The Primate Amygdala and the Neurobiology of Social Behavior: Implications for Understanding Social Anxiety

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The amygdala has long been implicated in the mediation of emotional and social behaviors. Because there are very few human subjects with selective bilateral damage of the amygdala, much of the evidence for these functional associations has come from studies employing animal subjects. Macaque monkeys live in complex, highly organized social groups that are characterized by stable and hierarchical relationships among individuals who engage in complex forms of social communication, such as facial expressions. Understanding the role of the amygdala in animals that display a level of social sophistication approaching that of humans will help in understanding the amygdala's role in human social behavior and in psychopathology such as social anxiety. Selective bilateral lesions of the amygdala in mature macaque monkeys result in a lack of fear responses to inanimate objects and a "socially uninhibited" pattern of behavior. These results imply that the amygdala functions as a protective "brake" on engagement of objects or organisms while an evaluation of potential threat is carried out. They also suggest that social anxiety may be a dysregulation or hyperactivity of the amygdala's evaluative process. Finally, recent data from developmental studies raise the possibility that, at least at some developmental stages, fear in social contexts may be subserved by different brain regions than fear of inanimate objects. Biol Psychiatry 2002;51:11–17

Key Words: Monkey, lesion, social behavior, fear, inhibition

Historical Overview

The amygdala is a complex of nuclei that resides in the anterior temporal lobe of the human and nonhuman primate brains (Amaral et al 1992). In recent years, work carried out primarily in the rat has implicated the amygdala in the mediation of emotional behavior, particularly fear (Ledoux 1995); however, the amygdala has also been implicated in the organization of social behaviors in a number of mammalian species (rats: Jonason and Enloe 1971, cats: Schreiner and Kling 1953, dogs: Fuller et al 1957, monkeys: Kling 1972, and humans: Adolphs et al 1998).

Among primates, the social function of the amygdala may be particularly important. Several primate species live in highly organized social groups that are characterized by stable, hierarchical relationships among individuals who engage in dynamic patterns of social interaction and subtle forms of communication. Although typically thought of as a "phylogenetically primitive" brain region, the composition and size of nuclei in the primate amygdala have advanced relative to the rodent, and the primate amygdala has capitalized on connections from the expanded association neocortex (Amaral et al 1992; Barton and Aggleton, 2001; Crosby and Humphrey 1941, 1944; Stephan et al 1987). Presumably this is due, in part, to the demand for the greater information processing capacity needed to subserve the more sophisticated social interactions of primates. Understanding the role of the amygdala in animals that display a level of social sophistication approaching that of humans may be helpful in understanding the amygdala's role in human social behaviors and in such psychopathologies as social anxiety and social phobia.

The first study to explicitly investigate the role of the amygdala in nonhuman primate social behavior (Rosvold et al 1954) found that high-ranking and previously aggressive rhesus monkeys fell in the dominance hierarchy and became extremely submissive following bilateral amygdalectomy. Kling and colleagues (Dicks et al 1968; Kling and Brothers 1992; Kling and Steklis 1976; Kling et al 1970) studied free-ranging vervet and rhesus monkeys who were prepared with bilateral damage of the amygdala and anterior temporal lobe and released back into their natal social groups. These animals did not re-establish contact with other group members, did not engage in social interactions, and usually remained socially isolated. In most cases, the amygdala-lesioned monkeys were

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Received May 7, 2001; revised September 25, 2001; accepted September 27, 2001.

0006-3223/02/$22.00
PII S0006-3223(01)01307-5

attacked and either died from their wounds, from predation, or from malnutrition. In contrast, when caged, amygdalectomized stump-tailed macaques were observed in a social group, they generally displayed a decrease in aggression and a reduction in positive social behaviors, such as huddling and grooming (Kling and Cornell 1971). Although results from studies using nonhuman primates suggest that the amygdala is important for socioemotional functioning, they also show that the consequences of amygdala lesions may be dependent on the environment in which the animals' social interactions are recorded, the size of the social groups, the particular species under study, and in some cases the sex of the animal receiving the amygdala lesion (Kling 1972).

The conclusions from this early era of nonhuman primate studies of social behavior must be viewed with some caution because of technological issues. For example, until recently all lesions were made using either radio frequency or suction ablation techniques. These techniques suffer from the "fiber of passage" problem, because they not only remove or destroy cell bodies in the lesioned nucleus but also damage axons that do not originate or terminate in the targeted brain area. They are also not completely selective in their targets, because they often destroy neighboring brain regions. Many of the early lesion studies, for example, employed the suction ablation technique that damaged the surrounding perirhinal cortex en route to the amygdala. It is now clear that the perirhinal cortex plays important roles in visual processing and perhaps other cognitive functions (Buckley and Gaffan 1997; Erickson and Desimone 1999; Miyashita et al 1998). Consequently, one can ask whether the changes in social behavior arise from damage to the amygdala, the fibers of passage, or areas adjacent to the amygdala, such as the perirhinal and entorhinal cortices. This question is further complicated by the fact that histological analysis was often not carried out and certainly not carried out in a quantitative fashion.

Finally, earlier studies used behavioral data collection methods that were often more subjective than objective, more qualitative than quantitative, and that generated little actual data that could be analyzed statistically. Often the investigators did not use an established ethogram or catalogue of social behavior. There were no direct comparisons between lesion and control groups; subjects were usually chosen at random from an established social group, and their behavior was recorded before and after the placement of the lesions. The subjects used were often of mixed age and gender, thereby complicating the picture through lack of control over, for example, neuroendocrine differences among subjects due to reproductive status or gender.

For the last 5 years, we have been re-investigating the effects of bilateral amygdala lesions on social behavior in mature male rhesus monkeys (Emery et al 2001). Rather than suction ablation or other destructive lesion techniques, we have employed the selective neurotoxin ibotenic acid, which is injected stereotaxically into the brain, causing minimal damage to adjacent areas. This toxin has the advantage of destroying only cell bodies and leaving fibers of passage through the amygdala intact. In addition, stereotaxic placement of every lesion was accomplished using an individual magnetic resonance imaging (MRI) atlas, and extensive, quantitative histological analysis was performed for each lesion. Subjects for these studies were adult male rhesus monkeys who were assessed pre-operatively to determine social status in their natal groups. One group of experimental animals sustained amygdala lesions while another group acted as control animals. To maintain some commonality of social experience, a third group of "stimulus animals" served as partners for members of both experimental groups for two of the dyadic interaction experiments. We investigated the changes these lesions produced to the responses animals made to inanimate objects as well as to their behaviors in various social contexts; an established list of social and nonsocial behaviors was used (Capitanio et al 1998). A full report of the alternations in dyadic social interactions has been published (Emery et al 2001).

A Brief Summary of Amygdala Neuroanatomy

Before describing some of the changes that take place in social behavior following bilateral lesions of the amygdala in macaque monkeys, it is perhaps worthwhile to briefly reiterate some of the main features of the connectional organization of the nonhuman primate amygdala. A detailed description of the cytoarchitectonic organization, chemical neuroanatomy, and connections of the monkey amygdala can be found in Amaral et al (1992). The monkey amygdala has at least 13 distinct nuclei and cortical areas. Much of the neocortical interaction is with the laterally situated nuclei: the lateral, basal, and accessory basal nuclei. As illustrated in Figure 1, the primate amygdala has extensive connections, not only with the hypothalamus and brain stem, but also with the basal forebrain, striatum, hippocampal formation, and neocortex.

Interestingly, the amygdala has substantial connections with many regions of the neocortex. Figure 2 illustrates the pattern of connections with the ventral stream or "what" pathway of the visual system. Very substantial inputs from the inferior temporal regions of the anterior temporal lobe terminate in the dorsal portion of the lateral nucleus. Earlier levels in the hierarchy of visual processing do not

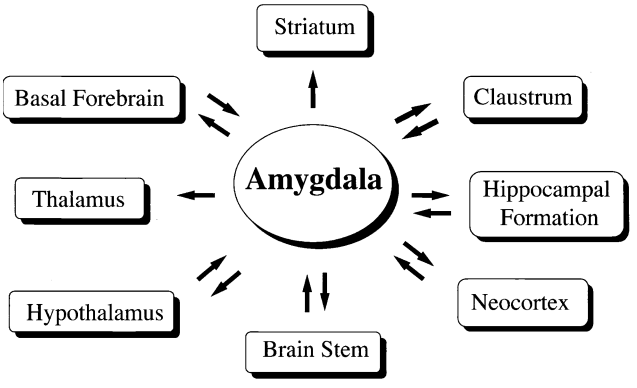

Figure 1. Block diagram that summarizes some of the main connections of the primate amygdaloid complex.

project to the amygdala. The lateral nucleus, in turn, projects onto the basal nucleus, which gives rise to "feedback" type connections, i.e., they terminate in layers I and II of the visual cortex. The initially surprising finding was that the return projections of the amygdala terminate not only in the anterior, higher-order visual areas, but also terminate throughout all ventral stream cortices, even including primary sensory cortex (Amaral and Price 1984). We have recently demonstrated (Amaral and Behniea, unpublished observations) that this return projection is not diffuse, because different populations of neurons in the basal nucleus project to different portions of the visual neocortex. Although the magnitude of the projections from other sensory modalities is lower, the amygdala is also

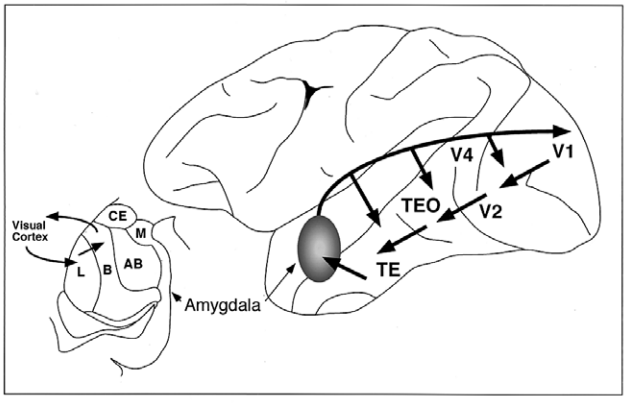

Figure 2. Diagram of the ventral stream "what" visual pathway that is involved in complex object recognition. The lateral nucleus of the amygdaloid complex receives input from the more rostral portions of the temporal visual cortex (area TE), but the basal nucleus gives rise to return projections to all levels of the temporal and occipital visual cortex. AB, Accessory basal nucleus of the amygdala; B, Basal nucleus of the amygdala; CI, central nucleus of the amygdala; M, Medial nucleus of the amygdala; TEO, Area TEO of the temporal lobe; V1, V2, V4, Visual areas.

privy to higher-order sensory information from auditory and somatosensory systems (Turner et al 1979).

The Function of the Amygdala—A Working Hypothesis

A working hypothesis of our laboratory, which has been supported by our recent lesion studies, is that the amygdala is a protection device; it is designed to detect and avoid danger. A primary function of the amygdala is to evaluate objects or organisms in the environment prior to interacting with them (or deciding not to interact with them). Based on the outcome of the evaluation, an appropriate species-typical response is coordinated by the amygdala. If, for example, a monkey were to encounter a predator, such as a snake, the amygdala would be involved in assessing the snake as a threat. Information for the assessment would presumably enter the amygdala via the temporal lobe visual system; however, a basic tenet of the hypothesis is that the emotional or social salience "the danger" of the stimulus is only appreciated once the amygdala is involved. Although the mechanism by which the amygdala assesses the threat of the stimulus is currently unknown, it could involve mechanisms ranging from pattern recognition based on innate templates of species-specific emotional elicitors (such as snakes) to interaction with trained networks that are perhaps associated with the primitive templates.

Once the assessment of salience is accomplished, one could imagine a whole series of actions initiated by the amygdala through its myriad connections. The projections to the neocortex, for example, could be involved in selective attention to the salient stimulus. Projections to the hippocampal formation could be involved in facilitating the encoding of the context in which the salient stimulus was encountered (Pare et al 1995); and the subcortical connections via the central nucleus to regions such as the dorsal motor nucleus of the vagus, the parabrachial nuclei, and the cervical sympathetic chain could mobilize appropriate visceral and autonomic responses to the stimulus. Although most of these ideas are speculative at this time, the scenario they portray is consistent with the results we have observed following bilateral lesions of the amygdala in macaque monkeys. We turn to a summary of those now.

Effects of Bilateral Lesions of the Amygdala in Macaque Monkeys

Mature, male monkeys with discrete ibotenic acid lesions of the amygdala demonstrate many of the same attributes of the Kluver-Bucy syndrome that results from much larger lesions of the temporal lobe (Emery et al 2001;

Kluver and Bucy 1938). For example, they demonstrate a distinct hyperorality. While exploring outdoor cages, they pick up any object that they can find and place it into their mouths. They also excessively mouth the chain link fence and any other objects that are within their grasp.

We have formally tested their responsiveness to inanimate items by bringing various objects of differing complexity to their cages in covered boxes (Mason et al, manuscript in preparation). A grape is placed in front of the object and the latency to take the grape is measured once the object is uncovered. The interactions with the objects, if any, are also recorded. We found that the normal animals took significantly longer to retrieve the grapes than the amygdala-lesioned animals, and there was an increasing latency as the objects became increasingly more complex. There were no significant differences in the latency to retrieve the grapes for the lesioned animals as the complexity of the objects increased. Even when the stimulus object was a realistic-looking rubber snake that the normal animals found quite aversive, the amygdala-lesioned animals readily took the grape and then began handling the snake. Thus, the major difference that was observed in this study was that the period of evaluation that normal macaque monkeys undertake when they are presented with a novel object was eliminated in the animals with amygdala lesions. Even normally very fear-inducing stimuli such as rubber snakes did not alter the approach behavior of the lesioned animals.

We also evaluated the amygdala-lesioned animals in a variety of social situations. These included various forms of dyadic interactions, as well as studies in which four animals (tetrads) were allowed to interact. A summary of the dyadic interactions is found in Emery et al (2001). It should be noted that at least some of the dyadic interactions were designed so that both the amygdala-lesioned monkeys as well as their age-, sex-, and dominance-matched normal control monkeys were observed in social interaction with other "common" or stimulus animals. These two male and two female macaque monkeys had the opportunity to interact with all 12 of our experimental animals and thus there was some commonality in the social experiences for all experimental animals. It should also be noted that an extensive ethogram of both affiliative and agonistic behaviors was used in these studies so as to detect either decreased or increased socioemotional behavior.

The alterations in social interactions observed in the amygdala-lesioned animals were consistent across all dyadic interactions. When normal macaque monkeys who are not familiar with each other are introduced into a common space, they take a considerable amount of time to evaluate each other (Figure 3). They maintain a cautious posture and typically do not come into close proximity. Much of their behavior is directed at preventing potential

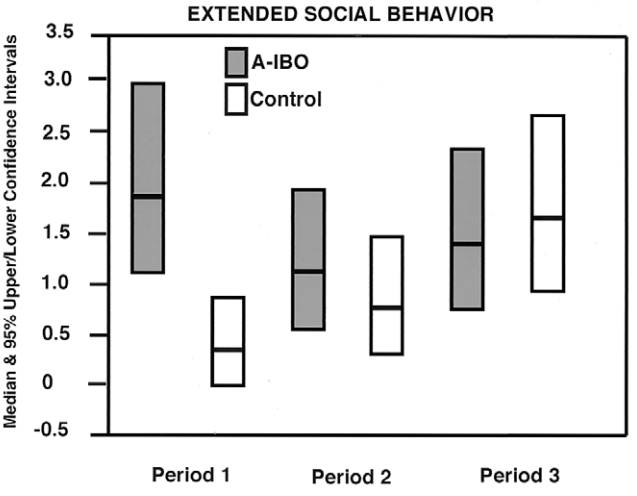

Figure 3. Illustration demonstrating the amount of "extended social behavior" (a group of affiliative social behaviors including grooming, sexual behavior, etc.) carried out by control and amygdala-lesioned (A-IBO) animals. In each of six sessions, every experimental animal (A-IBO and Control) was paired once with every stimulus animal for a 20-min test trial. Thus, every subject experienced a total of 24 trials. Period 1 presents data from the first eight trials, Period 2 from the next eight trials and Period 3 from the last eight trials. For each trial, the subject and stimulus animal were placed into their respective release cages, and on a signal the opaque doors were raised simultaneously, followed immediately by the metal grills. Once animals entered the test cage, the doors were lowered and latched. Behavioral data were collected using the Observer for the entire 20-min trial. The monkeys were then returned to their cages, and the release and test cages were cleaned and prepared for the next trial. Median (±95% confidence intervals) frequencies of behaviors for frequency of extended social behavior.

aggression, and there is relatively little social interaction during these initial encounters. As the social relationship is established, there tends to be an increase of positive social interaction, such as grooming and sexual behavior. This can be seen in Figure 3 where extended social behavior is at a low level during Period 1 (the first 8 interactions between experimental animals and stimulus animals) but progressively increases in Periods 2 and 3. The striking change in the amygdala-lesioned animals is that they do not go through the initial familiarization phase. They simply begin engaging in social activities as soon as they are placed with the novel stimulus animal.

Interestingly, the reciprocal behavior of the stimulus monkeys was also altered. One might imagine that the socially inappropriate behavior of the amygdala-lesioned monkeys would be upsetting to the normal stimulus animals who might attempt to escape from the premature sociality. However, just the opposite happened. The stimulus animals actually initiated more affiliative social behavior toward the lesioned animals than toward the

control animals, i.e., they found the amygdala-lesioned animals more "attractive" than the control animals.

The general conclusion from all of the dyadic encounters is that the amygdala-lesioned animals experience much less social stress and actively, and virtually immediately, engage in social interactions. Although there was little or no aggression in these controlled dyadic encounters, one expects that similar behavior might engender substantial retribution in more complex social situations with animals of higher dominance rank.

Lack of Social Stress Is Indicated by Analysis of Cortisol Levels

During the course of behavioral analyses, we were also in the position to evaluate the response of the hypothalamic–pituitary–adrenal (HPA) axis to various stressors (Ruys et al, unpublished data). In each of these experiments, baseline cortisol levels were compared with cortisol levels following either a social or physical stressor. As one example, the cortisol response to first interactions with novel animals was evaluated. Typically, the first meeting of novel animals is a highly stressful social interaction with substantial elevation of cortisol levels. The control animals in our study demonstrated a highly significant increase in cortisol levels following these social encounters. The amygdala-lesioned animals, in contrast, demonstrated only a modest and nonsignificant elevation of cortisol levels. The amygdala lesion, however, did not totally impair the ability of the HPA axis to respond to a stressor. We found that the response to a physical stressor, i.e., restraint in a primate chair, produced significant elevations in both the control and amygdala-lesioned animals.

These data are consistent with the conclusion that the amygdala-lesioned animals are not viewing the novel social interactions as a stressor. We believe this is the case because the amygdala is essential for making this evaluation. Without an amygdala, the animals simply do not appreciate the emotional salience of the situation, and their HPA axis is not activated.

The Effects of Neonatal Amygdala Lesions

One premise of the research program that we are undertaking is that the amygdala may be essential for gaining some facets of social knowledge but may not be the final repository of this information. By analogy with the hippocampal memory system, it is well known that the hippocampal formation and related medial temporal lobe structures are essential for the encoding of long-term episodic memories (Milner 1972); however, long-term storage appears to be in structures outside of the hippocampal formation.

If the amygdala is essential for gaining social knowledge, one would expect that early neonatal lesions of the amygdala would more seriously impair the social interactions of animals than lesions introduced into the mature animal. To investigate this, we have carried out a small number of experiments in which the amygdala is lesioned at 2 weeks of age in macaque monkeys (Prather et al, in press). These animals were returned to their mothers and were raised in a relatively normal fashion. We noted no striking alteration in the interactions between mother and infant. As with the mature lesioned animals, however, the infants appeared to be completely fearless of inanimate objects. They approached objects such as rubber snakes with absolutely no reluctance whatsoever. This was in stark contrast to age-matched control monkeys, who showed a robust fear of the rubber snakes.

The most unanticipated aspect of this study came when the weaned animals were allowed to have social dyadic interactions with novel animals. In this situation, the amygdala-lesioned animals demonstrated significantly more fearful behavior and engaged in significantly fewer social interactions. Thus, despite the fact that the amygdala was entirely absent in these animals, they were showing substantial social fear quite in contrast to the mature lesioned animals, who showed little or no social fear.

It is clearly too early to draw conclusions from these preliminary studies; however, it would appear that in the neonatal brain, regions other than the amygdala are capable of eliciting social fear reactions that are generally associated with amygdala function.

Conclusions

The overarching hypothesis governing, and thus far supported by, our program of studies is that the amygdala is a "protection device." Among its various functions, it appears to play a role in inhibiting an organism's approach to novel objects or other organisms. During the period of inhibited behavior, the amygdala participates in an evaluation of the environmental stimuli to ascertain whether there is something that is potentially dangerous. If so, the amygdala participates in the coordination of appropriate behaviors to avoid the danger. The "setpoint" of what is dangerous would certainly be governed both by innate predilections as well as learned associations.

Although it is clearly beyond the scope of this short review to summarize the increasingly larger body of literature on the role of the human amygdala, much of the available data are consistent with the hypothesis presented above. Phelps et al (2001), for example, have demon-

strated using functional MRI that the left amygdala is activated when a threatening stimulus is presented. Adolphs and colleagues (Adolphs et al 1999) have demonstrated that patients with bilateral damage of the amygdala are highly impaired in interpreting the facial expression of fear. These patients are also impaired in their ability to assess whether a person is trustworthy compared to normal control subjects (Adolphs et al 1998). Finally, Whalen and colleagues (Davis and Whalen 2001; Whalen et al 1998) have suggested that the amygdala is preferentially involved in the resolution of ambiguity. By their account, the amygdala is activated by a fearful face, because it is an ambiguous stimulus indicating that the face may be fearful of the observer or some other stimulus in the environment. In all of these hypotheses of amygdala function, it is portrayed as involved in an evaluative process particularly of situations that may be dangerous to the individual.

Given this scenario, it is relatively easy to imagine that social anxiety and social phobia might be related to hyperactivity or dysregulation of normal amygdala function. If the setpoint for determination that an object, individual, or situation is dangerous was set below what is normally beneficial to the individual, normally benign environmental stimuli might be judged dangerous and avoided. Although this hypothesis needs substantial additional experimental validation, it raises the prospect that selective pharmacologic manipulation of the amygdala might provide relief from social anxiety without many of the deleterious side effects of systemic antianxiety medications. A deeper understanding of the functions of the various amygdaloid nuclei, and of the particular molecular characteristics that identify neurons within these nuclei, may open the way to targeted therapies of anxiety-related disorders. The nonhuman primate is an ideal subject in which to pursue these highly valuable studies.

This work was supported, in part, by National Institute of Mental Health grants 41479 and 57502 and National Institutes of Health grant RR00169.

Aspects of this work were presented at the conference, "Social Anxiety: From Laboratory Studies to Clinical Practice," held March 22, 2001 in Atlanta, Georgia. The conference was supported by an unrestricted educational grant to the Anxiety Disorders Association of America (ADAA) from Wyeth-Ayerst Pharmaceuticals, and jointly sponsored by the ADAA, the ADAA Scientific Advisory Board, and the National Institute of Mental Health.

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