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Towards A Neurobiologically Based Unified Theory of Aggression

Vers un fondement neurobiologique de la théorie de l'agression : les circuits de la rage et de l'exploration sous-tendent des types de comportement agressif distincts.

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Abstract

Knowledge of the neurobiological mechanisms of aggression provides a foundation for a unified aggression theory. Neurobiological research in animals indicates that aggressive behavior can be divided into distinct subtypes which are aroused by different stimuli, serve different purposes, and have different behavioral manifestations. These distinctions, particularly between predatory aggression or SEEKING, and affective attack or RAGE, form the basis for a preliminary typology which accounts for the various categories of aggression that have been proposed. This typology must be refined by future research on the neurobiology of human aggression, particularly with regard to the difficult question of what underlies destructive behavior towards fellow humans.

Résumé

La connaissance des mécanismes neurobiologiques des comportements agressifs en permettent une vision théorique unifiée. La recherche neurobiologique chez l'animal indique que ces comportements peuvent être divisés en deux types distincts dont l'activation répond en effet à des stimuli différents. Ils sont aussi au service d'objectifs distincts et s'expriment de manière spécifique. Cette division, notamment celle entre aggression de prédation ou d'exploration et celle d'attaque affective dite de « rage », forme la base d'une typologie préliminaire intégrant les catégories variées d'agression qui ont été proposées jusqu'ici. À l'avenir, cette typologie devra être affinée grâce aux travaux d'orientation neurobiologique sur l'agression humaine, en particulier en référence à la question difficile des déterminants des comportements destructeurs à l'égard des congénères.

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Theorizing about aggression has led to a proliferation of categories and terms that need to be reconciled as we move towards a unified theory of aggression. A number of behaviors have been considered instances of aggression, including instinctual displays (such as hissing, baring of teeth and claws), fighting (wrestling, punching, pushing, etc.) and killing (as in predatory behaviors), as well as a range of human behaviors whose instinctual foundations are less clear, ranging from military actions to unwelcome sexual behaviors and everything in between, including arguing, yelling, sarcasm, teasing, and general assertiveness. The categories of aggression that have been proposed to account for these behaviors include impulsive, reactive, hostile, affective, explosive, irritable, defensive, controlled, proactive, instrumental, predatory, offensive, territorial, inter-male, and maternal. Which behaviors belong to which category? Which categories are valid? Are there any other behaviors or categories which should be included, or excluded?

Furthermore, an important consideration drives theorizing about aggression, as we seek to reduce the amount of destructive aggression which harms individuals, groups, or the cultural patrimony of the human race: to what extent is destructive aggression based on instinctual impulses that may be common to all humans? And if there are instinctual, genetic predispositions to destructive aggression, how might we successfully intervene?

We believe that a unified theory of aggression will move closer to developing universally accepted categories of aggression, and to answering the baffling and important question about the possible instinctual bases of aggression, when we are able to base theory on neurobiological evidence. Empirical evidence from animal studies already indicates that there are indeed distinct categories of aggression. Our aim in this chapter is to provide an overview of brain systems that mediate animal aggression in ways that may be useful for understanding human aggression. This evidence needs to be taken into account, and expanded upon through future research, as we segregate the aggressive tendencies that come down to us through evolution from an ancestral past, and get a clearer picture of how aggressive behavior is shaped in instrumental ways as we deal with a complex world. Our goal here is to focus more on the former than the latter.

This chapter will give a brief overview of some current neurobiological data on aggression and suggest a typology of human aggression based on that data, which is open to revision by further research. We need a generally accepted typology because research results have often been confused when distinct types of aggression are treated as one overall category. This paper focuses largely on two biologically disposed types of aggression -- predatory aggression and defensive/offensive rage -- which can be clearly dissociated in animal neurobiological research but have not been delineated with equal clarity as distinct motivations in humans. Although some researchers do make such distinctions (for example, Conner *et al.* [2002] and Weinshenker & Siegel [2002]), as long as distinct evolutionary sources of aggressive tendencies are not recognized by all researchers, long-standing questions about the nature and origin of aggression in humans, and the factors which influence it, will remain ambiguous. We acknowledge that recognizing the clear distinction between predatory aggression and defensive/offensive rage in other animals is just a first step towards developing a complete typology of human aggression, but it is an important first step in delineating the landscape to be explored, nonetheless.

It is important to note that basing a theory of aggression on neuroscientific findings does not automatically imply anything definitive about particular humans, genetically or otherwise. One concern about genetic research on aggression "is that, to the extent that it locates the springs of violence within the individual, it is an attempt to direct attention away from such social factors" as racism and poverty (Longino, 2001; p. 687). We do not agree that this is the case – while the evidence indicates that our brains have genetically-based, ancestral emotional processes shared by all mammals which show considerable individual variability, at the same time these processes are filtered through unique cortico-cognitive processes and cultural factors that vary enormously among species and human groups. A true neurobiological understanding means that we recognize that genes and environment are in complex, interdynamic relationships. On the one hand, genetic characteristics which have been linked to aggression can be highly influenced (suppressed or augmented) by environmental factors (Suomi, 2000). On the other hand, various

environmental influences can cause a person to react to a situation more aggressively than another person who did not experience those influences, regardless of their genetic predispositions, such as negative early childhood experiences which cause abused children to attend more selectively to threatening stimuli (Harvey, Fletcher & French, 2001), or social and emotional learning which create unconscious associations that are then activated by automatic social cognitions which increase aggressiveness (Todorov & Bargh, 2002).

Such heightened aggression which results from environmental events could make it *look* like a person is simply "naturally" more aggressive, but clearly an accurate explanation of an individual's aggressive behavior *must* include the many non-genetic factors that coax different individuals to navigate their social worlds in different ways. In fact, the more we understand the neurobiological circuits which underlie aggressive behavior, the more we will be able to describe how individual experiences interact with the more intrinsic functions of the brain. It may also bring us closer to understanding the internal affective dynamics that often fuels aggressive behavior. Ultimately this kind of comprehensive approach can only support the work against problems like racism and poverty. Only through a complete understanding can we imagine constructing safer societies that equally respect the cultural and the neurobiological sources of destructive aggression, and thereby have the means to address all relevant issues that deserve our attention.

Definition of Aggression

Let us start with some traditional thoughts about aggressions. Many researchers abide by the definition of aggression such as that advanced by Moyer (1976): *behavior which causes (or leads to) harm, damage or destruction of another organism*. A more expansive definition found in the Merriam-Webster Dictionary (online, 2003) is: "1: a forceful action or procedure (as an unprovoked attack) especially when intended to dominate or master; 2: the practice of making attacks or encroachments; especially: unprovoked violation by one country of the territorial integrity of another; 3: hostile, injurious, or destructive behavior or outlook

especially when caused by frustration." Notably, the derivation is from the Latin *aggressio* to attack, which comes from *ad-* + *gradi*, "to step, go."

Although these traditional definitions focus on physical harm and violence, we believe that a comprehensive definition of aggression should also encompass more subtle manifestations of effortful, goal-directed behaviors, including proactive aggression ("assertiveness") and competitiveness, as well as predatory aggression and defensive/offensive aggression. Some of these necessarily or indirectly lead to the physical harm or damage of things or people -- but some do only psychological harm, while others may involve no harm at all, as is the case with assertive actions to get one's needs met. However, we do believe that all of these behaviors still deserve to be grouped under one umbrella because of what they have in common: *effortful action*.

Neurobiological Circuits of Aggression

As mentioned earlier, two brain circuits have been identified in animals whose stimulation consistently yields aggressive behavior: predatory aggression (also called "quiet-biting attack") and affective attack (also called "defensive rage") (Panksepp, 1998; Weinshenker & Siegel, 2002). There is also some evidence for a distinct circuit that mediates inter-male aggression. The findings on these circuits will be summarized briefly.

Affective attack or defensive rage has, in recent years, been most thoroughly investigated by Allan Siegel and colleagues (for review, see Siegel, Roeling, Gregg & Kruk, 1999). In the cat, these investigators have identified key brain regions which are critical for the expression of defensive rage behavior, which is "characterized by marked sympathetic signs, retraction of the ears, baring of the teeth, growling, hissing, piloerection, arching of the back, unsheathing of the claws, salivation, and an explosive attack" (Bhatt, Gregg & Siegel, 2003). Siegel makes an important distinction between lower brain areas that *mediate* instinctual rage behaviors and higher brain areas that *modulate* such responses (Siegel, Roeling, Gregg & Kruk, 1999) -- those that mediate are responsible for whether the behavior is initiated and the basic pattern of activation; those that modulate will affect the

environmental and internal conditions under which the behaviors take place, the intensity of the behaviors, their durations, and other factors.

This brain operating system is simply labeled the RAGE system by Panksepp (1982, 1998), and we think it may at times be misleading to add the modifier "defensive" to the rage response since the system can operate in many contexts to promote active anger processes. Hence the RAGE system probably also operates in a more purely "offensive" fashion, and we will here typically refer to the outputs of this system as defensive/offensive aggression or affective attack. Aggression which may appear simply to be "defensive" from the outside might actually feel more assertive and "offensive" from the subjective emotional perspective of the angry organism.

This affective attack or RAGE response is mediated by neurons in the corticomedial amygdala, the rostro-caudal extent of the medial hypothalamus (MH) and the dorsolateral region of the midbrain periaqueductal gray (PAG); this is known because rage behavior can be reliably and consistently elicited through electrical stimulation of these regions. Many areas of the hypothalamus also modulate the behavior because of the capacity of this brain area to regulate the internal environment of the body and monitor various kinds of homeostatic imbalances, such as hunger, thirst, temperature, and sexual arousal. When stimulated, the medial hypothalamus projects information to the PAG. The PAG and surrounding tectal areas are the neural substrates for some of the essential sensory and motor representations of the core self, both visceral and somatic, and is centrally involved in pain perception.

The PAG projects to various brainstem nuclei, which mediate autonomic arousal such as increased blood pressure and respiration; the trigeminal nuclei that innervates the jaw and facial muscles; and a variety of others that mediate the bodily changes essential for coordinating instinctual action program (Bandler *et al*., 1988; Panksepp, 1998). The activation of specific medial hypothalamic and PAG circuits are currently envisioned as the necessary central triggers for the physiological and affective changes seen during enraged affective attack. In addition, the medial amygdala is an important input to the medial hypothala-

mus; this is where the primary sensory features of a situation are marked as "threatening" which then activates the core regions that give rise to affective attack. When there is abundant fear in a situation, the attack takes on a defensive character, which may, in part, reflect concurrent activation of the nearby FEAR system (Panksepp, 1998). The extended RAGE system also interacts with the prefrontal cortex, which plays an important inhibitory role in regulating many emotional behaviors, and other areas of the brain which feed into the amygdala, medial hypothalamus, and the PAG.

With regard to neurochemistries, the core of the RAGE system, as most other basic emotional systems, appears to be glutamatergic, with perhaps more cholinergic recruitment as the behavior gets more outwardly "defensive". Major inhibitory inputs to the system emerge from various brain opioid systems, GABA, and serotonin, and facilitation is provided by dopamine and norepinephrine (both of which can be deemed to reflect fairly generalized brain arousal effects). Among the neuropeptides Substance P is a key facilitator of the response (Siegel, *et al*., 1999). Cholecystokin administered into the PAG also facilitates rage, while reducing predatory aggression, highlighting the reciprocal interactions between these two types of aggression (Siegel *et al*., 1999; see below for further discussion).

In marked contrast to affective attack, predatory aggression (or "quiet-biting attack") is more methodical and appetitive. For instance, during arousal of this aggressive tendency, cats methodically stalk their prey, and pounce in a more focused way. Similar behaviors are evident in rats, but since rats are omnivores as opposed to carnivores, it is generally harder to evoke that behavior. We believe it is significant that in all species studied, predatory aggression is elicited by stimulating the lateral hypothalamus from sites where self-stimulation reward is typically evoked, and it is now well known that this brain system is confluent with the mesolimbic dopamine circuit which runs from the ventral tegmental area through the lateral hypothalamus to the nucleus accumbens and other forebrain zones. This overall circuit has been called the "reward system," the self-stimulation system, or the term that we prefer, the SEEKING system (see Panksepp, 1998 for an extensive discussion). This system sup-

ports expectancy, exploration, foraging and other appetitive activities aimed at meeting a large variety of bodily needs.

Because quiet-biting attack has been studied primarily in cats and self-stimulation studied in rats, the two behaviors have not been seen as sharing the same neurobiological substrate, but the evidence strongly supports the supposition that they are, in fact, highly overlapping systems. If this is true, it has great relevance for a unified theory of aggression, because if assertive, SEEKING behavior springs from the same source as predatory aggression in all mammals, we need to investigate further how that plays out in human beings. The evidence shows that quiet-biting attack and self-stimulation are both induced by stimulating the same lateral hypothalamic circuit. The behaviors induced are different: rats acquire self-stimulation much more readily than cats, and exhibit rapid, agitated behavior when they do, unlike cats; but this is most likely due to the fact that rats have a naturally vigorous foraging style, whereas food-acquisition in cats is more dependent on well-controlled hunting behaviors. Thus there are nuanced differences between these systems in different species, but the overall character of the resulting behavior is one of assertive goal-directedness, whether it be active foraging or methodical stalking. This similarity in the overall character of the behavior is likely to be based on their shared neurochemical controls (e.g., dopamine facilitation, albeit this neurochemical factor also can promote affective attack). Even though generally less is known about the neurochemistry of predatory aggression than affective attack (Panksepp, 1998; *Siegel et al*., 1999), one general prediction, well supported by existing data, is that drugs that increase self-stimulation in rats will tend to facilitate predatory aggression in cats, and vice versa for drugs that specifically reduce self-stimulation.

There is also preliminary evidence that indicates that a separate circuitry exists for the mediation of social competitive (typically inter-male), aggression, which probably interacts with both the RAGE and SEEKING systems in presently poorly specified ways. The arousability of the core circuits for this dominance type of aggression are controlled, in part, by testosterone receptors which run from the medial amygdala, through the preoptic, anterior hypothalamus area, and again down into the PAG. There may

be similar types of systems in females, albeit they are less well studied and may be less robust than the systems found in males. To some extent these circuits are distinct from the trajectory of the RAGE circuit, perhaps closely related to less well understood rough-and-tumble play systems, which may even promote competitive sports in human adults, but more work is needed to distinguish components. Another indication that inter-male aggression is a distinct neurobiological phenomenon is that some forms of damage to brain tissue, including the lateral septum, nucleus accumbens, medial hypothalamus, and raphe nuclei, intensify aggression towards experimenters and prey, but reduce fighting between males. In a thorough review of the topic, Albert, Walsh & Jonik (1993) have highlighted the importance of this system in animals but they question whether a comparable system influences human aggression. Aside from gonadal steroid modulation of this system in animal models, little is known about the key neurochemical controls of this system and it will not be discussed further.

Mapping Animal Findings onto Humans: Clarifying Categories of Aggression

Some might argue that human aggression is so complex that animal models are misleadingly simple or just irrelevant. Other researchers agree that animal models of aggression are necessary (Ramirez, 2000; Weinshenker & Seigel, 2002). As the senior author of this chapter has discussed (Panksepp, 1998), there are good reasons for using animal models for understanding human emotions and motivations. First, emotion and motivation emerge primarily from the subcortical, limbic circuits of the brain, which have substantially the same (i.e., evolutionarily homologous) structure, neurochemistry, and neurophysiology across all mammals. Whatever we learn about the neural nature of emotion in lower mammals is likely to be relevant to humans, although all findings must obviously be qualified since we have such a highly developed cortical/cognitive overlay that interacts with the older limbic circuits. Moreover, instinctual behaviors are easily measured in animals, and the underlying brain circuits can be manipulated with neurochemicals and electrical stimulation in

animals to a much greater degree than in humans (i.e., such manipulations are more ethically feasible and substantially more efficient and better controlled than research in humans).

Because at a basic neurobiological level aggressive urges are fundamentally ancient emotional and motivational phenomena shared by all mammals, animal investigations of the neurobiological substrates are bound to be relevant to humans. This type of knowledge may be especially pertinent for understanding the nature of inward aggressive feelings that may be expressed in and guide outward behaviors. Of course, in humans such inward feeling need not be outwardly shared with others since everything we do is filtered through higher cognitive activities, except in severe impulse-control psychopathologies (e.g., Pontius, 2002). By being able to project our psychological strategies forward and backward much more than other animals, through our great memory and foresight abilities, we can buffer our internal psychobehavioral urgesso that feelings are expressed symbolically. Still, it is likely that many of those higher cognitive actions are premised on the basis of how we envision that certain courses of action will make us feel in the long run. Our position is that those underlying affective projections are constructed from neuroemotional processes that lead to more impulsive behaviors in other animals.

The data on the neurobiology of aggression is already extensive, and yet much remains to be investigated. We have presented here only the briefest review of the basic neuroanatomy and neurochemistry, but much more is known about the details of the extended neural networks involved in each kind of behavior. Progress is also being made on understanding the interactions of various neurotransmitters, neuropeptides and hormones which mediate and modulate aggression, as well as the various internal states and external triggers. There is insufficient space to go into greater detail here, but all researchers on aggression would benefit from being familiar with the basic psychobiological literature (e.g., Panksepp, 1998; Siegel *et al*., 1999). Here we will simply elaborate further on one dimension that emerges rather clearly from the animal literature: the distinctions between two kinds of core operating systems that promote predatory and rage types of aggression. A unified theory of human aggression may find these

considerations useful. It is especially important that certain forms of aggression may have subjectively positive aspects, and this is most evident in the predatory aggression of animals.
Returning to the question proposed at the beginning: what do we make of all of the different kinds of behaviors and various categories that have been proposed for aggression? Based on the findings summarized above, there are three distinct categories which subsume most, if not all, of the categories suggested, as summarized in Table 1.

Category of Aggression	Types Included
SEEKING/assertive/predatory	controlled proactive instrumental predatory
Defensive/offensive RAGE; affective attack	irritable impulsive reactive hostile defensive explosive territorial maternal
Inter-male aggression	competitive offensive? territorial?

TABLE 1
A summary of three major types of aggression and the specific sub-types they may include.

Here we will focus on the first two categories, which so far have the clearer neurobiological findings. A recognition of these two types may be evident in the human psychological literature in various forms, such as the distinction between "proactive" and "reactive" aggression (Conner, 2002). There is also a long-standing debate within psychoanalysis about whether there is an aggressive drive that is "innately and primarily hostile and destructive toward the object" or whether "nonhostile striving in the service of mastery and adaptation [only takes on] hostile and destructive qualities in response to frustration and conflict" (Moore & Fine, 1995, p. 10; also see Akhtar, 1990, for extensive review). We believe such debates will be clarified by an understanding of the important differences in physiology, motivation, and subjectivity between the two types of aggression.

Two Distinct Types of Aggression

Predatory, assertive, or SEEKING aggression is mobilized in animals when they are wanting or needing something, and energizing activity is recruited by their brains to satisfy those desires or needs. While hunting is the evolutionarily and environmentally appropriate means for meeting needs in carnivorous species, it is also possible that this type of SEEKING behavior could be manifested in other species in ways that do not outwardly *appear* to be aggressive, but may still inwardly rely upon the same emotional power that leads to predatory intent. The expressions of this "psychic energy" can range from a rat exploring a maze for food, to a contractor enthusiastically building a house or a graduate student determinedly writing a dissertation. Although it could be argued that this type of "energy" does not deserve to be called aggressive, we contend that the main qualifier here should be the positive "assertiveness" of the behavior.

Assertive aggression can include walking into the boss's office to ask for a raise; making a proposal of marriage; redecorating one's living room; perhaps even working out the introduction to a paper in one's head, even though each of these activities can also be under the sway of less assertive motivations. Clearly in all these cases many other systems are involved in executing assertive/predatory aggression: motor programs, sensory perception, attention, memory, error detection, etc. But at the core of these behaviors is a dopamine-fueled interest, motivation, and energization to plan and execute the activity. And all of these activities are efforts directed towards the world, often quite forcefully, aimed at meeting one's needs and desires.

RAGE or affective attack, on the other hand, is a qualitatively different phenomenon. It is mobilized when an organism is frustrated, threatened or otherwise irritated, preparing it to remove an obstacle or subdue an attacker. Blood pressure rises, muscles are energized, attention is focused, and a special affective feeling is aroused that we commonly call anger. Interestingly, the behaviors in this category are more similar both within and across species than the diverse activities we group under assertive aggression – a rat, a cat, a monkey or a human jumping on or striking out at opponents resemble each other in many

ways, with species-typical differences depending largely on superficial anatomical issues or the unique emotional characteristics of different individuals. Affective rage is also involved in less archetypally violent forms, however, such as pounding on an uncooperative vending machine, letting out an exasperated sigh, or making a sarcastic comment. Our assumption is that the internally experienced motivation that we call anger is generated by the instinctual action apparatus that generates rage behaviors, but which can be inhibited, suppressed, or otherwise modulated in humans and other animals that have a sophisticated cortico-cognitive apparatus.

One central difference between these two forms of aggression is the level of autonomic arousal: it is comparatively low in predatory aggression, and very high in affective attack. This corresponds directly to the human finding that people prone to reactive aggression show increased autonomic arousal during aggressive acts, whereas those exhibiting proactive aggression do not have increased autonomic arousal (Conner, 2002). It is likely that the two types of aggression also have distinctive corresponding affective states. We can safely assume that predatory/assertive aggression is subjectively pleasurable, as the brain regions that elicit predatory aggression also elicit high levels of self-stimulation. In contrast, animals readily learn to turn off stimulation of RAGE sites (Panksepp, 1971), indicating that the experience is aversive. Furthermore, we believe that these subjective differences are inherent in the neurobiological nature of the circuits: SEEKING is aimed at resource acquisition, getting animals what they need, and it is reasonable that evolution has designed it so that this activation is positively valenced, whereas affective attack is designed more at resource maintenance, getting rid of something (e.g., threats and competitors) and hence would be accompanied more by aversive, or more ambiguously valenced, affective states. Presumably the state becomes more aversive (more defensive) as thwarting and counter-threats increase, so the overall affective valence of anger episodes is controlled by the recruitment of other emotions with the success or lack of success of angry behaviors.

Beyond the basic differences in affective valance, there may also be specific intentionality differences, which are very much related

to the aims of the behaviors in question. We presume that affective attack involves an emotional component of wanting to cause harm, mainly as a strategy of removing an obstacle, threat or challenger; causing pain or fear in another organism is an efficient means for making them withdraw. The evidence for this is the strong affective display component accompanying rage: in addition to the utilitarian physiological arousal which prepares the animal to fight, there are also supporting behavioral displays such as piloerection and teeth baring, which make animals appear more menacing to opponents. In contrast, an animal in the middle of stalking will try to remain undetected until the last possible moment. Contrary to affective attack, successful predatory aggression depends on not eliciting fear in the object (at least until it has no possibility of escaping). As such, we assume that the stalking cat does not "wish" to hurt the mouse, but merely to capture, toy with and consume it; although the mouse may feel pain when the cat pounces successfully, it does seem that nature recruits endogenous analgesics that may diminish such anguish when "the jig is up" (Kavaliers & Choleris, 2001).

Interestingly, although these circuits are substantially distinct and underlie different motivations and different behaviors, they are also interrelated in various ways. The most obvious are the overlapping neurochemical controls, such as the biogenic amines, which regulate all emotional behaviors, but there are also psychological interrelations. For instance, frustration is a major precipitant of anger; this emotion occurs when one does not receive an expected reward or is prevented from achieving a certain aim. The current model of frustration is that certain firing patterns of dopamine neurons in the SEEKING system normally indicate the availability (or expectation) of reward, and changes in those patterns indicate that reward has been received (Panksepp, 1998). When a reward is not received, altered firing will generate a representation of unfulfilled expectancies in the frontal cortex, where reward contingencies are computed (Rolls, 1999). This neural pattern of frustration would then be fed into the RAGE circuit; as such, it would be a separate cognitive input from the amygdala's processing of threatening and fearful stimuli.

This relation between the SEEKING and RAGE circuits may account for some of the challenges in studying aggression,

because RAGE can be triggered by either threat, frustration or other bodily irritations. In either case, the organism needs to be mobilized to confront the obstacle/irritant or the attacker. Therefore, without knowing the triggering agents, the affective attack aroused by frustration can be confused with affective attack provoked by threat. Likewise, the cascade of control from one aggressive system to another could be outwardly seamless. For instance, we might be able to visualize the continuum of SEEKING and RAGE as depicted in Table 2.

Assertiveness Proactive aggression SEEKING	**I like something and want to go after it** (but if I don't get it, I might start feeling that I need it)
–	**I need something and have to go after it** (but if I don't get it, I might start feeling very uncomfortable – hungry, lonely, frustrated)
	I'm irritated or uncomfortable with something and want to remove it (but if the source of irritation doesn't go away, it could start to feel threatening)
Anger Affective Attack RAGE	**I am being challenged/threatened/endangered**

TABLE 2
A Model: The cascade of assertive aggression into affective attack.

In addition, the circuits exert mutual inhibitory influences on each other. The medial hypothalamus (affective attack) sends GABA-mediated inhibitory projections to the lateral hypothalamus (SEEKING) and the lateral hypothalamus sends GABA-mediated inhibitory projections to the medial hypothalamus (Siegel et al, 1999). Hence arousal of one brain region tends to inhibit the other brain region. Therefore it is not really possible to be extremely angry and methodically goal-oriented at the same time. Indeed, "methodical" and "angry" are words which seem indisposed to exist in the same sentence, and certainly don't seem to co-exist in everyday experience. If one is able to recruit higher cerebral activities during emotional arousal, then affect tends to diminish as reflected in an emerging general finding in brain imaging of a reciprocal relationship between limbic and cortico-cognitive arousal (Liotti & Panksepp, 2004). As the saying goes, "I don't get mad; I get even." In other words, perhaps certain people (especially those with a sociopathic or alexithymic streak) are able to shift into a predatory aggressive or

SEEKING mode in response to irritations and frustrations, while more emotionally arousable people may tend to cope with them by remaining in the affective mode.

Aspirations to Integrate Animal Neuroscience with a Theory of Human Aggression

In the animals studied, aggressive behavior arises from at least two distinct neurobiological circuits which are aroused by distinct stimuli, and support distinct aims and behavioral/physiological attributes. Making these distinctions is a crucial first step towards a unified theory of aggression in humans, because we share the same basic neurobiological circuitry as the other mammalian species that have been studied. We therefore presume that the activity of the lateral hypothalamic SEEKING circuit and the medial hypothalamic RAGE circuit underlie distinct motivations that lead to aggressive behavior in humans, but this remains to be conclusively established.

Indeed, the point must be emphasized that we cannot apply conclusions from animal research unquestioningly to humans. What we have outlined here is a roadmap pointing towards future research, so that we can fully understand the neurobiology of distinct aggressions in humans. The most complicated question to be answered by this approach involves destructive behavior towards fellow humans. Some instances of human aggressions are undeniably impulsive and driven by affective attack, such as the murder of a spouse caught cheating (in fact, "crimes of passion" driven by affective aggression are even treated differently than pre-meditated aggression in many legal systems). In contrast, structured forms of assertive aggression (boxing, football) may reflect a mixture of predatory and inter-male. Finally, serial murders, as well as killing during warfare, seem to have some of the characteristics of predatory aggression. And yet we are not sure what drives this last form of violence in particular. Teasing out the similarities and differences among all of these forms of aggression should shed substantial light on the extent to which instinctual proclivities and/or environmental experiences induce or shape the expression of violence towards our fellows.

What does it say about our species that we have serial killers? What does it mean that we have planned social events where people purposely try to incapacitate each other? Could both ride upon a very similar primitive motivation, one an idiosyncratic and completely unsocialized form of aggression, and the other a fully socialized form, that only becomes troublesome when certain passions spill beyond the established constraints of rules and referees? Might both reflect an evolutionarily designed, "instinctive" impulse to prevail over others and sometimes to destroy them, an extension of predatory hunting or the primate urge for social dominance? We cannot yet be certain, but if we find specific chemistries (e.g., neuropeptides) that modulate each of these tendencies in humans, as they do in animals, we will be able to begin to judge the extent to which similar neurochemical "forces" control such seemingly distinct forms of human behavior, and how cognitive activities can bridge between their respective roles.

Moreover, even though we may in the future be able to dissociate predatory/SEEKING aggression from affective attack at the neurobiological level in humans, it also appears certain they can be mixed in various permutations, because our cortico-cognitive apparatus can reduce and regulate the impact of our limbic passions. As a trivial example, stalking an irritating fly with a flyswatter involves being irritated (RAGE circuitry) and methodical, predatory aggression (the SEEKING system). As a more relevant and disturbing example, a serial killer on the prowl, exhibiting predatory aggression, may use violence against a victim to express deep-seated rage against significant figures from childhood. One system may recruit the other: the irritation at the fly motivates controlled behavior aiming at getting rid of it, or the original RAGE-driven desire in a child to defend against an abusive adult during development may motivate the methodical pursuit of similar individuals later in life (the transference of being victimized to victimizing?). In either case, though, the mutually reciprocal inhibitions probably still apply at any given moment, with one circuit always predominating -- after all, you can't kill a fly if you're too enraged to wait for it to land anywhere. This type of emotional control may be due to other brain regions interacting with both aggression circuits, as part of the greater

complexity of the human brain and the higher degree of behavioral flexibility that we have.

However, the higher brain functions that allow us to be liberated from the immediacy of our limbic urges also allow us to become victims of obsessions that most other animals are not capable of, and probably create much more complicated temporal and subjective dynamics in human aggression. As we said earlier, we presume that the predatory animal has no particular wish to cause pain to its prey. Furthermore, under normal conditions in the animal world, an animal does not experience affective attack for extended periods; RAGE is terminated when opponents are beaten or have surrendered. But in distinct contrast, it appears that some human predators do wish to inflict pain or fear on their victims, and moreover that they nurture aggressive intentions over long periods of time, which would indicate some kind of extended, cognitively-mediated aggressive arousal. In this case, it may be that predatory aggression is working in the service of affective attack. Chronic activation of affective attack could lead to a desire to eliminate or injure the source of irritation or threat, and the prolonged RAGE arousal might recruit the predatory/SEEKING system. The outcome could then look like predatory aggression (an *instinctual* drive towards causing destructive harm) when the original cause was affective attack (an *environmentally-induced* responsiveness). Clarifying these questions on a neurobiological basis may contribute greatly to resolving such important question about the nature of human aggression.

Now that we have argued for a clear typology of aggression based on neurobiological substrates, let us share a few more examples of how neuroscientific data can provide a more robust foundation for a theory of aggression. Again, these are even more speculative that those in the previous section, focusing more on the inter-relations between some emerging neural details and depth-psychological aspects of human aggression. These closing thoughts are shared more as questions than as answers.

Knowing the full extent of the RAGE circuit, both its mediating and modulating brain regions and its neurochemistries -- the "machinery" of anger, as it were – will help us more fully understand the mental, emotional, and physiological dynamics of

human anger and the impulsive violence that often goes along with it: what triggers it, what influences how people regulate themselves, and what makes individuals different in terms of what makes them angry, how angry they get, what they do when they're angry, and their ability to control themselves. These influences are a complex interaction of genetically controlled dispositions and environmentally provided opportunities for action. In particular, further understanding in developmental neurobiology and developmental psychology will advance this area of aggression research, as we understand more about how early experiences with a child's significant love objects shape his or her ability to experience, express, and self-regulate strong emotions and impulses over the lifespan.

Major components of emotion and emotional regulation are highly influenced by early experiences (Panksepp, 2001; Suomi, 2000). For example, the neural infrastructure of the orbitofrontal cortex, an important area for emotion regulation, is especially affected by emotional transactions during a period of rapid growth in the first several years of life (Schore, 1994). Fear conditioning, based on experiences, is represented by changes in neuronal activity in the amygdala (LeDoux, 1998). And complex mental representations that are used to guide a person's behavior are developed within the emotionally charged attachment matrix between child and significant caregivers (Fonagy, 2001). In simplistic terms, then, what happens to you as a child shapes how you will get angry and what you will do about it as you mature.

In addition to giving us a framework to understand how experience can generate differences between individuals in how they manifest and regulate anger, neurobiological data also illuminates some of the essential features of anger that most of us are familiar with. For example, the core players in the RAGE circuit -- the medial hypothalamus and the PAG -- have mutual excitatory connections, meaning that the medial hypothalamus sends excitatory projections to the PAG, which initiate the somatic and autonomic changes associated with defensive rage, and the PAG also sends excitatory projections back to the medial hypothalamus. This "reverberating positive feedback circuit" (Bhatt, Gregg & Siegel, 2002) is most likely designed to prolong the expression

of defensive rage over a period of time (from seconds to minutes), which presumably keeps the organism in an aggressive stance as long as necessary, and certainly far beyond the initial stimulus which triggered the emotional response. In fact, these authors found that stimulation of the PAG facilitates defensive rage elicited from the medial hypothalamus. This means that when excitation is received from the PAG, the defensive rage is elicited more quickly from the medial hypothalamus, and may also be more intense.

This neurobiological dynamic underlies two things about anger that are widely experienced. First, this reverberating circuit may explain why anger is hard to turn off rapidly, and why people need time to "cool off" once they have been angered. Not only does the core RAGE circuitry initiate physiological changes in the body in response to environmental input, but these "generators" that are sending the signal for those changes remain in the "on" position for some extended period of time. A very similar line of reasoning could be applied to the cognitive aspects, where angry thoughts may have been encoded partly with the neurochemistries of anger, which would help sustain those cognitive activities as long as the anger facilitating chemistries remained active.

Secondly, because the PAG plays an important role in setting the "gain" of the defensive rage response, various factors which influence the PAG (and the rest of the RAGE circuitry) can affect whether someone has a hair-trigger to get angry, or maintains a laid-back demeanor well past the point where others would have lost their cool. Being hungry, tired, or already irritable from another cause can decrease the latency to anger, or increase the intensity of the response. Different variables, both environmental and genetic, can influence how likely someone is to get angry in a given moment, or how characteristic anger is as a general response. It is known that psychopaths, who are liable to exhibit aggression without remorse, exhibit diminished arousability of frontal lobe areas (Raine, 2002), but a critical and an unanswered question is whether such temperamental tendencies can be modified by early experiences. We know that in animals we can artificially induce irritability in aggression system that can last a long time (Adamec & Young, 2000), and such principles also

apply to humans (Pontius, 2002), but we do not know what can be done to diminish the impact of such kindled brain systems.

We also have much more to learn about the factors that influence the degree to which people have the capacity to inhibit their anger, which is based on the function of various areas in the prefrontal cortex and other brain structures. Clearly this is an important aspect of anger – as involuntary as the basic anger response may be, we still have an extensive capacity as a species to modulate it. In other words, anger does not necessarily need to lead to violence. We can restrain ourselves physically, use barbed or sarcastic language, or channel anger into constructive activities such as political protests or extended discussions with those with whom we are angry. In fact, our capacity to inhibit a violent response to possible danger may be one of the very foundations of the development of human civilization. The first humans lived in bands and tribes, groups which numbered only in the dozens or the hundreds, so that all members were familiar to each other and usually linked by blood or marriage. Indeed, it is well known that social bonding can diminish aggression, partly because bonding chemistries such as endogenous opioid are strong inhibitors of RAGE circuitry. However, with the rise of chiefdoms about 7,500 years ago, whose populations ranged from several thousand to tens of thousands, "people had to learn, for the first time in history, how to encounter strangers regularly without attempting to kill them" (Diamond, 1999, p. 273). A unified theory of aggression should ultimately be able to explain the factors influencing the inhibition or regulation of anger and defensive rage, as well as the environmental factors that can support people in developing more of these capacities.

Conclusion

The ancient animalian emotional forces which undergird the human spirit remain one of the great challenges for the mind sciences. The study of anger and aggression have a spotted history because such topics inflame long-standing cultural biases concerning the types of creatures that we are, part passionately animal and part rationally "angelic." Many would like to envision that our cultural overlay has completely transformed our minds

to ones where the animalian sources of our emotions are of secondary importance. That, we believe, is an incorrect and hence dangerous misrepresentation of the evidence. We are inheritors of brain systems that have a force in society that has to be recognized and acknowledged, and often it is. Thus, society commonly sees predatory aggression in humans directed at other humans as pathological, while the outcomes of brief instinctual arousals, as of anger, are more excusable. The legal system understands that there is some type of reciprocity between emotions and reason, and recent brain-imaging work is beginning to support that traditional vision of how our minds are organized (Liotti & Panksepp, 2004). Thus, a study of how our instinctual affective urges become integrated with cognitive strategies, both in the adaptive and maladaptive behaviors, remains one of the great challenges for the mind sciences (Panksepp, 2004).

In pursuing affective issues seriously, as evolved psychobiological processes, we must be wary of premature conclusions based on behavioral data. Let us just share one of many poignant examples. At one time cholecystokinin was thought to be a peripheral gastrically derived appetite control agent, but eventually it was found to mediate nausea, which will, of course, reduce one's desire to eat, but not in a normal way (i.e., not because one is satisfied). Injection of this hormone peripherally is now known to activate widespread nausea and stress systems of the brain (Billig, Yates & Rinaman, 2001), and anyone that administers this agent peripherally and finds a reduction in a behavior should be wary of interpreting it as a specific type of neurochemical control, while infusion of the same agent into the brain provides better evidence that it does participate in the genesis of rage (Siegel et al., 1999). In short, there is always room for better emotional education not only for the general public but also neuroscientists and other mind scientists as well. The nature of affect, in the present case the nature of the different aggressions, is the most important basic science issue that currently needs to be addressed to understand the ancestral sources of emotionally violent aggressive acts that are still so common in our highly cerebrated species. These are complicated questions that can only be clarified through interdisciplinary work that will require better integration among the behavioral, cognitive and affective neuro-

sciences along with developmental, social, and other depth psychological and psychiatric perspectives.

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