deal of caution is called for in making statements about behavioral loci or centers.

As an ex-engineer, I have always felt that the term "hard-wired" was a misnomer when applied to behavior. For example, Milgram, Devor, and Server (1971) found that there were spontaneous changes in the behaviors (feeding and drinking) elicited by lateral hypothalamic stimulation. Others have reported similar findings (Mogenson 1971; Valenstein, Cox, and Kakolewski 1969). Such plasticity is not what one expects in a "hard-wired" system. If phenomena such as this appear when the behavioral variables are relatively easy to measure (amount eaten or drunk), how much more "plastic" things are likely to be when the behavioral categories are offense, defense, and submission.

It is easy to point out difficulties and ambiguities in interpretation, and to be a curmudgeon. My main point is that the underlying assumption (or article of faith) of Adams's paper is that we can ultimately point to places in the nervous system that map onto behavior point for point. Adams ends with the speculation that the "master switch" may reside in the older parts of the cerebellum. While the shifts between offense, defense, and submission undoubtedly have neural correlates, and hormonal ones as well, to look for a single place where this happens seems to me to be misguided.

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Comments by Gary G. Berntson
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Cerebellar contributions to response selection. Adams's target article provides a very valuable and concise summary of an important literature on aggression. Further, the models presented in Figures 1-5, when viewed as conceptual flow charts rather than Markovian processors, provide a useful functional overview of the anatomical-physiological system involved in the control of certain classes of aggressive behavior.

Adams raises an important issue toward the end of his paper concerning the possible mechanisms involved in response selection. Sensory-releasing stimuli are certainly involved in this process, as we have argued elsewhere (Berntson and Micco 1976). The sequential appearance of relevant releasing stimuli within a behavioral chain most probably contributes highly to the serial coherence of motivated behaviors. Beyond this level of integration, however, one is struck by the apparent lack of response conflict between broader classes of motivated behavior, such as hunger and defense, for example. Some mechanism obviously allows an organism to put away concerns over energy balance in the face of a physical threat - even in the presence of releasing stimuli for both classes of behavior [see Toates: "Homeostasis and Drinking" BBS 2(1) 1979]. Adams suggests that such a process may be subserved, in part, by the paleocerebellum. I believe there is merit to this suggestion. Snider and Maiti (1976) and Heath (1976), among others, have documented the widespread functional interactions between the cerebellar fastigial nucleus and limbic mechanisms for motivated behavior. The fastigial nucleus also has been shown to have functional linkage with lower brainstem autonomic and behavioral substrates (Berntson and Paulucci 1979; Miura and Reis 1970; Snider 1975).

Several lines of behavioral research support the involvement of the paleocerebellum in behavioral function. In 1973 I reported that stimulation of the cerebellar fastigial nucleus in the cat could induce robust and coordinated eating and grooming behaviors (Berntson, Potolicchio, and Miller 1973). Comparable responses were independently obtained in Reis's laboratory (Reis, Doba, and Nathan 1973) and Martner's laboratory (Lisander and Martner 1975). Subsequently, similar effects have been demonstrated in the opossum (Buchholz 1976) and the rat (Ball, Micco, and Berntson 1974; Watson 1978a), although in the rat the responses demonstrated less behavioral specificity than in other species. In addition, electrodes in the fastigial nucleus of the rat were found to support self-stimulation (Ball, Micco, and Berntson 1974). These findings, together with reported changes in affective behavior with cerebellar stimulation or ablation (Berman, Berman, and Prescott 1974; Peters and Monjan 1971; Reis, Doba, and Nathan 1973; Zanchetti and Zoccolini 1954), strongly implicate the paleocerebellum in behavioral function.

It is recognized that the cerebellum participates in postural control and motor coordination through a relatively direct action on lower reflex mechanisms. More recently, there has been a great deal of interest in the participation of the cerebellum in the acquisition of skilled movements (Albus 1971; Gilbert 1975; Ito 1972; Marr 1969), perhaps through a response selection process (Eccles 1977). Comparable roles may well be played by other portions of the cerebellum in orchestrating sequences of species-characteristic behaviors and contributing to response selection and inhibition through actions exerted at higher levels of behavioral organization. Some preliminary data from our laboratory, gathered in collaboration with Professor David Hothersall and Kevin Schumacher, are consistent with this view.

Rats having medial cerebellar lesions were tested in an operent DRL (differential reinforcement of low rates) task, which is highly sensitive to behavioral inhibitory

processes. In this task the animal must press a bar for food and then wait a specified period before pressing again. A bar press during the time-out period will reset the timing clock and delay the subsequent availability of reinforcement. Thus, the animal must inhibit a high-probability response (bar press) for a period of time after reinforcement. When animals with cerebellar lesions were trained on this task, in the presence of a wood block that allows a collateral or "mediating" behavior to fill the time-out delay, their performance was as good as, or superior to, normal animals.

The fact that lesioned animals could achieve highly efficient performance indicates that timing and learning processes were not disrupted by the lesions. However, if the wood blocks were removed, performance of the lesioned animals deteriorated dramatically and did not recover as in normal animals. Rather, perseverative bar pressing continued at high levels, even though such responding precluded reinforcement. The lesioned animals were apparently deficient in the ability to withhold inappropriate high-probability responses - a deficiency that has previously been suggested to characterize animals with cerebellar damage (Buchtel 1970).

While these data are only suggestive, the robust and widespread behavioral effects of cerebellar manipulations (see Watson 1978b for review), including dramatic alterations in aggressive behaviors, plead for experimental and theoretical attention. In the context of the agonistic behaviors of offense, defense, and submission, the present view might predict specific types of changes after paleocerebellar lesions. While such lesions might alter the overall level of aggressive behaviors, a more interesting prediction would involve interactions among these classes of behavior. For example, it might be predicted that lesioned animals would have difficulty in shifting from offense to, say, defense or submission in accord with changing environmental conditions. Alternatively, components of one pattern may intrude into the behavioral performance of a different pattern. In view of these possibilities, it is likely that the detection of such alterations may require sophisticated behavioral testing and may not be apparent in casual observations. Indeed, it is perhaps for this reason that the cerebellum has not received greater attention in the behavioral literature.

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Neurobehavioral systems for attack and defense. **Major recent developments in the study of animal aggression center around the realization that there are two distinct but complementary neurobehavioral systems functioning in aggressive encounters. These systems are polarized in the attack of a dominant or alpha rat on a strange intruder into the alpha's territory; in this case the two combatants show virtually no overlap in behavior; their actions have diametrically opposite effects; and the strength of the two patterns, alpha attack and defense, are differentially influenced by major independent variables such as pain, the presence of threat stimuli, or pharmacological and brain manipulations. The point is that there are a number of areas of evidence providing consistent and absolute (rather than statistical) differentiation of attack (which Adams calls offense) and defense (see Blanchard and Blanchard 1977).**

Adams here proposes a further subdivision, creating categories of "defense" and "submission" from the original defense pattern. This distinction is clearly the major focus of the present treatment and constitutes one of our principal points of disagreement with Adams's review.

We do not believe there is a single area of evidence providing adequate grounds for a distinction between submission and defense "motivational systems". Adams cites a number of factors that he believes will differentiate the two, but not one of them is clear and consistent enough to serve as a criterion for the distinction. First, defense is described as characteristic of wild rats and rats with forebrain lesions, while submission is typical of the laboratory rat. In direct comparison of wild and laboratory Rattus norvegicus in conspecific encounters, we (Takahashi and Blanchard, submitted for publication) have

found consistent similarities between the two strains for both attack and defensive behaviors, including those behaviors constituting Adams's defense and submission categories. Both strains show ultrasonic vocalization and lying on the back (which Adams characterizes as solely submissive behaviors, in contrast to a larger group of behaviors that can be either defensive or submissive). In fact, the wild rats show somewhat higher levels of lying on the back (the "full submissive posture") than do lab rats. Both strains also show occasional instances of retaliatory biting attack (defense) to a conspecific who has just bitten them. The only area in which there is a substantial difference between wild and lab rats is that the former are more reactive to such stimuli as human experimenters and will bite if cornered or handled. However, these same animals will preferentially run away from a human if escape is possible. At present the most parsimonious interpretation of differences between wild and lab rats is that the process of domestication involved self-selection (through differential breeding rates), and perhaps caretaker selection, for animals with reduced defensive attack and flight behaviors. As to rats with forebrain lesions, which Adams also characterizes as more defensive and less submissive, the "septal syndrome" animal actually shows increases in "submissive" behaviors such as ultrasound, freezing, and boxing, which are at least as profound as its increased defensive biting (Blanchard et al 1979).

Adams further suggests that the distinction between submission and defense lies in the primary purpose of the constituent behaviors. Defense serves to protect through the infliction of pain or other noncommunicative actions, while submission serves primarily as communication. This reflects the traditional interpretation that certain behaviors, notably lying on the back, have no intrinsic defensive function but actively inhibit the attack of a conspecific through the signal of defeat or submissiveness. We have argued elsewhere (Blanchard and Blanchard 1977) that these behaviors do serve a specific function in protecting the primary targets of conspecific biting attack, and that there is little reason to believe that such behaviors of a rat can serve as unconditioned inhibitors of the attack of a conspecific. These arguments, based on detailed analyses of attacker and defender interactions during conspecific fights (Blanchard et al 1977), are much too long to recapitulate here. However, it should be noted that Adams does not present evidence or analysis contrary to this view, In fact, he appears to agree with our suggestion that such actions as boxing and lying on the back serve to protect the specific sites bitten during conspecific fights. But at the same time, he uses the "primary signal function" of these acts as a major criterion to distinguish them from acts that are purely defensive. We think this simply doesn't work: to an experienced animal, any consistent defensive (or offensive) act may have a learned signal value or function, by virtue of its previous association with a specific outcome. For example, an attacking rat faced with the skilled defensive behavior of an experienced intruder may cease attack more quickly than when a naive intruder is used. The initial attack latency and actions are the same, but a skilled defense reduces or eliminates the possibility of a successful bite by the attacker, and the experienced attacker appears to recognize this. The "signal" function of most, if not all, defensive acts may be based on association with the protection afforded by the defensive value of the act rather than standing in opposition to such a defensive function. Ultrasonic vocalization may

possibly be regarded as an exception to this statement, as it has no obvious primary defense function (except possibly as an alarm to conspecifics). However, the evidence that conspecific attack is actually inhibited by ultrasonic vocalization is indirect and unconvincing. Finally, Adams states that the present classification is based ultimately upon the neural circuitry involved, which we take to mean that major evidence for differences between submission and defense may be found in the extent to which brain manipulations differentially influence these behaviors. Since physiological studies of aggression and defense involve a heterogenous group of behavioral measures, there is necessarily much judgment and interpretation involved in attempting to evaluate effects on "submissive behavior" as opposed to "defense." We therefore examined four of Adams's citations listed in support of neural differentiation of these behaviors, in an effort to determine how compelling the evidence actually is.

Thus, Veening (1975) is described as having reported that "'if electrical stimulation is confined within the ventromedial hypothalamus, the effect is suppression of defense without fleeing." The actual Veening report is brief, the relevant portion (in its entirety) stating that "During stimulation not only feeding but also resting, sexual and aggressive behavior became clearly depressed in frequency and duration, whereas some other behavioral elements, such as sniffing and locomotion, showed increases..." There is absolutely no description of this aggressive behavior to indicate how Adams could decide that it constituted defense without fleeing, nor is there any indication that the locomotion increase reported had anything to do with flight.

Rodgers and Brown (1976) and Rodgers et al (1976) are both described by Adams as reporting that chemical stimulation of different amygdaloid nuclei "can increase boxing in response to footshock Without facilitating the lunge and bite response of defense." In fact, the sole response measure reported in each of these articles was the percentage of attack, which was defined as "lunging forward or... making a striking motion..." at the other animal. Boxing alone was measured separately, but the results were not reported. It was this "attack" measure that increased after stimulation, and that Adams has evidently interpreted as representing boxing without lunge and bite. I might add that we share Adams's implicit view that reflexive fighting depends on boxing, but if any bites or lunges at the opponent do occur, they are counted as fights; an increase in reflexive fighting certainly cannot be used as evidence that lunge and bite behaviors have not increased.

Finally, the results of Baxter (1967) are described as follows: "Whereas electrical stimulation in the hypothalamus of the cat produces defense carbachol from the same electrode produces submission when the chemical stimulation effect was blocked, the electrical stimulation effect remained intact..."

What is crucial here consists of two points made in the Baxter article but not mentioned by Adams. First, the carbachol effect was almost certainly not due to stimulation of the same site. It could be produced by stimulation of other structures, and especially after injection into the ventricular system, in which case the latency of the effect was less than for

hypothalamic injection. So an implied claim of differentiation of defense and submission by stimulation of different elements at the same site is not viable.

Second, Baxter noted that, although his cats retreated from a gloved hand after carbachol stimulation, they "vigorously clawed and bit the experimenter" if picked up, attacking so strongly as to necessitate the use of a transfer cage in moving them. This Obviously is not in accord with the claim that carbachol enhances submission but not defensive attack.

These examples were not the result of checking a large number of sources, then selecting those that showed poor agreement with Adams's interpretations, They were the first and only ones checked, and they were selected solely on the basis that Adams used them as support for his contention that defense and submission follow separate but parallel pathways in the brain. They provide consistent documentation for the argument that cleaner response measures are needed for studies of the neural basis of attack and defensive behaviors (Blanchard and Blanchard 1977). More to the present point, however, they indicate how weak is the case for neural separation of submission and defense "motivational systems."

Leaving this area of disagreement aside, it should be said that Adams has shown tremendous grasp of a range of physiological and behavioral phenomena, making a brave attempt to analyze a literature as complex and confusing as it is important. Much of this confusion is due to behavioral problems which (we hope) are well on the way toward solution. Another source of impending clarification concerns the pharmacology of the systems for which anatomic aspects were primarily discussed here. In sum, we foresee, in the near future, sizeable strides in understanding the physiological bases of attack and defensive behaviors in animals, and their human analogues of angry aggression and fear. Adams's review contributes greatly to this progress by bringing together a scattered literature, and by serving as a source of hypotheses for future work.

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Dividing up aggression and considerations in studying the physiological substrates of these phenomena. **I will forego commentary on the topography of the neural circuits that underlie aggressive phenomena - these will, no doubt, occupy other commentators. I have devoted considerable effort (Brain 1977; 1978; 1979) to creating a meaningful classification for the diverse phenomena included in the concept of "aggression" as it is applied to infrahumans. Again, the intention was ultimately to relate behavior to physiology. Adams's account is particularly useful in this respect. His categories seem likely to have wide**

application. For example, offensive and defensive forms of attack have been distinguished (on the basis of bite targets) in laboratory mice (Childs and Brain 1979a; b).

I propose to ask three basic groups of questions of this scheme. The first group concerns whether division of *all* intraspecific conflict behaviors into offense, defense, and subordination provides one with *sufficient* information for the eventual identification of the physiological bases ot these activities? Adams's basic separation is logical and defensible, but, are the categories too wide, and do they all refer to aggression? One may note that, while submission is clearly an important component of agonistic behavior (used in the ethological sense), it is not aggression (in the sense of relating to phenomena that involve the "intentional" direction of "noxious stimuli" towards conspecifics). It would not diminish the importance of submission to maintain that it is part of a continuum that incorporates offense. The distinction between offense and defense seems superficially clear, but aggressive encounters (which generally involve more than one animal) may involve fluctuations between activities that can be labeled as "offense, defense, and submission." Are defensive postures preliminaries to defensive attack or are they ambivalent components of offense? I concur with Adams that one should attempt to identify the major motivation involved in aggression, but I am less optimistic about their clear separation. Are separate neural mechanisms necessary, or even likely?

I would have liked Adams to have considered the neural basis of predatory responses in cats and rats. Although he dismissed them as being (largely) interspecific reactions, they do feature prominently in research on the neural control of aggression. The following categories of aggression may provide more information than Adams's scheme:

1. Self-defense ("fear"-mediated attack related to pain and escape).
2. Maternal aggression (attack in situations where litters, nest sites, etc, are threatened).
3. Predatory aggression (if it can be called aggression!).
4. Reproduction termination (in mammals this is the pup-killing response).
5. Social aggression (offensive attack, generally related to competition for mates, territories, or social status).

The first two are defense in Adams's scheme and the last is offense. The dubious nature of categories 3 and 4 has been emphasized elsewhere (Brain 1979), but they are currently used as models of "aggression."

A second group of questions concerns hormonal involvement in the phenomena described by Adams. Adams did not emphasize that endocrine influences on attack/defense/ subordination depend on the species, the behavioral context, the particular hormone, its dose, temporal relationships, and prior experience. For example, the statement that testosterone mediates offensive attack in these species is a gross simplification. Neural conversion of androgens may be implicated in some species (Bowden and Brain 1976; Brain and Bowden 1978). In other situations, offense seems inversely related to testosterone titer: group- housed, castrated mice show increased attack (compared to intact counterparts) on intruding lactating females (Haug and Brain 1976). This behavior is

suppressed by testosterone application. Pheromones (Brain and Haug, in press) mediating this response cannot be "dependent on testosterone production." Adams's models may benefit by considering (a) neural locations at which steroid-hormones can be shown to be autoradiographically concentrated, and (b) sites at which intracranially-administered hormones prove particularly effective, For example, the cited glucocorticoid influences on defense may be mediated by the postulated hippocampal modulation of septal activity, as the hippocampus is a prime target for these hormones.

The final fundamental questions relate to Adams's statements on the evolution of aggression. The view that defensive responses are derived from antipredatory reactions reminds one of Huntingford's (1976) distinction among "social aggression," "predation," and "antipredatory" reactions, Social aggression (perhaps the defensive components of these activities?) was said to have greater affinity with antipredatory responses than with predatory ones. This view is still highly controversial however (see Brain 1979), Some statements in the Adams account are circuitous, such as "the submission system evolved later to modify defense behavior when the animal was confronted with a conspecific whose offense behavior could be inhibited by particular submissive postures." The hope that "the primates can tell us something of the evolution of brain mechanisms for aggression in humans" is likely to prove particularly provocative. Do the similarities between the neuroanatomical areas that are involved in conflict in the small number of investigated species, imply that mammals have inherited "aggression circuits?" The alternative view is that aggression (in its various forms) is best viewed as a group of strategies that may be independently acquired by different species (see Eibl-Eibesfeldt 1977). Superficial similarities in neuroendocrine control may be generated by common requirements and limited possibilities; for example, as males are often required to show enhanced aggression in competition for breeding resources, a modulation by "androgens" and particular neural areas becomes especially likely. More obviously, as mammals have limited weapons of offense and defense (teeth, claws, etc,), this is likely to be reflected in similar motor control of aggression. More species and situations need to be systematically evaluated before one can chose between these alternatives.

I applaud the tone and content of Adams's paper. It provides a fair evaluation of some of the problems inherent in studies on the physiological control of aggression. There will no doubt be fierce debate concerning the existence and form of the neuro-endocrine mechanisms that motivate these activities, but it would help enormously if workers would (like Adams) pay more attention to behavioral intricacies. Slack use of the term aggression has generated enormous confusion.

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Neurotransmitter organization of aggressive behavior. **Adams rightly tries to reduce the possible aggressive behavioral forms to an acceptable minimum by dividing them into just two distinct groups: offense and defense (submission being the extreme manifestation of the latter). Such a simplified approach has likewise been chosen in the present commentary, in which an attempt is made to elucidate the neurotransmitter basis of various aggressive (emotional) behavioral reactions of the cat. Only two neurotransmitter systems (the**

cholinergic and the catecholaminergic) will be touched upon; the roles of other putative transmitters. such as serotonin, GABA, and so forth, lie beyond the scope of the present discussion.

Let us begin with one of Darwin's principal laws: self-preservation. Most probably, this drive is of utmost importance in determining the actual behavior of any individual. When consummatory behavior is not taken into account, the phenomenon of self-preservation can be reduced to mere defense. When looked at from an ethological point of view, and disregarding, again, consummatory or sexual drives, offense seems to be just a special defense strategy, one that is only manifest when the animal (or human?) is in a somewhat "desperate" situation.

Affective defense behavior. **This is the term used by Adams, and it seems to be roughly equivalent to the "affective defense reaction" (ADA) described by Hess and Brugger (1943) and by Macphail and Miller (1968), or to the behavior called "rage reaction' in some of our own earlier papers (Varszegi and Decsi 1967; Decsi, Varszegi, and Mehes 1969; Decsi 1974: Nagy and Decsi 1973). Eglin (cited by Maclean, 1969) was the first to describe direct cholinergic stimulation of the cat's hypothalamus as resulting in a characteristic emotional reaction. The response to cholinergic stimulation is very similar to, but not quite identical with, the response to electrical stimulation of the same locus (cf. Baxter 1967; Decsi, Varszegi; and Mehes 1969). Hunsperger and Bucher (1967) summarized the behavioral pattern seen alter electrical stimulation as'' characterized by lowering of the head, laying back ot the ears, hunching the back, accompanied by growling and hissing and signs of sympathetic discharge, such as pupillary dilation and pilo-erection".**

This is practically the same picture as that seen after chemical (muscarinergic) stimulation (Varszegi and Decsi 1967: Decsi, Varszegi, and Mehes 1969). Thus, the neurotransmitter background of this form of behavior should be cholinergic. This assumption is corroborated by several findings: e.g. topical carbachol (CCh) pretreatment decreases the threshold of the electrically-evoked rage reaction (hissing response), while atropine increases it (Decsi, Varszegi, and Mehes 1969); systemic pretreatment with atropine or t-hyosciamine fully counteracts the effect of intrahypothalamic CCh (Varszegi and Decsi 1967) Since this reaction pattern - i.e. the ADR - plays a crucial role in the behavior of the animal, investigations have begun in this laboratory to analyse it in detail. First, it has turned out that the reaction is not specific to the hypothalamus but can also be evoked from several other brain regions (Decsi 1974). This recognition led to the conception that a complex subcortical functional circuit should be responsible for the behavioral manifestation in question. Among others, this circuit comprises the hypothalamus, thalamus, and central gray matter (Nagy and Decsi 1977). The role of the limbic system in modifying this reaction pattern has been described in previous years (Nagy and Decsi 1973; 1974; Decsi and Nagy 1974; 1977b). The reaction can be started by cholinergic stimulation of any of the relay stations (but not from other parts of the brain Decsi 1974) and is inhibited by local atropine blockade (or electrocoagulation) of any of these stations. For instance, the ADR ("rage reaction') evoked from the hypothalamus can be prevented by

topical application of atropine in the thalamus, central gray matter, red nucleus, or septal region, but not by that in the amygdala.

Adams says: "A defense motivational mechanism is hypothesized to be located in the midbrain central gray." Not only there. Using the method of electrical stimulation, Hess and Brugger (1943), Hunsperger (1956), and many others had demonstrated long ago that this mechanism could be triggered from the hypothalamus and also from some other parts of the brain. Our own experiments with chemical stimulation have also demonstrated that this mechanism has a rather extended subcortical organization, and that the "mechanism" involves a complex cholinergic circuit (circuitry, in Adams's terminology).

Chemical stimulation is of more use than is electrical stimulation, for it is selective and only affects well-defined circuits. Moreover, chemical blockade is a more appropriate process than knife-cuts or electro-coagulation, which "knock out"' everything. Therefore, chemical manipulations in the brain surely give more information than do other more aggressive but less specific and less selective interventions. As a consequence, anatomically overlapping, but functionally quite differing, circuits can be distinguished rather easily by means ot specific chemical stimulation (or inhibition). And this may well be the reason why lesion and (or) electric stimulation experiments necessarily had to lead to Adams's sophisticated, but hardly informative, deduction that "motivating stimuli activate pathways that converge upon sets of homogeneous neurons, called the motivational mechanism, whose activity determines the motivational state of the animal". As a matter of fact, converging stimuli may be of quite opposite polarity: one may be the stimulus governing the reaction directly (a cholinergic one, in this case), while the other may be a modulatory one (adrenergic, in this case); and the latter will determine the final, already restricted, motivational output. According to our experience, a given behavioral pattern, actually the so-called aggressive behavior, can be "switched" from one form to another (e.g. from offense to defense or vice versa) in two ways:

Switching mechanism 1. **The neurotransmitter originally involved (acetylcholine, in this case) is also liberated in structures as yet unaffected by it; for instance, at receptors in the limbic system or at nonmuscarinergic receptors within the hypothalamus itself (T - receptors, see below).**

Examples of 1. **(a) Cholinergic stimulation of well-defined regions of the amygdala inhibits, or fully antagonizes, the ADR evoked by cholinergic stimulation of the hypothalamus (Decsi and Nagy 1974). (b) Stimulation of the hypothalamus with d-tubocurarine in one hemisphere counteracts the ADR evoked by simultaneous CCh-stimulation of the contralateral hypothalamus (Decsi, Varszegi, and Mehes 1969).**

Switching mechanism 2. **The action of the neurotransmitter originally involved (acetylcholine, in this case) will be modified (suppressed) by increased tone in another neurotransmitter system (adrenergic, in this case) in the region in question, or by that ot some other area also involved in circuitry of the reaction.**

Examples ot 2. **(a) Prior injection 01 noradrenaline in the thalamus suppresses the ADR evoked by subsequent CCh stimulation of the same region (Decsi and Nagy 1977a). (b) Injection of noradrenaline in the thalamus (intralaminary cell groups) counteracts the ADR evoked by cholinergic stimulation of the hypothalamus (Decsi and Nagy 1977b).**

Both mechanisms require very few "extra" inputs (e.g. local ones in the hypothalamus, red nucleus, etc), or additional inputs from, or through, the limbic system (e.g. hippocampus or amygdala; Nagy and Decsi 1974; Decsi and Nagy 1974).

Escape (fear) reaction ot the cat. **The term "fear and escape reaction" has been used by us to describe the characteristic behavior that can be evoked by direct topical stimulation ot the hypothalamus with d-tubocurarine (Decsi and Karmos-Varszegi 1969). Our first results have been corroborated by the experiments of Romaniuk, Brudzinsky, and Gronska (1973) The most characteristic external signs of the reaction were summarized as follows (Decsi and Karmos- Varszegi 1969) "High degree of restlessness, walking and running around in the cage Repeated attempts to escape from the cage; the animal tears the floor with the claws, jumps against the wall and even against the top of the cage Once out of the cage the cat hides at the most remote corner of the room and, when approached, runs terrifiedly away to look for some other hiding place"**

Since d-tubocurarine is a cholinomimetic drug, it is plausible to assume that this reaction is also cholinergic in character. However, the reaction is not brought about by stimulation of receptors belonging to either the M- or N-type; in this case a third type of cholinergic receptor had to be assumed to be present in the brain (T -receptors) resembling those found in the neuromuscular junction (Decsi, Varszegi, and Mehes 1969; in this connection, see also Myers 1974). Thus, the behavioral manifestation that we have called tear and escape reaction is cholinergic as to its neurotransmitter organization. This reaction seems to participate in the complex of "offense, defense, and submission" The d- tubocurarine-induced (cholinergic) escape should represent a part of the defense, but certainly not the last step of it, since the final stage of any defense must necessarily be an attack (or counter-attack), be it ever so hopeless. From a transmitter point of view, this might mean a switch from T -receptors to M- receptors, or releasing M-receptors from a continuous adrenergic modulatory control (Intraspecific submission is quite another question; it might represent increased catecholaminergic input to overpower the original cholinergic defense behavior).

Adams speaks about three motivational systems, with modulators, releasing and directing stimuli involved in each. At first sight, such a classification in terms of just three (or better two) groups seems to be an oversimplification; and, by themselves, the terms "modulators, releasing and dIrecting stimuli" do not say much either. However, when we try to reduce these systems to a common denominator on the basis of their possible neurotransmitter organization, they fit surprisingly well with the data obtained in neuropharmacological investigations. All the hypothesized motivational systems seem to work with cholinergic neurotransmission, while the chemical basis ot the modulatory, releasing and directing stimuli is most probably catecholaminergic.

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Cerebral play of forces in offensive-defensive mechanisms. **Dr Adams's excellent review of the specific neuronal circuitry of intraspecific aggressive behavior in mammals is most useful. I would, however, question his main working hypothesis about the anatomical identification of three motivational systems for offense, defense, and submission, and propose instead the involvement of a constellation of brain structures, with a continuous dynamic equilibrium, in the control of the various manifestations of agonistic behavior.**

To locate a defense motivational system in the midbrain central gray is controversial, because offensive-defensive responses depend on the play of forces in neuronal pools located not in one but in several structures, including the central gray, the tectum, some thalamic nuclei, and the amygdala, plus inhibitory influences from the septum, the caudate nucleus, parts of the amygdala, and other cerebral structures.

Electrical stimulation, chemical injections, and localized destructions of the brain may act upon the equilibrium of the whole system, which is also influenced by its past history, by present sensory inputs, and by individual interpretation of received information, We should not place functional labels on anatomical areas without taking into consideration the environmental context. Research should be oriented toward evaluating the role of specific

structures and factors involved in each type of offensive-defensive reactivity . Adams's reference to " ... points in the forebrain where there are inhibitory interactions between the defense and submission systems " appears to be in agreement with the idea of constellations and not centers.

Another controversial issue is Adams's concept of the "consociate modulator which switches the behavior of the animal when its opponent is familiar, from defense to submission," In our experience the essential factors determining an animal's response are hierarchical position and social context, not familiarity. This has been demonstrated in monkey colonies where the occurrence, direction, and expression of aggressive responses elicited by aversive radio stimulation of the brain or by radio-controlled skin shock depended on the social rank of the stimulated monkey, which would attack submissive monkeys but showed no aggression in the presence of a dominant partner. Exactly the same brain stimulation elicited aggression or submission, depending on the presence of a low- or high-ranking partner (Plotnik, Mir, and Delgado 1970).

In other experiments with gibbons, electrical stimulation of the same point in the central gray in the same animal produced aggressive behavior if the animal was in a laboratory colony but not when tested in a tree ecological situation on the island of Hall. In this last case the stimulated animal would run out of sight into the bushes without showing hostility against other gibbons. These studies also demonstrated the establishment of dynamic equilibrium between excitatory central gray and inhibitory caudate nucleus stimulations, with relative predominance dependent on relative strength of stimulation (Delgado, Sanguinetti, and Mora 1973).

In our opinion, e1ectrical stimulation of the brain acts as a trigger of preestablished cerebral functions, influencing the processing of sensory inputs and the release of previously acquired motor patterns and experiences. In some experiments the motor effects elicited by brain stimulation may be considered as fragments, often involved in offensive-defensive behavior, but lacking negative reinforcing properties (Delgado 1967).

As a working hypothesis we have proposed that emotional expression, including offense and defense, is composed of groups of fragments that comprise autonomic responses, vocalization, facial expression, and both tonic and phasic motor activity. Depending on the cerebral area stimulated, these fragments may be evoked as isolated or as organized sequences. Emotional states depend on the activation of constellations of brain structures determined by the decoding of specific sensory inputs. Brain stimulation may produce similar activation, allowing the artificial manipulation of emotions as well as of emotional reactivity (Delgado and Mir 1969).

Some of these ideas may coincide with Adams's distinction between motivating, releasing, and directing stimuli, with the difference that the distinction is not in the stimuli but in the cerebral frame of reference and in the social context, which may be decisive for the interpretation of sensory inputs. Hopefully Adams's thought-provoking review will provide

the basis for further studies to increase our understanding of the brain mechanisms involved in offensive, defensive, and submissive behavior.

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Comments by Burr Eichelman

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Brain mechanisms of aggression: Dilemmas of perspective. Adams has made a significant contribution to the aggression literature with his attempt to catalogue various experiments in terms of their modulation of offense, defense, and submission. The attention to behavior as offense, defense, or submission has been overly long in coming to the aggression literature. This categorization must be taken into account by current researchers and integrated with Moyer's (1968) groupings and Reis's (1974) separation ot affective and predatory aggression. It is important for defensive behaviors to be included within the rubric of aggression, since many defensive attacks inflict severe physical damage.

The attempt to categorize aggressive behavior as offensive, defensive, or submissive has limitations. The behavioral repertoire for a given animal in an agonistic encounter may encompass all three behavioral groupings. Intermale aggression in previously isolated mice illustrates this point. Both mice may initially tail-rattle, labeled by Adams as defensive behavior, but they both also move in and out ot offensive sideways and upright postures and the bite-and-kick attack. Only over time. as a dominance is established, does the distribution of the behaviors change, Similarly, in the model of shock-induced fighting in the rat, it becomes perhaps a semantic argument to determine whether an increased frequency of boxing (noted as a component of both defensive and submissive behavior) in this paradigm is an increase in defensive behavior (lacking the lunge-and-bite attack) or an increase in submissive behavior (eg, in Rodgers and Brown 1976). And there are further

subdivisions of these groupings which need to be made. Beleslin and Samardzic (1977) describe behaviors induced in the cat with muscarin and carbachol, which they divide into fear and irritable aggressive behaviors, yet both of these seem to be represented by Adams's defense category.

In relation to neurophysiology and the integration of multiple experiments, Adams himself notes inherent limitations in his paper. Some of these limitations should be underscored, though they need not detract from the utility of the paper in cataloging data and generating new experiments. First, the brain mechanisms proposed are essentially "one-way" mechanisms of either inhibitory or excitatory neuronal systems. Reciprocal inhibitory systems and cross-talk between various brain regions is acknowledged but finds little expression in the conceptualization. Not only are neuronal systems bidirectional between various regions, but they also include multiple neurotransmitter systems (e.g. the nigro-striatal system with reciprocal interaction involving dopamine, acetylcholine, and GABA). Secondly, the model deals with aggressive behaviors induced with the onset of brain activity. There is a whole class of behaviors in which both the environmental and neural antecedents are the cessation ot a stimulus. Aggression (defense) can be induced by morphine withdrawal (Boshka, Weisman, and Thor 1966); or by extinction in an operant paradigm (Azrin, Hutchinson, and Hake 1966); or by the cessation of reinforcing brain stimulation (Hutchinson and Renfrew 1978). This class of behaviors needs amplification within Adams's conceptual framework.

The lesion and stimulation studies reviewed must also be subjected to some cautionary interpretation. Most of the stimulation studies relating to defensive behavior can be interpreted on the basis of inducing pain with the concomitant induction ot boxing or other defensive postures, much the same as with footshock-induced fighting. Perhaps certain operant methods might be used to assure that defensive aggressive behavior induced by central grey stimulation was not the animal's response to centrally induced pain. Conversely, lesion effects must also be cautiously interpreted when large lesions in the brain stem are required to abolish defensive behavior. Careful attention should be given to the specificity of these lesions in relation to other behaviors, particularly in terms of activity level and general motor coordination. Some of the studies'reported by Adams have made such attempts, while others have only superficially described the general state of the lesioned animals. Finally, within the cataloging of experiments as carried out by the author, there is some selectivity with respect to data contrary to the thesis expressed. One such example is noted regarding the cingulate cortex. Adams suggests that lesions of the cingulate should increase defense, and he cites such an example in the cat, However, lesions of the cingulate cortex in the rat decrease the frequency of shock-induced fighting composed of defensive postures (Blanchard and Blanchard 1968; Eichelman 1971).

The neural mechanisms of longitudinal neural circuits as set forth by Adams do not encompass another alternate (and equally biased) view of the central nervous system often utilized in pharmacology. Such a view deals with central "tone" or levels of excitability and irritability. The widespread distribution of noradrenergic neurones originating from the

locus coeruleus can aid in illustrating this view. Stimulation of the locus and firing of its cells must have effects that involve many brain nuclei and regions. Further, such "tonal" modulation may even involve nonneural elements. Glial cells have been reported to contain binding sites for putative neurotransmitters and pharmacologic agents such as diazepam (Henn and Henke 1976) These elements may significantly influence whether any aggressive behavior occurs, or they may provide an alternative "consociate modulator" system much more diffuse than the postulated VMH entity of Adams's thesis.

Lastly, the brain mechanisms postulated within Adams's paper, and the experiments that they suggest, remain preponderantly within the area of preclinical, nonhuman research. For the clinician it is very difficult to conceptually transfer data relating to vibrissae and defensive behavior to patients with a diagnosis of Explosive Personality Disorder. Considerably more conceptualization must be applied to human aggressive behavior to meaningfully integrate this murine and feline model with clinical experience. Much more ethological work must be carried out to categorize human agonistic behavior as offensive or defensive. Clinicians have instead tended to describe dystonic human aggressive behavior in terms of impulsivity (DSM III, 1978) or an inability to inhibit aggression (perhaps a deficit in passive avoidance), rather than in terms of offensive or defensive behavior patterns. Human behavior is also much less accessible to modulation by brain stimulation or lesioning. Thus the transfer of knowledge from the rodent or cat to man may be severely limited; this limitation may even extend to the asking of similar questions in the human. One is hard pressed to draw parallels with limbic function when septal lesions or stimulation in the rodent induce marked behavioral change in contrast to minimal effects in the primate or human. It may be that more productive modeling for clinical research will develop from the analysis of environmental antecedents to aggression and perceptual differences between eggressive and nonaggressive individuals (e.g. Kinzel 1970), or through pharmacological manipulations, both as tools for understanding the central nervous system and as beneficial modulators of dystonic behaviors.

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Motives: Metaphors in motion. **Robert Frost used to challenge his students, and critics, with the query "What is a meta for?" As a poet who had mastered clean lines of description he was keenly aware of both the power afforded by language that transcends direct observation and the dangers of taking one's symbols too literally. The behavioral and brain sciences face a similar problem with their meta language. Motivation is a case in point.**

Adams apparently presumes that we (readers) agree on what motives are and that they are real. I suggest that motivation is a construct of diverse definition designed to help us gain awareness of changing relations among observed fragments of input and output as they operate in the intact organism. This is its power in setting problems, and its limitation in solving them. Brains have cells, not drives, and strict localization of network transfer functions defined for the intact organism may not be possible (e.g Luria 1976). Like the Cheshire cat, we are left with but a faint smile of our original image.

The image in the first instance reflects a distinction between stimuli that motivate behavior and those that merely release it . Adams is perfectly right that this is the classical view of early ethology, but it now appears much too simple. The distinction is relative not absolute, and multidimensional rather than unitary Two illustrations will suffice.

First, the dichotomy between "functional groupings" and "discrete" motor patterning mechanisms is imperfect and a matter of perspective. The single swipe of a cat's paw may be a descriptively discrete component of the overall melee, but no doubt contains a finely tuned, and functional, grouping ot cooperative and antagonist muscles, excitatory and inhibitory circuitry, and so on. Adams's early implication, from which he retreats at the end, that such an act can be generated "without regard to its functional or temporal relationship to other motor patterns" cannot stand for a variety of considerations, ranging from Sherrington's research on reflex integration to modern research on coarticulation in human speech (e.g. Studdert-Kennedy 1976).

A second division is the time course of action, with "releasing" functions being short and "motivating" functions somewhat longer. This is fine if it leads the investigator to examine explicitly different time courses of action, but it fails as a dichotomy. For example, repeated application of "releasing" stimuli can reveal both incremental and decremental consequences upon subsequent readiness to respond that have quite protracted decay functions (e.g. Heiligenberg 1976). Adams gets himself into some related difficulties in logic when, for example, he distinguishes low latency threat from longer latency attack; is the former "released" and the latter "motivated?" Similarly, with examples such as escape in response to footshock I am not certain whether I am supposed to think "motivate" or "release." The author does point out that the same stimulus can have both consequences, but that only serves to weaken his distinction further. Occasionally Adams bypasses the problem by using other terminology, such as "activate." His brief attempt to superimpose modalities upon the division is fortunately abandoned. As a final point, if a stimulus initiates a behavior it is easy to think of a releasing function, whereas if the same stimulus facilitates the same behavior already in progress one may be more inclined to think of it as motivational, even though it might be operating through the same principles in each case. The distinction has difficulties (cf. Fentress 1977).

One is led next to the division among offense, defense, and submission as distinct motivational systems. This is an obvious releaser (or motivator - take your pick) for comment. It does not help much to define, for example, the defense motivational system as that which contains defense motor patterns such as attack and defense. Nor is the distinction helped when the same motor patterns are found to be shared in pairs of systems. Neither does the contextual definition always help, as in the case of "offense" shown by the rat defending its territory, and "defense" by the rat on the offensive (i.e. invading the territory). Maternal aggression, we are told, is a mixture of offense and defense, and data that are apparently contradictory to the model are viewed suspiciously as resulting from an understandable mislabeling by the investigator. Function, evolution, genetics, and learning join forces to clarify the picture, but the result is not always clear. Offense is reportedly

seen more often in males (but see Ryon 1979), and is often communicative (undefined) along with submission, where defense is not, since the latter evolved earlier as a response to predators (presumably this goes for wolves and grizzly bears as well). and submission apparently evolved in the lab - and so on. I think some of the metaphors are mixed.

While it is difficult to construct a logically tight package from the present survey, one positive idea that emerges is that "offense" and "submission" represent ends of a continuum, whereas "defense" is something in between (and thus, e.g., shares motor patterns with each of the others) That is a nice notion, albeit contradictory to the idea that one is dealing with three discrete systems Indeed, if one watches a boxing match between human opponents it is often quite impossible to sort out individual actions as offense, defense, and so on, and even football coaches are fond of stating that the best offense is a good defense (and sometimes the reverse) - just to confuse us all Some of our animal subjects may have the same mentality!

Finally, there is the issue of hardware (or software, depending on one's bias). Ideas such as "homogeneous" neurons lend a ring of reality, but by what criteria are they defined? In some instances they seem to mean "same," as in relating motor mechanisms of defense and submission, but in other instances they refer to the separation of these very systems, as in their action through a "consociate modulator." This last construct relabels the fact that animals sometimes behave differently when they recognize one another than when they do not. Without worrying about problems of localization the "modulator" may have logical troubles, such as when a stimulated animal flees ("submits?") upon the absence of its perceived opponent. Modulate normally means change in degree rather than kind, but this raises the interesting question of how one distinguishes between two different states of the "same" system and two "different" systems. Adams does use the construct of intensity on both the input and output side, but does not separate factors such as current, pulse width, and pulse frequency on the one hand, and amplitude, speed, completedness, interruptability, and so on of behavioral acts on the other (cf Fentress 1973). The summary arguments rest upon a complex mixture of (a) stimuli and their consequences (e.g. "defense and submission inputs"), (b) anatomical loci (e.g. "midbrain central gray"), (c) hypothetical constructs (e.g. "submission motivational mechanism"), and (d) behavioral outputs (e.g. "fleeing"). These are very different logical orders that cannot be boxed together in any simple way (e.g. Figure 2).

We (investigators) obviously attempt to patch coherent pictures out of very incomplete glimpses of nature, and as Frost said about poets, a meta language can provide a powerful ally - as long as we do not forget what we are doing. Where I suspect I differ from Adams is that I view behavioral "systems" as tongue in cheek conveniences for clustering some observations together in distinction from other observations; but I do not take them as being really real beyond that. This leads to the heuristic that one continues to look for signs that favorite systems of the moment are neither unitary (e.g. watch out for "homogeneous neurons") nor totally separate (e.g. watch out for "distinct" and "localized"), and also that their defining boundaries may shift in time as a consequence of both "intra" and "inter"

system dynamics (e.g. Fentress 1976a). The most we can do at this stage is to look at how things (processes) cluster under different specified circumstances. Adams has given us one framework for doing just this, and, even though I suspect his solution has difficulties, the effort is precisely what a "meta is for."

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***Is there anything new in the neurophysiology of aggression for social psychologists?* Since my competence is limited to the field of personality and social psychology, I would prefer that ethologists and neurophysiologists take over the task of deciding whether Adams's models of offense, defense, and submission are supported by empirical data or whether they should rather be viewed as heuristic constructs. On the other hand, my lack of competence does not imply disinterest. In fact, my involvement in research in human aggression requires some orientation in new theoretical and methodological propositions evolved in various**

sciences. During the course of exploring new facts concerning aggression, I will, nevertheless, view them from the perspective of a social psychologist. That is also how I came to comment on Adams's models. What are the general presuppositions of Adams's models? In my opinion, two important implicit assumptions might be distinguished.

First, several specific statements reflect a tendency to treat the brain as a complex system, whose function is to integrate behavior and regulate individual/environment relations. Such an assumption is neither original nor revealing, since it has been presented in detail earlier by Seczenow and Pawlow. Luria's studies in neuropsychology were explicitly based upon this assumption; Konorski's theory is also built upon it. Still, experimental neurophysiology more or less ignored this concept. There was no attempt to develop a general model of neurophysiological mechanisms regulating behavior. The consequences are evident the gap between neurophysiology and the behavioral sciences (especially those concerned with human behavior in social interactions) is broadening In this context. Adams's proposal seems to be valuable, as an attempt to describe the brain's functions and to construe some general models of behavior on the grounds of recent findings in neurophysiology.

Secondly, the functionalism in Adams's concepts is evident. This presupposition is not new in ethology, in which theoretical constructs such as regulative mechanisms are defined in terms of their functional characteristics rather than structural elements. Such theoretical constructs are developed to explain overt behavior. Functionalism is also a familiar presupposition in contemporary social psychology. A stable, reoccuring behavioral pattern, observed during an Interaction between an individual and his physical or social environment, will serve as a basis for seeking the underlying hypothetical functional regulative "structures" or "mechanisms." Although the origin of the functionalism underlying Adams's hypothetical motivational systems and the "consociate modulator" differs from the roots of functionalism found in psychological constructs, still, the idea of a functional definition is the same.

Are the three hypothetical motivational systems and the "consociate modulator" useful in understanding human interpersonal behavior and specifically, human aggression? First of all, I shall try to point out the differences between traditional definitions and models of infra specific aggression in animals and the definition of human aggression. Natural sciences describe aggression as physical attack. Many studies have been aimed at identifying brain centers of attack, withdrawal, and escape responses. This biological model is applicable to traditional definitions of human aggression, according to which attack responses are released by anger. Fear will produce opposite behavioral effects.

The traditional model of aggression analysis developed in the natural sciences - i.e. the attack/escape model - is of limited utility in social psychology, mainly because aggressive actions in human beings are a form of interpersonal contact and it is seldom that simple aversive stimuli or basic biological needs are at their source. In interpersonal relations, aggression consists in the transgression of socially established and historically developed rules of coexistence. Hence in the human world aggression ought to be analyzed in opposition to prosocial action - i.e. actions that serve others and are beneficial to them.

Adams's conceptions are far from similar to my understanding of human aggression. Still, they are probably much closer to the model of human aggression defined as a socio-psychological phenomenon than the attack/escape model. Adams argues that the hypothetical motivational system for offense remains unexplored. Thus, the attack/escape model has yet to be neurophysiologically verified. The discovery of the defense (with elements of aggression) and submission systems with their neurophysiological bases further demonstrates the inadequacy of the traditional approach (There is also the problem of specifying aggression as a form of interpersonal behavior, which will not be discussed here).

I would also like to emphasize another aspect of Adams's models specifically, the reasons for introducing the "consociate modulator." The psychology of aggressive behavior distinguishes several such systems. For example, laboratory studies indicate that the effects of stimulation on the magnitude ot aggression depends on the need for exposure to stimulation (or level of reactivity). Such a modulator is, certainly, a fundamental one compared to the "consociate modulator," which reflects social experience. Neurophysiological processes are obviously not sufficient in explaining shifts from defensive to submissive behavior. One must assume that some system of accumulated experience is responsible for behavioral changes. We can, however, find some "modulators" at higher levels of behavioral organization. Information processing, which provides anticipations - i.e. the foreseeability of negative consequences of one's own behavior for another person - might inhibit or facilitate aggression [see Toates "Homeostasis and Drinking" *BBS* 2(1) 1979]. Another group of "consociate modulators" in human aggression is connected with central elements of personality, like the self-image, beliefs concerning the world, and so forth. The level of guilt-feelings, understood as a stable personality factor, will mediate fhe relation between stimulation and aggressive responses. The above examples suggest that Adams's "consociate modulators" construed on the basis of his neurophysiological experience have also been "discovered" (or described) within the domain of psychological processes. The modulators developed during personal experience not only mediate external stimuli (like a telephone switchboard), but also generate their own power or motivation (like a turned-on nuclear reactor).

The only thing we should do now is wait for neurophysiologists to confirm and add to our knowledge concerning "central or higher level modulators" and "generators" of particular forms of social behavior.

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Comments by Ronald Gandelman
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Androgens and aggression. **I would like to make two comments regarding Dr Adams's thoughtful paper. The first is technical in nature and involves the statement that maternal aggression is enhanced by prolactin. Although this may be true for the hamster (see Wise and Pryor 1977), it is not the case for the rat and mouse. With regard to the former, Erskine, Barfield, and Goldman (1979) report the presence of postpartum aggression in the hypophysectomized rat. In addition, postpartum aggression remains intact in female mice administered ergocornine hydrogen maleate, a compound that blocks the release of prolactin from the pituitary (Gandelman, unpublished data). Thus, Adams's comment that "The effect of the prolactin upon defense is probably due to the suppression of the hypothetical 'consociate modulator and release of defense from its inhibitory Influence" must be clarified.**

My second comment concerns Adams's discussion of fhe motivating stimuli for offense. It was correctly pointed out that in many muroid rodents one of the principal stimuli for the initiation of aggressive behavior is olfactory in nature, is testosterone-dependent, and is effective primarily in males. Moreover, it was speculated that the "sensory filters" or CNS receptor mechanisms for these stimuli are dependent upon testosterone.

The notion that testosterone is involved in both the emission of the cue and its reception is an important one, In that it may shed light upon the manner in which androgen initiates or activates fighting behavior. The study of aggressive behavior in rodents has taken two ostensibly divergent paths. European investigators, for the most part, have been concerned primarily with specifying the stimuli that elicit fighting behavior. Some of the major findings have been that males emit an odor found in the urine that elicits aggression from other males, and that this olfactory cue or releaser pheromone is androgen-dependent, being absent in castrate male mice and females and present following the administration of testosterone (Mugford and Nowell 1970a; 1970b; 1971). If has also been reported that ablation of the olfactory bulbs, as well as masking of natural odors by adulterating mice with odorants reduced fighting behavior (Ropartz 1968).

In the US many investigators have been interested in the hormonal involvement in aggression and in the influence of androgen in particular. The finding that castration abolishes aggression in male mice. and that testosterone replacement is restorative (Beeman 1947), was followed nearly twenty years later by reports demonstrating that the administration of testosterone to females can cause them fo fight (Edwards 1968, Bronson and Desjardins 1970; Svare, Davis, and Gandelman 1974).

Summarizing the two principal paths taken by researchers interested In aggressive behavior, we have those who have shown that an androgen-dependent olfactory stimulus plays an important role in triggering fighting behavior, and others who have reported that testosterone is responsible for establishing the propensity of an animal to fight. One of the

major questions posed by behavioral endocrinologists concerns the manner in which hormones affect behavior The data cursorily summarized above suggest that androgen affects the CNS in such a way as to render it uniquely responsive to the aggression-promoting olfactory stimulus emitted by the opponent. In other words, in the absence of relatively high levels of androgen, the olfactory stimulus is either not "perceived," or it lacks certain "information value." It is unlikely that animals devoid of gonadal androgen fail to detect the olfactory stimulus. It is more likely that this olfactory cue possesses little if any information relevant to the status of the other animal (the potential opponent) in the absence of testosterone at a level normally found in the male. In the parlance of Adams, the sensory filters would not be activated in the absence of testosterone. However, it would appear that the term "sensory filter" should be replaced by "sensory analyzer."

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Comments by Raymon M. Glantz
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The advantages of simple systems in neuroethology. **The central issue in the Adams's paper is best described as the localization of neural structures associated with defensive end offensive fixed-action patterns. In general, lesion and stimulation experiments can only suggest that neural activity at certain loci may be necessary or sufficient to elicit behavior. The validity of these interpretations depends upon the additional constraints that (a) the neural deficit due to lesions be confined to the target structure, and (b) that selective electrical stimulation ot a neural locus reasonably mimics physiological activity at the same site when the behavioral activity is elicited naturally. Were these tests performed, the results would indicate the localization of the above mentioned mechanisms. To extend our understanding beyond a gross structural association, it is necessary to establish the degree of functional heterogeneity of the neurons at a stimulated locus, the character of physiological activity in the relevant neurons, the patterns of functional connectivity within and between loci, and the dynamics of synaptic and ensemble interactions that release and control the behavior These are the issues that address the neural "mechanisms."**

The circuitry that Adams postulates for the release of defense behavior could be constructed with about twenty neurons This is several orders of magnitude fewer than the number of participating nerve cells indicated by lesion studies. These considerations imply that the methodology or the conceptual approach may be seriously wanting.

I believe that. in our present state of ignorance, the invertebrates and lower vertebrates provide more appropriate subjects for the study of the neural basis of fixed-action patterns. Behavioral and neurophysiological analyses indicate that these systems exhibit a significant economy in the number of neurons controlling a behavior and substantially less variability in the behavioral output. Individual neurons at all levels of escape and defense pathways can be uniquely identified (Zucker 1972; Auerbach and Bennett 1969; Getting 1977) and connectivity patterns and synaptic actions are amenable to direct cellular analysis. Although there are still some obstacles to a completely safisfactory description of these systems (Kupferman and Weiss 1978; Davis 1977; Glantz 1977), the limitations do not appear to be essential aspects of either the methodology or the conceptual approach. Experiments can be performed to determine whether the physiological activity of a single neuron is necessary or sufficient to release a given behavior (Glantz 1977; Taghert and Willows 1978). Furthermore, the interpretation of these experiments does not require questionable assumptions, such as the homogeneity of a large population of synchronously activated cells, nor the ambiguity inherent in the unphysiological nature of such a manipulation .

A few of the more salient, emerging generalizations are discussed in several recent symposia (Stein et al 1973; Fentress 1966b; Galun et al 1976, Hoyle 1977). Briefly, they are as follows (i) The decision process for defense or escape is invested in a small number of high-threshold, rapidly conducting, multisegmental interneurons. (ii) The interneurons

project to all of the segments participating in the action pattern. (iii) The intersegmental interneurons either synapse directly with the relevant motoneurons or with local premotor interneurons (iv) Speed and synchronization are optimized by the presence of electrotonic connections between interneurons and motoneurons, between synergist interneurons, and between synergist motoneurons (v) The sensory filters are composed of a small number of primary afferents or sensory interneurons, whose input requirements are precisely tuned to the requirements of the behavior (vi) Adaptive variations of the behavior pattern arise from the activation of different subsets of sensory neurons (vii) Lability (e.g. habituation) arises in the afferent limb of the neural pathway. (viii) Concurrent with the onset of behavior, the afferent input may be inhibited by the decision-making intersegmental interneuron.

All of these generalizations apply to the escape systems of the crayfish and hatchet fish, and instances of all of these phenomena have been observed in escape and defense systems of other arthropods, molluscs, annelids, and fish These generalizations provide an important foundation for further research into the neuronal mechanisms of defensive fixed-action patterns. At present, however, we lack an appropriate methodology to address these issues in mammalian systems.

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Comments by Robert L. Isaacson
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Are we ready to localize motivational systems? **In his target article Dr. Adams undertakes to provide a basis for understanding varieties of behaviors related to aggression, flight, and "submission." What makes his analysis unusual is that its basis is hypothetical neural circuitry. In his theory the presumed neural circuitry ties actions together into meaningful patterns. At least, this is the ultimate goal of his work.**

Adams deserves credit for his concern with the ethological significance of behavioral sequences or acts. Too frequently, neuroscientists study behaviors without regard to their role in an animal's life patterns. We need to be concerned with the role of behavioral acts. Many times similar motor patterns in one species or in different species are considered to be "the same" when, in fact, they may be components of behaviors with different significance to the animal. A rat standing on its hind legs could be engaging in behaviors that may be related to aggression, defense, or exploration. The standing erect of a great ape on its hind feet is an entirely different matter.

The article is in the tradition of the distinguished group that was assembled at Yale in the 1950s (i.e. John Flynn, Jose Delgado [qv], and Paul Maclean) These men invented a new approach to the study of the central nervous mechanisms responsible for behaviors - one that was sufficiently broad for the neurobehavioral data they were collecting. Subsequent research has been directed at an increasingly fine-grained analysis of related neural and behavioral systems. Adams's large-scope theory may be too general to encompass the specific physiological, anatomical, and behavioral evidence now available.

He analyzes three types of "motivational systems": offense, defense, and submission. In connection with the last two, a "consociate modulator" is proposed that determines which of the two will occur at any moment in time. This is necessary because both types of systems are thought to share portions of the same neural system: the central gray of the midbrain. This modulator acts to facilitate submissive acts if an intruder is familiar. The intruder need not be an animal of the same species. The consociate modulator is thought to

be the ventromedial nucleus of the hypothalamus. As mentioned, both the defense and the submission behavioral systems are thought to be located in the central gray, although separate neuronal pools are associated with each. The modulator selects one or the other for expression. The neural mechanisms responsible for offense reactions are not specified, but they are also thought to lie in the midbrain. All systems receive influences from forebrain regions and are regulated by hormonal influences as well.

The motivational systems are said to be made up of "homogeneous" neurons. The actions of these homogeneous neurons determine the motivational state of the organism. It is not clear what "homogeneous" means. Should the neurons be thought ot as identical in terms of anatomical structure, in terms of input or output relationships, or in their biochemical nature or their responsiveness to circulating neuromodulators?

In this theory the motor systems ultimately responsible for all behavioral as executed by an animal are located in the brain stem and the spinal cord, Few would object to this. Specific facilitation of any particular act is thought to occur when motor mechanisms are "activated" by "simultaneous" input from motivational mechanisms and from "sensory filters responsive to releasing and directing stimuli specific to that motor pattern." The entire complex of specific motivational stimuli, sensory filters, and motor mechanisms is also referred to as a specific motivational system. This seems to add confusion to an already complex theoretical structure.

Stimuli can have motivational or directional functions. They function in one or the other capacity, depending on how the information they convey is processed by the central nervous system - that is, on the reactions they induce. All that we know, however, is thaI stimuli can serve both types of function.

An intruder can elicit aggressive reactions related to defensive or submissive responses. Adams believes that laboratory animals are more prone to exhibit submissive reactions than are their wild brethren, but even they can exhibit defensive reactions that will occur in a particular situation. Stimuli, especially olfactory ones in the rat, activate the "consociate" motivator. This mechanism biases responsiveness toward submission instead of aggressive defense.

While there may be a bias toward submissive acts in laboratory rodents and tame cats, the evidence is less than convincing. Indeed, it is rather difficult to imagine just what sort of evidence might be convincing. Every species undoubtedly has rather special releasing stimuli for such behaviors whose efficacy, both absolute and relative, may not be known. Consequently, how could legitimate comparisons be made? Further, are all submissions the same within a species or across them? There is no question but that most species have a range of behaviors that are associated with defense and submission. They can be observed in free-roaming and laboratory-bred and-reared animals. They can occur in response to a variety of threats or environmental events. The issue is whether "tame" animals or strains selectively bred for docility show enhanced amounts of submission compared to wilder members of their species.

How stable are the defensive, submissive, and offensive reactions? Are they really hard-wired? It is curious that submissive behaviors tend to be ones appropriate to young animals, or those with sexual significance.

However, the real issue is whether there is scientific value in associating defense and submission reactions to specific regions of the central nervous system in certain species with implied generality to other mammals or in the notion of a "consociate modulator."

Both the defensive and submissive reactions are assigned locations in the central gray of the midbrain, although each is said to reside in different subpopulations of the region. Therefore, each of the behavioral patterns has a location in the brain stem - defense in A, submission in B Under some conditions the diencephalic "consociate modulator" selectively facilitates neural region B at the expense of A. But it can't be concluded that these are the only areas involved with motivational systems, since Adams indicates that these must include sensory "filters" and the lower motor mechanisms as well. Even if these centers are not "motivational," some component of them must facilitate the appropriate motor pattern, and others must influence sensory systems. If this is correct, how can these areas be "homogeneous?"

In Adams's theory various forebrain regions, especially those of the limbic system, are thought to funnel their influences via tracts passing in lateral hypothalamic areas to the midbrain centers. These include the septal area, the amygdala, the cingulate cortex, and so forth. A number of lesion and stimulation studies in cats and rats are cited to show their relevance to the motivational states. Many of these are classics of the literature, but there are rather difficult problems of interpretation. Lesion studies cannot be interpreted as reflecting the effect of loss of tissue per se. The effects of any lesion are partly the effects of secondary alterations in remaining neural tissues. These effects change over time. Furthermore, those effects produced by a lesion depend on the genetic background of the animals, the prelesion experiences of the animal, the post- lesion environment, and the testing procedures used. Many of these same considerations apply to electrical stimulation studies as well, but, in addition, factors relating to frequency, current density. amount of current, spread of current, and the like must be considered as important determinants of effects.

At our present state of knowledge about the brain and behavioral patterns, it seems premature to localize motivational systems in particular regions of the central nervous system. What is needed is more understanding about how motivational systems, localized or diffuse, exert their influences to govern behavior, and not where they are.

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Comments by Pierre Karli

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Emotional responsiveness and relevant history of reinforcement are important determinants of social behavior. In keeping with the basic concepts of ethology, Adams's three hypothetical motivational systems are characterized by narrowly specific, rather rigid (with little change over time), linear, and unidirectional relations between sensory input and behavioral output. Since our data concerning the rat's mouse-killing behavior point to emotional responsiveness and prior social experience as essential determinants, we are led to imagine brain manipulations and to interpret the results obtained in a somewhat different perspective. More concretely, each time we elicit or facilitate, suppress or abolish a given behavior (attack, defense, submission, avoidance, escape, flight) by means of some brain manipulation, the behavioral effect thus induced is not interpreted straight off in terms of altered functioning of a specific motivational mechanism. We rather try to take into account all the possible effects of the brain manipulation on the organism's level of overall responsiveness, on its capacity to integrate an affective significance with the sensory input so as to make the behavioral response coherent with previously shaped social-emotional adaptations, on the functioning and interactions of the systems of positive and negative reinforcement, on the bringing into play of these systems by some of the consequences of behavior that are anticipated or actually derived from it.

With regard to the existence of a rather rigid relation between specific motor-patterning mechanisms and a specific motivational mechanism, it might be pointed out that the mouse-killing response does not seem to be based on the same motivational state in the experienced killer-rat as when the rat is being presented with a mouse for the very first time (Karli et al 1974). The rather stereotyped and "cold-blooded" killing response displayed by the experienced killer-rat can be regarded as an appetitively motivated attack-behavior. When presented with a mouse for the first time(s), the rat mostly displays a killing- behavior that is rather of the "affective" kind - a behavior that can easily be elicited in the natural nonkiller by electric stimulation of medial hypothalamic and periaqueductal sites. The effective stimulation sites are in every case "switch-off" sites. Once the rat has learned to stop the brain stimulation, either by fleeing or by pressing a lever, it is much more difficult to induce it to kill a nearby mouse, This "affective" kind of mouse-killing might be considered a defense-behavior - a kind of active-avoidance behavior - killing being a way of putting an end to aversive experience. If the motivational state underlying the rat's mouse-killing behavior changes over time, some misinterpretations of experimental results are bound to occur, since initiation of mouse-killing in the natural nonkiller and abolition of killing behavior in the experienced killer-rat should no longer be regarded as two mirror-image processes.

It is both tempting and hazardous to try to specify precise and delimited locations for motivational mechanisms assumed to underlie behavior. A tempting project, since we are fully aware of the almost insuperable difficulties we would run against it we were to specify such mechanisms in terms of rather widely distributed and diffusely imbricated neuronal networks with complex reciprocal interactions. But also a hazardous project, since we are almost inevitably led to bestow upon a delimited brain structure or substructure an unduly global role

with regard to the generation of a specific motivation. More concretely, let's consider the following two facts (1) an experimentally produced hyperreactivity (eg following a septal lesion) facilitates initiation of mouse-killing if and only if the rat did not previously develop a stable inhibition of interspecific aggression on the basis of repeated contacts with mice; (2) destruction of the corticomedial amygdala or interruption of the stria terminalis interferes with the development of such an inhibition on the basis of prior experience with mice (Karli et al 1977). In other words, whether or not a rat kills the mouse with which he is presented depends in an essential way on mechanisms in which the septum and the corticomedial amygdala are deeply implicated. Could it then be meaningful to search tor a "motivational mechanism" that would, for instance, be limited to the central gray and would thus comprise neither the septum nor the amygdala?

The search for narrowly specific mechanisms, in which the central gray or the medial hypothalamus may be deeply implicated, should not lead to overlooking the more general functional role played by the periventricular system in the generation of aversive experience, and of one of the two basic attitudes of the living organism towards its environment (i.e. retreat or escape, as opposed to approach) as well as in the negative reinforcement of approach behavior. As a matter of fact, the stimulation of a great many sites located in both the central gray and the medial hypothalamus induces one common effect: i.e, the rat readily learns to switch off such a stimulation by means of a variety of behavioral sequences. Furthermore, when two "switch-off" sites are simultaneously stimulated, the resulting escape speed corresponds most often to the sum of the escape speeds induced by stimulating either site alone (Schmitt and Karli 1979). With regard to this more general role of the periventricular system in motivational processes, there are complex interactions between central gray and medial hypothalamus on the one hand, and between these structures and the reward- approach system on the other hand. Central gray lesions depress hypothalamically-induced escape behavior (Schmitt, Paunovic, and Karli 1979), and one records in each of the two periventricular structures unit activities that are correlated with the escape speed induced by stimulating sites located in the same structure or in the other one (Sandner, Schmitt, and Karli 1979). On the other hand, central gray lesions provoke not only a decreased responsiveness to fear-inducing aversive stimulations and situations, but at the same time a general facilitation of various appetitively motivated behaviors (Chaurand, Vergnes, and Karli 1972; Karli et al 1974). It is clear that much further research is needed in order to better understand the part taken by these periventricular structures in general motivational processes and in more specific ones, respectively.

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The risks of using descriptive ethological models in brain research. **The ultimate goal of many biological and psychological studies is to understand the way in which an animal is organized to survive in its natural habitat. In this article Adams focuses on the central nervous organization of agonistic behaviour - i.e, behaviour that can be observed in encounters between two male animals of the same species. On the basis ot ethological studies, three subsystems of this agonistic behaviour can be distinguished, namely offense, defense, and submission. It is to Adams's credit that he reviews, with this classification in mind, a large number ot studies in order to construct a general outline of the neural circuitries involved .**

Alhough one may express some doubts on ethological grounds about the value of this classification for the purpose of analysing brain mechanisms (see also commentary by Wiepkema), offense, defense, and submission are nevertheless recognisable behavioral strategies in the rather extreme situations in which the animals are usually tested.

The problem now is to analyse how the brain is able to process these strategies. For this, Adams uses descriptive hierarchical models from ethology. In such models a critical role is played by a common causal factor or the motivational mechanism. In Adams's terminology, this motivational mechanism is a hypothetical set of homogeneous neurons whose activity is held to be responsible for the motivational state of the animal. In my opinion, Adams makes a fundamental mistake in trying to give a unitary interpretation of such a common causal factor. Of course, the ethologically observable organisation of behaviour is embodied in the structure of the brain. Its units are groups of neurons, so organized as to produce the

various behaviours at the right times. However, such an organization need not necessarily be identical with the boxes posited in a hierarchical model. One box in the model - for example, the motivational mechanism - may be represented in the brain by many structures, possibly the whole limbic system, or even the whole brain.

In his attempt to find neural structures related to the boxes in the model, the author uses somewhat forced arguments. He states, for example, that the midbrain central gray meets the criteria for the motivational mechanism for defense, without explicitly mentioning these criteria. It is argued that neurons in the central gray are specifically active during shock-elicited defense. The original article (Pond, Sinnamon, and Adams 1977), however, only shows that these neurons are most active during shock-induced upright posture. If these units represent the motivational mechanism for defense, one might expect them to be active during other defensive behaviours as well - i.e. during lunge-and-bite attack, squealing, freezing, fleeing, and so forth. Also, if these units are specifically for defense, one might expect them to fail to be active during offense or submission. The original study does not give any answer to these questions, and therefore one cannot argue that these central gray neurons are specifically active during shock-induced defense. Moreover, these units are also activated by vibrissal stimulation, which is thought to be a releasing stimulus for defense. According to the model presented by Adams, such stimuli may not affect the motivational mechanism but the motor-patterning mechanism.

Some of the evidence for the role of the central gray as the motivational mechanism for defense is based upon fear or escape measured under nonsocial conditions (shock-food conflict task, one-way two-way avoidance). The relation of these measures to defensive behaviour in a social situation is far from clear.

Reading the target article, I realized more and more that it is too early to ascribe functions to the various neural structures involved in agonistic behaviour. In the first place, the flexibility of an animal in a social situation has been insufficiently explored ethologically. This means that it is difficult to express any expectancies about the internal organisation of behaviour (see also Wiepkema). Secondly, the effects of brain manipulations have been tested in a wide variety of test situations but rarely in a social setting. If our ultimate goal is to understand how an organism is adapted to its natural environment, knowledge about the relationship between the behaviour in our test situation and that under more natural conditions is a condition sine qua non.

At most we know, for some types of agonistic behaviour, which brain manipulations alter the probability of occurrence of that behaviour. However, questions like why, under what circumstances, and how specifically that probability is changed are often unanswered. In order to answer this, we have to test a wide variety of input variables known to affect the agonistic behaviour of an intact animal These are variables like previous experience, hormones, day-night rhythms, stimuli from the opponent, priorities for other behaviours, and so forth. Manipulation of each of these variables might alter the probability of occurrence, latency, sequential structure, and other mechanisms of agonistic behaviour.

The brain should accordingly contain mechanisms for processing information about these variables. My approach to the function of brain structures in agonistic behaviour is to test, with each brain manipulation, whether it affects one or more of these mechanisms. Not until a number of brain structures have been tested in this way can we speculate about functional organization.

In fact, the main part of this discussion concerns the problem of tuning the size of brain manipulation to the level of integration of brain functioning for agonistic behaviour. Adams has started this discussion, and I do hope that it will lead to a number of fruitful studies resulting in better understanding of the brain mechanisms in agonistic behaviour.

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Action-inhibiting system (AIS) vs. submission system. **I believe that the work of Adams is very interesting; I agree with the majority of the facts he joins together and, broadly speaking, with his general model for the neural circuitry that triggers offense, defense. and submission. Indeed, his model and my own published work have many points of similarity (see Laborit 1975. p 578).**

I have named what he calls the "submission system" the "action-inhibiting system" (AIS), and I have distinguished it, as he has, from the fight and flight system. But I think that this system is also distinct from the "conservation withdrawal" system, which little rodents and some human babies are able to use (Engel and Smale 1972).

Although Adams mentions the importance of memory, and of the animal's prior experience, for the functioning of this circuitry, I believe that this factor is not sufficiently emphasized. We have shown experimentally that the AIS is triggered by the memory of the inescapability of punishment, or, in others words, by the memory of the impossibility of coping with a previously experienced situation. The animal's response is not innate, in our opinion, but learned (Kunz, Valette, and Laborit 1974).

I regret, too, that the role of endocrine regulation in the behavior was not discussed (see Laborit 1976). It is possible to differentiate the systemic response to electrical stimulation of dorsomedial and basolateral amygdala, as well as that of dorsal and basal hippocampus (Laborit and Baron 1977; and Laborit, Baron, and Laurent 1977). The effect of

hydrocortisone on AIS activity and of adrenocorticotrophic hormone on the action-activating system (periventricular system and medial forebrain bundle) is also very interesting,

In the same way, some results of experiments involving intracerebroventricular administration of neuromediatory substances (dopamine, norepinephrine, muscarinic or nicotinic antagonists or agonists, and serotonin synthesis inhibitors or precursors) casts some light on the relations between neuroendocrine systems and behavior.

Perhaps the most fundamental criticism that will be made of this work concerns the basic approach to the problems exclusively by way of the stimulus-response concept. In particular, in our opinion, "motivation" arises from an internal drive, a homeostatic perturbation [see Toates "Homeostasis and Drinking" BBS 2(1) 1979]. External stimuli in particular give rise to imprinting, or, later, the encoding of pleasure, discomfort, or pain, which trigger reinforcement or inhibition. Therefore, learning processes use the innate final common neural circuitry of action or inhibition to control behavior (Laborit 1975-1978). But, aside from this light criticism, which only expresses my opinion, the Adams paper is a very full report and will be useful for every research worker in this field.

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Are neurophysiological techniques adequate to account for agonistic behavior? **David B. Adams's attempt to review and organize the available data concerning the brain mechanisms of agonistic behavior is admirable for its consistency and its mastery of methods and material. Since the data are still far from complete and the gaps in our knowledge are all too apparent, no one could at this stage succeed in creating an overall picture to everyone's satisfaction. Hence, I will simply state my general agreement with most of Adams's efforts and briefly outline my main reservations.**

These concern three points (1) an apparent bias, (2) an apparent overconfidence in the adequacy of neurophysiological methods and techniques, and (3) a misapprehension of some ethological views and behavioral phenomena.

1. Adams appears to be one of the last believers in the idea that a rigid stimulus-response hypothesis provides an adequate explanation for all behavior. Thus, since there is motivation, there must be motivating stimuli, and there cannot be stimulus-independent motivation. This leads to the demand for unitary motivational mechanisms consisting of "sets of homogeneous neurons." For reasons given later, I doubt the existence of such homogeneous motivational mechanisms. Moreover, while no one doubts that stimulation can bring about changes in the motivational state of animals, there is equally no doubt that internal, stimulus-independent changes of that state are continuously going on, and that the internal, autonomous generation of motivation, variously named "drive," "instinct," or "propensity," is not only equally important but constantly "setting" the peripheral and central afferent mechanisms concerning which kinds of stimuli are to be reacted to at any given time, and how, and which stimuli are to be disregarded. A stimulus is not a stimulus unless the internal motivational mechanisms make it one.

Consequently, I cannot agree with the distinction made between the motivating and the releasing function of stimuli. Nor does it make any difference whether a stimulus releases a motor pattern or its directional mechanisms (taxes), which are also motor mechanisms. The function of the stimulus is merely to touch off and sustain this motor apparatus, which in turn does all the orienting. Therefore, strictly speaking, there is no such thing as a directing

or orienting stimulus. In conclusion. and in accordance with N Tinbergen, who first made this plain, there are releasing (or eliciting), supporting, and consummatory (switch-off) stimuli, and that is all.

2. Adams states: "Despite the fact that electrical stimulation of the hypothalamic ventromedial nucleus produced affective defense, the neurons of that nucleus are not active during affective defense elicited in a seminatural situation. Puzzled, I came to the conclusion *that our behavioral control was not adequate to the demands of of neurophysiological techniques*" (commentator's emphasis). Apart from finding the second sentence somewhat cryptic, I should like to reverse what I take it to mean: Neurophysiological techniques certainly are still far from adequate to the control of behavior as exercised by the organism itself, notwithstanding all modern refinements. Even a micro-electrode is too clumsy a tool, compared with the cellular mechanisms with which it rather brutally interferes. And however tiny the electrodes used may be, they are not even micro-electrodes in most electrostimulation experiments, and certainly not in the type of experiment Adams is referring to here.

Furthermore, all of the more complex behavioral patterns that may be elicited by electrical brain stimulation can be elicited from a wide rage of loci, usually extending from the amygdala down to the midbrain and even the medulla. This is in keeping with the wide variety of internal and external factors that contribute, separately or jointly, toward the manifestation of the behavior in question. To assume that in any given case all should always be active seems to me quite out of the question. It would be most interesting to provide all the loci from which, say, attack can be elicited with electrodes in the same experimental animal and then vicariously use one for stimulation and all others for recording. To my knowledge, this has never been done so far; but my prediction would be that the number and kind of loci from which recordings could be obtained would vary greatly with the site chosen for stimulation, stimulation intensity and duration, and the interval between stimulations.

The systems we try to study are of a rather complex nature and certainly farther from being unitary or homogeneous than Adams seems to allow. Much as the stimulation experiment is by far the most useful tool at present available for such investigations, we must not hope that a point-by-point stimulation procedure, as originally followed by W.R. Hess and more or less by all his successors, can provide us with a true picture of all the organisational complexities involved in behavior such as defense or rival-fighting. Even less may we expect this from the results of lesion experiments, which are notoriously misleading when used to interpret the neuronal basis of complex behavioral patterns. Here. I feel, it would have been more valuable if Adams had attempted to trace clearly the still extant gaps rather than to depict a fairly complete system.

3. When a cut-off tail end of an earthworm is attached to its former front end with a piece of thread, it will follow the front with well-coordinated creeping movements. Thus was it demonstrated that the locomotion of an earthworm is a chain reflex. Later, when E. von Holst (1932; 1933) treated the middle of an earthworm with an acid that ate away all the

tissue except the nerve fibers, that preparation also showed well-coordinated locomotion in both front and tail, although the nerve fibers were too tender to exert any pull on the tail end; thus it was demonstrated that earthworm locomotion is controlled by a central nervous automatism The whole case highlights the danger of thinking in alternatives where biological systems are concerned.

Likewise, agonistic behavior is produced by both endogenous and exogenous factors in parallel, the endogenous producing the generalized or "ideal" pattern, the exogenous modulating and adapting it to suit the irregularities of the here-and-now conditions of the actual situation. This is one reason why it is not possible to subdivide agonistic behavior into rigidIy closed subsystems of offense, defense, submission and flight. The other is that these subsystems have a varying number of behavioral elements in common. In fact, the subsystems of offense (including .'aggression") and defense have most of them in common, regardless of whether they are being directed toward a conspecific or heterospecific adversary or toward a prey animal. The difference lies not so much in the few elements specific to each of them but in the relative order and intensity in which the elements are performed. Even within each subsystem, that order varies according to the situation and the individual and actual motivational state of the individual before the releasing situation arises. It is this hierarchically organized, endogenously coordinated subsystem, created anew each time, which is the "motivational mechanism," and it quite certainly is not unitary, nor is it permanent: it is a disposition, a potentiality, rather than a fixed structure, and for all we know it may use a fairly large number of brain structures and mechanisms vicariously. The ethological analysis of these complexities and my interpretation of the results I have given in detail elsewhere (Leyhausen 1965: 1979 a. b).

E. von Holst and U. von St Paul (1960) have demonstrated that even such complex problems of behavioral organization can be attacked successfully using electrical brain stimulation. It is to be deplored that no other investigator has followed their example.

Reviews such as Adams's are both useful and necessary, not in spite of but because of their at least partly controversial nature. Apart from this. I am inclined to think that, in that part of neurophysiology which attempts to deal with complex behavior, the rule of parsimony leads to oversimplification more often than not, and proves itself a hindrance rather than a help to progress.

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Comments by Klaus A. Miczek
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What are the chemical characteristics of brain mechanisms for aggression? **Adams is to be complimented on a well-presented integration of behavioral and neurophysiological data on the brain mechanisms for offense, defense, and submission. Unlike the popular classification schemes, many of which were presented by nonexperimental, armchair theoreticians during the last decade, Adams's proposal is founded on his own experimental experience and that of others. The majority of the references and the largest amount of discussion is devoted to describing the brain mechanisms concerned with defensive behavior. And, in fact, most experimental work on neural mechanisms of "aggression" actually involves defense. Whatever caused Hess's observation of "affektive Abwehr" to be called "affective aggression"? Considerably less information is available on brain mechanisms of submissive behavior and even less on those Systems regulating offense.**

Although it is difficult for me to present a critique, given that I am acknowledged by the author for earlier discussions of his ideas and sharing so many of his views, I offer the following four points:

1 The proposed outline of brain mechanisms regulating offense, defense, and submission follows the format and logic of traditional physiological psychology, composed basically of (1) sensory input. (2) "motivational" mechanisms, and (3) motor output ("patterning mechanisms" plus discrete acts, postures, and movements) with a few feedback and feedforward loops. Even if one accepts this type of flow of information as possible, a neural network subserving such functions has to be demonstrated. Most importantly, convincing

identification of "motivational mechanisms," particularly that for offense, in the form of actual neural activity, has for the most part eluded investigators. I do not think that a single study in cats, reporting on four neurons active during "affective defense," is sufficient (although it is highly intriguing) evidence for a "motivational mechanism." Similarly, large-size destruction of midbrain structures and the subsequent observation of behavioral dysfunctions can hardly be considered cogent proof for motivational mechanisms. The postulated higher-function "motivational mechanisms" and "master switches." which ultimately determine whether movement A or B is exhibited, at present exist only as blocks in flow charts, not as "real" data.

2. Adams's neural circuitries describe mostly descending information flow. It is a great pity that he has chosen not to consider neurochemical and neuropharmacological data on anatomical systems as they relate to offense, defense, and submission. Many of the dopamine-, norepinephrine-, serotonin-, and GABA- containing pathways are ascending from mid- and hindbrain to subcortical and cortical terminations. There is, of course, a great deal of information relating the activity of these neurotransmitters to various modes of agonistic behavior (see, for recent reviews, Miczek and Barry 1976; Miczek and Krsiak 1979). The histochemical evidence suggests strongly that the brain structures portrayed in Adams's charts cannot be considered "functional anatomical units.' I think lesion and stimulation studies are of limited value in finding functional units. Describing behaviorally relevant neural networks in terms of anatomy as well as chemistry appears to be a prerequisite for modeling brain mechanisms.

3. Adams's distinction between submission and defense is, so far, the most convincing discussion of these modes of agonistic behavior. Yet, I am puzzled by the lack of discussion concerning flight. What is the relationship of submission and defense to flight? How is it possible to discuss submission and flight without reference to the form of social life characteristic for a particular animal species? For example, is submission prevalent in species that live in groups? And, do species whose members live a solitary adult life tend to rely on flight? The decision as to whether an organism engages in defense or submission is postulated to be made by a "consociate modulator" at the level of the ventromedial hypothalamus. An alternative view would predict that an organism exhibits defensive or submissive behavior mainly as a result of previous fighting experience and the intensity of the attack to which it is subjected.

4. Along with other authors. Adams emphasizes that a sensory comparator mechanism decides on the familiarity of a conspecific. If the comparator reads "unfamiliar," this signal becomes an important determinant for offense to occur. My own recent experimental experiences with mice, rats, and squirrel monkeys (Miczek 1978; 1979; Miczek and O'Donnell 1978) substantiate the great significance of the "stranger" as a proximal cause for offensive behavior. Yet, what are the releasing and directing stimuli for intragroup conflict? Offensive behavior toward a familiar conspecific or consort does occur even without apparent competition for food, mate, or shelter. The rather complex and multiple causes of fighting within established groups of rodents or primates appear largely ignored by Adams.

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Comments by Jaap Panksepp
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Offense and defense vs. rage and fear: A matter of semantics? **The manner in which events are classified determines the manner in which they are studied. Adams's conceptualization that agonistic behaviors are controlled by distinct offense, defense, and submission motivation systems is capable of organizing a great deal of neuropsychological data and accordingly provides a neurotaxonomy that begins to combine distal evolutionary and proximal neurophysiological causes into a viable model of aggressive behavior. The categorization of agonistic behaviors with reference to the deep structure of brain circuits is probably more useful for understanding aggression than taxonomies that focus merely on eliciting conditions or on specific kinds of aggressive acts. Different eliciting conditions (releasing stimuli) probably converge upon common control systems, and these intervening "command systems" can probably initiate a number of related behavior sequences. which are then filtered and molded by their outcomes.**

In conceptualizing discrete offense, defense, and submission systems, Adams is concerned that the postulation of separate defense and submission systems may be gratuitous; but in the end he remains convinced ot the utility and reality of the distinction. I am not. Might not offense and defense suffice as primary systems, with submission being merely a conditional, and hence a diminutive, form of defense, in the same way that anxiety may be a learned diminutive form of fear? In Adams's system, submission becomes a separate category, because the defense system (as used by Adams) can instigate certain offensive gestures (lunge and bite attack), and hence a separate category is required for the tendency of animals to remain totally defensive. However, if one assumes that defensive threat and aggression - which can be evoked from cornered animals or induced by brain stimulation (so-called affective attack) - take the form they do because both defense (fear?) and offense (rage?) systems are concurrently activated, then the need for a separate "submission motivation" system disappears.

I believe that the available data concerning aggressive behavior can be handled as readily by two primary motivational (or command) systems as by three. For instance, the argument that medial hypothalamic lesions increase defense and reduce submission, while amygdala lesions decrease defense and increase submission, may be semantically recast to conclude that medial hypothalamic lesions increase both offense and defense (leading to a high incidence of affective attack or defensive threat), while amygdala lesions reduce offense (hence all forms of aggression), leaving defense (and hence submission) processes without opposition. Of course, it must be emphasized that, within such a conceptualization, defense has a somewhat different connotation (namely fear) than might be agreed to by Adams.

Although I take issue with the distinction between defense and submission, I strongly agree with Adams's postulation of a "consociate modulator." All specific social acts are expressed in broad affective contexts - of social comfort or social unease - and through some kind of neural representation of such processes, whether they be called "consociate modulators" or simply "social affect" (Panksepp et al, 1978), the probability of offensive or defensive emotions is heightened or diminished. Although Adams's system is useful for analyzing aggressive behavior, on a more general level, the proper taxonomy for the basic neural systems that subserve instinctual emotive behavior patterns remains an open issue. Thus, one potential problem with Adams's taxonomy is its exclusive focus on emotive behaviors analyzed in the context of aggression. Potential behavioral controls exerted by these same systems in nonsocial contexts are ignored. Although it is possible that these systems act solely or primarily in the context of aggression, my opinion is that offense and defense systems are really generalized emotive systems that can control behavior in a variety of situations. Accordingly, I have recently used the label "rage sensory-motor command system" and "fear sensory-motor command system" (Panksepp 1980) to describe what t think Adams is referring to in his "offense" and "defense motivational systems" [see also Kupfermann & Weiss: "The Command Neuron Concept" BBS 1(1) 1978].

My use of everyday emotional terminology is specifically intended to convey the possible generalized roles of hard-wired emotive systems in the brain. Thus, I would assume that the neural representations of "frustration"(i.e. the failure of expectancies to be met) would "enrage" animals and thus activate the same primitive command circuits that are active when a male rat intrudes on the homeground of another male. This kind of seemingly minor semantic distinction can have marked experimental repercussions. For instance, rather than simply looking at the activity of ventrolateral hypothalamic cells during aggression, one might also be led to look at their activity during extinction of appetitive tasks, with the prediction that certain cell populations that fire when rats attack intruders would also be active during extinction. Simply viewing such cells as part of an "offense" circuit may not lead as readily to such a broader analysis.

To take another example I would predict that threat to the bodily integrity of an animal, whether arising from a laboratory shock-generator or the blood-lust of a predator, would trigger common limbic command systems. A "fear" designation for such a system could, I think, more readily coordinate experimental results derived from different threatening situations. I think such distinctions are important, and under a different verbal disguise they have led to the very real controversy as to whether the hypothalamus contains "specific" control systems that orchestrate behavioral acts or .'nonspecific " emotive command systems for biassing classes of behavioral tendencies (see Valenstein 1973) Although this controversy still remains unresolved, my evaluation of the evidence is that the limbic "command" systems that can sustain "stimulus-bound" behaviors are generalized emotive circuits (Panksepp 1971; 1980). Since Adams analysis is implicitly based on the existence of such "stimulus-bound" behaviors, it might be useful if he clarified his stance on the issue.

In the final accounting, what matters in the selection of terms is the success of the research that various conceptualizations generate. If the underlying brain processes are nonspecific, then it would be useful if the labels for the systems reflected the nature of the generalized processes rather than a more limited class of behaviors that can be provoked. Of course, these issues can only be resolved empirically, but I think the nonspecificity notion can accommodate more of the existing data then approaches that seek a more articulated phrenology in the executive systems for emotional behaviors (Panksepp 1980). In any case, our own working hypothesis is that the limbic "command" systems that sustain "stimulus-bound" behaviors are normally active during broad classes of environmental events that have required similar types of adaptive responses in the evolutionary history of the species. Thus, emotive "command" systems may activate sets of related behaviors in a variety of situations, and contiguous reinforcement processes may provide the selective force for determining which acts become habits in the behavioral repertoire of animals. Thus, I see submission as a habit that arises from repeated activation of defense systems, as well as perhaps from repeated nonreinforcement or punishment of offensive gestures. Rather than being a primitive behavior control system in its own right, submission may be the final behavioral outcome of the consequences of offensive and defensive acts.

In any case, I think Adams's contribution is a substantial step in the right direction. Brain systems are provided with conceptual tags that can guide further work into the deep functional structure of neural circuits controlling emotive behavior. However, I would be tempted to take an even larger step in the same direction - to see whether we can make sense of behavior by categorizing the command systems that appear to run through the limbic system in terms of certain old emotional constructs that have been avoided in behavioral research for the better part of this century. Although the surplus subjective connotations of terms such as rage and fear can easily lead to anthropomorphic excesses, I suspect that such a set of terms can help us come close to the reality of limbic organization than more conservative concepts. Indeed, by thus identifying and labeling neural systems in terms of distal evolutionary causes rather than ongoing neurophysiological processes, we may begin to make better sense of the myriad facts that the fabric of the brain continues to yield in abundance.

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Changing methodology in aggression research. **David Adams provides a long overdue review of recent research on the neurophysiology of agonistic behaviour. However, to simply refer to this work as "a review" does not do justice to the conceptual framework within which research findings are presented. The author has adopted a "neuroethological" approach to**

the problem and, as such, has placed considerable emphasis upon thorough behavioural analysis. This awareness of the value of ethological methodology has not been a characteristic feature of neurophysiological/ psychopharmacological investigations of agonistic behaviour over the years.

Scott (1966), in pointing out that "aggression is a poor scientific term," suggested that fighting behaviour cannot be fully analyzed without also studying the alternative patterns of escape, threat, "freezing," defensive posture, dominance, and subordination. To cover this behavioural system, composed of behavioural patterns having the common functions of adaptation to situations involving physical conflict between members of the same species, he coined the term "agonistic behaviour." His own studies and those of Grant and Mackintosh (1963) provide detailed accounts of the social postures of the most commonly used laboratory animals: rats and mice. Unfortunately, the significance of these contributions has largely been ignored, with most research relying heavily upon rather simplistic (and perhaps somewhat dubious) models of "aggression." However. within the past five years, several dominant trends have become apparent in the literature, which indicate that methodology in aggression research is undergoing a metamorphosis. I believe that these trends may be summarized as follows:

1. The development of more "naturalistic" test paradigms This approach, exemplified by the work of Blanchard's [q.v.] group on colony reaction to intruders (Blanchard et al 1975; Blanchard and Blanchard 1977; Blanchard, Takahashi, and Blanchard 1977; and Blanchard et al. 1977c) and Miczek's [q.v.] group on fighting generated by frustrative nonreward (Miczek 1974; Miczek and Barry 1974; 1977) facilitates the detailed observation of the effects of physiological/ pharmacological manipulation on the full repertoire of agonistic response patterns in rats.

2 The recognition of differential behavioural effects of manipulation, depending upon the status of the treated animal. Miczek (1974) has shown quite different effects of amphetamine given to dominant or subordinate animals: In the former, low doses of the drug enhance all elements of attack, whilst in the latter, drug treatment results in exaggerated defensive and submissive postures.

3. The realization that the behaviour of an untreated animal may alter in the presence of a treated animal: Early work by Krsiak and Steinberg (1969) showed changes in the social behaviour of undrugged rats in the presence of chlorpromazine-treated rats. More recently Miczek (1974) has reported that subordinate animals, under the influence of amphetamine, provoke more attacks and threats from undrugged dominants.

Recent research, through the application of this methodology, has highlighted the fallibility of some "established facts" in aggression literature. The minor tranquilizer, chlordiazepoxide, long thought to reduce aggression, has recently been shown to dramatically increase attack and threat in rats (Miczek 1974). Factors such as drug dose, status of the injected animals, and the test paradigm appear to be critical determinants of behavioural response. Septal lesions, often reported to induce "hyperaggressivity," have

recently been found to reduce elements of attack in both colony (Blanchard et al 1977b) and food competition (Lau and Miczek 1977) situations. Results such as these tend to cast doubt upon the conclusions reached in earlier studies on the physiology of aggression, and they stress the necessity for at least a reformulation of ideas in the light of detailed behavioural analysis.

The most often used laboratory model of intraspecific fighting in rats has been shock-induced "aggression." Various authors have questioned the use at the term "aggression" in this context, suggesting that pain-elicited fighting more closely resembles defense (Scott 1966; Reynierse 1971; Powell 1974). In a recent thorough analysis of fhis issue, Blanchard's group has provided considerable support for these earlier suggestions (Blanchard, Blanchard, and Takahashi, 1977; Blanchard et a11977a; Blanchard, Blanchard, and Takahashi, 1978), They have demonstrated many parallels between the responses of shocked animals and those of colony intruders, and they have concluded that the behaviours displayed in response to shock are primarily defensive, not aggressive. In view of this detailed appraisal, it would seem imperative to revise many of the conclusions concerning the neural/neurochemical bases of "aggression" that have resulted from the use of this model. Of course, this suggestion would not necessitate a "scrap and start again" policy, but rather a reclassification in terms of mechanisms of defense. Examined in this manner, existing research findings on shock-induced defensive fighting might be correlated with results from future studies on the physiological analysis of intruder behaviour in a colony paradigm.

In his article, Adams makes an important distinction between patterns of defense and submission, but he seems rather uncertain concerning the classification of data from the shock-induced fighting literature. At one point he refers to defensive upright posture and boxing whilst in another context he considers that this posture is submissive, In view of the above discussion of Blanchard's studies, it would appear that the present formulation requires some revision in order to maintain internal consistency. This type of problem, I believe, reflects the fact that most researchers have not previously addressed themselves to specific questions concerning the physiology of agonistic behaviour. As a result, Adams, whilst achieving a remarkable synthesis of the data, has been forced (in many instances) to disentangle from the literature exactly which behaviours had been studied.

Adams has now provided us with preliminary, yet detailed, "models" of the neural circuitry involved in offensive, defensive, and submissive behaviour patterns. In the light of this invaluable service, and bearing in mind the foregoing discussion, surely the moment has come to argue for the precise evaluation of these "models" in relation to specific elements of agonistic behaviour?

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Tentative analysis of apomorphine-induced intraspecific aggressive behavior in the rat according to Adams's classification. **Administration of apomorphine in rats induces signs of intraspecies aggression. Three categories of rats have been thus distinguished: (a) those in which these signs are severe, long-lasting, and reproducible; (b) those in which these signs are moderate, briefer, and inconstant; and (c) those in which these signs are absent (Senault 1968; 1970).**

During these aggressive manifestations the animals rear on their hind legs, vocalize, and lunge and paw at the opponent's face with their front paws or teeth. This behavior may be quite damaging, and seems to correspond to the "defensive behavior" among the different categories described by Adams.

Experiments performed on two extreme aggressive versus non aggressive animals (Senault 1973; 1977) have shown the following effects of destruction by electrolytic lesions (or aspiration in case of olfactory bulbs);

(1) Septum, putamen and ventromedial hypothalamus; no affect on aggression; There is no inhibition in the aggressive rats, nor is there facilitation in nonaggressive rats.

(2) Lateral hypothalamus, globus pallidus, substantia nigra and amygdala can reduce or sometimes inhibit completely the occurrence of aggressive behavior in aggressive rats.

(3) Olfactory bulbs, caudate nucleus can elicit appearance of these signs in nonaggressive rats.

A comparison of these results with the schema for the neural circuitry of defense and offense proposed by Adams, shows that apomorphine-induced aggressive behavior differs:

(i) from both schemes in terms of the absence of the role of septum.

(ii) from the defense scheme by the fact that the ventromedial hypothalamus has no role in it. We have shown (Senault 1977) that the absence of influence of the ventromedial nucleus distinguishes this aggressive behavior from that induced by electric shocks, which is favored by lesions of this structure (Panksepp 1971a; Eichelman 1971; Grossman 1972).

(iii) from the offense scheme by the fact that the influence of olfactory bulbs seems the opposite: facilitative in offense behavior, inhibitory in apomorphine- induced aggressiveness. This is also the case because of the essential role of the globus pallidus in this aggressive behavior: apomorphine seems to have an attack function at this site since injections of this substance into this structure can elicit aggressive behavior (Senault 1977). The results, in agreement with those reported by MacLean (1978), provide one example of the role of the globus pallidus in intraspecies aggressive behavior in the rat.

These data would be an argument for considering this behavior as an offense behavior. It is also this offense system that represents the neural ciruitry the most similar to that of apomorphine-induced behavior. The observed differences are referred to globus pallidus, septum and olfactory bulbus: As a result of our studies and those of McLean, the globus pallidus might be included in the "offense" system; and as Adams stated, the data reported in the literature related to the septum are not univocal. The same holds true for the olfactory bulbs In point of fact, considering only rats, and setting aside the behavior of mice-killers - which is favored by olfactory bulbectomy (Vergnes and Karli 1963) as well as hypermotility syndrome, similar to the septal syndrome induced by bulbectomy (Douglas, Issacson. and Moss 1969; Kumadaki, Hitomi, and Kumadi 1967: Ueki, Nurimoto, and Ogawa 1972) - the variability of the effects obtained after this operation is striking. It can depend on the size of the lesion (Bandler and Chi 1972), the strain of the experimented animal (Thorne and Linder 1971), and the aggressive behavior under study (intra or extraspecies aggressiveness in regard to manipulator or object) (Bernstein and Moyer 1970; Bugbee and Eichelman 1972) - which means that aggressiveness is not a unitary phenomenon and that the neural structures involved vary as a function of the observed behaviors.

In his tentative study Adams has dealt only with intraspecies aggression, but we should note that apomorphine-induced aggression is indeed a type of intraspecies aggression, one that seems to be akin to the behavioral postures observed in the defense scheme on the one hand and to the neural profile of the offense scheme on the other. This form of aggression does not seem to integrate with Adams's proposed classification, and may well call into question its underlying principles.

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Comments by Holger Ursin
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Aggression and the brain: Reflex chains or network? **Adams bases his classification of intraspecific aggressive behavior on a postulated neural circuitry of social behavior. His system is interesting and challenging. However, acceptance of the model rests on acceptance on the neural circuitry. The flow diagrams are meant not only as intervening variables, representing a hypothetical flow of information; they are also assumed to represent structures, and therefore they assume the power of hypothetical constructs (MaCorquodale and Meehl 1948). Adams suggests that the information flow is really from the stimulus through several structures, ending up in responses.**

My first difficulty is with this basic assumption of the brain functioning as a reflex chain, since we know that the brain is built as a network. There are complex feedback controls, gates, and a substantial source of variance intervening between stimuli and responses. In his discussion Adams mentions the fact that many of his arrows of postulated information flow may be reciprocal. I would suggest that all arrows on his flow diagrams are reciprocal, and that there is really no good reason, except tradition, to stick to one of these directions, If this is true, simple feedforward models for brain functions become hard to accept, in particular when stimulus "control" of behavior is assumed. Adams states that he finds ethological stimulus concepts like "releasing" and "motivating" stimuli helpful and meaningful.

At least my own neuroethological work on cats with frontal, cingulate, and septal lesions fails to agree with his statements. Flight and defense behavior in feral cats (= defense in Adam's definition) are not affected by these lesions (Ursin 1969; Ursin and Divac 1975). Also, I do not know what is lost due to a lesion affecting 90% of the mesencephalic central gray, but I doubt that the resulting behavioral deficits are explained satisfactorily as a loss of defense. The direction of information flow is particularly doubtful for this area. Ascending systems from very small areas within the central gray and in neighboring areas have a profound influence on a variety of behaviors: for instance, pain (see Liebeskind 1976), sexual behavior (Sodersten, Berge, and Hole (1978), and also defense behavior in a more strict sense (Hole, Johnson, and Berge 1977). For amygdala there is also a quite specific disagreement between Adams and other authors. This is due at least in part, to different behavioral terms.

The author postulates three main motivational systems defense, offense, and submission . There is a growing consensus that it is necessary to discriminate between offense and defense. However, Adams's defense concept differs markedly from the ordinary ethological one, as well as trom the general use of terms in psychology. Adams defines defense as the behavior or "motivational system" in "wild" animals, regardless of whether this is flight, defensive postures, or threats. When the identical behavioral pattern occurs in intraspecific behavior, it represent "submission." If it occurs as a response to an object or an unidentified stimulus source, like uncertainty itselt in open-field behavior, or a shock prod,

it is impossible to classify the motivational state following this system. If the behavior is elicited by brain stimulation, it is likewise impossible to classify, since the stimulus condition is now bypassed.

The classification differs from conventional ethological definitions. Leyhausen's [q.v.] descriptions of cat defensive behavior (Abwehr - "defense sensustriction" - SS) is from a cat-cat situation, but identical behavior is observed in cats confronted with dogs or humans (Ursin 1964). Adams's classification is not based on neural circuitry, as was his original goal. The differentiation he suggests is not supported by such data. He assumes "parallel pathways" to explain why he postulates more than one system where only one has been demonstrated. The system also fails to account for the differentiation that has been found for flight and defense (SS) (see Kaada 1967). I believe that a cautious, nontheoretical approach based on ethological and neuroethological observations leaves us with three aggressive categories (defense, attack, and prey-killing) and one type of fear behavior (flight). In addition, freezing is a fear category that seems to be generally accepted In neuropsychological lesion work, freezing and flight have proven to be fruitful concepts for explaining learning deficits (see Kaada 1967; Ursin 1969).

The terms "aggression" and "fear," as used here, follow conventional definitions in psychology. Behavior is aggressive it an object or other individual is damaged or is threatened with damage. Fear is also related to threats, but the individual is now avoiding contact with the stimulus, either "passively" (freezing - passive avoidance) or "actively" (flight - active avoidance). This conservative set of definitions has the advantage of relating to experimental psychology and learning theory as well as neuropsychological data from limbic structures.

Before we conclude that a structure plays a crucial role for a certain behavior, several criteria should be satisfied;

1. The particular behavior should be readily identified in the naturally occurring behavior of the animal, and the terms used should be as close to ethological terms as possible.

2. The behavior should be elicited by electrical or chemical stimulation of that particular structure.

3. Units in that structure should change their activity during execution of that particular behavior.

4. The behavior should be reduced by a lesion to this particular structure.

5. Lesions should produce a handicap in learning problems where this particular action pattern is important for the execution of the instrumental behavior.

6. Electrical or chemical stimulation should "jam" the ordinary stimulus control of that behavior.

7. Pharmacological manipulations should elicit, eliminate, or jam this type of behavior.

8. At least some phylogenic homology should exist across species.

9. The behavioral changes produced should be fairly specific for !his particular response, and not be a general effect on all kinds of motivational systems.

The amygdala control of flight and defense behavior seems fairly well established, even with these criteria (Ursin 1965; 1972). For the other areas in Adams's flow diagrams. this strict criteria-set is not met. However, it should be admitted that it is much more difficult to fulfill these strict criteria for brain stem structures, where an anatomical localization is difficult due to the many ascending and descending systems. Adams's work is extremely interesting and challenging, but it does not resolve or eliminate controversies in this field. This was probably not the intention of the author, nor is it achieved by my comments.

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Offense, defense, submission, and attack: Problems of logic and lexicon. **In attempting to bring coherence to the unruly mass of data on the brain mechanisms of aggressive behavior, Adams has taken on a thankless task. I was pleased to read that he was arguing for both a more precise behavioral categorization of responses and a more thorough behavioral analysis. However, his subsequent acknowledgement that the response categories (offense, defense, and submission) were not mutually exclusive was perplexing. It is commonly thought that such categorizations are useful only to the extent that they are based on a logical set of behavioral distinctions. Adams instead suggests that the neural circuitry of behavior can be used as the basis for behavioral categorizations. As a specific example, Adams maintains that "offense consists of those behaviors under the control of an offense motivational system." Because I believe there are problems with this approach. and because these considerations are basic to the neurological model presented, I shall restrict my comments to this area.**

Adams's suggestion that a physiological substrate can be used as the basis for behavioral categorization is not new. Such a strategy is used in those investigations that classify stimuli as stressful if they increase corticosterone. Many physiological psychologists hold that while such a categorization is formally logical, it is inappropriately focused. The basis for its logical acceptability Is clear; however, corticosterone can be measured independently of behavior. Unfortunately, a behavior-independent measurement of brain motivational systems is not possible. The interrelationship between the behavior being defined and the construction of the defining mechanism makes the logic associated with a neural categorization of behavior circular. To make this clearer, imagine the task facing a new investigator using the Adams schema to determine which of the so-called aggressive responses should be placed in the offense category. Because Adams maintains that behavioral categorization occurs as the result of manipulating the brain mechanisms, the new investigator's research is logically assured of failure. He cannot begin to localize the brain mechanism, because he has no way of knowing when he has mapped out the appropriate mechanism. Normally this would not be a problem, because manipulations of brain are related to changes in responses already classified according to an independent set of criteria.

Although Adams maintains that his behavioral categorization is "ultimately based on neural circuitry," it is possible that he actually means that neural circuitry data can be used to supplement and strengthen a categorization schema based on behavioral descriptions. In this context, however, the strategy is not helpful, because response topographies appear in more than one category. Possibly, and hopefully, Adams uses undescribed situational variables to specify the appropriate categorization of a response. In the apparent absence of such objective guidelines, responses appear to be shifted between categories merely to support the argument of the moment. Adams provides examples of shifting of responses from one category to another where fleeing is first claimed to be a

submissive response, then a defensive behavior. Shifting is again found where the boxing response is first seen as defensive and then as submissive. Another problem with the present categorization is that it cannot constrain the extent of post hoc analyses. At one point, for example, it is suggested that data inconsistent with the model could be made consistent by arbitrarily shifting a response from one category to another. A more widely accepted strategy would be to accept the data as discordant and consider changing the model.

A major difficulty for reviewers of this area is that there is no standardization of terms used to describe behavior. At selected points Adams apparently feels compelled to translate the work of original authors into his own terminology. For example, "attack" is translated by Adams to defense. The legitimacy of this translation is open to question, however, because the term attack is commonly "opposed to defense" (Webster's New Collegiate Dictionary). A rereading of the original work in question (Baxter 1967) makes it fairly clear that the author meant to connote offense with his choice of the term. A similar translation problem arises where "fear" is translated to submission. The experiment Adams refers to here has an interesting interpretive history and typifies the problems of the area. The original author reported that electrical stimulation of the thalamus elicited a low-profile posture with side-to-side head movements and occasional low-profile forms of locomotion. The posture was provisionally termed crouching. At this point the original author created the opportunity for misinterpretation when he noted that the response topography was similar to that seen in what he called "fear producing situations" (Roberts 1962). The use of the term "fear" allowed attention to move from the observable elicited behavior and towards its inferred motivational or emotional basis. Because inference easily becomes acceptance, the term fear became reified, and the original experiment was interpreted as indicating that electrical stimulation of the thalamus produces "fear." Adams takes this sequence further when he translates fear to submission. As a result, the original work is interpreted as indicating that electrical stimulation of the thalamus elicits, if not submissiveness, at least submissive behavior. One begins to wonder how many translations can occur before interpretations are completely divorced from data. It is clear from this example that the area needs to abandon terms referring to inferred mental or emotional states. Instead, clear and simple descriptions of observed responses should be used.

On the matter of behavioral models, I would like to point out that there is an alternative to the "and gate" formulation presented by Adams. The "and gate" model requires that the motivating and releasing stimuli be simultaneously present before a response can occur. Such a model grows out of laboratory experience with solid-state modules and helps to defocus our attempts at understanding behavior. The alternative model is simpler and more focused in its behavioral analysis. It does not distinguish between motivating or releasing stimuli but instead views all behavior as response chains. The length of the chain and the nature of the component responses may, of course, vary between behaviors. The questions for bio-behaviorists is straightforward. What variables (external and internal stimuli) control fhe various components of the chain, and what aspects of brain respond to, or process, these stimuli?

A recent study on mouse-killing behavior has exemplified this approach (Waldbillig 1979). It was found in this work that lesions of the area adjacent to the rat mesencephalic central gray, an area where electrical stimulation elicits mouse-killing in natural nonkillers (Waldbillig 1975), blocked this behavior. Interestingly, however, the lesions blocked only the killing bite. Orienting towards the mouse, tracking the mouse's position, and lunges toward the mouse were not affected by the lesion. Clearly, it would be inappropriate to interpret the lesions' effects as due to the loss of a mesencephalic "motivational mechanism." Instead, a discrete portion of the behavioral chain was altered. Such an interpretation leads naturally toward determining exactly what variables control the killing bite and how this area of the brain is involved in processing these stimuli.

When biopsychology focuses on both the stimuli that naturally control behavior and how the brain responds to these stimuli, it will have taken a major step away from the speculation inherent in intervening variables and virtually unverifiable hypothetical constructs. By avoiding post hoc speculation, and by staying close to the behavioral data, we will ultimately be able to specify the neurological interactions involved in these important behaviors.

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On the specification ot motivational systems. **In brain/behaviour studies an ultimate question is how the brain organizes and regulates entities like reproductive behavior, food intake, and the like as they occur under natural conditions. In spite of much research, the answers to this question are still very preliminary. It is the merit of Adams's paper to survey the relationship between one such entity- intraspecific aggression - and a number of brain structures, and to speculate about the functional significance of these neural circuits.**

Although Adams's paper raises many questions, I want to comment mainly on the concept of motivational systems and the way the author has translated this into defense, submission, and offense. We have to be as clear as possible about what we mean by such concepts, when these are used to elucidate brain. behaviour relationships.

Although not stated explicitly by the author, a motivational system comprises not only a set of specific stimuli and internal motivating and motor mechanisms, but also a specific overt behavioural output. Otherwise, Adams could not have made the distinction between three behavioural entities such as defense, submission, and offense. An overriding question is, then, what precisely the characteristics of such motivational systems are, and whether these can be distinguished from each other. Since Adams's motivational system is very similar to behavioural systems in ethology, it is worthwhile to mention briefly a present-day view on the characteristics of such behavioural systems (cf Baerends 1976).

Overt behaviour consists of stereotyped elements (movements, postures, etc.) that enable a quantification of that behaviour within a species These elements do not occur at random, and this can be demonstrated by different techniques (cf Colgan 1978: Hazlett 1977).

Associations of behavioural elements in time are often similar for many conspecifics under comparable conditions and are called behavioural systems Such systems have often been represented as hierarchical models in which a number of behavioural elements show some common causal factor. Although there is a regular warning against a unitary interpretation of this common factor, it has often been interpreted as a recognizable internal unity such as aggression, flight, or hunger. Although it is not quite clear what Adams means by homogeneous neurons underlying defense, submission, or offense, he strongly suggests that these neurons partially represent the internal hierarchy that ethologists have been looking for. Such an idea is strengthened by the hierarchical models that Adams presented in a recent paper (Lehman and Adams 1977).

However, although hierarchical models are helpful in understanding and describing the organisation of overt behaviour, they may facilitate sham explanations, First, most if not all behavioural observations used to define behavioural systems have been made in a limited set of well circumscribed situations. The observations cited by Adams are no exception to this rule - for instance, the behaviour of a dominant male meeting an unknown intruder male in its home cage, or the behaviour of two males next to each other while receiving unexpected pain shocks. At least after some experience all animals react to such a situation in a more or less stereotyped manner; say offense or defense.

However, such a behavioural stereotype does not mean that system A (e.g. offense) and system B (e.g. defense) must also have their own specific underlying internal mechanisms. Such a conclusion would be tenable only if it could be demonstrated that under varying conditions the animal uses either A or B but never mixtures of both. If, however, the latter would be found, then a more likely model is that under different conditions an animal may use different behavioural patterns, composed out of elements of one basic system. The separate behavioural patterns are then the average responses of an animal adapted to a

specific situation. If such reasoning is correct, one would expect different patterns among individuals dealing with a similar behavioural situation, or that elements of, say, defense, submission, and offense can be intermingled in different ways by conspecifics in different situations. The last expectation is supported by the fact that lactating females may show such a mixture of offense and defense.

Therefore, before we can categorize aggressive behaviour of a species into three motivational systems, suggesting the existence of three hierarchies (defense, submission, and offense), we have to investigate how robust these systems are under varying and more complex conditions than hitherto investigated.

A second drawback of hierarchical models is that they do not specify whether or not the behavioural elements belonging to a particular system show specific sequences or patterns in time. The statistical procedures used all emphasize the presence of a common factor and are less explicit about a specific patterning of behaviour in time and space. This may lead to a loose handling of such systems. For instance, if it is stated that as a result of a given brain lesion defense does not disappear, one is inclined to conclude - as Adams does - that for the occurrence of defense that particular brain structure is not necessary. The real and often unanswered question, however, is whether the entire original pattern of the defense response is still intact (normal latencies, durations, frequencies, sequences, goal directedness). Only very sensitive behavioral measurements can inform us about possible changes in behaviour in the limited number of behavioural situations used.

I really doubt, for instance, that forebrain structures fail to form an essential part of a defense motivating mechanism (as Adams suggests). The answer seems to be given when, in simple experimental conditions, no changes are recorded. What really has to be investigated is how such lesioned animals behave in much more complex situations; for instance, in natural colony conditions.

In my opinion the existing literature is either far too imprecise to support, say, a yes/no relationship between certain brain structures and defense or submission, or else the definitions of the latter two concepts are too vague to be useful.

In this context it is somewhat surprising that the author does not refer to newer approaches to the problem of the organisational structure of behavioural systems. These approaches emphasize the specific characteristics of behavioural programs. The point is that behavioural systems like feeding. drinking. sexual behaviour, different forms of aggressive behaviour, and so on, are all conceived as regulatory systems (Archer 1976: Baerends 1976; Toates and Archer 1978; Wiepkerna 1978). All these systems are behavioural programs directed at the homeostasis of specific aspects of the internal and/or external environment.

To realize this, each organism has at its disposal a limited set of behavioural elements that can to a certain extent be arranged and rearranged according to individual experience with a particular situation. Such an approach is attractive not only in that It assumes a similar basic organisational structure of all gross behavioural systems, but also because it

accentuates the need for precision in describing time and space patterns of the behavioural programs involved.

In order to know what is regulated, one has to know the differences between and within individuals in terms of aggressive behavioural programs. This brings us to one of the most intriguing fields of present behavioural research; the question of the flexibility of behavioural programs or systems (cf. Hinde and Stevenson-Hinde 1973). At the moment we hardly know anything about the flexibility of "systems" like defense, submission, or offense.

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Neural circuitry for motivational systems. **Specifying the sets of neurons and interconnections that integrate motivated behaviors furnishes a useful framework for further considering the neuropsychological basis of aggression. This commentary will discuss the defense, submission, and offense circuits of the septum and amygdala and their connections, and particularly the effects of localized lesions within these systems.**

Defense. **The exclusion of the septal area from the diagram for the neural circuitry for defense is surprising, in view of the well known hyperdefensiveness of septal rats toward their human experimenters. Perhaps the exaggerated form of the reaction or the lack of data on the motivating stimuli for the septal syndrome precludes inclusion of these behaviors as part of the animal's "natural" behavioral repertoire. The following experiment gives food for thought concerning septal hyperreactivity and the stimulus properties that evoke it.**

Max, Cohen, and Lieblich (1974) noted the reactions of septal rats when approached from above or below by the gloved hands of the experimenter. In rats approached from below, resistance to capture, vocalization, and biting occurred much less frequently than in those approached from above. These authors interpreted approach from above as representing the sort of stimulation that might come from a dangerous predator, or, in Adams's terminology, dorsal tactile stimulation capable of activating the defensive motivational system in muroid rodents. According to this interpretation, the septum might be viewed as a sensory link between peripheral sensory stimulation and the midbrain defense motivational mechanism.

A somewhat more complicated role for the septum may be inferred from its relation with the amygdala. King and Meyer (1958) discovered a reciprocity between the two areas, in that septal hyperreactivity was totally abolished by a subsequent lesion of the amygdala. Perhaps this experiment should now be repeated, but in a more elegant variant made possible by the proposed neural circuitry for defense. The replication and extension would involve making lesions in the ventromedial amygdala (defense zone), or in the perifornical hypothalamus, in hyperreactive septal. rats, to determine whether interruption of the pathways facilitating defense would block !he septal syndrome. Several cautions are indicated in an experiment of this sort. First the amygdalo-hypothalamic defense pathway is apparently inferred from data in cats, not rats. Second, while the amgydala defense/zone may be identified using electrophysioiogical criteria, later research (Ursin 1965) involving lesions of the defense zone in cats did not result in reduction of defensive behavior. Finally, lesions of the perifornical hypothamus that abolished defense reactions from the amygdala stimulation in one experiment (Hilton and Zbrozyna 1963) appeared to encroach upon the lateral hypothalamic-medial forebrain bundle area. The latter area, when damaged in rats, has been found to severely limit the expression of all forms of behavior (LHA syndrome).

Defense and submission. **The nature of the influence upon consociate modulation of the ventromedial hypothalamus may also be considered in connection with septal and amygdaloid functions. Jonason and Enloe (1971) found that pairs of septal rats, following an initial period of fighting, spend significantly more time on socially cohesive behavior in an open field than did normal control pairs. Amygdaloid-amygdaloid pairs averaged about one-half the contact time of normals. Thus it appears that septal rats display defense reactions to conspecifics in this situation but then settle down to a more docile contact-seeking form of behavior, whereas rats with amygdaloid lesions show no defense and much less contact-seeking behavior.**

Does contact-seeking between rat pairs in an open field represent submissive behavior? To this commentator's knowledge, a detailed analysis of posturings between pairs of septal or amygdaloid rats has not been published. One might assume that obvious submissive encounters are not likely to be seen as consequences of attacks, since very few attacks indeed occur in this situation. Meyer, Ruth, arid Lavond (1978) have presented an interesting analysis of contact-seeking behavior that may be relevant to the present discussion. Recall from Adams's model that familiarity with the individual is a prerequisite tor consociate submissive behavior. Meyer, Ruth, and Lavond maintain that "because septal rats are likely to fight when they are first put into the open field, [they] require substantial periods of time to establish their social contacts" (p. 1028). Furthermore, based on the data with septal rat pairs and with septal rats, choices among other rats, furry rabbits, or cats, or nothing, Meyer, Ruth, and Lavond have concluded that social cohesiveness in the septal rat may represent the release of a contact-comfort motive akin to that postulated by the Harlows for infant monkeys [see Rajecki et al "Toward a General Theory of Infantile Attachment" BBS 1(3) 1978]. To return once again to the combined-lesion paradigm, consociate behaviors of septal rats should be abolished by a subsequent lesion to the ventromedial hypothalamus (consociate modulator). The specific effect should either be a failure to give up defensive fighting or flight (septal-lesion effect) or a tendency to engage in less contact-seeking behavior (amygdaloid-lesion effect).

Offense. **Paradoxically, septal rats appear to be neither offensively aggressive nor contact-seeking in reaction to a strange intruder in a colony situation (Blanchard et al 1977b). Whether hippocampal lesions would produce a similar effect, due to the loss of spatial recognition, as Adams suggests, is a question awaiting investigation. Surgical interruption of hippocampal-septal connections by fornix lesions prevents the occurrence of hyperdefensiveness to handling when septal lesions are subsequently made in the same animal (Olton and Gage 1974). Whether fornix lesions would also block hypo-offensiveness in septal rats would seem a paradox on a paradox and is not reasonable to expect if spatial appreciation of territory is an essential element of the offense motivational system.**

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Author's Response:
Introduction and Motivational systems

My remarks will address three types of fundamental issues that are raised by the commentaries: (1) the general conceptual framework and terminology of motivational systems as I have defined them; (2) other levels of analysis that are alternatives to the neuroethological analysis employed here; and (3) specific questions concerning the offense, defense, and submission systems.

On the most general level, I am gratified that some of the commentators, especially the Blanchards, Miczek, and Panksepp, seem to agree that a classification of behaviors ought to be based upon the neural circuitry involved. This has not been a common view in the past.

A "neural" classification of behaviors, such as I propose, does not necessarily correspond to traditional logical distinctions, as Miczek points out. Nor does it necessarily provide an exhaustive classification of behaviors that have been grouped together traditionally. For example, Brain, in the useful schema he provides in his commentary, includes categories of predatory aggression and reproduction termination that are not included in my classification. And, as Brain notes, submission is not a category of aggression, although it is related to aggressive behaviors. However, I am pleased to see that there is considerable correspondence between his "self-defensive" category and my "defense," his "social aggression" and my "offense," and his "maternal aggression" with what in my classification consists of both offense and defense. I am also pleased to see considerable correspondence between my categories and those of Ursin. Ursin distinguishes fear, defense, attack, and prey-killing. These appear to correspond to submission, defense, offense, and predatory aggression in my terminology.

Conceptual framework and terminology of motivational systems. I have provided a new and, I hope, clearly defined terminology for a conceptual framework for the brain mechanisms of social behavior. These terms include "motivational system," "motivational mechanism," "motivating stimuli," releasing and directing stimuli," "motor patterning mechanism," and "motor pattern." The commentators are free, of course, to disagree with this choice of terminology, but it is also important that my own use of it should be represented accurately.

There is considerable confusion in the commentaries in the use of the terms "motivational mechanism" and "motivational system." I have arbitrarily defined a *motivational mechanism* as a hypothetical set of homogeneous neurons responsible for the motivational state of the animal (Lehman and Adams 1977) and I have in the present target article supported this definition with data on a hypothetical defense motivational mechanism. In the terms of the Baenninger commentary, it is a "neural center" with one function and one anatomical locus. In terms of Isaacson's question, such a mechanism is hypothesized to be homogeneous in all of his categories: anatomical structure, input, output relations, biochemical nature, and responsiveness to circulating neuromodulators. I have arbitrarily

defined a *motivational system*, on the other hand, as a more complex assemblage of neural mechanisms involving many anatomical structures, of which a motivational mechanism is only one component, albeit the defining component. In Figures 1, 2, and 4, the motivational mechanisms are shown as one box per figure, while each motivational system consists of the *entire* figure. Many commentators do not accept this distinction; instead, they often use the term "motivational mechanism" to represent all or part of what I have called the "motivational system." Thus, Decsi & Nagy say that a defense motivational mechanism is "not only there," meaning in the midbrain central gray. Delgado and Isaacson do not follow my definitions when they maintain that a defense motivational system is not confined to the central gray, and similarly, Karli cannot accept the idea that a motivational mechanism could be limited to the central gray. Koolhaas suggests that a motivational mechanism must be represented by many structures in the brain. Leyhausen asserts that motivational systems are far from being unitary or homogeneous; although he uses the word "system," he appears to be criticizing my concept of "motivational mechanism." Wiepkema also cannot accept the idea that forebrain structures fail to form an essential part of a defense motivational mechanism.

There are two sources for the confusion surrounding the use of the terms "motivational mechanism" and "motivational system," one factual and one semantic. The factual question can only be answered by future research. Are there homogeneous sets of neurons in a single anatomical locus responsible for the motivational state of the animal? I have hypothesized and given data to support the existence of such neurons. 'The semantic question is also important, however. If the existence of such neurons is confirmed; should they be called a motivational mechanism? Many of my commentators obviously wish to use this term for more complex neural assemblages. What term would they like to use for a homogeneous set of neurons? I am not dogmatically committed to "mechanism," but I cannot, at the moment, think of a better term.

What are the criteria for a motivational mechanism? And do I give a convincing example? These questions are raised explicitly by Koolhaas and by Miczek. Although I imply the criteria in the first three paragraphs of the section on defense, I am not sufficiently explicit. In fact, the criteria are best expressed by Ursin in his commentary, although I am not sure that stimulation must "jam" the stimulus control of a behavior. The Ursin criteria define a motivational mechanism if one restricts them further to insist that a lesion of the structure would *totally* and *irreversibly* abolish *all* of the behaviors of the motivational system, that is, all of its motor patterns except those that are "ambivalent" when they are activated by other motivational systems as well. Although Ursin does not acknowledge it, I think that these criteria are met for the midbrain central gray and immediately adjacent tegmentum as the motivational mechanism of defense: (1) the behaviors of defense may be described in ethological terms; (2) defense may be elicited by both electrical and chemical stimulation here; (3) units here change their activity during defense - in this case I grant Koolhaas's objection that the unit data on the rat are weak, but I invite him to consider my unit data from the cat, which are much stronger; (4) the behaviors of defense are all permanently abolished by total lesions here; (5) an animal will no longer learn a response to escape from

stimulation after lesions here; (6) the question of jamming is controversial as stated above; (7) pharmacological manipulations such as those involving morphine have their effects upon defense here; (8) there are complementary data from animals as widely removed phylogenetically as chickens, rats, cats, and monkeys; and (9) the behavioral effects of manipulations here, while not confined to defense, may be quite different for other behaviors, for example, facilitation of "appetitively motivated behaviors" as noted by Karli in his commentary. It should be noted that Ursin's criteria need not refer exclusively to a motivational mechanism. If one substitutes the term "motor pattern" for Ursin's "behavior," then the criteria would define a motor patterning mechanism. The criteria apply to a motivational mechanism only if the term "behavior" stands for *all* of the motor patterns of a particular motivational system, and if the behaviors are irreversibly abolished by a lesion. By these criteria, for example, the amygdala cannot contain a motivational mechanism for defense, because defense survives removal of the entire forebrain.

My assumption that a vertebrate neural mechanism would consist of a set of homogeneous neurons is criticized by Glantz from the standpoint of his work with invertebrate nervous systems. The question he raises is interesting, but I do not agree with his answer that neural systems must consist of circuits of small numbers of heterogeneous neurons. Glantz and many other invertebrate neurophysiologists bias their analysis by recording only from a few large neurons. For example, in the abdominal nerve cord of the crayfish studied by Glantz the giant escape command cells have the two largest axons, as shown in the figure from Krasne and Wine on page 276 in the Hoyle (1977) volume. What about the hundreds and perhaps thousands of smaller neurons in the figure that have not been categorized because they are too small for intracellular recording? Are they homogeneous or heterogeneous? It is true that invertebrate nervous systems have smaller numbers of neurons than those of vertebrate but Davis in the Fentress (1976b) volume still concedes that there are at least 10,000 neurons in the central nervous system of a snail and 100,000 in a crayfish. Most of these neurons are small and no easier to study than the neurons of a vertebrate. Vertebrates, in some cases, also have a few giant neurons like the Mauthner neuron of fish and the Muller cells of the lamprey. But, as in invertebrates, it is not appropriate to characterize their nervous systems by these few large neurons, Instead, these neurons are exceptions that have evolved to handle peculiar functions for which large size is useful. There is only one complex animal that I know in which all neurons are large, and that is the vertebrate *Necturus*. (This does argue, by the way, that there should be more intensive study of the behavior and nervous system of this organism.)

There are several arguments suggesting, but not demonstrating, that vertebrate neuronal aggregates consist of pools of homogeneous neurons. The best studied neural system, the vertebrate retina of *Necturus*, does consist of populations of homogeneous neurons of only five (or a few more) classes of neurons, each class homogeneous in terms of its anatomical location, inputs, outputs, and types of synapses. However, the retina, one may argue, is a sensory system and should not be taken to represent an integrative system such as that described here. Another simple consideration argues for a great degree of homogeneity in vertebrate neural populations, no matter what system; the absolute size of the vertebrate

nervous system, and its total number of neurons, are determined less by function than by size and metabolism of the species (Blumenschine, Mink, and Adams 1978). For example, a rat brain has about ten times as many neurons as a mouse brain, yet surely no one would argue that a rat brain has ten times as many classes of neurons; the difference must be in the degree of redundancy in homogeneous pools of neurons. Finally, there is a point of view, to which I adhere, that the actual genetic instructions for neurons are both few in kind and quite limited in number. Strumwasser (1967) has suggested that there may be as few as eight kinds of information encoded in a neuron, and data from my own work in behavior genetics suggest that the genetic factors underlying differences in behavior among strains of rats may be quite small in number (Adams 1978).

The distinction between motivating and releasing/directing stimuli is central to the motivational systems analysis presented here. Yet, despite the fact that I drew the distinction between motivating and releasing/directing stimuli from Tinbergen (1951, pp. 122-23), Leyhausen, who quotes Tinbergen in his commentary, says that he "cannot agree with the distinction between the motivating and releasing function of stimuli." Waldbillig also questions my proposal that many motor patterns do not occur until their patterning mechanisms receive simultaneous motivating and releasing/directing inputs. Waldbillig suggests that there is an alternative model consisting of what he calls "response chains," but he does not describe this model in detail in his commentary or in the papers to which he refers. The example he gives, the abolition of biting and the retention of approach following midbrain lesions in the rat, is similar to the example of selective abolition of hissing in cats with midbrain lesions that I have cited as an instance of destruction of a pathway from a motivational mechanism to one particular motor patterning mechanism. (See the fourth paragraph in the section on motor patterning mechanisms for defense in the target article.)

My proposal that motivational mechanisms are usually activated by motivating stimuli does not rule out the possibility of activation by "internal" stimuli as well. Laborit, Leyhausen, and, by implication, Ursin consider my model to depend too much upon the former and to ignore the latter. It is true that I have emphasized the role of motivating stimuli in offense, defense, and submission, but I think that this reflects the fact that intraspecific aggression is usually confined to specific and transitory environmental situations. There are at least two exceptions, however. One is aggressive play, which is not considered in this review. The other is the offense component of maternal aggression, which I have argued is activated by prolactin in the absence of motivating stimuli (but cf. Gandelman commentary for a contrary view). Other motivational systems may be more dependent upon internal stimuli. I have argued elsewhere (Adams, submitted for publication) that the activation of the motivational mechanism of exploration/marking can occur from hormonal stimulation alone, that is, in the absence of motivating stimuli. Therefore, in Leyhausen's terms, I do not consider myself to be "one of the last believers" in a rigid stimulus-response model of behavior.

Motor patterns, according to the analysis I present here, may be "ambivalent," that is, activated by more than one motivational mechanism. This can lead to some logical

confusion. If an animal flees or adopts an upright posture, the observer cannot determine from that alone whether its defense or submission motivational mechanism is activated. This explanation is in reply to the question of Miczek about the significance of flight and of Rodgers concerning the upright posture.

The proposal that there may be a "master switch," located perhaps in the paleocerebellum, that chooses among contradictory motor patterns, has been elaborated by Berntson. Although I would emphasize motor patterns rather than motivational mechanisms, I appreciate not only the discussion of his own original data, but also the clear analysis of the existing literature. It was in a personal discussion with him that I became sufficiently emboldened with the idea to commit it to print. Berntson's commentary is the best reply I can give to the disbelief expressed by Baenninger that there might be one locus in the brain with such a powerful function.

Author's Response Continued: Some alternative levels of analysis

The questions raised in my target article can be analyzed on many levels. I have chosen to use a neuroethological approach in this case. Other levels of analysis are also appropriate, however, as the commentators point out. These include a strictly ethological approach, an analysis of the role of learning, an analysis of the role of hormones, a pharmacological approach, and a regulatory systems approach.

Although I have used ethological concepts in the present paper, I have not presented much ethological data. Yet any complete description of behavioral systems must conform to the behavior of animals observed under natural conditions, as correctly emphasized by Koolhaas and Wiepkema. For this reason, I have recently reviewed the literature on aggressive behavior in naturalistic as well as laboratory settings for all species of muroid rodents (Adams, submitted for publication). The present neural analysis conforms to the results that derived from the comparative study of naturalistic behavior.

An ethological approach is important, among other reasons, for its insistence upon the accurate description of motor patterns of behavior. As the Blanchards, Waldbillig, and Rodgers point out, failure to provide such accurate descriptions has plagued much of the previous literature, especially in psychology and physiology. I propose that workers in the field of muroid rodent aggression should base their descriptions of behavior upon those of Grant and Mackintosh (1963) and those working with cats should use the descriptions of Leyhausen (1956, 1979a).

An analysis of the role of learning in aggressive behavior is also important, as many commentators point out. Andrew invokes learning to account for stability of behavior patterns; Baenninger suggests that aggressive behavior must be even more "plastic" than feeding; Isaacson asks regarding offense, defense, and submission, "Are they really hard-wired?"; Karli emphasizes learned changes in mouse-killing by rats; Koolhaas and Wiepkema emphasize the "flexibility" of behavior; Laborit believes that I do not give

sufficient emphasis to the role of memory; and Panksepp speaks of submission as a "habit" based on "contiguous reinforcement." But, contrary to the impression one might gain from this, the literature on learning effects upon aggression, at least in cats and rodents, is remarkably sparse. In the target article, I list the points in the motivational systems where 1 think there are ontogenetic changes as a function of experience that might be called learning. This brief list is considered in greater detail in another recent. review (Adams, in press). In fact, I would challenge anyone to be more specific than I have been here as to how learning affects aggression and where its effects take place in the nervous system. Learning does not take place in a vacuum or in a Skinnerian "black box"; rather, it represents changes in the functioning of neural circuitry. Only when we have some notion of the neural circuitry can we begin to pin down these effects specifically.

The neural circuitry of aggression is greatly affected by hormones, as well as by learning, as pointed out by Brain, Gandelman, Koolhaas, and Laborit. Hormonal effects are even more profound than learning, in my opinion, and are so complex (See Brain and Gandelman commentaries) that I have chosen to review them in another paper, submitted for publication, entitled "Hormone influences on motivational systems of social behavior in muroid rodents and their significance for reproductive states." As in the case of learning, I submit that these hormonal effects may be analyzed most effectively in terms of their action upon specific types of neurons of the circuitry outlined in the target article.

The pharmacology of aggression has a rich literature, but because I have no experimental experience with it, I neglected it in the target article. As noted by the Blanchards, Decsi & Nagy, Eichelman, Laborit, Miczek, and Senault, an analysis of motivational systems could be strengthened greatly by consideration of these data. As in the case of learning and hormonal effects, ideally one would analyze the pharmacology of aggression in terms of actions at specific synapses and sets of neurons of the circuitry outlined here. Senault doubts the usefulness of this approach, however, and points to apomorphine-induced fighting as an example. In my opinion, the apomorphine-induced fighting in rats that he mentions is simply defense-activated upright posture and boxing. The relevant neural circuitry, as well as the motor pattern, resembles that of defense, and it is quite fortuitous, that the involvement of the globus pallidus parallels the involvement of that structure in the penile displays of squirrel monkeys. As in defense, apomorphine-induced fighting is not increased by septal, ventromedial, or olfactory lesions. These lesions can shift the behavior of an animal from submission to defense, but if an animal is already showing defense, no further change should he expected. On the other hand, lesions of the lateral hypothalamus and amygdala may decrease defense, presumably by interrupting the forebrain defense pathway. The involvement of the putamen, globus pallidus, and substantia nigra in apomorphine-induced fighting is its only unique feature, at least among those listed by Senault.

Another important approach to motivational systems is to consider them as regulatory and to analyze the temporal sequencing of their motor patterns. Wiepkema mentions several recent reviews that emphasize such an approach. I have made some preliminary

observations relevant to the question of aggression as a regulatory system in a recent empirical paper (Lehman and Adams 1977). For example, we found that while most behavioral sequences can occur in either direction, that is, they are symmetrical, the bite-and-kick attack usually terminates an offense sequence, suggesting that it is a consummatory response. Unfortunately, there are insufficient data from any one mammalian species to make such an approach feasible at the present time, and before we can analyze regulatory systems across species, we must understand one better in a single species. Any such analysis also ought to account for the intriguing relation between aggressive behavior induced by brain stimulation and the phenomena of negative and positive rewarding characteristics of the stimulation, as discussed by Karli and Eichelman.

Author's Response Continued: Specific questions concerning offense, defense, and submission

Several commentators agree that the distinction between offense and defense is a legitimate one, and the Blanchards and Brain provide new observations that strengthen the basis for the distinction. The Blanchards note that offense and defense are quite distinct along a number of dimensions and that in a fight between a dominant rat and a strange intruder, there is virtually no overlap between the offense of the former and the defense of the latter. Brain adds new data to the effect that offensive and defensive attacks may be distinguished on the basis of bite targets in mice.

Other commentators continue to doubt that offense and defense may be clearly distinguished. Leyhausen, for whom I originally learned the distinction between offense and defense, seems to consider that 1 have overdrawn the distinction. Panksepp wants to consider aggressive behavior as the result of "nonspecific" systems in contrast to my "specific" systems. He is right that I consider that offense and defense may be activated simultaneously, as in the case of the lactating female. Yet, even so, the result is not a hybrid behavior, but an alternation of offense and defense motor patterns. Panksepp also suggests that one can see a combination of offense and defense in cats during brain stimulation, but I am doubtful of this. As Miczek points out, most of the experimental work on neural mechanisms of aggression in cats actually involves defense, and Flynn (1976) also points out that offense is not normally seen in response to brain stimulation in cats. In my own experience with brain stimulation in cats (Adams and Flynn 1966; Adams 1968; Adams, Bacelli, Mancia, and Zanchetti 1969), I have never seen behavior corresponding to the "Angriffsreaktion" described by Leyhausen, which I have translated as offense. With this in mind, I cannot agree with Decsi & Nagy that "offense" characterizes the behavior of cats in a desperate situation. Rather, I think they refer to what I am calling defense. Wiepkema also does not believe that offense and defense are separate systems, but considers that they may be "intermingled" in different situations. As I mention above, his example of offense and defense by a lactating female does not in my opinion demonstrate such intermingling.

The distinction between defense and submission is particularly important in the present analysis. Therefore, I will take some care to respond to objections by a number of

commentators, and especially by the Blanchards, who have contributed greatly to the literature on the behaviors of these systems.

The ultimate criterion for distinguishing defense and submission must be their neural circuitry, but since that is still in dispute, I must rest my claim on five types of less direct evidence. Because they are indirect they are open to the charges by Waldbillig and Brain that the definition is circular. No one of these types of evidence is indispensable. For example, contrary to Ursin's interpretation, I did not intend to base the distinction upon only one criterion such as the difference in behavior between wild and tame animals, and it is a misinterpretation when Fentress says that submission "apparently evolved in the lab." Rather, defense is observed more often in wild animals and submission more often in tame or laboratory animals. The distinction is based on five types of evidence: (1) differences between wild and tame animals; (2) an evolutionary argument on differential responding to conspecifics and to predators; (3) distinctions in the cat between affective defense (defense) and flight (submission); (4) an analysis of the function of forebrain structures in the emotional behavior of the rat; and (5) differential hormonal effects. Unfortunately, the hormonal evidence, which I think is most convincing of all, could be presented only briefly in the target article. As mentioned above, a more extensive treatment has been submitted for publication.

The Blanchards doubt that wild and laboratory rats differ in the relative strengths of defense and submission. They cite new unpublished evidence that wild and domestic rats have "consistent similarities" in defense and submission. My own unpublished observations are quite different; wild rats were much more likely than domestic rats to show a lunge-and-bite defense when attacked by another rat, and they were much less likely to show the full submissive posture. Ursin (1964) has reported that in response to provocation tame cats show only flight, which I interpret as submission, while wild cats show a lunge-and-bite attack and flight, which I interpret as defense. The situation in rats needs further clarification.

As to the evolutionary argument that submission inhibits conspecific but not predator attacks, the Blanchards doubt that ultrasound and full submissive posture inhibit conspecific bite-and-kick attack in the rat. They doubt the experimental evidence that ultrasound inhibits attack. In reply, I point to my own data (Lehman and Adams 1977) as well as those of others (Lore, Flanelly, and Farina 1976). At one point the Blanchards concede that ultrasound might inhibit conspecific attack; if so, would they expect it to inhibit the attack of a predator as well? As part of the same discussion, they raise what I consider to be a straw-man objection as to the way that the full submissive posture inhibits bite-and-kick attack by a conspecific. Their interpretation is no different from my own, as implied in the target article and as explicitly stated in an earlier paper (Adams 1976). In other, words, contrary to their statement, I also consider that the full submissive posture inhibits the bite-and-kick by denying the opponent access to the dorsal surface, which is a necessary releasing stimulus for the bite-and-kick attack. Returning to the original question, would they expect the same posture to inhibit the attack of a predator?

The traditional opinion that one can often distinguish between affective defense and flight as two separate but related behavioral systems in the cat is not questioned by the Blanchards, nor do they deny my proposal that this reflects the differences between defense and submission. They do question one of my quotations, however, from Baxter (1967). Baxter's results are open to several alternative explanations, as the author himself pointed out. On the one hand, the different behavioral effects of electrical and chemical stimulation may have taken place upon two separate neural systems near the site of chemical stimulation, as I have chosen to interpret the data. On the other hand, the chemical stimulation may have taken place at a remote site, which was a "hypothesis" considered by the author and raised by the Blanchards to the level of "almost certainly." In any case, Baxter himself concluded that his data support the case of different neural substrates for escape, on the one hand, and attack and threat, on the other. I relate these systems to submission and defense, respectively.

Finally, my analysis of the function of forebrain structures in the rat is questioned by the Blanchards, The crux of my analysis deals with the ventromedial hypothalamus as a structure that facilitates submission and inhibits defense. With this they do not quarrel, except to point out correctly that the evidence from Veening (1975) is very weak because the behavior is so poorly described. With regard to the septum, they cite new data recently published from their laboratory, which they consider to support a unitary concept of defense rather than a distinction between defense and submission (Blanchard, Blanchard, Lee, and Nakamura 1979). I am not convinced. Whereas the motor patterns characteristic of defense, such as lunge-and-bite, boxing, and jumping, were all greatly increased following septal lesions, the two motor patterns characteristic of submission did not increase in most cases and sometimes did not appear at all. Rates of full submissive posture were not mentioned, presumably because they remained very low. And rates of ultrasonic vocalization were paradoxical. On the first day of experiment 1 and the preshock tests of experiment 2 they were elevated, but on the other days of experiment 1 they were, if anything, decreased, and on the postshock tests of experiment 2 they were not reliably different from controls, despite continued elevations in rates of defense motor patterns. The appearance of ultrasound *at all* is inconsistent with the simple model that I present in the final figure, but as I point out in the last paragraph in the section on submission and show with dotted lines in Figure 3, there is evidence that septal lesions not only affect the hypothetical consociate modulator, but also disinhibit the forebrain afferent pathway for both defense and submission without involving the consociate modulator (see Albert commentary).

The role of the septum in aggressive behavior is very complex, as indicated in the target article. In muroid rodents it appears to facilitate the consociate modulator, which can explain why lesions release the lunge-and-bite attack. But, as noted above in discussing the Blanchard critique, it may also have a tonic inhibitory effect upon the forebrain defense pathway, so that lesions may also disinhibit inputs for both submission and defense and stimulation may directly suppress defense. Furthermore, septal lesions may affect offense behaviors. Adding to the complications, the effects of septal lesions on defense are

transitory and may not be observed at all in some species such as the mouse, opossum, and primates. Although septal lesions occasionally enhance defense-in- the cat (see, e.g., Spiegel, Miller, and Oppenheimer 1940, quoted in the target article), they have also failed to enhance defense in the cat in other studies, such as the one that Ursin has performed, which he mentions in his commentary.

Part of the complication may be related to the fact that the septum is a complex structure and lesions may involve a number of different neural systems; thus, Albert, who has studied the matter in detail, notes in his commentary that the most effective site for lesions that enhance defense is not in the septum itself, but ventral to the lateral septum. Another aspect of the complication is the time course of lesion effects; thus Yutzey mentions the fact that after rats recover from the initial period of hyperdefensiveness caused by septal lesions, they show a curious behavior called "contact-seeking" in which they approach other animals, including predators, and seem to be in a state of abnormal "fearlessness" (Meyer, Ruth, and Lavond 1978). It would appear that following an initial period in which the function of the consociate modulator is depressed there follows a rebound period in which it is overactive and even predators are treated like consociates. Finally, Yutzey asks if I consider the septal syndrome (i.e., the initial hyperdefensiveness following septal lesions) to represent natural behavioral repertoires. The answer is yes, since I point out in the target article that "although the lesion-induced defense is excessive in comparison to that of laboratory rats and house cats, it may appear normal if compared to that of wild-trapped rats and feral cats."

The role of the cingulate and frontal cortex in aggressive behavior is much more complicated that I indicate in the target article. Although cingulate lesions have been reported to enhance defense in laboratory cats (Koridze and Oniani 1972), Ursin found no changes in defense following cingulate lesions in wild cats. Eichelman points out that cingulate lesions in the rat can decrease the frequency of shock-induced fighting, although in neither of the studies that he quotes were the effects statistically significant (Blanchard and Blanchard 1968; Eichelman 1971). Also, electrical stimulation of the cingulate cortex in the rat does not affect defense and submission in the same way as does septal stimulation (see Albert). A report that cingulate lesions in monkeys can decrease defense, which I quote in the target article (Glees, Cole, Whitty, and Cairns 1950) was not replicated by a later study (Pribram and Fulton 1954). The role of the frontal cortex in aggression is also more difficult to assess than I have indicated. Although the data from frontal lesions in the rat and monkey could be interpreted to mean that the frontal cortex normally inhibits the consociate modulator, Ursin points out that he and Divac showed several years ago that there were no effects of frontal cortex lesions upon the defensiveness of wild cats. About all that one can conclude at this time is that these structures are somehow involved in the defense and submission motivational systems.

My terminology for offense, defense, and submission is questioned, or, at least, not accepted by a number of commentators. The Blanchards and Ursin prefer the term "attack" as a synonym for "offense." I use the term "offense" partly in deference to the traditional use of

the term by Grant and Mackintosh (1963). In addition, I do not like the term "attack" in this context because it implies that defense cannot include attack and, as Eichelman points out in his commentary, defensive attacks may inflict severe physical damage. Panksepp would use the terms "rage" and "fear" rather than offense and defense. The term "rage" implies 1ack of control and extreme violence, whereas offense behavior, as l understand it, can occur in a wide range of intensities and can be quite well directed and controlled. The term "fear" does not imply action as much as does "defense." And since 1 think that the motivational systems are primarily programs for action, I think that the latter is a more appropriate term.

Semantic differences also arise in Waldbillig's commentary. He suggests that Baxter (1967) meant offense rather than defense when he said "attack." I disagree, since, as I mention above, I consider that practically all of the aggressive behavior reported from brain manipulations in cats is defense, not offense. Waldbillig also considers that I have been misled by the use of the term "fear" by Roberts (1962) when I call the behavior of cats during dorsomedial thalamic stimulation a form of submission. In the words of Roberts, "In the crouching response, the animal crouched 1ow; looked around its environment, and engaged in occasional locomotor activity which was maintained close to the floor at all times with absence of any climbing upward. If given the opportunity, the animal would run over to a dark corner or a box and crouch as if hiding." Roberts further notes that the crouching and lack of climbing upward distinguishes this response from the behavior induced by hypothalamic stimulation. The crouching and fleeing that Roberts reports, and the absence of striking or lunge-and-bite attack, fit generally with the hypothesis that these lesions enhance submission rather than defense. I have based .my analysis upon the motor patterns described in this case; not upon Roberts' use of the word "fear," which is what Waldbillig suggests. My model cannot, however, explain why following one type of stimulation the cat flees into dark corners and following the other it flees by climbing.

There is another behavior called "submission" that, in my opinion, is totally different from the one described here. It is characterized by Laborit as "the memory of the inescapability of punishment. ..or impossibility of coping with a situation." I do not think that it necessarily represents activation of the submission motivational system. Instead, I conceive of it as an artificial laboratory problem that arises when an animal is given motivating stimuli for submission and defense, such as pain, but is not given any releasing stimuli so that it can produce an appropriate motor pattern in response. It is not given releasing stimuli from a conspecific which would release defensive upright posture, lunge-and-bite attack, full submissive posture, and so on. And it is not given releasing stimuli for escape. Under such a circumstance, which would rarely arise under natural conditions, the animal is left to express the only motor patterns that do not require releasing stimuli, freezing and vocalization.

The consociate modulator is crucial to my distinction between defense and submission. Therefore, it is particularly important to clarify that just because I give the consociate modulator an anatomical locus, I do not make the inference that it is completely hard-

wired, as Miczek attributes to me. Instead, as I point out as the third item in the list of learning effects on aggression, the forebrain inputs to the consociate modulator are developed as a function of experiences that may be considered as a form of imprinting. The data supplied by Karli in his commentary support my contention that structures that project to the ventromedial hypothalamus should play a critical role in these imprinting effects. As Karli points out, lesions of the amygdala or stria terminalis interfere with the inhibition of attack upon a familiar consociate (in this case, a rat against a mouse with whom it has become familiar), and the effects of septal lesions depend upon whether or not such familiarity has developed prior to placement of the lesion.

Frustration or extinction-induced aggression is mentioned by several commentators (Eichelman, Andrew, and Panksepp) as if I had not discussed it. I intended to say that I consider these types of aggression as aspects of competitive fighting, which is a type of offense. I use the term "competitive fighting" in deference to its historical usage by Fredericson (1950), rather than using the term "frustration," which is rather anthropomorphic, or the term "extinction-induced aggression," which is restricted in application to artificial laboratory techniques.

Author's Response: Conclusion

The ultimate test of the present model is the extent to which it can serve to stimulate and guide future experiments that can further our understanding of the brain mechanisms of behavior. Having already seen a great deal of progress in the field since I began my research fifteen years ago, I am confident that we will be that much further ahead in another fifteen years. I am proud to have participated with my many distinguished commentators in this process. New data are mentioned in the commentaries that begin to force revision of, the model. Gandelman mentions evidence that prolactin is not the critical hormone for maternal aggression in mice. And the Blanchards have found that septal lesions, contrary to what I might have predicted, do not abolish ultrasound during shock-induced fighting. Berntson proposes new and intriguing experiments on the role of the paleocerebellum in the integration of behavior. Yutzey proposes new experiments to clarify the role of the septum and hippocampus in submission and defense. And Karli has undertaken a renewed attack upon the brain mechanisms of aggression at the single unit level.

I am particularly intrigued by the possibilities of extrapolating from the behavior of rats and cats to that of other vertebrates. But such extrapolation is hazardous as several commentators correctly observe. I appreciate the comments of Andrew that I have "effectively re-interpreted" his data on the chick, although the effect of midbrain lesions is much more complicated than I had indicated. From recent unpublished work on motivational systems in stumptail macaques, I agree with Delgado that in primates "the essential factors determining an animal's response (defense and submission) are hierarchical position and social context, not familiarity," However, I hope to show in a future publication that this difference between rodents and primates reflects a rather simple change in the nature of the inputs to the consociate modulator. And regarding

extrapolation to humans, I agree with Eichelman and Fraczek that human aggressive behavior is far more complex than that of other vertebrates. Human aggression has been transformed by many cultural factors such as the development of institutions and economic systems and the elaboration of motor patterns with tools and language. Knowing this, we have a moral obligation to avoid oversimplified phylogenetic extrapolations (which may be "particularly provocative" as noted by Brain), and we should make it clear that such human phenomena as crime, revolution, and war are not the inevitable results of neural circuitry.

References

References for this section are included in those listed on pages 31-43.