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The Effects of Bilateral Lesions of the Amygdala on Dyadic Social Interactions in Rhesus Monkeys (*Macaca mulatta*)

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The role of the amygdala in dyadic social interactions of adult rhesus monkeys (*Macaca mulatta*) was assessed after bilateral ibotenic acid lesions. Social, nonsocial, and spatial behaviors of amygdalectomized and control monkeys were assessed in 3 dyadic experiments: constrained, unconstrained, and round robin. Lesions produced extensive bilateral damage to the amygdala. Across all experiments, the amygdalectomized monkeys demonstrated increased social affiliation, decreased anxiety, and increased confidence compared with control monkeys, particularly during early encounters. Normal subjects also demonstrated increased social affiliation toward the amygdalectomized subjects. These results indicate that amygdala lesions in adult monkeys lead to a decrease in the species-normal reluctance to immediately engage a novel conspecific in social behavior. The altered behavior of the amygdalectomized monkeys may have induced the increased social interactions from their normal companions. This is contrary to the idea that amygdalectomy produces a decrease in social interaction and increased aggression from conspecifics.

The amygdala has long been implicated in the organization of social behaviors such as affiliation, aggression, and parental and sexual behaviors in a number of mammalian species (rats: Jonason & Enloe, 1971; cats: Schreiner & Kling, 1953; dogs: Fuller, Rosvold, & Pribram, 1957; monkeys: Kling, 1972; and humans: Adolphs, Tranel, & Damasio, 1998). Among primates, however, the function of the amygdala may be particularly important. Members of many primate species live in complex, highly organized social groups that are characterized by stable relationships among known individuals, dynamic patterns of social interaction, and subtle forms of communication. 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 processes, including both normal functioning and dysfunctional patterns such as autism, sociopathy, or schizophrenia (Bachevalier, 1994).

The connectional anatomy of the primate amygdala supports the idea that this structure is involved in processing social information and in contributing to social responding. The macaque amygdala is located within the anteromedial temporal lobe and consists of 13 individual nuclei or cortical regions (Amaral, Price, Pitkanen, & Carmichael, 1992). It receives extensive inputs from brain areas associated with different sensory modalities, including the inferotemporal cortex, superior temporal gyrus, and the somatosensory, gustatory, and olfactory cortices (Amaral et al., 1992). Outputs from the amygdala are likewise extensive, with projections back to the striatum, hypothalamus, hippocampus, brainstem, and areas of the neocortex—structures that are associated with the control of social behavior, homeostasis, hormonal state, and physical action (Amaral et al., 1992; Franzen & Myers, 1973).

Behavioral studies in primates have provided the most direct evidence that the amygdala is a critical structure involved in normal social and emotional functioning. The first study to directly investigate the relationship between the amygdala and nonhuman primate social behavior (Rosvold, Mirsky, & Pribram, 1954) found that high-ranking and previously aggressive rhesus monkeys with amygdala lesions fell in the dominance hierarchy and became extremely submissive after surgery. Kling and colleagues (Dicks, Myers, & Kling, 1969; Kling & Cornell, 1971; Kling & Dunne, 1976; Kling, Lancaster, & Benitone, 1970) conducted a research program in which free-ranging vervet and rhesus monkeys that

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sustained damage to the amygdala and uncus were released back into their social groups. These monkeys did not reestablish contact with other group members, did not initiate social interactions, and usually remained socially isolated and withdrawn, lacking appropriate positive socioaffective responses to solicitous behavior and physical contact from others (Dicks et al., 1969; Kling et al., 1970). In most cases, the amygdala-lesioned monkeys were attacked and died from their wounds, from predation, or from malnutrition. In contrast, when caged amygdalectomized stumptailed macaques were observed in a social group, they displayed either a decrease in aggression (adult male) or an increase in aggression (adult female), hypersexuality (adult female), and a reduction in positive social behaviors, such as huddling and grooming (Kling & Cornell, 1971). Some of the symptoms of the Kluver–Bucy syndrome (Kluver & Bucy, 1939) were also observed in caged vervets with amygdala lesions, such as coprophagia, hyperorality, a reduction in fear responses directed toward the human observers, and a decline in interanimal aggression (Kling, Dicks, & Gurowitz, 1969). Although results from studies in 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).

Notions about the role of the amygdala in the social behavior of nonhuman primates are based almost entirely on older methodological approaches. For example, until recently all lesions were made by either radiofrequency 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, as they often destroy neighboring brain regions. For example, many of the early lesion studies that used the suction ablation technique 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 & Gaffan, 1998). Thus, 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. In the experiments described in this article, the selective neurotoxin ibotenic acid was injected stereotaxically into the brain, causing minimal damage to adjacent areas while destroying only cell bodies and leaving fibers of passage through the amygdala intact. In addition, stereotaxic placement of every lesion was accomplished with an individual magnetic resonance imaging (MRI) atlas, and extensive quantitative histological analysis was performed for each lesion.

Earlier studies used behavioral data collection methods that were often more subjective than objective, more qualitative than quantitative, and which generated little actual data that could be statistically analyzed. The investigators often did not use an established ethogram 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 sex, thereby complicating the picture through lack of control over, for example, neuroendocrine differences among subjects as a result of reproductive status or sex.

In this series of experiments, subjects were adult male rhesus macaques that were assessed preoperatively to determine social competence in their natal groups. One group of experimental subjects sustained amygdala lesions; another group acted as controls. So that some commonality of social experience could be maintained, a third group of stimulus monkeys served as partners for members of both experimental groups in two of the experiments. An established catalog of social and nonsocial behavior was used (Capitanio, 1984; Capitanio, Mendoza, Lerche, & Mason, 1998), as were measures of the monkeys' attitudes (Capitanio, Bond, & Mason, 1997) and relative spatial locations.

General Method

Subjects and Living Arrangements

Twelve adult male, experimentally naive rhesus monkeys (*Macaca mulatta*) were randomly assigned either to receive bilateral ibotenic acid lesions of the amygdala (A-IBO group, $n = 6$) or to act as unoperated controls (control group, $n = 6$). The mean age of the A-IBO monkeys at the time of removal from their natal group was 6.15 ± 0.43 years, and their mean weight was 11.65 ± 0.36 kg. The mean age of the control monkeys was 5.93 ± 0.31 years, and their mean weight was 9.81 ± 0.50 kg. The monkeys were each born and raised in one of 12 half-acre enclosures that contained approximately 70 monkeys each, at the California Regional Primate Research Center, University of California, Davis. The monkeys were chosen from a larger sample after trained behavioral observers watched each monkey in its natal cage for two 30-min sessions over a 2-week period and ascertained that all the monkeys displayed a moderate level of social behavior and that none displayed unusual or inappropriate aggressive behavior or stereotypies. All selected subjects were mid-ranking in their cages' dominance hierarchies. Each subject was raised in a different corral from the other subjects and did not encounter the other experimental subjects until Experiment 3.

Four rhesus monkeys served as stimulus monkeys for the first two experiments. So that initial unfamiliarity between experimental subject and stimulus monkeys was ensured, the two stimulus males and two stimulus females (mean age at time of removal from natal group was 5.13 ± 0.39 years; mean weight was 7.17 ± 1.16 kg) were selected from different corrals. Both females had previously had one successful pregnancy; no contraceptive treatment was used during these studies.

Subject and stimulus monkeys were relocated to individual housing in rooms (9.0 m long, 3.4 m wide, with a sloping ceiling 2.7–3.2 m high) with automatically regulated lighting (12-hr light–dark cycle) and temperature (75–85 °F). The subjects could not see other monkeys from the same project. The subjects were fed on a diet of Monkey Chow (Ralston-Purina, St. Louis, MO) supplemented with fruit and vegetables, and water was freely available. Subjects were transported from their living cages to the test cages in aluminum transport cages measuring 0.5 m × 0.3 m × 0.4 m.

MRI Imaging and Stereotaxic Injections of Ibotenic Acid

Three weeks after relocation to indoor housing, small glass beads filled with copper sulphate (visible with T1-weighted imaging) were cemented to the skull at known stereotaxic coordinates to serve as fiducial marks for MRI. The monkey was tranquilized, a tracheal cannula was inserted, and the monkey was placed into an MRI-compatible stereotaxic apparatus (Crist Instruments, Damascus, MD). The subjects were anesthetized with isoflurane (1%–2%), and a midline incision of the scalp was made. Two glass beads (3 mm diameter) filled with copper sulfate (2% solution) were

attached to the skull with dental acrylic, at predetermined positions (A24 and A18 from interaural 0).

Two weeks after the bead implant surgery, the location of the amygdala in each monkey was determined by MRI (Alvarez-Royo, Clower, Zola-Morgan, & Squire, 1991; Rebert, Hurd, Matteucci, De LaPaz, & Enzmann, 1991; Saunders, Aigner, & Frank, 1990). The subjects were anesthetized with Telazol (10 mg/kg), and the brain was imaged with a Phillips 1.5T Gyroscan magnet (Phillips Medical Systems, Best, the Netherlands). Sections were taken with a T1-weighted inversion recovery pulse sequence (TR = 2084, TI = 708, TE = 20 NEX 2, FOV = 18 cm, matrix = 154 × 256). An interleaved coronal series of 3-mm-thick sections was obtained with both beads centered, and a similar series of sagittal 3-mm-spaced sections was also acquired with one bead centered. The MRIs were developed onto X-ray film and scanned. The scanned MRI was then exported to the Canvas graphics program (Version 5, Deneba Systems, Miami, FL) for stereotaxic analysis. The amygdala was outlined, and a 1-mm-spaced grid was overlaid onto the MRI image (calibrated to marks on the X-ray film, see Figure 1). For the coronal images, the grid was aligned with the midline; for the sagittal images, the grid was aligned with the glass bead. The coordinates for injection sites, which were typically separated from each other by 2 mm, were measured from the grid superimposed on the MRI image.

Before surgery, each monkey was anesthetized with ketamine hydrochloride (8 mg/kg), intubated with a tracheal cannula, and anesthetized with isoflurane (1%–2%). Because of concerns for morbidity and mortality with one-stage lesions, 5 of the 6 experimental monkeys received two-stage lesions of the amygdala. The interval between the two surgeries was 7 days. Because all surgical sequelae, such as respiratory arrest and lethargy, were manageable through veterinary intervention, the 6th monkey (26085) underwent bilateral amygdala injections. Recovery for this monkey was similar to that of monkeys that received unilateral lesions.

Throughout surgery, the monkeys' vital signs were monitored. A midline incision was made, and craniotomies were performed over the amygdala. The predicted dorsoventral location of the amygdala was verified electrophysiologically by making extracellular recordings with a tungsten microelectrode lowered into the amygdala along a trajectory estimated to be at a mid-rostrocaudal and mediolateral position within the amygdala.

Once the dorsoventral position of the amygdala was defined, the electrode was withdrawn. Ibotenic acid injections were then made with a 10-μl Hamilton syringe (26 gauge beveled needle). In the amygdala, 1.0 μl of ibotenic acid (Biosearch Technologies, Novato, CA, 10 mg/ml in 0.1 M phosphate buffered saline) was injected into each site. To allow diffusion of the ibotenic acid and to reduce potential tissue damage, the injections were made at a rate of 0.2 μl/min. A complete unilateral amygdala lesion required injections at 20–24 sites, with two to three rostrocaudal levels, three mediolateral levels, and three or four dorsoventral levels at each mediolateral level. In the 6th monkey, which received a two-stage bilateral lesion, the injections were made with two identical Hamilton syringes to simultaneously inject ibotenic acid at the same location within each amygdala. After the injections, the dura was replaced (and in some cases sutured), the temporalis muscle tissue was repositioned and sutured, the craniotomy was filled with Gelfoam (Pharmacia & Upjohn, Peapack, NJ), and the wound was sutured in three layers. After surgery, the monkeys' vital signs and general condition were monitored continuously for 24 hr by veterinary staff. Postsurgical recovery varied substantially from subject to subject. In all cases, complete recovery from anesthesia appeared to be prolonged by the neurotoxin. In some monkeys, recovery was so advanced by 2 hr postsurgery that the monkey was returned to recovery observation cages. In other monkeys, postsurgical lethargy continued for 6 hr or more, and in 2 cases, the monkeys required postsurgical mechanical ventilation because of a lack of spontaneous breathing. All monkeys received prophylactic doses of the antibiotics Cefazolan (20 mg/kg three times daily) and Baytril (BVP, 5 mg/kg once daily) and the analgesic Oxymorphone (0.15 mg/kg three times daily; all from Endo Pharmaceuticals, Chadds Ford, PA) as needed, and they were allowed to recover for at least 3 months after the second lesion before behavioral testing commenced. All surgical procedures were performed under an approved University of California, Davis Institutional Animal Care and Use protocol and strictly adhered to the National Institutes of Health guidelines on the use of nonhuman primate subjects.

Lesion Analysis

So that the extent of the amygdala lesions could be quantitatively evaluated, the volumes of the entire amygdala and of the lateral, basal, accessory basal, and central nuclei were measured in the left hemisphere of five unlesioned rhesus monkeys of approximately the same age and weight as the A-IBO subjects. Sections from these monkeys were kindly provided by Dr. Peter Rapp, Mount Sinai School of Medicine, New York. Although the brains were fixed and processed in a similar way, two differences are worth noting. First, the brain was not blocked in the coronal plane but at an angle of 13° so that sections could be cut more perpendicular to the plane of the longitudinal axis of the hippocampus. Second, sections were cut at 40 μm rather than 30 μm, and the series was 1-in-10 rather than 1-in-8. Because sections were measured throughout the full rostrocaudal extent of the amygdala and cross-sectional areas were multiplied by the appropriate rostrocaudal distance that they represented, these histological processing differences should not have markedly affected our estimate of amygdaloid volumes. For each control and A-IBO subject, a Leica Stereomicroscope and camera lucida (Wetzlar, Germany) were used to make drawings of each section that contained the amygdaloid complex. The cross-sectional areas of the entire amygdala and the lateral, basal, accessory basal, and central nuclei were digitized with a SummaSketch III digitizing tablet (Summagraphics, Seymour, CT) connected to a PC with Sigma Scan software (Version 3, Jandel Corp., San Raphael, CA). To compute the volumes, the cross-sectional areas were multiplied by the distance represented by each coronal section (240 μm for the A-IBO subjects and 400 μm for the control subjects).

Histology

After completion of all behavioral testing (Experiments 1–3 and further experiments not reported here), all A-IBO subjects were immobilized with ketamine hydrochloride (8mg/kg), deeply anesthetized with Nembutal (50–100 mg/kg iv), and perfused intracardially. Perfusates included 4% paraformaldehyde in 0.1 M sodium phosphate buffer (pH 7.2 at 4 °C), 250 ml/min for 10 min and 100 ml/min for 50 min. The brain was then blocked stereotaxically, removed from the skull, and postfixed for 6 hr. The brain was then cryoprotected overnight in a solution containing 10% glycerol and 2% dimethylsulfoxide, followed by 3 days in a solution containing 20% glycerol and 2% dimethylsulfoxide. The brain was frozen by the isopentane method (described by Rosene et al., 1986) and stored at –70 °C until cut. Frozen sections were cut on a sliding microtome in the coronal plane at a thickness of 30 μm (1-in-8 series) and placed into a cryoprotectant tissue-collecting solution (30% ethylene glycol, 25% glycerin in 0.005 M sodium–phosphate buffer). The sections were stored at –20 °C until they were processed for Nissl staining. A 1-in-8 series of sections was mounted onto gelatin-coated slides and stained with thionin.

Behavioral Data Collection

Social and nonsocial behaviors were recorded for all experiments by means of a catalog of behaviors commonly used for this species (e.g., Capitanio, 1984; Capitanio et al., 1998). Behavioral categories (listed and defined in Table 1) differed slightly depending on the experiment. All behavioral data collection was done on standard PCs with The Observer (Noldus, 1991) behavioral data collection software.

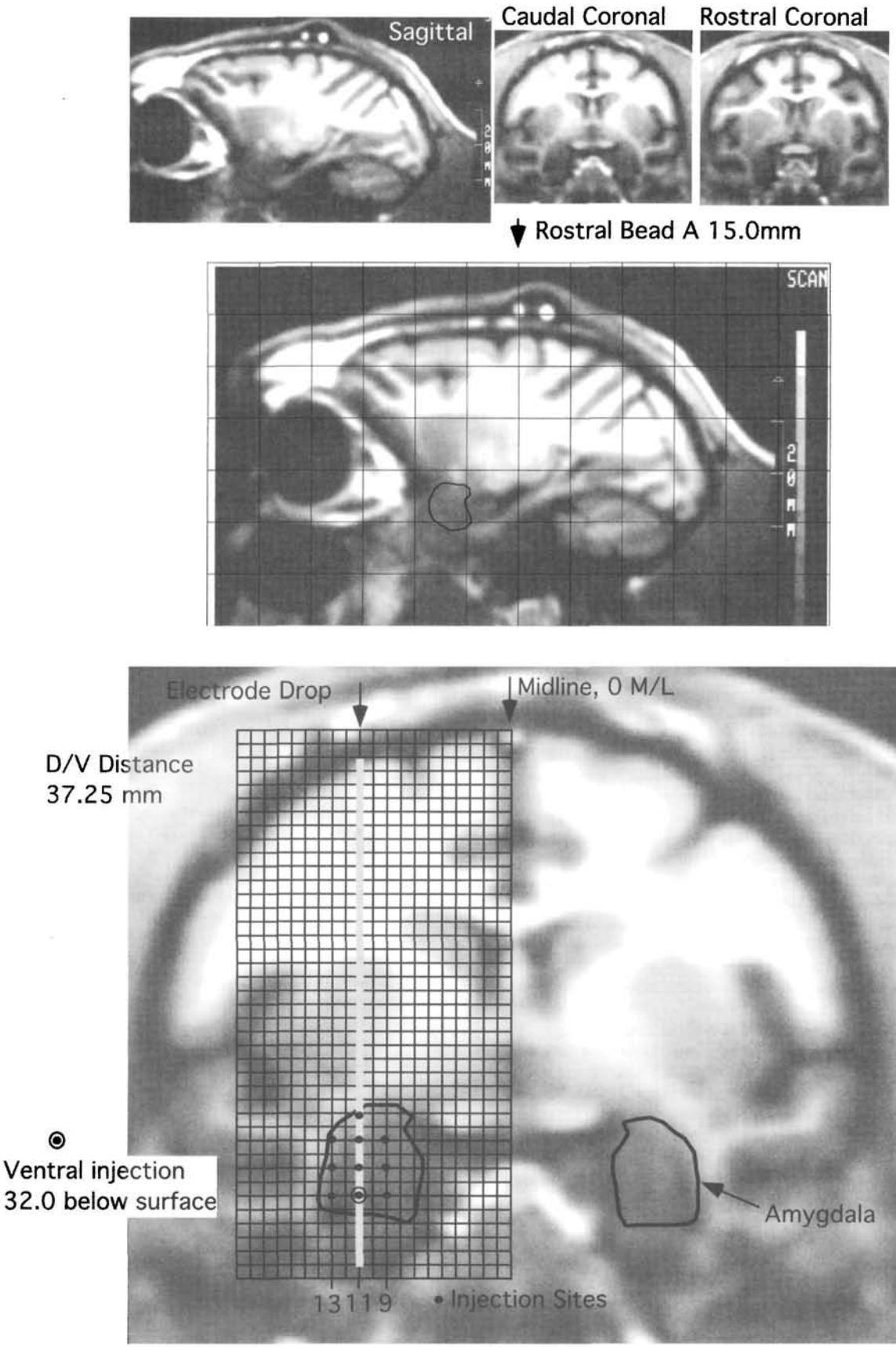
Sagittal
Caudal Coronal
Rostral Coronal
Rostral Bead A 15.0mm
SCAN
Electrode Drop
Midline, 0 M/L
D/V Distance
37.25 mm
Ventral injection
32.0 below surface
Amygdala
13119
Injection Sites

Table 1
Descriptive Behavioral Ethogram (States and Social and Nonsocial Events)

State or behavior	Description
	Behavioral states
Proximity	Within arm's reach of one another at start of trial only.
Contact	Monkeys are in direct physical contact for 3 s. Contact implies proximity.
Grooming	Monkey is picking or licking another's fur for 3 s. Grooming implies proximity.
Extended social	Play, aggression, chase, or mount longer than 3 s. Extended social implies proximity.
Nonsocial stationary	Out of arm's reach while remaining stationary (constrained and unconstrained dyads only).
Nonsocial locomotion	Out of arm's reach while in motion (constrained and unconstrained dyads only).
Nonsocial	Out of arm's reach at start of trial only (round robin dyad only).
Proximity zone	Located within the quadrant in front of the stimulus cage (constrained dyad only).
	Behavioral events: Social behaviors
Walk by	Moving into and out of proximity in less than 3 s.
Aggression	Grab, hit, bite, or slap.
Threat	Two or more of open mouth stare, lunge, head bob, bark vocalization.
Fear grimace	Large grin, exposing teeth.
Lipsmack	Rhythmic lip movements, often with pursed lips.
Vocalization coo	High-pitched, soft vocalization.
Vocalization scream	High-pitched, high-intensity vocalization or alarm call.
Vocalization bark	Long, guttural bark vocalization.
Vocalization grunt	Soft, bubbly, guttural sound, made in affiliative situations.
Crooktail	Dominance display in which monkey struts with tail held up in "?" shape.
Anogenital exploration	Sniffing, touching, or licking anogenital area of other monkey.
Present sex	Stiff four-point stance, tail up, rump toward partner.
Present groom	Rigid presentation of body part for grooming.
Mount	Double foot clasp, hands on back, thrusting.
Incomplete mount	Missing double foot clasp, hands on back or thrusting.
Play	Rough and tumble play, grappling, with play face.
Chase	Quick, hostile movement after another monkey.
Displace	Monkey takes other monkey's spot for 3 s.
Approach	Movement into arm's reach of another monkey for 3 s (round robin dyad only).
Withdraw	Movement out of arm's reach of another monkey for 3 s (round robin dyad only).
	Behavioral events: Nonsocial behaviors
Tooth grinding	Audible rubbing of lower premolars on upper canines.
Cage aggression	Dominance display, including cage shaking and body slams.
Yawn	Fully open mouth, with lips fully retracted and teeth showing.
Self-clasp	Abnormal grasping of the torso.
Self-bite	Hair-plucking, self-biting, or other self-mutilation.
Self-groom	Picking or licking at one's own fur or nonfur body part.
Self-sex	Manual or oral manipulation of one's own genitals.
Tactile exploration	Use of the hands to explore the physical environment.
Oral exploration	Use of the mouth to explore the physical environment.
Urine drinking	Drinking of one's own urine from penis, hand, or ground.
Scratch	Crude, rapid, hand movements, using fingers to scratch.
Motor stereotypies	Abnormal motor movements: bucking, bouncing, circling, etc.

Note. Behavioral states are continuous acts of behavior lasting longer than 3 s; behavioral events are single instances of behavior; social behaviors are behaviors that can only be performed with a partner; nonsocial behaviors are behaviors that do not require the interaction of a partner.

Figure 1 (opposite). Magnetic resonance images of 1 experimental monkey used to determine coordinates for ibotenic acid injections. The top panel shows a parasagittal section (left) and two coronal levels through the amygdala (center and right). The middle panel shows an enlarged view of the parasagittal section in which two glass fiducial beads filled with copper sulfate solution can be seen on the surface of the skull. In the bottom panel, a 1-mm grid is superimposed over one of the coronal sections through the amygdala. Dots indicate the locations (separated by 2 mm) at which 1-μl injections of ibotenic acid were made. D/V = dorsoventral; M/L = medial–lateral. See text for more details.

In addition to recording frequencies and durations of discrete behaviors, observers also noted the spatial location of subjects during each trial and, at the end of every trial, rated the attitude of each subject. Attitude ratings provide summary impressions on the part of the observers that describe qualities of the subject's behavior and interactions that are not easily captured through coding of discrete behaviors (Capitanio et al., 1997). The procedure is an adaptation of one commonly used for assessing personality (e.g., Capitanio, 1999), with the principle difference being the time-frame involved: Attitude tends to reflect states, whereas personality reflects traits. Each monkey was rated on either three (Experiment 1) or five (Experiments 2 and 3) adjectives (nervous, confident, avoidant, aggressive, and affiliative; for descriptions of adjectives and which experiment they were recorded on, see Table 2), on a 5-point scale that was anchored by the endpoints "not at all descriptive" and "extremely descriptive." Raters were trained and instructed to ensure that their ratings for a given trial were based only on the immediately preceding interaction.

Spatial position was recorded for each monkey at regular intervals during every trial by using grids located on the floor of the test cage. Details of this data collection are found in the *Method* sections of the specific experiments.

For all observational procedures (behaviors, attitude, spacing), interobserver agreement data were collected before the start of each experiment by observation of comparably aged, nonstudy monkeys in the test situation. For behavioral data, observers demonstrated better than 80% agreement (agreements / [agreements + disagreements]) for each category, with most categories greater than 85% agreement. Measurement of duration of behaviors between observers correlated at $r > .98$. For spatial position, all observers were trained to a criterion of greater than 95% agreement. During reliability sessions, observer ratings of attitude correlated at $r >= .85$, except for avoidant ($r = .69$).

During Experiments 1 and 2, three trained observers recorded the behavioral categories and performed the attitude assessments, and a second group of trained observers recorded the relative spatial location. During Experiment 3, two trained observers recorded the behavioral categories and relative spatial location and performed the attitude assessments.

We should note that, as with other studies that have examined the social behavior, emotional responsiveness, and cognitive responsiveness of animals with amygdala lesions, our behavioral observers were not unaware of the lesion status of our amygdalectomized and control subjects. Although this knowledge could have influenced our results, we consider this possibility to be remote for three reasons. First, social testing commenced approximately 6 months after the surgeries, by which time there were no visible differences in appearance between lesioned and control subjects. Thus, during the actual data collection process, which involves intense concentration on the behavior (not the appearance) of the monkeys and second-by-second inputting of data into the Observer program, there were no overt cues by which the observers might have been reminded on a moment-to-moment basis of which monkey was in which group. Second, at all stages of the test series, observers were trained and interobserver reliability data were obtained with nonstudy monkeys. This was likely to have prevented observers from associating lesion status with particular patterns of behavior for specific subjects. Last, and perhaps most important, many of the results we obtained were strikingly counter to our expectations. As we report, rather than the subjects being hyposocial, as would have been predicted by the previous literature, they generated increased social activity. It is unlikely that this result would have been obtained had there been a systematic bias on the part of our observers.

Statistical Analyses

In general, analysis of variance (ANOVA) procedures were used for the statistical analysis, with subject nested within group (A-IBO vs. control) as a random blocking factor to reduce the effects of variation within the subject groups (Sokal & Rohlf, 1995). Follow-up contrasts were made with the Bonferroni post hoc test (significance set at $p < .005$ for a total of 10 comparisons; $p < .05/10$). In most cases, the frequency and duration data were not normally distributed, occasionally displayed heterogeneous variances, and often contained a number of zero values. Therefore, a $\log_{10}(x + 1)$ transformation (Howell, 1997) was used to transform the data. For display purposes, the transformed median was then converted back by

Table 2
Attitude ("Personality") Assessment Ethogram

Assessment	Description
Confident	Behaves in a positive assured manner, not restrained or tentative. The subject's attitude is characterized by free movement about the cage; the movements will be fluid, not furtive, and the subject may be strutting with a crooktail posture.
Nervous/anxious	Uncomfortable or tense with the situation. The subject's attitude is characterized by fidgeting, picking at the cage or floor, stereotypies, fear grimacing, yawning, or jerky movements.
Avoidant	Refrains from interaction with other the monkey by exhibiting evasive behavior or gaze aversion. The subject's attitude is characterized by refraining from engaging in any social interactions (i.e., no grunts, sex presents, etc.), moving away, gaze averting, or ignoring social attempts by the other monkey, and is often accompanied by fear grimaces.
Affiliative	Sociable, friendly, seeks out the companionship of the other monkey. The subject's attitude is characterized by actively seeking to be near and/or in friendly contact with the other monkey or by facilitating contact with the other monkey. Typical behaviors displayed are lipsmack or pucker face, grunts, groom or present groom, present sex, social play, mount, or attempted mount.
Aggressive	Causes harm or potential harm to the other monkey. The subject's attitude is characterized by forms of vigorous physical contact such as bites, slaps, grabs, chases, threat face, and/or bark vocalizations. Crooktail, tooth grinding (audible), exaggerated and prolonged chewing movements, lunges toward others, head bobs toward others, and ear flaps are also expressions of an aggressive attitude, particularly when they occur in combination.

Note. Confident, nervous/anxious, and avoidant ratings are reported for constrained, unconstrained, and round robin dyads only. Affiliative and aggressive ratings are reported for unconstrained and round robin dyads only.

using exponential $(x) - 1$ with 95% upper and lower confidence intervals (Sokal & Rohlf). Interanimal distance was not transformed because it was normally distributed, and subject was not nested as a random factor for this analysis because the relative spatial location was not dependent solely on the behavior of 1 monkey.

Histological Results

Histological Analysis

Defining the cross-sectional area of the amygdala—even of the normal animal—is not without difficulties. The border between the dorsal amygdala and the substantia innominata, for example, is difficult to set precisely. This task becomes even more problematic with the distortion that is produced after long-term survival of the lesioned monkeys. In Table 3 and Table 4, we have listed the measured volumes of the amygdala and subnuclei in the control and A-IBO monkeys. In Table 5, we have indicated the percentage of loss of the amygdala relative to the mean value obtained from the control monkeys. Although we feel confident in the volumes attributed to the entire amygdala in the lesioned monkeys, volumes assigned to the major nuclei must be viewed as approximations. We have also included the volume of the entorhinal cortex. This ventrally subjacent region was damaged in several of the cases, and we have indicated in Table 3 the volume of the residual entorhinal cortex located at the same levels as the amygdala. In no case was the entorhinal cortex located caudal to the amygdala (accounting for approximately half of its rostrocaudal extent of the region) damaged. This part of the entorhinal cortex was not measured in either the control or lesioned monkeys. The average bilateral loss of entorhinal cortex located subjacent to the amygdala was about 50%, or approximately 25% of the entire volume of the entorhinal cortex.

Third, we have also provided an indication of the extent of damage to structures surrounding the amygdala. Table 6 provides a qualitative summary of the amount of damage located in all regions that were either directly involved in the ibotenic acid lesion or that may have been damaged by the passage of the Hamilton syringe during the lesion procedure. Fourth, photomicrographs of six levels through the amygdala of A-IBO Subject 25468 (left and right sides), along with sections from similar levels in 1 of the control subjects, are presented in Figure 2. This case represents both a relatively complete amygdaloid lesion and indicates the types of extraneous damage seen in these subjects.

Table 3
Volume (in Cubic Millimeters) of Total Amygdala (AMYG), Individual Amygdala Nuclei, and Entorhinal Cortex in 5 Unlesioned Control Monkeys

Subject ID	AMYG	L	B	AB	Ce	EC
24080	282.49	54.63	71.70	37.80	15.97	130.70
25014	281.70	62.18	66.76	37.16	16.94	115.38
24712	264.61	52.38	63.73	35.63	15.14	110.83
25616	251.46	54.78	51.13	34.42	12.97	103.94
24047	309.62	61.75	69.31	44.44	15.47	147.07
Mean	277.98	57.15	64.53	37.89	15.30	121.58

Note. Control monkeys had histories similar to those of the experimental subjects and were raised at the California Regional Primate Research Center, University of California, Davis. Only the left hemisphere was available for comparison. L = lateral; B = basal; AB = accessory basal; Ce = central; EC = entorhinal cortex.

Control Brains

The overall volume of the adult male rhesus monkey amygdala is approximately 280 mm^3. This average volume is consistent with MRI measurements indicating that the amygdala is roughly cube-shaped (with a caudal tail mainly comprising the central nucleus and the amygdalohippocampal area) and measures approximately 6–7 mm in each direction. There was relatively low variability in the measurements of total volume, with a range of 251–309 mm^3. The basal nucleus was the largest of the deep nuclei, with a mean volume of approximately 65 mm^3, the lateral nucleus was somewhat smaller (57 mm^3), and the accessory basal (38 mm^3) and central nuclei (15 mm^3) were smaller yet. The entorhinal cortex located beneath the amygdala has a total volume of approximately 122 mm^3.

A-IBO Brains

The ibotenic acid injections produced substantial lesions in each of the experimental monkeys. The average of the left and right side cell loss in different monkeys ranged from 66% in Subject 25942 to 84% in Subject 26085. More important than the volume of cell loss was the region that suffered the greatest cell loss. We found that the residual amygdala consisted primarily of the medial or superficial nuclei, such as the periamygdaloid cortex, the medial nucleus, and the central nucleus. These results were expected because we purposely avoided syringe placements that were too medial in order to prevent infarction of blood vessels that lie adjacent to the amygdala. We also were cautious about injecting ibotenic acid into the caudally placed central nucleus in order to prevent inadvertent damage to the hippocampal formation.

Our lesions were designed to maximally affect the lateral, basal, and accessory basal nuclei, that is, the nuclei that have the most direct association with the neocortex. The injections were quite successful in achieving this goal. Again, as indicated in Table 5, cell loss in the lateral nucleus was nearly complete in several cases. The average bilateral cell loss ranged from 84% in Subject 25942 to 99% in Subject 26085. Four of the 6 monkeys had losses exceeding 90%. The basal nucleus demonstrated equally complete cell losses, whereas there was a somewhat greater sparing of the accessory basal nucleus. As noted above, the central nucleus demonstrated substantial sparing. Whereas the average bilateral loss of the central nucleus in Subject 25571 was 83%, loss in the other monkeys ranged from 55% to 71%.

Given that there was some variability in the amount of damage to the amygdala and to surrounding structures, we will describe the lesion extent for each of the experimental monkeys individually.

Subject 26085. The lesion in this subject was both the most complete and the most discrete of the experimental group. The lateral, basal, and accessory basal nuclei lesions were all greater than 90% complete bilaterally (Table 5). The central nucleus lesion was at least 60% complete bilaterally. The major extraneous damage in this case was to the rostral entorhinal cortex, which was reduced by 73% on the left side and by 53% on the right side. The extent of damage to surrounding areas is indicated in Table 6. This case demonstrated bilateral damage to the piriform cortex (both

Table 4
Volume (in Cubic Millimeters) of Total Amygdala (AMYG), Individual Amygdala Nuclei, and Entorhinal Cortex in the 6 Experimental Subjects With Bilateral Ibotenic Acid Lesions of the Amygdala

	AMYG		Lateral		Basal		Accessory basal		Central		Entorhinal cortex	
Subject ID	L	R	L	R	L	R	L	R	L	R	L	R
26085	46.51	42.50	0.85	0.00	1.82	0.40	2.67	2.03	6.18	5.29	33.10	57.70
25468	67.75	34.16	3.90	0.28	3.09	0.00	7.19	0.16	6.20	2.38	83.56	83.54
24349	107.88	28.70	11.95	0.00	14.29	0.00	19.08	0.00	8.24	4.89	49.27	40.67
25627	78.23	54.71	0.85	4.27	1.84	2.06	15.82	4.15	7.97	5.56	57.31	66.56
25571	12.50	82.35	0.00	6.20	0.00	6.26	0.00	11.29	1.40	3.69	49.89	77.98
25942	73.35	113.17	3.16	14.93	3.59	19.43	12.89	20.76	6.09	5.92	60.16	77.91

Note. L = left; R = right.

frontal and temporal portions), to a short rostrocaudal extent of the ventral claustrum, to area 35 of the perirhinal cortex below the amygdala (but not rostral or caudal to the amygdala), and to the fundus of the superior temporal sulcus (STS) for approximately 3 mm through the rostral amygdala, as well as minor punctate cell loss at the most rostral levels of the hippocampus. No other bilateral damage was observed in this case.

Subject 25468. Photomicrographs of the amygdala from this subject are shown in Figure 2. We have illustrated six levels that represent the full rostrocaudal extent of the amygdala, as well as the types of extraneous damage, which in this case is typical of what was observed in the other cases. This subject also had greater than 90% bilateral removal of the lateral and basal nuclei (Table 5). The accessory basal nucleus was almost entirely eliminated on the right side, but there was approximately 19% savings on the left. The central nucleus had somewhat greater savings bilaterally. As with Subject 26085, there was relatively minor extraneous damage in this case. The subjacent entorhinal cortex demonstrated approximately 31% loss bilaterally. There was bilateral damage in the temporal lobe portion of the piriform cortex, rostral levels of the ventral claustrum, some cell loss in the substantia innominata, complete loss of area 35 through the rostral amygdala (with little or no damage to area 36), and bilateral damage to the fundus of the STS for a distance of less than 1 mm. There was no other extraneous damage observed in this case.

Subject 24349. The lesion in this case was less symmetric than in the previous 2 cases. It appeared that the lesion on the left side of Subject 24349 was placed somewhat too ventrally. For this reason, there was less damage to the amygdala and more damage to the perirhinal cortex. The lesion of the right side was very successful, with complete removal of the lateral, basal, and accessory basal nuclei. The central nucleus on the right side was also reduced by 68%. The lateral and basal nuclei on the left side had greater than 70% tissue loss, though the accessory basal nucleus was only reduced by 50%. There was slightly less than 50% loss of volume in the central nucleus. As indicated, the extraneous damage was greater on the left side than on the right. The rostral entorhinal cortex demonstrated about 60% bilateral damage. The piriform cortex demonstrated only patchy cell loss on the left side but was markedly shrunken on the right. There was also patchy cell loss in the substantia innominata. The rostral ventral claustrum demonstrated bilateral damage, and both areas 35 and 36 subjacent to the amygdala were damaged. Area 36 through the caudal half of the amygdala appeared to be normal. There was damage to the fundus of the STS for a short rostrocaudal distance, and there was minor cell loss at the most rostral level of the hippocampus. This subject also had a small infarct located in the left caudate nucleus. This was likely due to the passage of the syringe needle during the injection procedure.

Table 5
Percentage Volume of Amygdala (AMYG), Individual Amygdala Nuclei, and Entorhinal Cortex Damaged After Bilateral Ibotenic Acid Lesions

	AMYG		Lateral		Basal		Accessory basal		Central		Entorhinal cortex	
Subject ID	L	R	L	R	L	R	L	R	L	R	L	R
26085	83.27	84.71	98.51	100.00	97.18	99.38	92.95	94.64	59.61	65.42	72.78	52.54
25468	75.63	87.71	93.18	99.51	95.21	100.00	81.02	99.58	59.48	84.44	31.13	31.29
24349	61.19	89.68	79.09	100.00	77.86	100.00	49.64	100.00	46.14	68.04	59.48	59.48
25627	71.86	80.32	98.51	92.53	97.15	96.81	58.25	89.05	47.91	63.66	52.86	45.26
25571	95.50	70.38	100.00	89.15	100.00	90.30	100.00	70.20	90.85	75.88	58.97	35.86
25942	73.61	59.29	94.47	73.88	94.44	69.98	65.98	45.51	60.20	61.31	50.52	35.92

Note. Each side (left [L] and right [R]) was calculated separately, and the percentage of loss was determined by using the mean volume of each structure from unlesioned control monkeys (n = 5).

Table 6
Extraneous Damage to Nonamygdala Brain Regions in Ibotenic Acid-Lesioned Monkeys

	26085		25468		24349		25627		25571		25942	
Brain region	L	R	L	R	L	R	L	R	L	R	L	R
Frontal cortex	0	0	0	0	0	0	0	0	0	0	0	0
Temp. pole (medial)	0	0	0	0	0	0	1	0	1	1–2	0	0
Temp. pole (lateral)	0	0	0	0	0	0	0	0	0	0	0	0
Pir. crtx. (frontal)	2	2	1	0	0	0	0	0	2	2	2	2
Pir. crtx. (temp.)	2	2	3	3	1	3	1	0	3	3	2	2
Claustrum (ventral)[a]	2	2	2	2	2	2	1	1	2	2	1	1
Substantia innominata	0	0	1	1	1	1	0	0	1	1	0	0
Area 35[b]	3	2	3	3	2	2	3	2	3	3	3	0
Area 36r[b]	2	0	0	0	2	2	0	0	0	0	0	0
Area 36c	0	0	0	0	0	0	0	0	0	0	0	0
TE (ventral)	0	0	0	0	0	0	0	0	0	0	2	0
STS fundus[c]	3	3	3	3	2	2	3	1	2	2	0	0
STS lateral	0	0	0	0	0	0	0	0	0	0	0	0
Hip. form. (rostral)	1	1	0	0	1	1	1	1	1	0	1	0
Hip. form. (caudal)	0	0	0	0	0	0	0	0	0	0	0	0
Striatum	0	0	—[d]	0	—[d]	0	0	0	—[d]	0	—[d]	0

Note. L = left; R = right; 0 = no cell loss; 1 = minor cell loss; 2 = extensive cell loss; 3 = region completely gone; Temp. = temporal; Pir. crtx. = piriform cortex; TE = inferotemporal cortex; STS = superior temporal sulcus; Hip. form. = hippocampal formation.
[a] Damage to claustrum restricted to levels of the amygdala. [b] Area 35 or 36 damage restricted to area ventral to the amygdala. [c] Damage less than rostrocaudal distance of the amygdala. [d] These subjects had infarcts of the striatum.

Subject 25627. This was another case that had a very discrete lesion of the amygdala. Both the lateral and basal nuclei were reduced by greater than 90%. The lesion of the accessory basal nucleus was 89% complete on the right side but removed only 58% of the nucleus on the left. The central nucleus demonstrated 48% loss on the left and 64% loss on the right. As in the other cases, the rostral entorhinal cortex was reduced by 53% on the left and by 45% on the right. Unlike the other cases, however, there was no damage to the piriform cortex, no cell loss in the substantia innominata, and only minor cell loss in the ventral claustrum. Area 35 had bilateral cell loss through the rostral half of the amygdala but was normal on the right side through the caudal half of the amygdala. The fundus of the STS demonstrated substantial cell loss for a short rostrocaudal distance on the left side, but the right side was largely intact. There was patchy cell loss in the rostral hippocampus that was more extensive on the right side.

Subject 25571. The lesion of the amygdala in this case was somewhat asymmetric. The left amygdala was almost completely eliminated by the lesion, whereas there was about 30% sparing of the right amygdala, mainly because of preservation of the medially situated structures. There was close to 90% or greater loss of the lateral and basal nucleus. The accessory basal nucleus was completely eliminated on the left side, but there was 30% savings on the right side. The bilateral extraneous damage in this case included mild neuronal loss in the medial aspect of the temporal polar cortex (area 36), both the frontal and temporal portions of the piriform cortex, portions of the ventral claustrum, and in the substantia innominata. There was near total cell loss in area 35 ventral to the amygdala (but not area 36 through these levels) and the fundus of the STS at the level of the amygdala. This subject also had an infarct of the left caudate nucleus that extended into the internal capsule and was somewhat gliotic.

Subject 25942. The lesion in this case was complicated by the presence of infarcts on both sides of the brain. This subject demonstrated the smallest lesion, with 74% loss on the left side and 59% loss on the right. As indicated in Table 5, there was a substantial saving in both the basal and accessory basal nuclei, as well as in the medial or superficial nuclei. The lesion was smaller on the right side. It appears that one or more of the injections may have been placed too rostrally and/or laterally in this case. There was an infarct that was first noticeable at a level just rostral to the amygdala and that extended into the white matter lateral to the amygdala for its full rostrocaudal distance. Although this infarct would have undoubtedly damaged some of the fibers extending between the amygdala and temporal neocortex, it is also likely that it disrupted some associational or projection fibers originating from the temporal lobe. There was also an infarct on the left side located just medial to the ventral claustrum. In addition to the extraneous damage caused by the infarct, there was bilateral cell loss in the piriform cortex and light cell loss in the ventral claustrum. Areas 35 and 36 were completely intact on the right side, and only area 35 was damaged on the left side ventral to the amygdala.

Summary of Histological Evaluation

In general, the A-IBO lesions produced substantial loss of neurons in the amygdala, with the most consistent and extensive loss in the lateral, basal, and accessory basal nuclei. The central nucleus was less affected, and superficial areas like the periamygdaloid cortex were generally much less affected by the neurotoxin. Nonamygdala areas in which cell loss was consistently observed included the rostral entorhinal cortex and perirhinal cortex. Damage in these areas was confined to the region ventrally adjacent to

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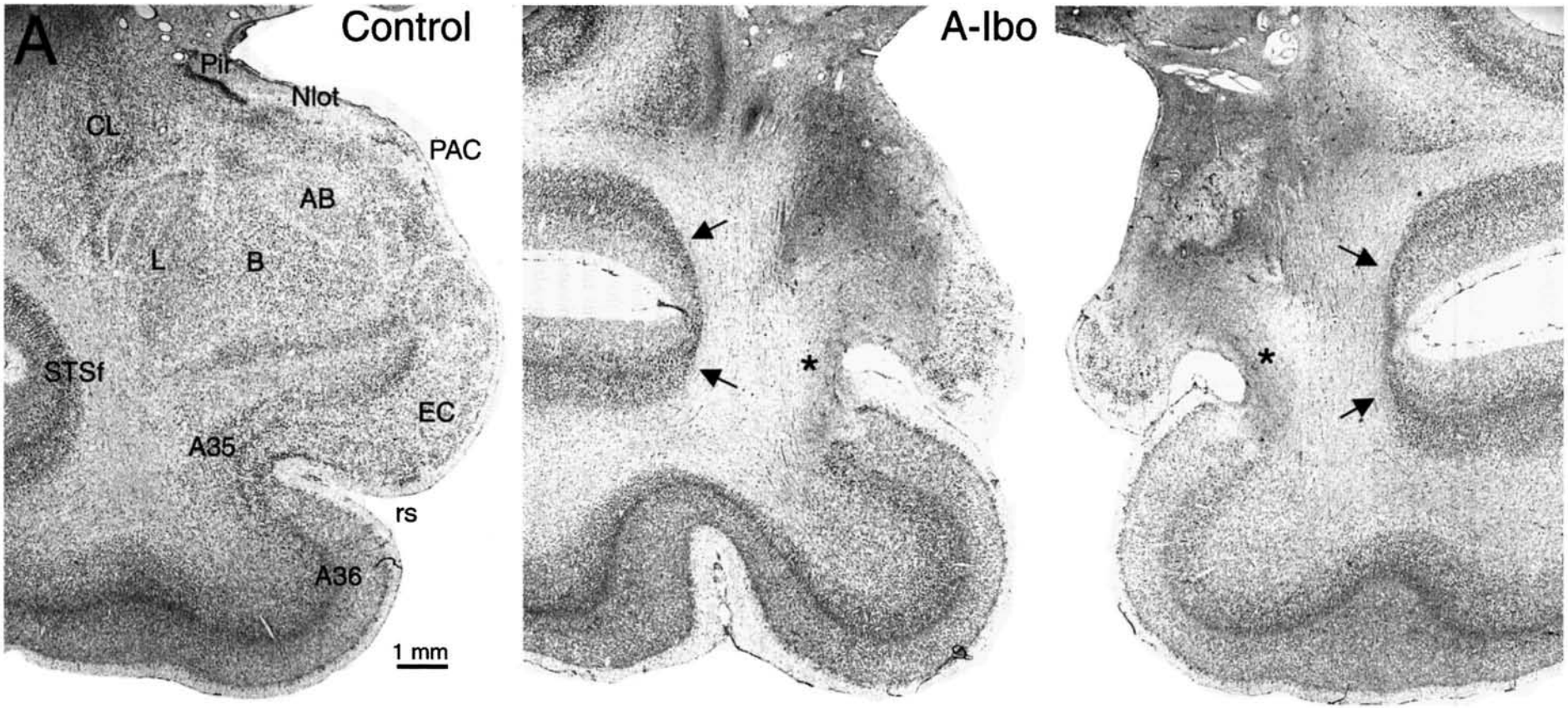
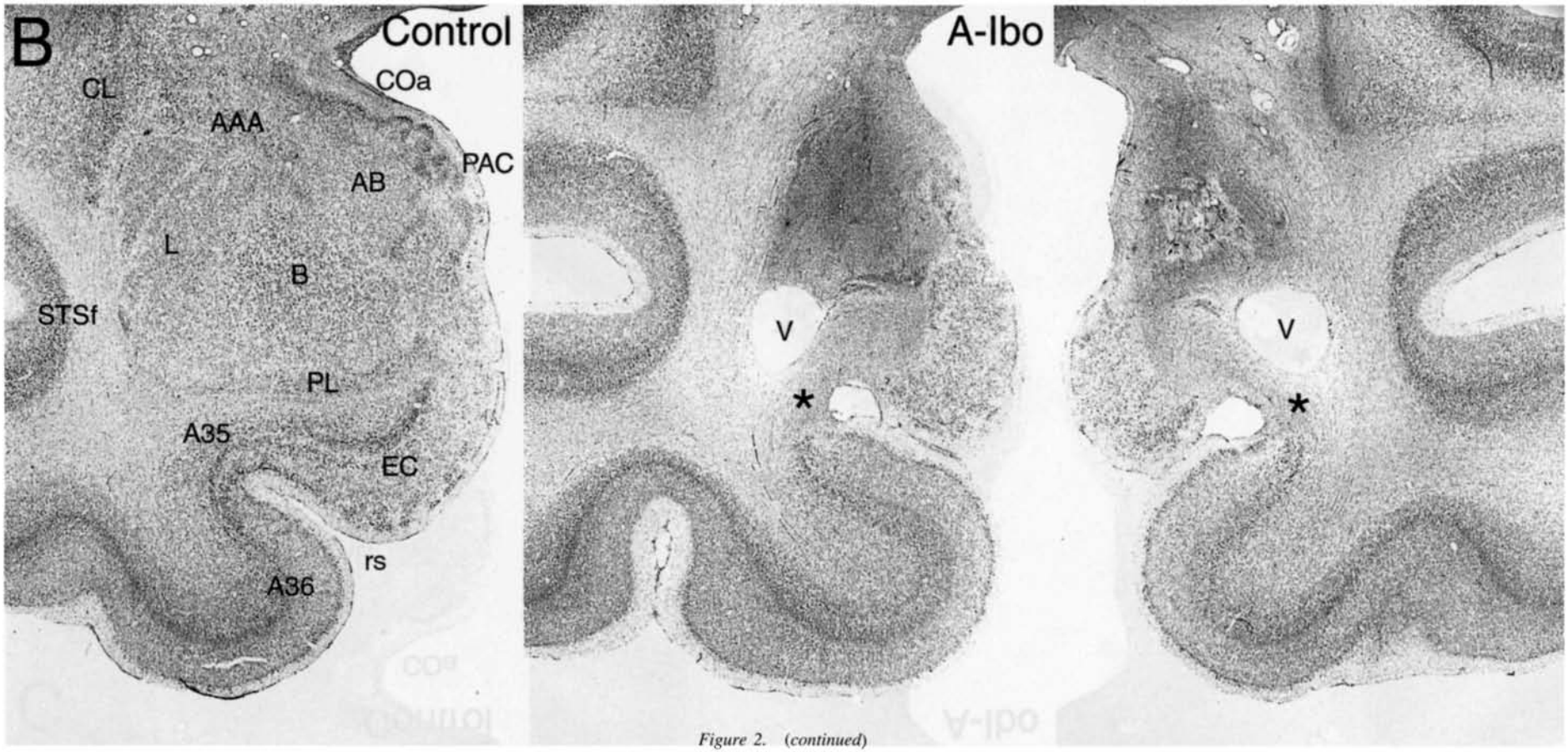


Figure 2 (following pages). Photomicrographs of representative coronal sections through the rostrocaudal extent of the amygdaloid complex in a control monkey and ibotenic acid-lesioned (A-Ibo) Subject 25468. The sections are arranged from rostral (A) to caudal (F). Calibration bar in Panel A equals 1 mm and applies to all other panels of this illustration. See text for more detailed description of the extent of the lesion. AAA = anterior amygdaloid area; AB = accessory basal nucleus of the amygdala; A35 = area 35 of the perirhinal cortex; A36 = area 36 of the perirhinal cortex; B = basal nucleus of the amygdala; CE = central nucleus of the amygdala; CL = claustrum; COa = anterior cortical nucleus of the amygdala; EC = entorhinal cortex; H = hippocampus; L = lateral nucleus of the amygdala; M = medial nucleus of the amygdala; Nlot = nucleus of the lateral olfactory tract; PAC = periamygdaloid cortex; Pir = piriform cortex; PL = paralaminar nucleus of the amygdala; PU = putamen; rs = rhinal sulcus; S = subiculum; STSf = fundus of the superior temporal sulcus; V = ventricle. Asterisk indicates damage in the fundus of the rhinal sulcus; arrows indicate areas of damage in the STSf.



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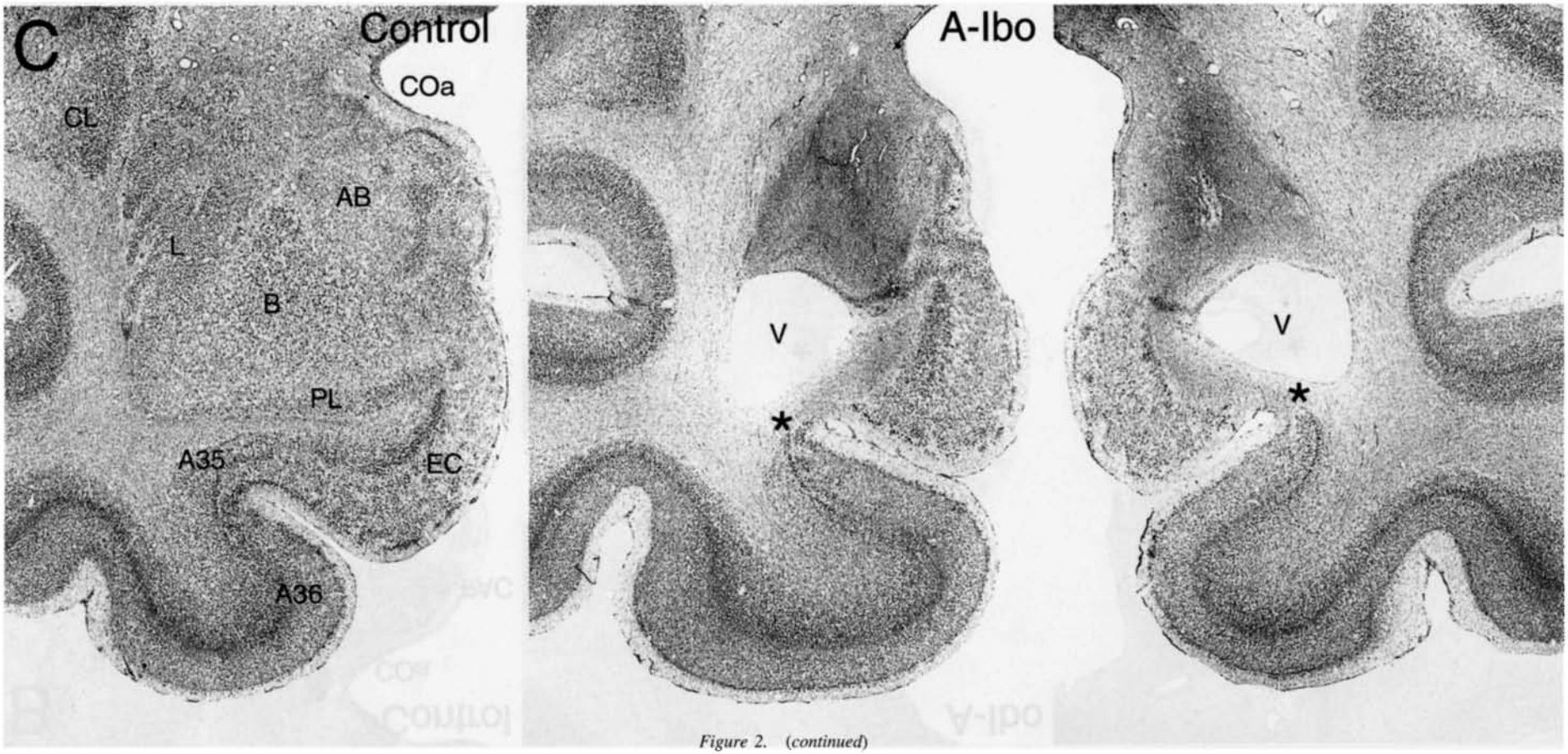

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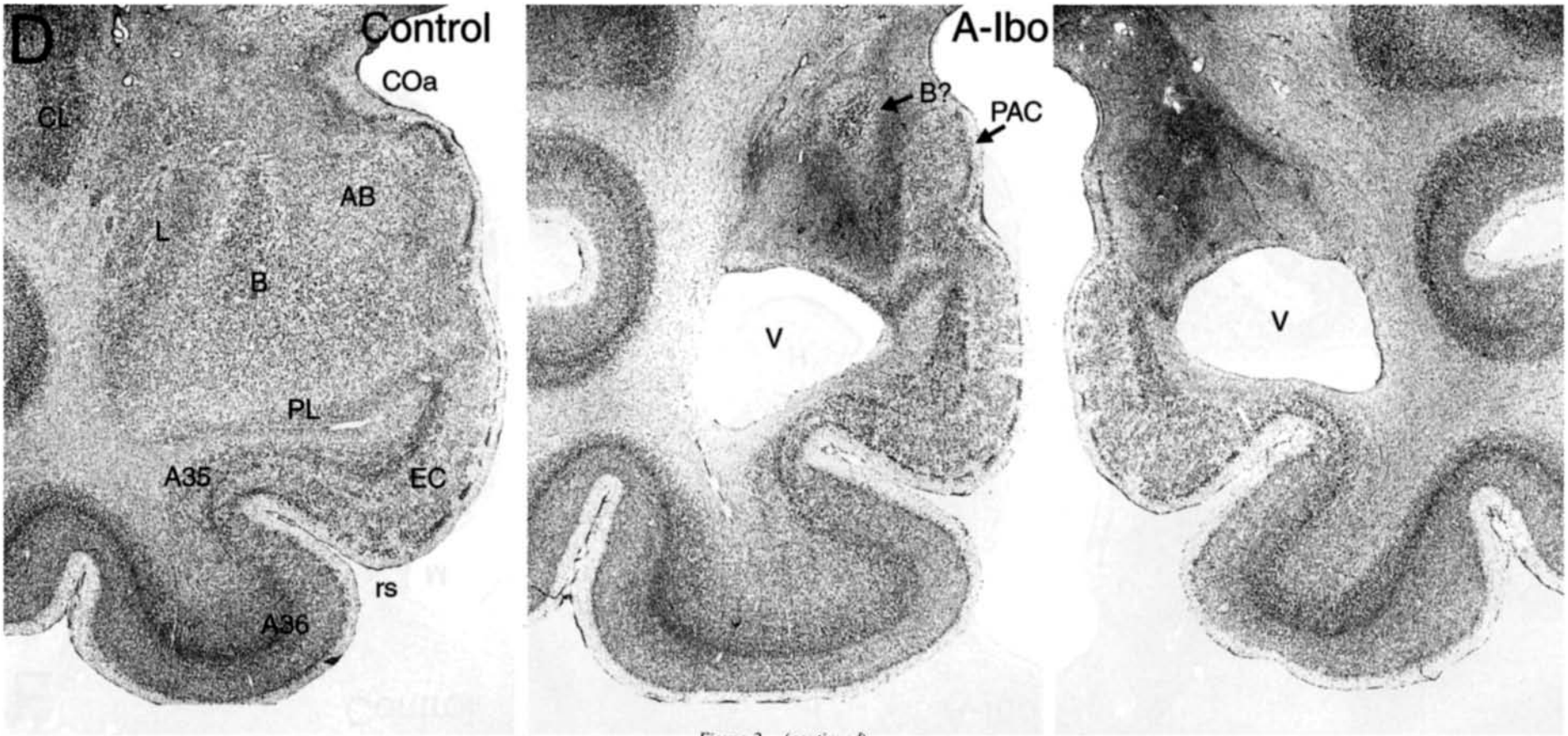

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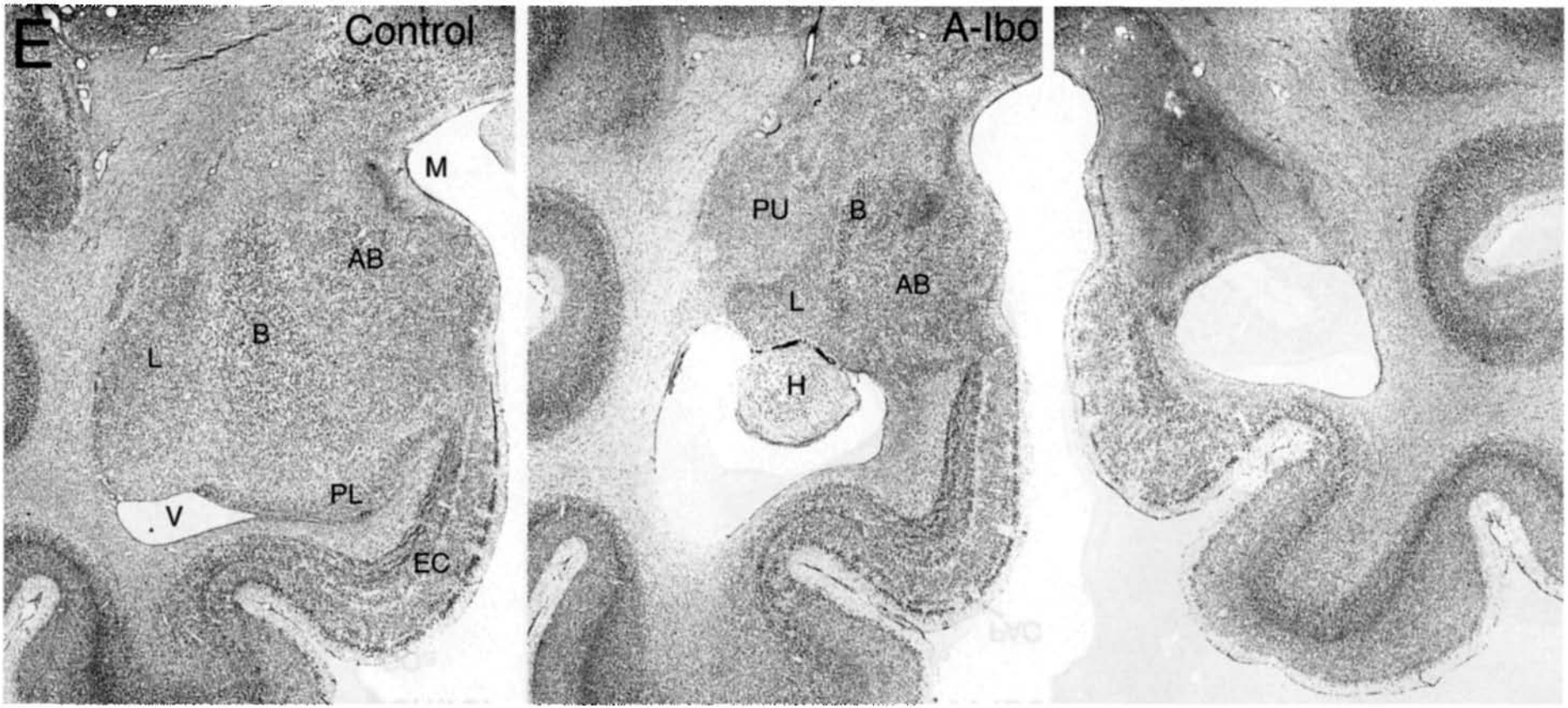

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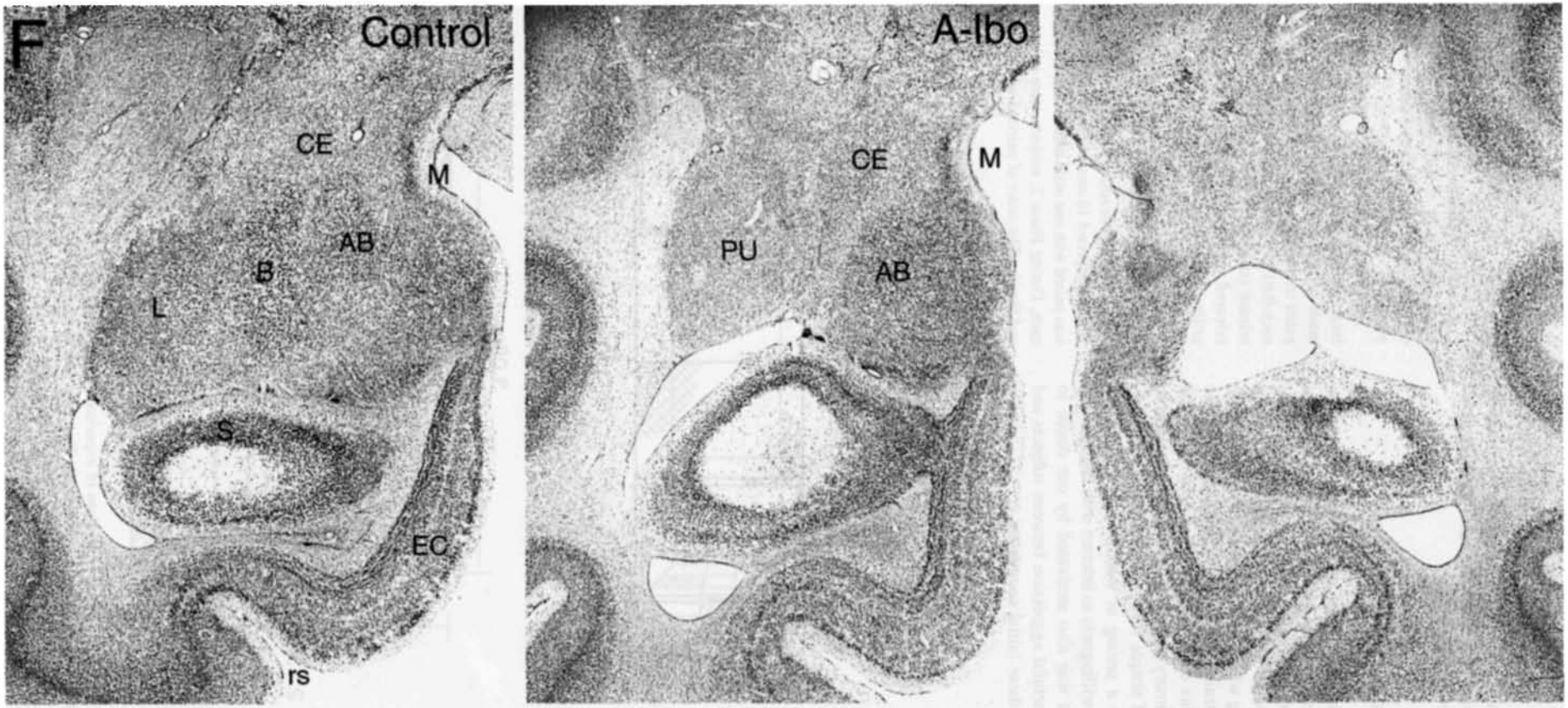

Figure 2. (*continued*)

the amygdala. Both areas appeared normal caudal (or rostral in the case of area 35) to the amygdala. Other damaged areas included the piriform cortex and ventral claustrum.

Experiment 1: Constrained Dyad

This experiment assessed the effect of amygdala lesions on dyadic interactions between amygdala-lesioned subjects or unoperated controls paired with unfamiliar male or female stimulus monkeys. Each dyadic session comprised two consecutive 10-min observation periods, during which either the subject monkey or the stimulus monkey was restrained in a small cage adjacent to the experimental cage (stimulus cage) and the other was free in the experimental cage. The primary purpose of this experiment was to assess the development of simple forms of social interaction between two individuals in a setting in which each was able to independently indicate its willingness to interact with the other. In addition, this arrangement was also motivated by our desire to reduce the likelihood of harmful aggression between subjects and stimulus monkeys during these initial encounters.

Method

Subjects. At the beginning of this experiment, A-IBO subjects were a mean age of 6.79 ± 0.42 years and control subjects were a mean age of 6.58 ± 0.30 years. Stimulus monkeys were a mean age of 5.75 ± 0.39 years.

Apparatus and materials. Behavioral testing was performed in a large outdoor enclosure (5.56 m long × 1.91 m wide × 2.13 m high) made of pipe and chain-link fencing, with a cement floor and a corrugated iron roof (see Figure 3). The floor was marked into 27 quadrates, each 0.61 m × 0.64 m. At either end of the test cage was an aluminum release (stimulus) cage with an opaque door and metal grille, which released the monkey into the larger enclosure. Both the door and grille could be raised separately by means of a remote pulley system. All subject and stimulus monkeys were trained to enter the release cage and to enter the test cage when the opaque door and metal grille were raised.

Design and procedure. Testing was conducted in two phases, each of which comprised 12 sessions. In Phase 1, every subject (A-IBO or control) was paired with one male and one female stimulus monkey for six sessions each. During Phase 2, subjects were paired with the other stimulus male and female. The order of pairings was counterbalanced, and every session

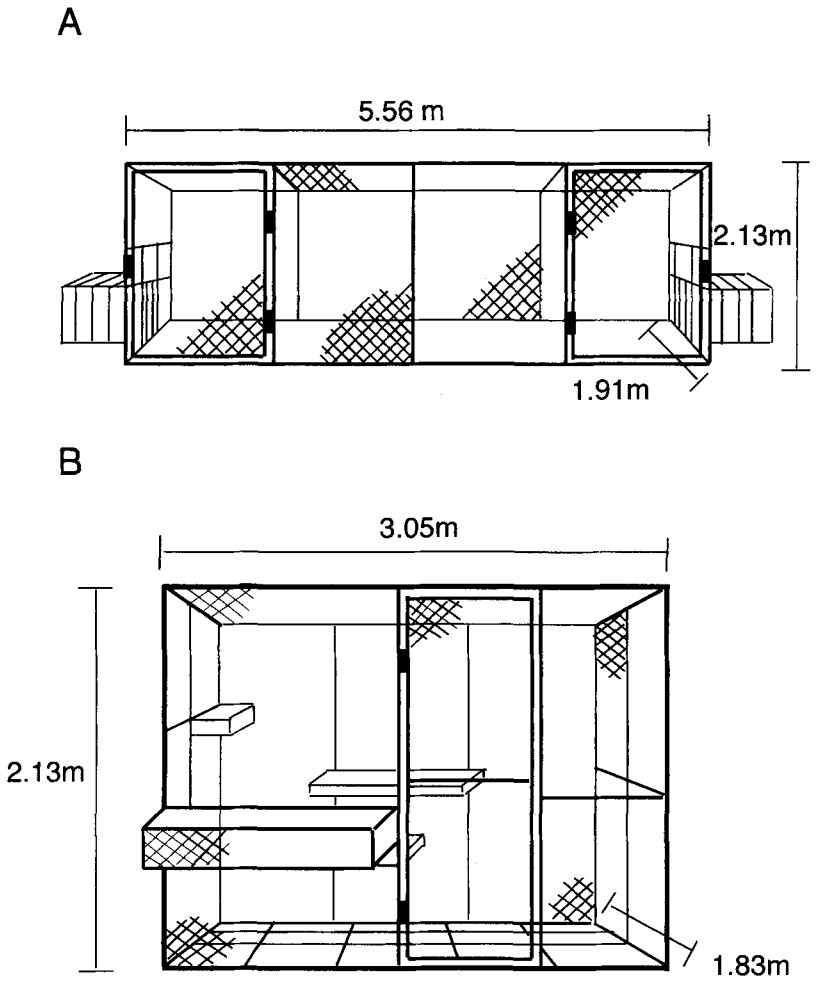

Figure 3. Schematic representations of experimental test cages. A: Test cage used for Experiments 1 and 2 (constrained and unconstrained dyads). B: Test cage used for Experiment 3 (round robin dyad).

comprised two 10-min trials. For one trial, the subject remained in the stimulus cage (i.e., was constrained), and the stimulus monkey was allowed to freely move about the large test cage (i.e., was unconstrained or free). For the other 10-min trial, conducted immediately after the first, the conditions were reversed. For the first session with a new stimulus monkey, the stimulus monkey was always constrained on the first trial. For subsequent sessions, identity of the constrained monkey was balanced. On all trials, the 2 monkeys were able to interact through the metal grille situated between the release and test cages. Two outdoor test enclosures were used, and each subject was always tested in the same test enclosure. Each subject was released from the same stimulus cage on all sessions, and the order of the sex of the stimulus monkey was pseudorandomized. Subjects experienced one session per day.

Each trial began with the opaque door and metal grille being raised for the unconstrained monkey, simultaneous with the raising of the opaque door only for the constrained monkey. Once the unconstrained monkey was released, behavioral data collection began. Social and nonsocial behaviors were recorded continuously for the entire 10-min trial. In addition, the spatial location of the unconstrained monkey was also recorded every 15 s by noting the location of the monkey's head. Position was recorded in three dimensions, defined by the quadrate on the floor plus notation of whether the monkey was on or off the floor (i.e., on the wall or ceiling). These data were used to calculate the mean distance (in meters) between the 2 monkeys during the session. At the conclusion of the trial, the unconstrained monkey was allowed to enter the release cage, opaque doors were lowered for both monkeys, and the observers rated the experimental subjects' overall attitude, as described above after the second trial. The monkeys were removed from the test apparatus, which was cleaned and prepared for the next pair of monkeys.

Statistical analysis. Behavioral data (frequency and duration) were arranged by period and sex of the stimulus monkey. Periods were designated as the first two sessions with a stimulus monkey (male or female; Period 1), the third and fourth sessions (Period 2), and the fifth and sixth sessions (Period 3). Frequency and duration of social and nonsocial behaviors were $\log_{10}(x + 1)$ transformed (as described earlier) and analyzed with three-way ANOVA, with group (A-IBO or control) as a between-subjects variable and sex (male or female stimulus monkeys) and period (1–3) as within-subjects variables.

Results

When the subjects were in the free condition and the stimulus monkeys were constrained, the A-IBO subjects displayed greater interest in the stimulus monkeys than did the controls. Compared with controls, A-IBO subjects had higher frequencies of being in the proximity zone (the quadrate located directly in front of the stimulus cage), $F(1, 10) = 5.22$, $p < .05$ (see Figure 4); higher frequencies of the affiliative vocalization coo, $F(1, 10) = 108.62$, $p < .0001$ (Figure 4); higher frequencies of walk by (locomotion to within proximity), especially in Period 1: Group × Period, $F(2, 10) = 5.37$, $p < .01$ (Figure 4); and a shorter interanimal distance with the stimulus monkeys, $F(1, 10) = 6.28$, $p < .05$ (see Figure 5). Although the A-IBO subjects displayed significantly lower frequencies and durations of nonsocial locomotion than did the controls, $F(1, 10) = 16.43$, $p < .0005$, and $F(1, 10) = 7.58$, $p < .01$, respectively, the shorter interanimal distance did not appear to be due to the fact that the A-IBO monkeys simply remained inactive in close proximity to the release cage. In fact, there were no significant differences in the mean number of quadrates visited, $F(1, 10) = 0.56$, $p = .47$, nor were there group differences in the closest distance to which 1 monkey (either A-IBO or controls) would approach a stimulus monkey during a trial, $F(1, 10) = 0.03$, $p = .87$.

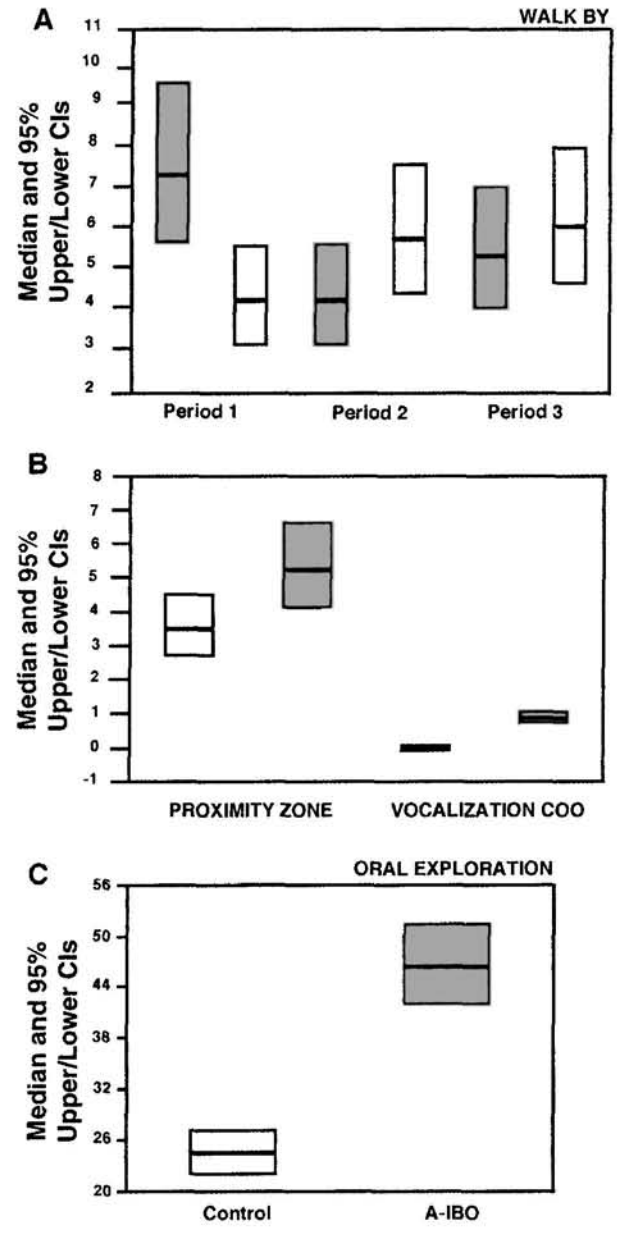

Figure 4. Median (± 95% confidence intervals [CIs]) frequencies of behaviors initiated during Experiment 1 (constrained dyad). A: Group × Period interaction for frequency of walk by. B: Group effects for frequency of proximity zone and vocalization coo. C: Group effect for frequency of oral exploration. For all post hoc comparisons, a Bonferroni test ($p < .005$) was performed. Ibotenic acid lesioned (A-IBO, shaded bars), $n = 6$; control (open bars), $n = 6$.

In general, the A-IBO subjects appeared less tense than did the controls when in the free condition. This was indicated by the A-IBO subjects' lower frequencies of tooth grind, $F(1, 10) = 4.84$, $p < .05$; cage aggress, $F(1, 10) = 8.44$, $p < .01$; and yawn, $F(1, 10) = 9.07$, $p < .01$; and by significantly lower ratings on the adjective anxious, $F(1, 10) = 17.44$, $p < .01$; and higher ratings of

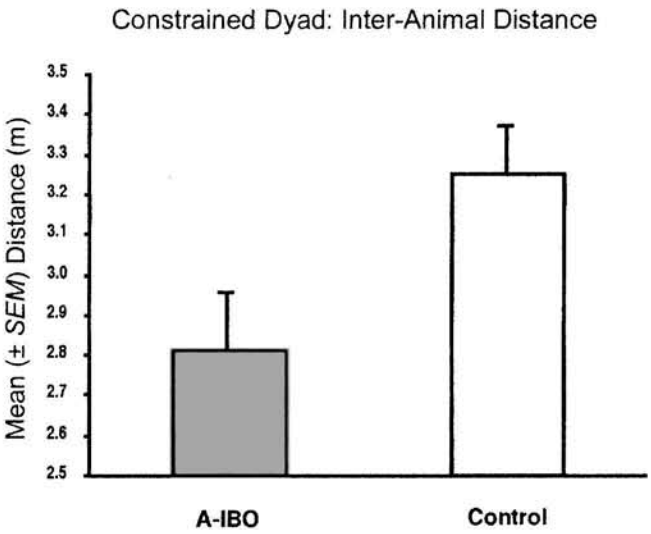

Figure 5. Spatial proximity. Mean (± *SEM*) distance between subject and stimulus monkeys during the 20-min test period for constrained dyad (subjects free and stimulus monkeys constrained). Ibotenic acid lesioned (A-IBO), $n = 6$; control, $n = 6$.

confidence, $F(1, 10) = 6.09$, $p < .05$. The A-IBO subjects also displayed less self-groom, $F(1, 10) = 8.39$, $p < .01$; more scratch, $F(1, 10) = 34.82$, $p < .0001$; and substantially more oral exploration of the environment, $F(1, 10) = 76.84$, $p < .0001$ (Figure 4), compared with controls.

When the subjects were in the constrained condition and the stimulus monkeys were unconstrained, only one group difference was found: A-IBO subjects received more present sex than did controls, $F(1, 10) = 4.61$, $p < .05$ (see Figure 6). In this condition, the female stimulus monkeys were located closer to the stimulus cage than were the male stimulus monkeys, $F(1, 10) = 60.29$, $p < .05$. They did not discriminate between the A-IBO monkeys and the control monkeys, however. The female stimulus monkeys also displayed a shorter distance from the A-IBO and control monkeys than did the male stimulus monkeys, $F(1, 10) = 21.55$, $p < .05$, but again did not differentiate between lesioned and control subjects.

Discussion

Although the potential for physical social interaction was very limited, there were nonetheless some clear differences between the A-IBO and control subjects in the social behaviors they displayed. Compared with controls, the A-IBO subjects sat next to the stimulus monkeys more frequently, displayed higher frequencies of affiliative coo vocalizations, and walked by the stimulus monkeys more frequently (though only during the first period). The increased frequency of these three behaviors indicates that the A-IBO subjects retained some of their social skills and were actively participating in social interaction (to the extent possible in this constrained situation). In addition, behaviors suggesting tension (tooth grinding, cage aggression, yawning) were less frequent among A-IBO subjects, as indicated by both the analysis of behavior categories and the ratings made by the observers.

The stimulus monkeys also surprisingly altered some aspects of their social behavior on the basis of the lesion status of the subject they were paired with, suggesting a greater attraction to the lesioned subjects. The stimulus monkeys, when free in the large cage, made more sex presentations toward the A-IBO subjects than toward the control subjects. Moreover, contrary to our expectation, there were almost no examples of aggressive behavior directed toward the A-IBO subjects.

Finally, we found that the A-IBO subjects engaged in more oral exploration of the cage and cage contents (twigs, leaves, insects, etc.) than did the control subjects. This hyperorality (compared with unlesioned monkeys) is a classic symptom of the Kluver–Bucy syndrome (Kluver & Bucy, 1939) first seen in rhesus monkeys with large lesions of the entire anterior portion of the temporal lobe. Kluver's monkeys excessively mouthed the cage, the experimenters, and any objects found within their cage, such as metal bolts or toys. Hyperorality has commonly been described after lesions of the amygdala (and subnuclei of the amygdala) and/or disconnection of inputs to the amygdala (Aggleton & Passingham, 1981; Horel, Keating, & Misantone, 1975). Meunier, Bachevalier, Murray, Malkova, and Mishkin (1999) also reported hyperorality after ibotenic acid lesions of the amygdala. The differences between the two experimental groups in the social behaviors directed toward the stimulus monkeys indicated that the amygdala lesions had altered social behavior. However, contrary to previous reports, the alterations led to a subtle increase in positive social behavior. We explored this result further in Experiment 2 by removing any barriers to physical interaction and allowing the subjects to interact with the stimulus monkeys in the large social enclosure.

Experiment 2: Unconstrained Dyad

In this experiment the effect of amygdala lesions on dyadic interactions between amygdala-lesioned monkeys or unoperated controls and male and female stimulus monkeys was assessed during 20-min observational periods, when both the subjects and stimulus monkeys were able to interact freely in the experimental cage.

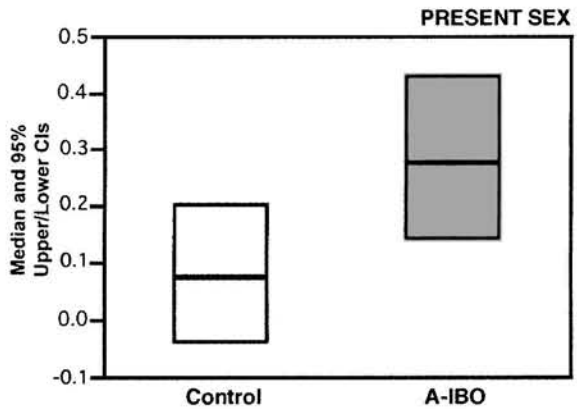

Figure 6. Median (± 95% confidence intervals [CIs]) frequencies of sex solicitations received during Experiment 1 (constrained dyad). For all post hoc comparisons, a Bonferroni test ($p < .005$) was performed. Ibotenic acid lesioned (A-IBO), $n = 6$; control, $n = 6$.

Method

Subjects. At the beginning of this experiment, the A-IBO subjects were a mean age of 7.07 ± 0.43 years, and the control subjects were a mean age of 6.83 ± 0.30 years. The 4 stimulus monkeys were a mean age of 6.04 ± 0.39 years.

Apparatus and materials. Behavioral testing was performed in the same outdoor enclosure as was used for Experiment 1.

Design and procedure. In each of six sessions, every experimental monkey (A-IBO and control) was paired once with every stimulus monkey for a 20-min test trial. Thus every subject experienced a total of 24 trials. Subjects were assigned to the same test cage and release cage as in Experiment 1, and this assignment was constant across trials. Gender order of the stimulus monkeys was pseudorandomized. Each subject experienced no more than one trial per day.

For each trial, the subject and stimulus monkeys were placed into their respective release cages, and on a signal the opaque doors were raised simultaneously, followed immediately by the metal grilles. Once the monkeys entered the test cage, the doors were lowered and latched. Behavioral data were collected with The Observer for the entire 20-min trial, with spatial location recorded (as described above) at 15-s intervals during the first, middle, and last 5 min of the 20-min trial. At the conclusion of each trial, the monkeys returned to their release cages and attitude assessments were completed. The monkeys were then returned to their cages, and the release and test cages were cleaned and prepared for the next trial.

Statistical analysis. Frequency and duration data was again $\log_{10}(x + 1)$ transformed and analyzed in a way similar to that used in Experiment 1: a three-way ANOVA for group (A-IBO vs. control), sex (male or female stimulus monkeys), and period (Sessions 1–2, 3–4, and 5–6), with subject nested within group as a random factor.

Results

The A-IBO subjects displayed a substantially greater frequency and duration of positive social behavior directed toward the stimulus monkeys than did the controls. This was reflected in significantly higher frequencies for the A-IBO subjects for the behaviors mount, $F(1, 10) = 8.86$, $p < .005$; incomplete mount, $F(1, 10) = 8.19$, $p < .01$ (see Figure 7); present sex, $F(1, 10) = 20.83$, $p < .0001$; proximity, $F(1, 10) = 13.07$, $p < .001$ (Figure 7); present groom, $F(1, 10) = 13.92$, $p < .0005$ (Figure 7); walk by, $F(1, 10) = 28.02$, $p < .0001$ (Figure 7); and vocalization coo, $F(1, 10) = 18.69$, $p < .0001$ (Figure 7); and for both frequencies and durations of the behaviors contact: frequency, $F(1, 10) = 18.17$, $p < .0001$ (Figure 7); duration, $F(1, 10) = 10.48$, $p < .01$; and extended social behavior: frequency, $F(1, 10) = 4.40$, $p < .05$; duration, $F(1, 10) = 7.53$, $p < .005$. Mean interanimal distance was smaller between the A-IBO subjects and the stimulus monkeys than between the control subjects and the stimulus monkeys, $F(1, 10) = 8.68$, $p < .05$ (see Figure 8). Group differences in sexual behavior were most apparent during the early part of the experiment, as demonstrated by significant Group × Period interactions, with follow-up analysis revealing group differences only during Period 1. This was evident for the behaviors mount, $F(2, 10) = 3.12$, $p = .05$ (Figure 7); extended social (which mostly comprised extended mounts), $F(2, 10) = 3.61$, $p < .05$ (Figure 7); and present sex, $F(2, 10) = 3.28$, $p < .05$ (Figure 7).

Controls displayed higher frequencies of behaviors that suggested tension. Although there were very few occurrences of chase, controls did show a higher frequency than did A-IBO subjects, $F(1, 10) = 4.18$, $p < .05$. Compared with the A-IBO subjects, controls also displayed higher frequencies of yawn, $F(1, 10) = 6.86$, $p < .05$; and motor stereotypies, $F(1, 10) = 8.87$, $p < .005$; and were rated as more nervous, $F(1, 10) = 24.44$, $p < .001$; avoidant, $F(1, 10) = 5.61$, $p < .05$; and less confident, $F(1, 10) = 5.12$, $p < .05$. There were no significant group differences in the frequencies of aggression, threat, displace, fear grimace, or lipsmack ($ps > .20$).

Group differences were found for many nonsocial behaviors as well. A-IBO subjects displayed more frequent oral exploration, $F(1, 10) = 45.64$, $p < .0001$; and tactile exploration, $F(1, 10) = 12.11$, $p < .001$; as well as higher frequencies of self-sex, $F(1, 10) = 6.34$, $p < .05$ (Figure 7). Controls showed higher frequencies of self-groom, $F(1, 10) = 15.62$, $p < .0005$. A-IBO subjects also tended to be more active than controls. When the subjects were not in proximity or contact with the stimulus monkeys, controls displayed higher durations of stationary behavior, $F(1, 10) = 8.52$, $p < .01$. A-IBO subjects, in contrast, had higher durations of locomotion, $F(1, 10) = 61.67$, $p < .0001$, particularly during Period 2: Group × Period, $F(2, 10) = 20.21$, $p < .0001$.

There were some differences between controls and A-IBO subjects on the basis of the sex of the stimulus monkeys, as indicated by significant Group × Sex interactions. Control monkeys directed more lipsmacks toward females than did A-IBO subjects, but there was no group difference in lipsmacks directed toward male stimulus monkeys, $F(1, 1) = 18.26$, $p < .0001$ (Figure 7). A-IBO subjects made more sex presents toward male stimulus monkeys than did controls; there was no difference in sex presents directed toward females, $F(1, 1) = 7.15$, $p < .01$ (Figure 7).

Stimulus monkeys displayed more affiliation toward A-IBO subjects than toward controls, as indicated by higher frequencies of groom, $F(1, 10) = 20.07$, $p < .0005$; walk by, $F(1, 10) = 23.91$, $p < .0001$; proximity, $F(1, 10) = 51.34$, $p < .0001$ (see Figure 9); contact, $F(1, 10) = 22.95$, $p < .0001$ (Figure 9); and sex present, $F(1, 10) = 40.77$, $p < .0001$ (Figure 9); as well as longer durations of proximity, $F(1, 10) = 14.19$, $p < .0005$; contact, $F(1, 10) = 6.71$, $p < .05$; groom, $F(1, 10) = 14.22$, $p < .0005$; and extended social, $F(1, 10) = 7.01$, $p < .05$. Significant interactions were found for two behaviors: The A-IBO subjects received more groom from the stimulus monkeys during Period 3 but not during Periods 1 and 2: Group × Period interaction for frequency, $F(2, 10) = 4.60$, $p < .05$ (Figure 9), and A-IBO subjects received more walk bys than controls during Period 1 but not during Periods 2 or 3: Group × Period, $F(2, 10) = 4.21$, $p < .05$ (Figure 9). Stimulus monkeys also displayed higher frequencies of tooth grind and tactile exploration when paired with A-IBO subjects than when paired with controls: group, $F(1, 10) = 26.03$, $p < .0001$, $F(1, 10) = 5.41$, $p < .05$, respectively.

Male and female stimulus monkeys showed evidence of differentiation of lesion condition, as demonstrated by significant Group × Sex interactions. The A-IBO subjects received a greater frequency of proximity, $F(1, 1) = 28.60$, $p < .0001$ (Figure 9); contact, $F(1, 1) = 12.11$, $p < .005$ (Figure 9); and present sex, $F(1, 1) = 7.92$, $p < .05$ (Figure 9), by the female stimulus monkeys than did the controls. Male stimulus monkeys displayed a greater frequency of walk by, $F(1, 1) = 48.75$, $p < .0001$ (Figure 9), and self-sex, $F(1, 1) = 8.05$, $p < .05$ (Figure 8), when paired with the A-IBO subjects than when paired with the controls.

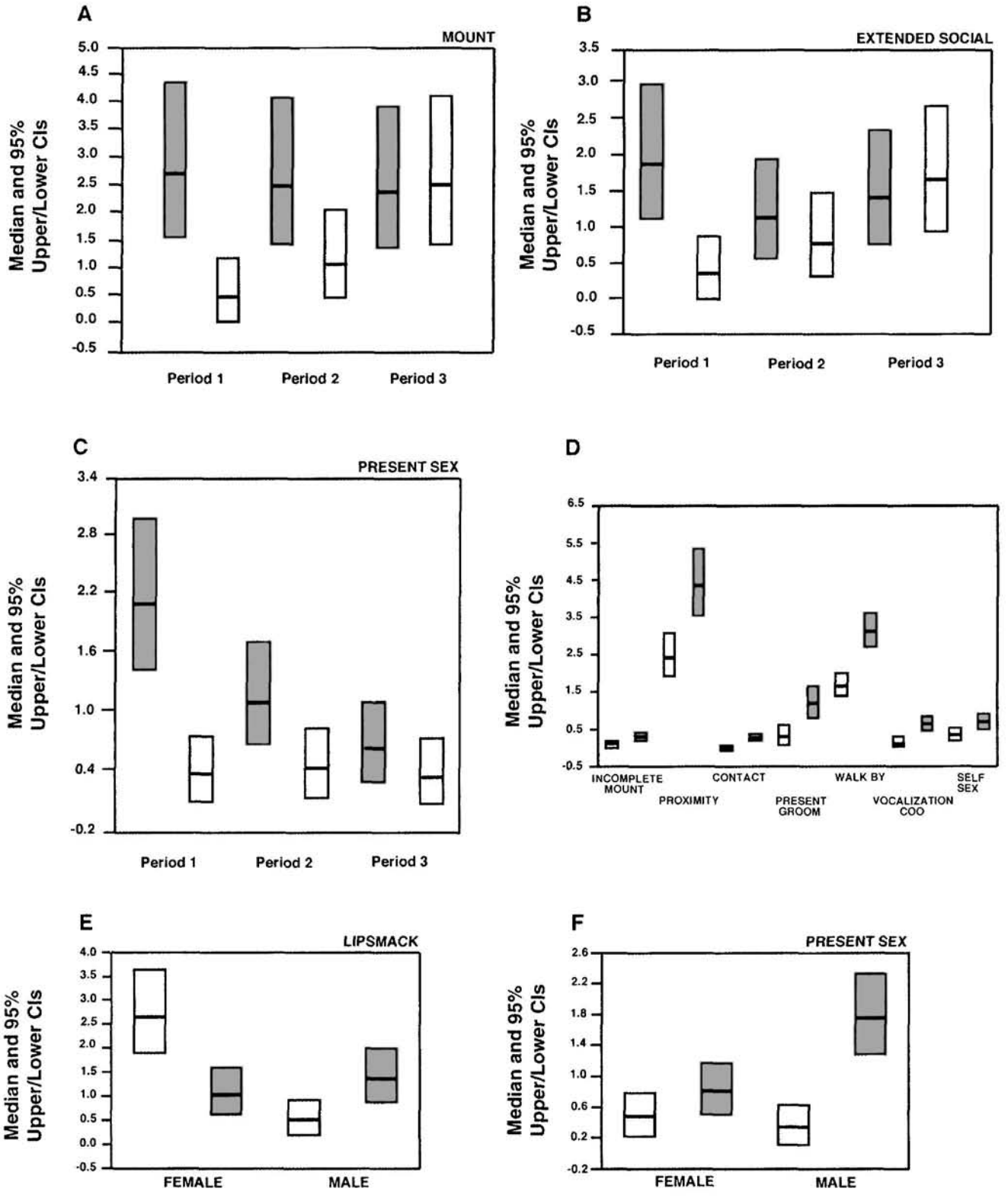

Figure 7. Median (± 95% confidence intervals [CIs]) frequencies of behaviors initiated during Experiment 2 (unconstrained dyad). A: Group × Period interaction for frequency of mount. B: Group × Period interaction for frequency of extended social behavior. C: Group × Period interaction for frequency of present sex. D: Group effects for frequency of incomplete mount, proximity, contact, present groom, walk by, vocalization coo, and self-sex. E: Group × Sex interaction for frequency of lipsmack. F: Group × Sex interaction for frequency of present sex. For all post hoc comparisons, a Bonferroni test ($p < .005$) was performed. Ibotenic acid lesioned (A-IBO, shaded bars), $n = 6$; control (open bars), $n = 6$.

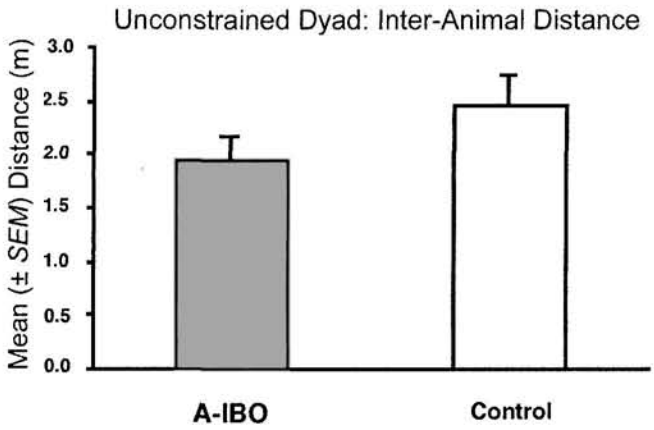

Figure 8. Spatial proximity. Mean (± *SEM*) distance between subject and stimulus monkeys during the 20-min test period for unconstrained dyad (both subjects and stimulus monkeys free in the test cage). Ibotenic acid lesioned (A-IBO), $n = 6$; control, $n = 6$.

Discussion

The A-IBO and control subjects displayed very distinct differences in their social interactions with the stimulus monkeys. In general, the A-IBO subjects could be characterized as more affiliative, as demonstrated by their higher frequencies of many positive social behaviors and the lack of group differences in most agonistic behaviors. A surprising result from this experiment was the finding that the stimulus monkeys responded very differently to the A-IBO subjects than to the controls. We discuss these results below.

Greater affiliation by A-IBO subjects. A common response pattern when unfamiliar rhesus monkeys (particularly adult males) are formed into a group is initial wariness often accompanied by aggression (contact and noncontact, including threats and chases) that lasts for a relatively brief period of time under many circumstances (although see Mendoza, 1993). The wariness continues, but sociosexual behavior (presenting and mounting) replaces agonism, until ultimately affiliation (e.g., grooming) is displayed (e.g., Bernstein, Gordon, & Rose, 1974; Bernstein & Mason, 1963). In Experiment 2, control subjects displayed more evidence of wariness than did the A-IBO subjects, as reflected in their greater interanimal distances from the stimulus monkeys, higher frequencies of yawn, and ratings by the observers as more avoidant and nervous. Levels of all forms of aggression were low, and, with the exception of chase, group differences were not evident. This was not surprising, as the subjects were familiar with the stimulus monkeys from the earlier participation in Experiment 1. In fact, a common strategy for introducing new animals into an existing social troop is to place the animal in an enclosure adjacent to the social enclosure (Reinhardt, 1989). Apparently, the opportunity to see and have constrained social interactions leads to a level of familiarity that precludes overt aggressive interactions. Thus, because of the low level of aggression displayed by the subjects, we are unable to state whether amygdala lesions cause disruptions in the processing and production of aggressive behavior.

A considerable amount of sexual behavior was seen in Experiment 2, largely performed by the A-IBO subjects. The frequencies of mounting, extended mounting, and mount solicitations (present sex) in the A-IBO subjects were significantly higher during Period 1, when the subjects experienced their first trials with each pair of stimulus monkeys (Figure 7). This result, combined with those just described, suggests that the behavior of the A-IBO subjects did not conform to the usual pattern seen during initial encounters with (relatively) unfamiliar monkeys; the initial wariness seemed absent (or greatly reduced). Presumably, this early period of inhibited interaction affords normal monkeys the opportunity to evaluate the potential threat or propensity for affiliation of the new social partner. The inability of amygdala-lesioned subjects to inhibit the natural tendency to interact with novel social stimuli, which we term *social disinhibition*, has not been reported previously for monkeys with amygdala lesions. In fact, early studies of the amygdala and monkey social behavior suggested that lesions of the amygdala cause dramatic reductions in positive social behavior (see review by Kling & Brothers, 1992), resulting in social isolation and heightened aggression from other group members. Kling (1968) studied the effects of amygdala lesions in juvenile rhesus monkeys in dyads and described increases in positive social behavior similar to those reported here. We subjected Kling's data to statistical analysis (Emery, 2000, not performed in Kling's original paper) and found that the differences between amygdala-lesioned and normal monkeys were only clear for mounts. Our data replicate and extend these earlier findings using modern neuroscientific and behavioral techniques.

Hypersexuality is one of the core symptoms of the Kluver–Bucy syndrome, and we did see higher frequencies of sexual behavior (including self-sex) in our lesioned monkeys. Unlike the monkeys described by Kluver and Bucy (1939) and cats described by Schreiner and Kling (1953). The A-IBO subjects in this study did not mount inappropriate objects (although the possibility for this was admittedly limited). Although the A-IBO subjects may have been expected to display inappropriate mounting of objects or excessive masturbation during the constrained dyad study (Experiment 1), which was more comparable to the testing environment of Kluver and Bucy's early studies, no differences between the groups were found in any category of sexual behavior. We suggest that lesions of the amygdala do not cause hypersexuality but do profoundly influence male (and probably female) sexual behavior. Mounting, thrusting, sexual presentations, and inspection of the genitalia of a member of the opposite sex often occur in other social contexts aside from sex (Dixson, 1998). Although such behaviors can occur in concert with dominance relations, they primarily serve an affiliative or tension-reducing function (Dixson, 1983). Such sociosexual behavior, in comparison with primary sexual behavior, may not be influenced by testosterone as it is in heterosexual behavior (Hanby, 1978). Castrated talapoin monkeys (an Old World monkey), for example, continue to mount and present to other males, but there are very few instances in which they attempt to mount with females (Dixson & Herbert, 1977). It is worth noting that the amygdala contains the second highest accumulation of testosterone receptors in the primate brain after the medial preoptic area (Rees & Michael, 1982), and it has also been suggested that the medial nucleus of the amygdala (which projects to the medial preoptic area; Amaral et al., 1992) and potentially the basal nucleus (McGregor & Herbert, 1992) are important components of the neural circuit that controls male sexual behavior (Dixson, 1998). The amygdala may therefore be a

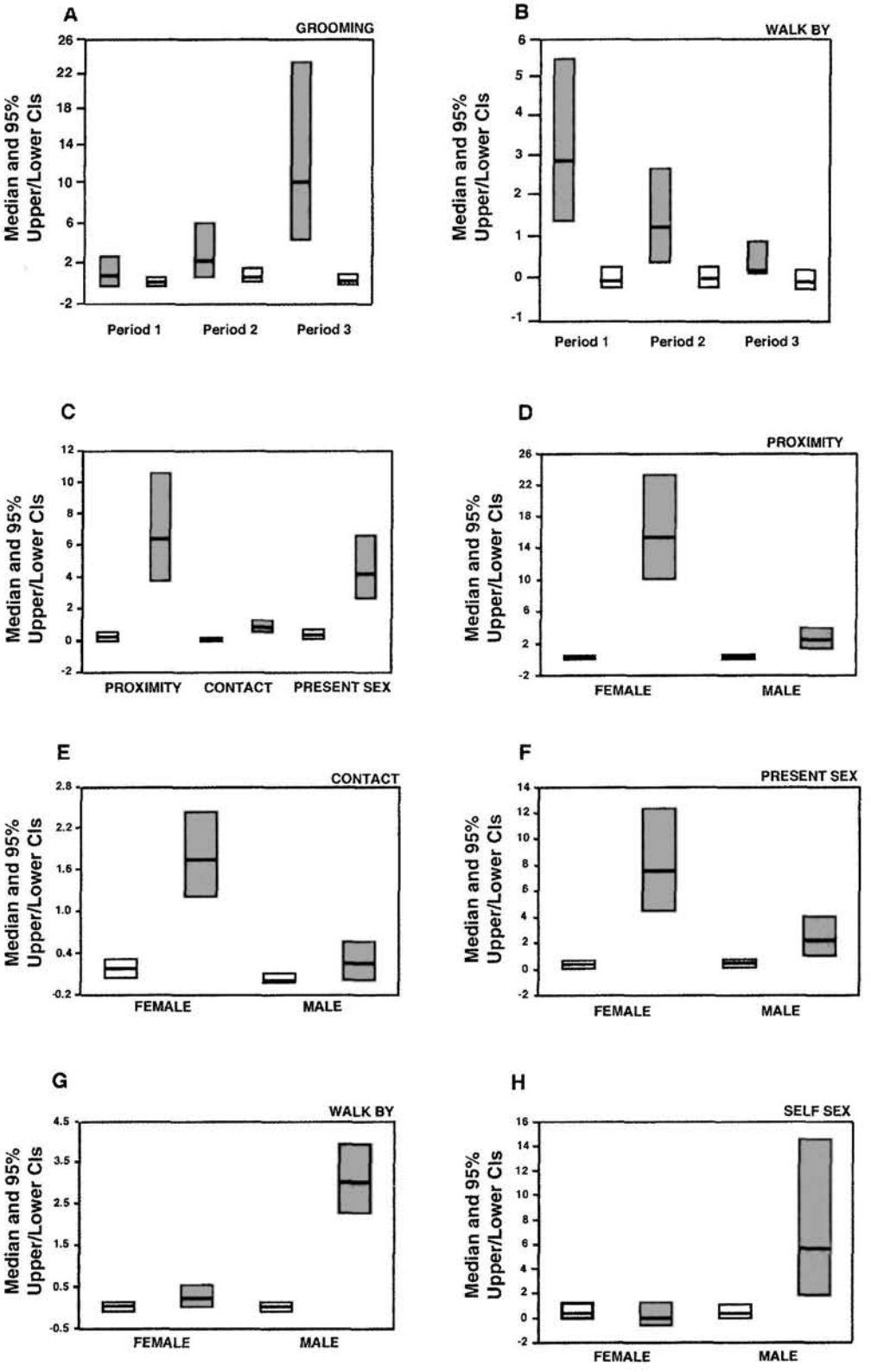

Figure 9. Median (± 95% confidence intervals [CIs]) frequencies of behaviors received during Experiment 2 (unconstrained dyad). A: Group × Period interactions for frequency of grooming. B: Group × Period interactions for frequency of walk by. C: Group effects for proximity, contact, and present sex. D: Group × Sex interactions for proximity. E: Group × Sex interactions for contact. F: Group × Sex interactions for present sex. G: Group × Sex interactions for walk by. H: Group × Sex interactions for self-sex. Ibotenic acid lesioned (A-IBO, shaded bars), $n = 6$; control (open bars), $n = 6$.

potential target by which sociosexual stimuli are modulated by certain neuroendocrine states, and therefore influences the expression of male sexual behavior (Rees & Michael, 1982). The amygdala lesions in the present subjects may have increased the frequency of sociosexual behavior, as the A-IBO were no longer constrained by the influence of testosterone, as would be the case for the initiation of heterosexual behaviors. This explanation is clearly speculative at this point, however.

Greater attractiveness of the A-IBO subjects. Perhaps the most surprising finding from this study was the reaction of the stimulus monkeys toward monkeys in the two subject groups. The A-IBO subjects received higher levels of proximity, contact, grooming, sex present, and walk by from the stimulus monkeys than did the controls. The high level of proximity, contact, and groom received by the A-IBO monkeys was apparent from the beginning of the unconstrained social interactions, although the greatest difference in grooming received occurred during Period 3, suggesting that the stimulus monkeys required some previous experience with the A-IBO subjects before determining that they were appropriate to groom. It is not known whether this decision was based on the high level of sexual and/or positive social behavior displayed by the amygdala-lesioned subjects during early interactions (and sustained over the testing sessions). There is some evidence that sexual behavior (including mounting) functions to establish, and then maintain, affiliative relationships in adult monkeys (Smuts, 1987; Wallen & Tannenbaum, 1999) and vice versa (Smuts, 1985). This may be especially important for adult male monkeys entering a new troop. This usually occurs during the breeding season, when sexual consortships with females may facilitate establishment of dominance within the male hierarchy (Wallen & Tannenbaum, 1999).

Consistent with the results from Experiment 1, data generated in the unconstrained dyad study are compatible with the interpretation that the A-IBO subjects were perceived by the stimulus monkeys as more socially attractive than the control subjects. It is reasonable to speculate that this resulted from the early heightened positive (especially sexual) behavior initiated by the A-IBO subjects toward the stimulus monkeys. This ability of the stimulus monkeys to differentiate between the two groups must have occurred very rapidly, as the frequency of some behaviors, such as walk by, were higher toward the A-IBO subjects during Period 1. These findings prompted us to determine whether normal monkeys could differentiate amygdala-lesioned monkeys during a single 20-min social interaction. We were also interested in determining whether the A-IBO subjects could also discriminate between other A-IBO subjects and normal control subjects. We evaluated these issues in Experiment 3.

Experiment 3: Round Robin Dyad

This experiment assessed the effects of amygdala lesions on the dyadic interactions of amygdala-lesioned and control subjects in a round robin design. Every subject (6 A-IBO and 6 controls) experienced a single 20-min interaction with every other subject. It is important to emphasize that these monkeys had never previously met (nor had they seen each other in their home cages) before this first social encounter.

Method

Subjects. At the beginning of this experiment, the A-IBO subjects were a mean age of 7.71 ± 0.42 years, and the control subjects were a mean age of 7.49 ± 0.30 years.

Apparatus and materials. Testing occurred in a smaller indoor test cage (3.05 m long × 1.83 m wide × 2.13 m high) made from pipe and chain-link fencing, with a mesh roof and floor (Figure 3). The cage contained three perches made of PVC-coated galvanized pipe, at different locations (at the front of the cage, 0.91 m in length and 0.43 m from the floor; at the back of the cage, 1.5–2.0 m in length and 0.81 m from the floor; and on either the right or left wall, 1.31 m in length and 1.09 m from the floor; Figure 3). The floor of the cage was marked into 15 equally sized quadrates.

Design and procedure. All experimental subjects were paired with all other subjects (A-IBO and control) once, in a round robin design. Thus, each subject had five sessions with subjects from the same condition (A-IBO or control) and six sessions with subjects from the opposite condition. Two pairs were tested per day, and no subject was tested on consecutive days. Order of pairings was pseudorandomized and balanced insofar as possible.

Members of each dyad were released into the test cage in quick succession via a chute attached to the front of the cage. If a monkey did not enter the test cage within 5 s, a technician provided assistance. Once both monkeys were in the test cage, behavioral data were collected with The Observer for the entire 20-min session, with spatial location recorded at 15-s intervals during the first, middle, and last 5 min of the 20-min session. At the conclusion of each session, the monkeys were returned to their transport cages and attitude assessments were completed. The monkeys were then returned to their living cages, and the test cage was cleaned and prepared for the next session.

Statistical analysis. Statistical tests similar to those used in Experiments 1 and 2 were used in this experiment. For each behavior category, every subject had two values, one reflecting the mean for sessions when the subject was paired with controls, and the second reflecting the mean for sessions when the subject was paired with A-IBO subjects. The data were transformed with a $\log_{10}(x + 1)$ transformation, and effects of group and partner were analyzed with a two-way mixed model ANOVA with subject nested within group as a random (blocking) factor. Again, post hoc comparisons were made with the Bonferroni correction (10 planned comparisons, $p < .005$). Fisher's least significant difference post hoc test was used to test for significant differences in mean interanimal distance between the four conditions (A-IBO with A-IBO, A-IBO with control, control with A-IBO, and control with control).

Results

The A-IBO subjects initiated and received greater frequencies and durations of positive social behaviors compared with controls, regardless of whether they were paired with another A-IBO monkey or a control monkey. This was reflected in a greater number of mounts, $F(1, 10) = 6.25$, $p < .05$, and present groom, $F(1, 10) = 9.14$, $p < .05$ (see Figure 10), initiated by the A-IBO subjects; by higher frequencies of present groom, $F(1, 10) = 17.13$, $p < .005$, and extended social behaviors received, $F(1, 10) = 6.36$, $p < .05$ (see Figure 11); and by a greater duration of extended social behavior received, $F(1, 10) = 12.85$, $p < .005$. Although there were no group differences in the frequency or duration of proximity, A-IBO subjects exhibited higher frequencies of withdraw, $F(1, 10) = 24.73$, $p < .001$ (Figure 10).

Although there were few instances of aggression during the 20-min interactions, the control subjects displayed higher frequencies of aggression, $F(1, 10) = 18.24$, $p < .005$ (Figure 10) and

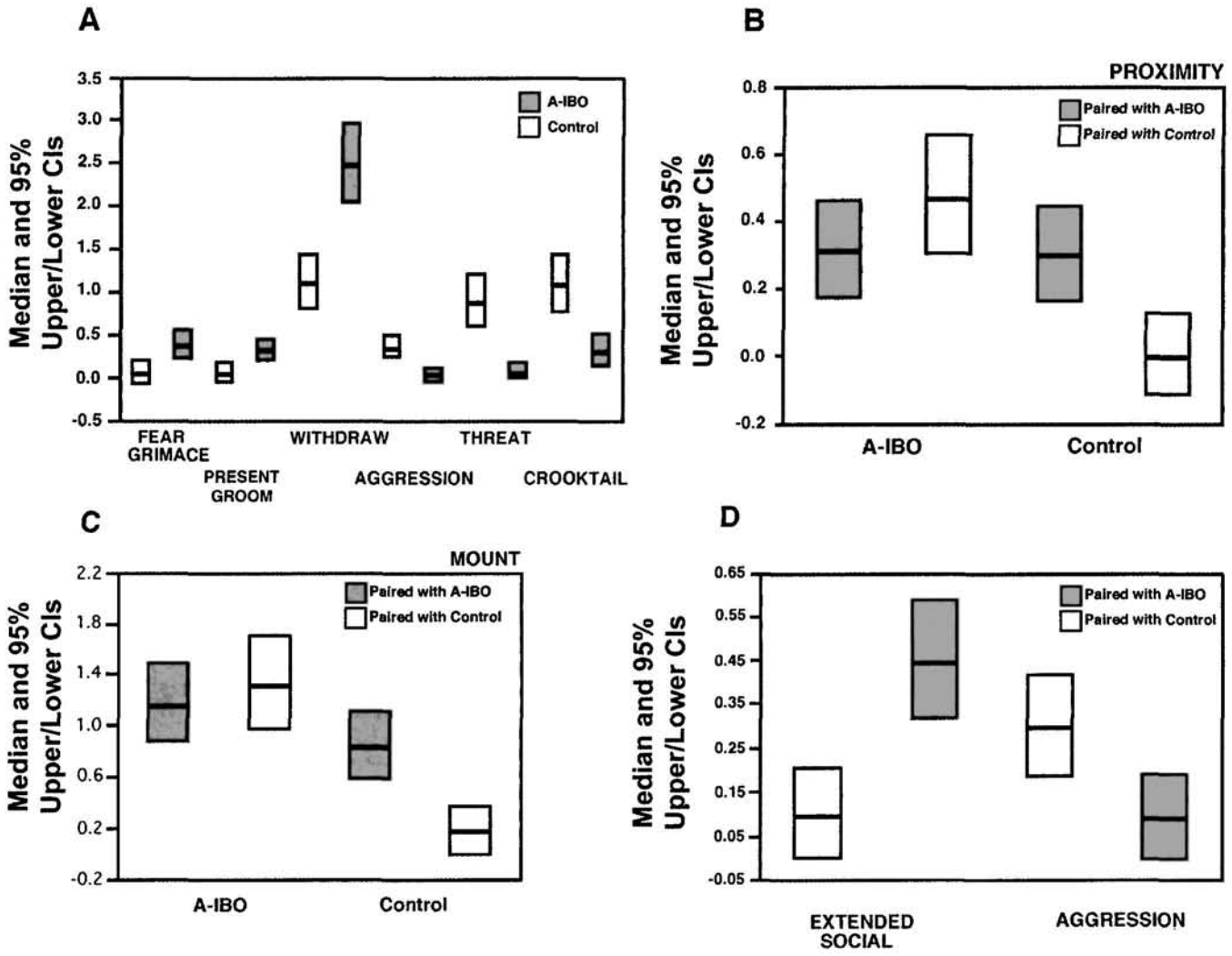

Figure 10. Median (± 95% confidence intervals [CIs]) frequencies of behaviors initiated during Experiment 3 (round robin dyad) between homogeneous (ibotenic acid lesioned [A-IBO] with A-IBO or control with control) and heterogeneous (A-IBO with control) pairs. A: Group differences between A-IBO and control subjects for frequency of fear grimaces, present groom, withdraw, aggression, threat, and crooktails. B: Group × Partner interactions for frequency of proximity. C: Group × Partner interactions for frequency of mount. D: Partner effects for frequency of extended social behavior and aggression. A-IBO, $n = 6$; control, $n = 6$. All experimental monkeys paired with each group.

threat, $F(1, 10) = 29.67$, $p < .0005$ (Figure 10), than did the A-IBO subjects. The control subjects also displayed more crooktail locomotion than did the A-IBO subjects, $F(1, 10) = 17.05$, $p < .005$ (Figure 10). The A-IBO subjects, in contrast, displayed a higher frequency of fear grimaces, $F(1, 10) = 9.09$, $p < .05$ (Figure 10), and they received more chases, $F(1, 10) = 6.04$, $p < .05$ (Figure 11).

In addition to being more affiliative, A-IBO subjects appeared more attractive and less threatening as partners, as indicated by significant main effects for partner. When paired with A-IBO subjects (in contrast to when paired with controls) both A-IBO and control subjects displayed a greater frequency of mount, $F(1, 1) = 30.28$, $p < .005$; proximity, $F(1, 10) = 11.47$, $p < .01$; and extended social behavior, $F(1, 10) = 16.86$, $p < .01$ (Figure 10); and a longer duration of extended social behavior, $F(1, 10) = 16.12$, $p < .005$. Also, when paired with A-IBO subjects, subjects (both A-IBO and controls) received fewer threats, $F(1, 10) = 7.52$, $p < .05$ (Figure 11), and aggression, $F(1, 10) = 7.52$, $p < .05$ (Figure 10), and were rated as more confident: partner, $F(1, 10) = 11.67$, $p < .01$; more affiliative: partner, $F(1, 10) = 19.80$, $p < .001$; and less avoidant: partner, $F(1, 10) = 10.64$, $p < .01$.

The affiliation and attractiveness of the A-IBO subjects were more apparent when the identities of partners were considered. For example, A-IBO subjects mounted controls and controls mounted A-IBO subjects significantly more than controls mounted controls: Group × Partner interaction, $F(1, 1) = 11.55$, $p < .01$ (Figure 10). Controls also displayed a greater frequency and longer duration of

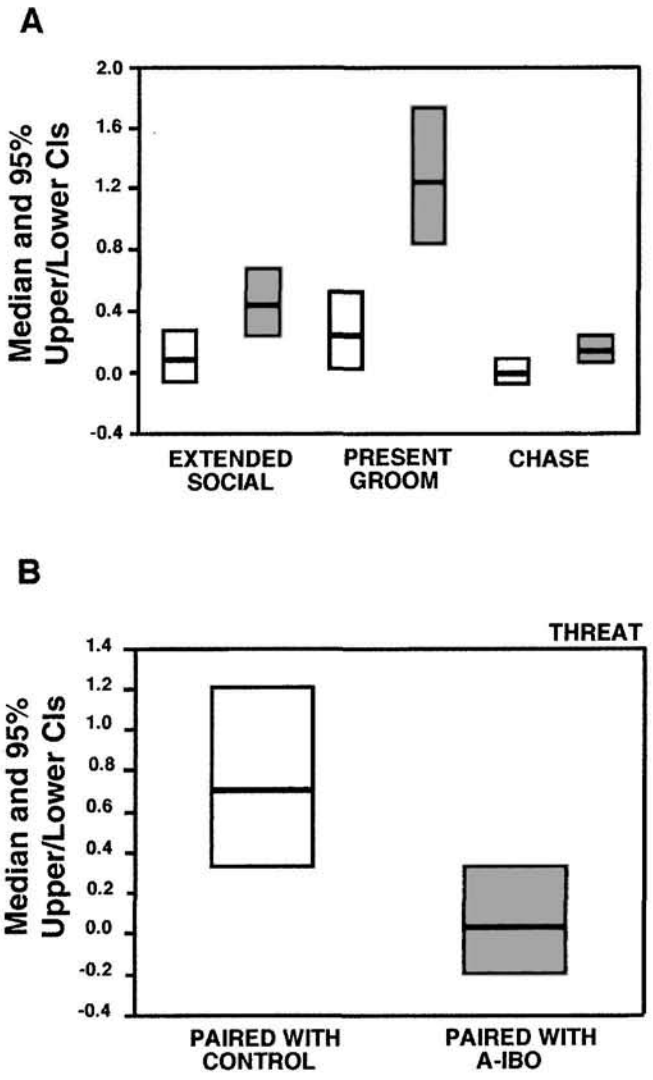

Figure 11. Median (± 95% confidence intervals [CIs]) frequencies of behaviors received during Experiment 3 (round robin dyad) between homogeneous (ibotenic acid-lesioned [A-IBO, shaded bars] with A-IBO or control [open bars] with control) and heterogeneous (A-IBO with control) pairs. A: Group differences between A-IBO and control monkeys for frequency of extended social behavior, present groom, and chase received. B: Partner effects for frequency of threat received. A-IBO, $n = 6$; control, $n = 6$.

proximity: Group × Partner interaction, frequency, $F(1, 1) = 10.42$, $p < .05$; duration, $F(1, 1) = 10.73$, $p < .01$ (Figure 10), and a shorter interanimal distance, $F(1, 1) = 5.04$, $p < .05$ (see Figure 12), with A-IBO subjects than with other control subjects. Finally, A-IBO subjects were rated as more affiliative when paired with control subjects than were control subjects paired with other control subject: Group × Partner, $F(1, 1) = 22.06$, $p < .001$.

Several group differences were noted for nonsocial behaviors. Control subjects displayed more cage aggression, $F(1, 10) = 5.24$, $p < .05$; self-grooming: group, $F(1, 10) = 13.25$, $p < .01$; and motor stereotypies: group, $F(1, 10) = 7.39$, $p < .05$; and less scratching, $F(1, 10) = 13.27$, $p < .01$, than did the A-IBO subjects.

Discussion

Experiment 3 provided the first opportunity for the A-IBO and control subjects to interact together. The main goal of this study was to determine whether the control and A-IBO subjects would differentiate between the A-IBO subjects and unlesioned controls within a single 20-min social session. The results of this study suggest that the control subjects did differentiate the A-IBO subjects from the control subjects, whereas the A-IBO subjects were not as good at the same differentiation. The control subjects, for example, were more often in proximity, generated more mounts, and had smaller interanimal distances when paired with the A-IBO subjects than when paired with other controls, whereas the A-IBO subjects did not differentiate clearly between A-IBO and control partners.

The potential for aggression was increased in Experiment 3 compared with the previous experiment, probably because of the unfamiliarity of the monkeys with each other, and perhaps because of the smaller size of the cage. Indeed, the controls displayed a higher number of threats, more crooktail dominance displays, and initiated more aggression than did the A-IBO subjects. The A-IBO subjects, however, received a greater number of chases than did the control subjects. These data suggest that the increased opportunities for aggression increased the behavioral differences between the A-IBO subjects (who displayed no aggression) and the controls (who displayed higher levels of aggression). This may have enhanced the ability of the control subjects to differentiate monkeys within the two groups.

The patterns of behavior described here are very similar to those described for the unconstrained dyad. The A-IBO subjects displayed higher levels of positive social behavior, which was recip-

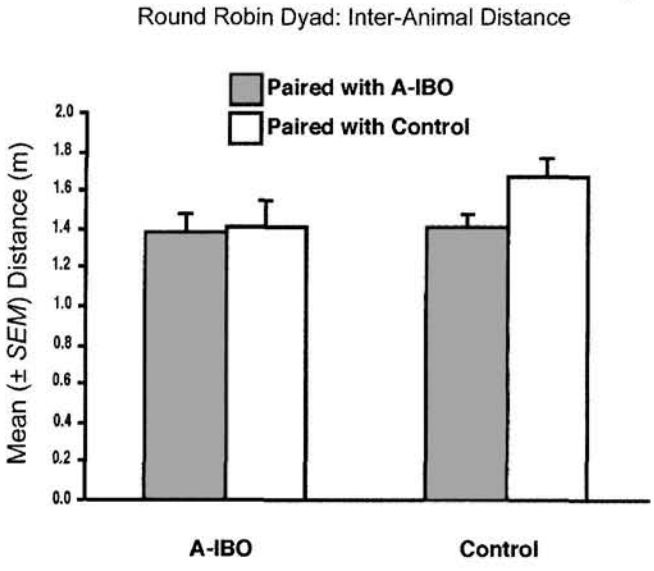

Figure 12. Spatial proximity. Mean (± *SEM*) distance between ibotenic acid-lesioned (A-IBO) and control monkeys during the 20-min test period for round robin dyad. Post hoc comparisons were made with Fisher's least significant difference test ($p < .05$). A-IBO, $n = 6$; control, $n = 6$.

rocated. The only aggression displayed was by the control subjects, and it was displayed predominantly when the partners were other control subjects. As with the previous two experiments, the controls also displayed a higher frequency of tension-related behaviors than did the A-IBO subjects. The similar results between the two experiments occurred despite many differences: Subjects in Experiment 3 had a shorter time for interaction, the monkeys were unfamiliar with each other, the test cage was smaller, and the partners were much more variable (i.e., comprised both normal and lesioned monkeys).

We believe these data suggest that the A-IBO subjects are impaired in their ability to discriminate between other A-IBO subjects and controls. Their increased social behavior (social disinhibition), then, may be unrelated to their ability to discriminate between individuals with or without amygdala lesions. Perhaps the control subjects were responding to the increased positive social behavior displayed by the A-IBO subjects as the primary indicator of their lesion status, compared with the relatively antisocial behavior displayed by the controls. Although the social behavior of the A-IBO subjects was unusual for adult male rhesus monkeys paired with other unfamiliar adult males, their behavior did not appear detrimental; although they did receive a greater frequency of chases, the occurrence of this behavior (and all other agonistic behaviors) was very low.

General Discussion

Although the potential for social interaction was different in all three dyad experiments, similar patterns of behavior were apparent when the A-IBO and control subjects were compared. In all three experiments, the A-IBO subjects displayed more positive social behaviors and lower levels of aggressive behavior compared with unlesioned subjects. This was most evident during the unconstrained dyad, in which the amygdalectomized monkeys displayed what we refer to as *social disinhibition*, an increased tendency toward uninhibited positive interaction with a novel social partner. This occurred at a time when normal monkeys usually show cautious assessment of the social intentions of an unfamiliar monkey.

One of the most surprising findings from these studies was that the behavioral reactions of the subjects and stimulus monkeys differed depending on the lesion status of their partner. The reaction of normal monkeys to lesioned monkeys has rarely been examined (although see Deets, Harlow, Singh, & Blomquist, 1970 for an important exception). It is unclear from our studies which cues the monkeys were using to judge that the amygdala-lesioned monkeys were different from the unlesioned monkeys, although it is likely that the increase in positive social behavior by the A-IBO subjects might have contributed to this assessment. Data from Experiment 3 also suggest that this assessment occurs very rapidly, within the first 20 min of the initial encounter.

These studies suggest that monkeys with amygdala lesions do not become isolated and socially withdrawn but appear to contribute positively to social interactions, at least in the simple social configuration of the dyad. We recognize that the conditions under which testing occurs can influence behavioral outcomes, however. Kling and colleagues have previously suggested that differences in the environment in which their subjects were tested (single-caged, small group in small cage, small group in large cage, seminatural group in artificial island setting, and group in natural habitat) led to differences in the effects of amygdala lesions on social behavior (Kling, 1972; Kling & Mass, 1974). Kling (1968), for example, found that juvenile monkeys with amygdala lesions displayed similar increases in mounts and groom/mount solicitations as displayed by the amygdalectomized monkeys in the present study (however, see earlier discussion of this article). These behaviors were not displayed in larger social groups (Dicks et al., 1969; Kling & Cornell, 1971). Hyperorality was also very apparent in the dyads (Kling, 1968) but was very rare or absent in the larger groups (Kling & Mass). Although there was considerable consistency in our results across test situations (constrained, unconstrained), cages, and social partners (novel or familiar), it remains to be seen whether similar results will be found in more complex social situations.

The Amygdala and Normal Social Behavior

What is the role of the amygdala in governing the social behavior of intact adult male rhesus monkeys? During normal social interaction, a *social rule system* controls the behavioral actions and reactions of social beings, including monkeys, apes, and humans. In rhesus monkeys, this rule system involves the status (e.g., dominance relations) of the interactants and the social environment in which they encounter each other. A single dyad within a constrained environment (such as a cage), for example, limits the potential for social interaction, as compared with a free-ranging environment that permits virtually unlimited interactions with different individuals in different social contexts. Under normal conditions, a subordinate animal might respond to a threat from a larger, higher-ranked animal with submissive gestures, such as fear grimaces, perhaps accompanied by lipsmacks (Redican, 1975), mount solicitations, and fleeing. An inappropriate response by the subordinate animal, such as a threat, would likely elicit aggression from the dominant animal. It has previously been suggested that different nuclei of the amygdala are important for the perception, evaluation, and initiation of appropriate behavioral and physiological responses to social (and nonsocial) stimuli (Emery & Amaral, 2000). Therefore, removal of different parts of the amygdala might disrupt different components of the social rule system.

On the basis of the data presented in this article, we propose that one role of the amygdala is to function as a "brake" on behaviors, both social and nonsocial, that are dependent on the processing and evaluation of biologically important stimuli. The amygdalectomized monkeys produced increased social behavior (including heightened sexual behavior), whereas the normal, unlesioned monkeys were (appropriately) apprehensive in initiating social interaction and more frequently displayed indicators of anxiety or tension. This suggests to us that the amygdala in the intact animal inhibits it from interacting positively or negatively with the individual it is paired with, until an appropriate evaluation of the social partner has been made and appropriate social responses can be initiated. This evaluation is dependent on sensory information about the social partner and the social context and past history of encounters with this particular individual, or individuals with similar characteristics. Removal of the amygdala removes this brake, and information about social context is lost. Therefore, the amygdalectomized animal responds impulsively, that is, before appropriate evaluation.

Is there any evidence from the human neuropsychological literature that the amygdala is essential for interpreting biologically important stimuli, such as faces within a specific social context? Adolphs et al. (1998) recently asked 3 patients with bilateral amygdala lesions to rate a collection of unfamiliar faces on two potentially important social attributes, approachability and trustworthiness. These faces had previously been rated by a number of control participants and had been separated into high-rated and low-rated on both measures. The patients with amygdala damage tended to rate all faces as being more trustworthy and approachable than did the control participants. The amygdala-damaged patients also tended to rate positively the faces that the control participants had rated as being most untrustworthy and unapproachable. This suggests that the human amygdala may be required for perceiving specific attributes of the face, such as facial expressions (Adolphs, Tranel, Damasio, & Damasio, 1994; Young, Hellawell, Van de Wal, & Johnson, 1996), and for attaching and retrieving the social meaning of facial stimuli. These data may also be explained by the "amygdala as a brake" hypothesis, as the natural tendency of humans with an intact amygdala is to perceive some faces as trustworthy and approachable and others as not. The amygdala-lesioned individuals treat all faces as approachable and trustworthy because the natural brake on responding inappropriately is missing. (For examples in other mammalian species in which the amygdala may be described as a brake, see Fleming, Vaccarino, & Luebke, 1980, and Numan, Numan, & English, 1993, for maternal behavior in rats, and Karli, Vergnes, Eclancher, & Penot, 1977, for mouse-killing in rats.)

Differences From Earlier Studies

There were a number of methodological innovations used in the present research program that may explain the differences found between these results and those from earlier published studies. First, we used adult males of approximately the same age as experimental subjects. Kling (1974) has suggested that there are important sex differences in the effects of amygdala lesions in monkeys. Some females with amygdala lesions become hyperaggressive, in contrast to males, which uniformly display hypoaggression after amygdala lesions. Sex differences in gonadal steroids may be important for this sex difference. Kling (1968), for example, found that juvenile rhesus monkey males with amygdala lesions displayed some of the increases in social behavior described in this study (mounting, grooming, and sexually receptive behavior), and injections of testosterone tended to increase the frequency of these behaviors.

Second, in the present study, a prelesion behavioral evaluation of all experimental monkeys was performed to aid in choosing suitable monkeys for study. This method largely eliminated the possibility of using monkeys that displayed extremes (in either direction) in aggressive, sexual, or social behaviors that may have either skewed the results in predictable directions, or increased behavioral variability in our lesioned monkeys after surgery. A hyperaggressive male, for example, may have displayed more dramatic alterations in its behavior after an amygdala lesion than the mid-ranking submissive males used in the present study.

A third difference between the present study and past studies involves experimental design. Previous studies compared the pre- and postlesion effects within the same monkey from an established social group. The behavioral changes displayed after returning the lesioned monkey to its group may have been due to disruption of the established group social dynamics, rather than the effects of the amygdala lesions themselves. In the current study, we chose to compare lesioned subjects with unlesioned control subjects in the same social situations, and to assess group differences in experimental settings that involved brief encounters with unfamiliar monkeys (Experiment 1 and Experiment 3). All subjects (A-IBO and control) received the same number of exposures with the stimulus monkeys (constrained and unconstrained dyads) and to one another (round robin dyad). Normal social interactions are a series of encounters, not single events. Although the subjects experienced the stimulus monkeys in the constrained dyad, they could not physically interact with them, therefore the potential for habituation was reduced. However, habituation is not necessarily detrimental in a study that concerns the formation and maintenance of social relationships. It is precisely the differences in the evolution of social interactions (the rapidity of getting to know each other) between the A-IBO and control monkeys that proved most interesting in this series of experiments.

Our procedures for making the amygdala lesions also contrast with most of the previous research on the role of the amygdala in primate social behavior. Earlier studies involved either suction ablations (aspiration) or radio frequency lesions of the amygdala, which destroy cell bodies and fibers passing through from the temporal cortex to the frontal cortex via the amygdala (i.e., fibers of passage). The neurotoxin ibotenic acid, used to produce the lesions in this study, selectively destroys cell bodies but spares fibers of passage. The neurotoxin is injected in very small amounts, which increases the selectivity of the effect and reduces damage to cortical areas adjacent to the amygdala.

A number of studies have compared the effectiveness of aspiration versus neurotoxic lesions on tests of memory (e.g., Murray & Mishkin, 1984, 1998) and found very different results depending on the method used. The effects of amygdala lesions on other behaviors have also been shown to be more subtle than previously thought (Malkova, Gaffan, & Murray, 1997). The first published study (Meunier et al., 1999) to compare aspiration and neurotoxic lesions of the amygdala on emotional responsiveness demonstrated similar deficits in emotionality between the two methods. However, the effects of the neurotoxic lesions were more subtle than those of aspiration lesions. Meunier et al. suggested that the extra effects were probably due to the extraneous damage to surrounding cortical areas. The results of the present study similarly suggest that ibotenic acid lesions of the amygdala produce different effects on social behavior than do lesions made by the aspiration and radio frequency techniques, not just subtle versions of similar behavioral effects.

Other methodological differences between earlier studies and the present one reflect attempts at precise lesion placement and detailed analysis of the resulting lesions. In this study, the amygdala for each monkey was located before surgery by using MRI, which permitted the creation of a stereotaxic atlas for each subject, thus increasing the accuracy of the ibotenic acid injections. After the experiments, the extent of the amygdala lesions was verified histologically, and a quantitative analysis of the percentage of damage to the amygdala and other cortical and subcortical areas was used to determine the precise extent of the lesions and potential influences of extraneous damage on the social behavior defi-

cits. Again, this is the first study, to our knowledge, that provides a comprehensive analysis of the amygdala lesions in individual subjects, including quantitative analyses of the extent of the lesions of specific areas (lateral, basal, accessory basal, and central nuclei of the amygdala, and entorhinal cortex) and a detailed qualitative description of the extraneous damage to other subcortical and cortical areas surrounding the amygdala.

The behavioral inventory used in the current studies was comprehensive and included a variety of social and nonsocial behaviors. Additional indicators of behavioral expression, such as assessment of attitude and the measurement of relative spatial proximity (interanimal distance), also provided valuable insight into the differences between the two experimental groups. This level of behavioral detail has not been presented before in earlier studies of the amygdala and social behavior.

A Note on the Lesions in Our Amygdalectomized Subjects

We note that in several of the A-IBO subjects, the ibotenic acid failed to remove all of the central nucleus and some of the superficial regions such as the periamygdaloid cortex. Emery and Amaral (2000) have suggested that because complex social information (involving faces and communicative gestures) enters the lateral nucleus (see also Stefanacci & Amaral, 2000) and is passed on to the basal nuclei, lesions of these structures alone should be sufficient to severely affect the perception of social signals in amygdala-lesioned subjects. The pathways between the amygdala and orbitofrontal cortex would also be destroyed by this lesion, thereby eliminating the influence of the orbitofrontal cortex on the outputs of the amygdala to effector structures that produce behavioral, physiological, and hormonal reactions to social stimuli. Therefore, it may be the case that relatively complete lesions of the lateral, basal, and accessory basal nuclei, such as those produced in the present study, should have the same detrimental effects on social functions as do complete amygdala lesions (i.e., that include the central nucleus). Lesions of the central nucleus alone should not dramatically effect the perception and evaluation of social signals but are likely to disrupt the physiological, endocrine, and visceral reactions to these stimuli. Therefore, although the central nucleus demonstrated significant sparing , it is unlikely that a more complete central nucleus lesion would have significantly altered the present results.

There is some evidence in rats that there are different effects on socioemotional behavior after different amygdala lesions. Central nucleus lesions, for example, tend to affect fear-related behaviors, in particular, fear conditioning (Davis, 1992; Killcross, Robbins, & Everitt, 1997). Basolateral nuclei lesions tend to affect aggression (McGregor & Herbert, 1992) and medial nucleus lesions affect sexual behavior (Masco & Carrer, 1980; McGregor & Herbert, 1992). Aggleton and Passingham (1981) also reported differences in aspects of the Kluver–Bucy syndrome after subamygdala and total amygdala lesions in the monkey: The total amygdala lesions produced the greatest level of hypoemotionality, coprophagia, and increased tendency to approach objects. The subtotal lesions (lateral nucleus, basal nucleus, corticomedial nuclei, and white matter alongside the amygdala), in contrast, did not significantly produce the symptoms of the Kluver–Bucy syndrome.

In the present study, there was some damage sustained to other brain regions extraneous to the amygdala. The main regions damaged (Table 6) were the piriform cortex, entorhinal cortex, claustrum, area 35, fundus of the STS, and the striatum. In the literature, there are no clear relationships between these structures and social behavior in monkeys. Green, Clemente, and De Groot (1957) reported that piriform cortex lesions alone were sufficient to increase sexual activity in cats, although similar effects were not seen in dogs with combined amygdala and piriform cortex lesions (Fuller et al., 1957). The effects of piriform lesions on sociosexual behavior in monkeys is unknown. A few single neurons within the entorhinal cortex have been shown to respond to social stimuli (Brothers, Ring, & Kling, 1990). Neurons within the fundus of the STS also respond to facial stimuli (Perrett, Rolls, & Caan, 1982). However, when the latter are lesioned, face processing deficits are not apparent except for gaze discrimination. Given that the patterns of extraneous damage differed in the A-IBO monkeys and that similar behavioral alterations were observed in the monkeys that had little or no extra-amygdaloid damage, it does not appear likely that the extraneous damage contributed greatly to the changes in social behavior that we have reported.

Summary and Conclusion

The present study suggests that monkeys with lesions of the amygdala display greater affiliation, suggesting social disinhibition, and, perhaps as a consequence, are considered more attractive to normal, unlesioned monkeys. We propose that the amygdala may function as a brake on behavior, which would normally prevent the monkey from interacting with an unfamiliar animal or object until the animal or object had been thoroughly evaluated. Further research, involving other social situations and other assessments of responsiveness, is necessary to determine the validity of these ideas.

References

Adolphs, R., Tranel, D., & Damasio, A. R. (1998). The human amygdala in social judgment. *Nature, 393*, 470.

Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. R. (1994). Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature, 372*, 669–672.

Aggleton, J. P., & Passingham, R. E. (1981). Syndrome produced by lesions of the amygdala in monkeys (*Macaca mulatta*). *Journal of Comparative and Physiological Psychology, 95*, 961–977.

Alvarez-Royo, P., Clower, R. P., Zola-Morgan, S., & Squire, L. R. (1991). Stereotaxic lesions of the hippocampus in monkeys: Determination of surgical coordinates and analysis of lesions using magnetic resonance imaging. *Journal of Neuroscience Methods, 38*, 223–232.

Amaral, D. G., Price, J. L., Pitkanen, A., & Carmichael, S. T. (1992). Anatomical organization of the primate amygdaloid complex. In J. P. Aggleton (Ed.), *The amygdala: Neurobiological aspects of emotion, memory and mental dysfunction* (pp. 1–66). New York: Wiley-Liss.

Bachevalier, J. (1994). Medial temporal lobe structures and autism: A review of clinical and experimental findings. *Neuropsychologia, 32*, 627–648.

Bernstein, I. S., Gordon, T. P., & Rose, R. M. (1974). Aggression and social controls in rhesus monkey (*Macaca mulatta*) groups revealed in group formation studies. *Folia Primatologica, 21*, 81–107.

Bernstein, I. S., & Mason, W. A. (1963). Group formation by rhesus monkeys. *Animal Behaviour, 11*, 28–31.

Brothers, L., Ring, B., & Kling, A. (1990). Response of neurons in the macaque amygdala to complex social stimuli. *Behavioural Brain Research, 41*, 199–213.

Buckley, M. J., & Gaffan, D. (1998). Perirhinal cortex ablation impairs visual object identification. *Journal of Neuroscience, 18*, 2268–2275.

Capitanio, J. P. (1984). Early experience and social processes in rhesus macaques (*Macaca mulatta*): I. Dyadic social interaction. *Journal of Comparative Psychology, 98*, 35–44.

Capitanio, J. P. (1999). Personality dimensions in adult male rhesus macaques: Prediction of behaviors across time and situation. *American Journal of Primatology, 47*, 299–320.

Capitanio, J. P., Bond, J. C., & Mason, W. A. (1997). "A state of mind or feeling": Assessment of the attitudinal domain of social interaction. *American Journal of Primatology, 42*, 98–99.

Capitanio, J. P., Mendoza, S. P., Lerche, N. W., & Mason, W. A. (1998). Social stress results in altered glucocorticoid regulation and shorter survival in simian acquired immune deficiency syndrome. *Proceedings of the National Academy of Sciences, USA, 95*, 4714–4719.

Davis, M. (1992). The role of the amygdala in fear and anxiety. *Annual Review of Neuroscience, 15*, 353–375.

Deets, A. C., Harlow, H. F., Singh, S. D., & Blomquist, A. J. (1970). Effects of bilateral lesions of the frontal granular cortex on the social behaviour of rhesus monkeys. *Journal of Comparative and Physiological Psychology, 72*, 452–461.

Dicks, D., Myers, R. E., & Kling, A. (1969). Uncus and amygdala lesions: Effects on social behavior in the free ranging rhesus monkey. *Science, 165*, 69–71.

Dixson, A. F. (1983). The hormonal control of sexual behavior in primates. In C. A. Finn (Ed.), *Oxford reviews of reproductive biology* (Vol. 5, pp. 131–219). Oxford, England: Clarendon Press.

Dixson, A. F. (1998). *Primate sexuality: Comparative studies of the prosimians, monkeys, apes, and human beings.* Oxford, England: Oxford University Press.

Dixson, A. F., & Herbert, J. (1977). Gonadal hormones and sexual behavior in groups of adult talapoin monkeys (*Miopithecus talapoin*). *Hormones and Behavior, 8*, 141–154.

Emery, N. J. (2000). *Evaluating the contribution of the amygdala to primate social behaviour: A re-analysis of the data.* Unpublished manuscript.

Emery, N. J., & Amaral, D. G. (2000). The role of the primate amygdala in social cognition. In R. D. Lane & L. Nadel (Eds.), *Cognitive neuroscience of emotion. Series in affective science* (pp. 156–191). New York: Oxford University Press.

Fleming, A. S., Vaccarino, F., & Luebke, C. (1980). Amygdaloid inhibition of maternal behavior in the nulliparous female rat. *Physiology & Behavior, 25*, 731–743.

Franzen, E. A., & Myers, R. E. (1973). Neural control of social behavior: Prefrontal and anterior temporal cortex. *Neuropsychologia, 11*, 141–157.

Fuller, J. L., Rosvold, H. E., & Pribram, K. H. (1957). The effect on affective and cognitive behavior in the dog of lesions of the pyriform-amygdala-hippocampal complex. *Journal of Comparative and Physiological Psychology, 50*, 89–96.

Green, J. D., Clemente, C. D., & De Groot, J. (1957). Rhinencephalic lesions and behavior in cats. *Journal of Comparative Neurology, 108*, 505–545.

Hanby, J. P. (1978). Social factors affecting primate reproduction. In J. Money & H. Mustaph (Eds.) *Handbook of sexology: Vol. II. Genetics, hormones and behavior* (pp. 461–484). New York: Elsevier.

Horel, J. A., Keating, E. G., & Misantone, L. J. (1975). Partial Kluver-Bucy syndrome produced by destroying temporal neocortex or amygdala. *Brain Research, 94*, 347–359.

Howell, D. C. (1997). *Statistical methods for psychology* (4th ed.). Belmont, CA: Duxbury Press.

Jonason, K. R., & Enloe, L. J. (1971). Alterations in social behavior following septal and amygdaloid lesions in the rat. *Journal of Comparative and Physiological Psychology, 75*, 286–301.

Karli, P., Vergnes, M., Eclancher, F., & Penot, C. (1977). Involvement of amygdala in inhibitory control over aggression in the rat: A synopsis. *Aggressive Behavior, 3*, 157–162.

Killcross, S., Robbins, T. W., & Everitt, B. J. (1997, July 24). Different types of fear-conditioned behaviour mediated by separate nuclei within amygdala. *Nature, 388*, 377–380.

Kling, A. (1968). Effects of amygdalectomy and testosterone on sexual behavior of male juvenile macaques. *Journal of Comparative and Physiological Psychology, 65*, 466–471.

Kling, A. (1972). Effects of amygdalectomy on socio-affective behavior in non-human primates. In B. E. Eleftheriou (Ed.), *Neurobiology of the amygdala* (pp. 511–536). New York: Plenum Press.

Kling, A. (1974). Differential effects of amygdalectomy in male and female nonhuman primates. *Archives of Sexual Behavior, 3*, 129–134.

Kling, A., & Cornell, R. (1971). Amygdalectomy and social behaviour in the caged stump-tailed macaque. *Folia Primatologica, 14*, 91–103.

Kling, A., Dicks, D., & Gurowitz, E. M. (1969). Amygdalectomy and social behavior in a caged group of vervets (*C. aethiops*). *Proceedings of the Second International Congress of Primatology: Vol. 1. Behaviour* (pp. 232–241). New York: Karger.

Kling, A., & Dunne, K. (1976). Social-environmental factors affecting behavior and serum testosterone in normal and amygdala lesioned *M. speciosa. Primates, 17*, 23–42.

Kling, A., Lancaster, J., & Benitone, J. (1970). Amygdalectomy in the free-ranging vervet. *Journal of Psychiatric Research, 7*, 191–199.

Kling, A., & Mass, R. (1974). Alterations of social behavior with neural lesions in nonhuman primates. In R. L. Holloway (Ed.), *Primate aggression, territoriality, and xenophobia: A comparative perspective* (pp. 361–386). New York: Academic Press.

Kling, A. S., & Brothers, L. (1992). The amygdala and social behavior. In J. P. Aggleton (Ed.), *The amygdala: Neurobiological aspects of emotion, memory and mental dysfunction* (pp. 353–378). New York: Wiley-Liss.

Kluver, H., & Bucy, P. C. (1939). Preliminary analysis of functions of the temporal lobes in monkeys. *Archives of Neurology and Psychiatry, 42*, 979–1000.

Malkova, L., Gaffan, D., & Murray, E. A. (1997). Excitotoxic lesions of the amygdala fail to produce impairment in visual learning for auditory secondary reinforcement but interfere with reinforcer devaluation effects in rhesus monkeys. *Journal of Neuroscience, 17*, 6011–6020.

Masco, D. H., & Carrer, H. F. (1980). Sexual receptivity in female rats after lesion or stimulation in different amygdaloid nuclei. *Physiology & Behavior, 24*, 1073–1080.

McGregor, A., & Herbert, J. (1992). Differential effects of excitotoxic basolateral and corticomedial lesions of the amygdala on the behavioural and endocrine responses to either sexual or aggression-promoting stimuli in the male rat. *Brain Research, 574*, 9–20.

Mendoza, S. P. (1993). Social conflict on first encounters. In W. A. Mason & S. P. Mendoza (Eds.), *Primate social conflict* (pp. 85–110). Albany: State University of New York Press.

Meunier, M., Bachevalier, J., Murray, E. A., Malkova, L., & Mishkin, M. (1999). Effects of aspiration versus neurotoxic lesions of the amygdala on emotional responses in monkeys. *European Journal of Neuroscience, 11*, 4403–4418.

Murray, E. A., & Mishkin, M. (1984). Severe tactual as well as visual memory deficits follow combined removal of the amygdala and hippocampus in monkeys. *Journal of Neuroscience, 4*, 2565–2580.

Murray, E. A., & Mishkin, M. (1998). Object recognition and location memory in monkeys with excitotoxic lesions of the amygdala and hippocampus. *Journal of Neuroscience, 18*, 6568–6582.

Noldus, L. P. (1991). The Observer: A software system for collection and analysis of observational data. *Behavior Research Methods, Instruments & Computers, 23*, 415–429.

Numan, M., Numan, M. J., & English, J. B. (1993). Excitotoxic amino acid injections into the medial amygdala facilitate maternal behavior in virgin female rats. *Hormones and Behavior, 27*, 56–81.

Perrett, D. I., Rolls, E. T., & Caan, W. (1982). Visual neurons responsive to faces in the monkey temporal cortex. *Experimental Brain Research, 47*, 329–342.

Rebert, C. S., Hurd, R. E., Matteucci, M. J., De LaPaz, R., & Enzmann, D. R. (1991). A procedure for using proton magnetic resonance imaging to determine stereotaxic coordinates of the monkey's brain. *Journal of Neuroscience Methods, 39*, 109–113.

Redican, W. K. (1975). Facial expressions in nonhuman primates. In L. A. Rosenblum (Ed.), *Primate behavior: Developments in field and laboratory research* (Vol. 4, pp. 104–194). New York: Academic Press.

Rees, H. D., & Michael, R. P. (1982). Brain cells of the male rhesus monkey accumulate ^{3}H-testosterone or its metabolites. *Journal of Comparative Neurology, 206*, 273–277.

Reinhardt, V. (1989). Behavioral responses of unrelated adult male rhesus monkeys familiarized and paired for the purpose of environmental enrichment. *American Journal of Primatology, 17*, 243–248.

Rosene, D. L., Roy, N. J., & Davis, B. J. (1986). A cryoprotection method that facilitates cutting frozen sections of whole monkey brains for histological and histochemical processing without freezing artifact. *Journal of Histochemistry and Cytochemistry, 34*, 1301–1315.

Rosvold, H. E., Mirsky, A. F., & Pribram, K. H. (1954). Influence of amygdalectomy on social behavior in monkeys. *Journal of Comparative and Physiological Psychology, 47*, 173–178.

Saunders, R. C., Aigner, T. G., & Frank, J. A. (1990). Magnetic resonance imaging of the rhesus monkey brain: Use for stereotaxic neurosurgery. *Experimental Brain Research, 81*, 443–446.

Schreiner, L., & Kling, A. (1953). Behavioral changes following rhinencephalic injury in cat. *Journal of Neurophysiology, 16*, 543–659.

Smuts, B. B. (1985). *Sex and friendship in baboons.* New York: Aldine De Gruyter.

Smuts, B. B. (1987). Sexual competition and mate choice. In B. B. Smuts, D. L. Cheney, R. M. Seyfarth, R. W. Wrangham, & T. T. Struhsaker (Eds.), *Primate societies* (pp. 385–399). Chicago: University of Chicago Press.

Sokal, R. R., & Rohlf, F. J. (1995). *Biometry: The principles and practice of statistics in biological research* (3rd ed.). New York: Freeman.

Stefanacci, L., & Amaral, D. G. (2000). Topographic organization of cortical inputs to the lateral nucleus of the macaque monkey amygdala: A retrograde tracing study. *Journal of Comparative Neurology, 421*, 52–79.

Wallen, K., & Tannenbaum, P. L. (1999). Hormonal modulation of sexual behavior and affiliation in rhesus monkeys. In C. S. Carter, I. I. Lederhendler, & B. Kirkpatrick (Eds.), *The integrative neurobiology of affiliation* (pp. 101–118). Cambridge, MA: MIT Press.

Young, A. W., Hellawell, D. J., Van de Wal, C., & Johnson, M. (1996). Facial expression processing after amygdalotomy. *Neuropsychologia, 34*, 31–39.

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