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Early Amygdala or Hippocampus Damage Influences Adolescent Female Social Behavior During Group Formation

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This study continues a longitudinal analysis of rhesus macaque social behavior following bilateral neonatal ibotenic acid lesions of the amygdala or hippocampus, or sham operations. The social behavior of female subjects was evaluated at a critical developmental time point—the transition to adulthood. At approximately 4 years of age, female subjects were housed in small groups with other female subjects and reproductively viable adult males. As compared with neurologically intact control animals and animals with early amygdala damage, animals with early hippocampal damage were more social with their female peers. In contrast, as compared with control animals, animals with early amygdala damage spent less time with the males, engaged less frequently in behaviors typical of reproductive consortships, had higher frequencies of self-directed stereotypies, and became pregnant later. Males also generated fewer communicative signals toward animals with early amygdala damage than to control animals and animals with early hippocampus damage. Rates of sexual behavior were generally low for all animals, and there were no lesion-based differences in their frequencies. Discriminant function analyses demonstrated that patterns of affiliative social behaviors differed across the 3 experimental groups, both in terms of the social behaviors directed to the males, and the social behaviors generated by the males toward the females. In 4 of the 5 social groups, amygdala-lesioned animals were lowest ranked, potentially contributing to reduced sociability interactions with males. Other potential mechanisms and the experiments needed to elucidate them are discussed.

Keywords: Macaca mulatta, neurodevelopment, nonhuman primate, rhesus macaque, sexual behavior

More than a century of research demonstrates that damage to the nonhuman primate amygdala during adulthood results in stereotypic alterations to social and affective behavior (e.g., Aggleton & Passingham, 1981; Brown & Schafer, 1888; Emery et al., 2001; Izquierdo, Suda, & Murray, 2005; Kling, 1968; Kling, Lancaster, & Benitone, 1970; Kling & Cornell, 1971; Kling, 1974; Klüver & Bucy, 1939; Machado et al., 2008; Machado, Kazama, & Bachevalier, 2009; Mason, Capitanio, Machado, Mendoza, & Amaral, 2006; Meunier, Nalwa, & Bachevalier, 2003; Mirsky, 1960; Schreiner & Kling, 1956; Stefanacci, Clark, & Zola, 2003; Zola-Morgan, Squire, Clower, & Alvarez-Royo, 1991). The role of the amygdala in normal social and affective development, however, has received less attention as only a few laboratories have undertaken developmental studies following early amygdala damage (e.g., Bachevalier, Alvarado, & Malkova, 1999; Bachevalier, Beauregard, & Alvarado, 1999; Bauman, Lavenex, Mason, Capitanio, & Amaral, 2004a; Bauman, Lavenex, Mason, Capitanio, & Amaral, 2004b; Bauman, Toscano, Mason, Lavenex, & Amaral, 2006; Bauman, Toscano, Babineau, Mason, & Amaral, 2008; Beauregard, Malkova, & Bachevalier, 1995; Bliss-Moreau, Toscano, Bauman, Mason, & Amaral, 2010; Bliss-Moreau, Toscano, Bauman, Mason, & Amaral, 2011; Bliss-Moreau, Bauman, & Amaral, 2011; Bliss-Moreau, Moadab, Bauman, & Amaral, 2013; Goursaud & Bachevalier, 2007; Goursaud, Wallen, & Bachevalier, 2014; Raper, Stephens, Sanchez, Bachevalier, & Wallen, 2014; Stephens, Raper, Bachevalier, & Wallen, 2015; Thompson, Schwartzbaum, & Harlow, 1969; Thompson & Towfighi, 1976; Thompson, Bergland, & Towfighi, 1977). As part of an ongoing study, we evaluated social behavior at a critical developmental time point—the transition to adulthood—in a cohort of female rhesus macaques who received neonatal damage to the amygdala. In addition to evaluating the social and sexual behavior of the neonatal amygdala-lesioned animals relative to neurologically intact control animals, we also evaluated the behavior of animals that sustained neonatal hippocampus damage who served as operated controls.

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Previous work evaluating the effects of early damage to the amygdala in macaques demonstrated variable impact on social and affective behavior (Goursaud et al., 2014; Raper et al., 2014; Thompson et al., 1969; Thompson & Towfighi, 1976; Thompson et al., 1977)—with variation across studies potentially being related to lesion technique and, or, social rearing conditions. Thompson and colleagues, for example, (Thompson et al., 1969; Thompson & Towfighi, 1976; Thompson et al., 1977) demonstrated that animals with early ablation of the amygdala who were subsequently reared alone were less reactive to novel stimuli (Thompson et al., 1969) and markedly more reactive to peers during social interactions (Thompson & Towfighi, 1976). More recent studies, in contrast, have demonstrated that when animals sustain early damage to the amygdala but are reared in large social groups, the impact of amygdala damage is very subtle (Goursaud et al., 2014; Raper et al., 2014). The only notable difference observed in these more recent studies was that animals with early amygdala damage spent less time in physical contact with their mothers than neurologically intact control animals (Goursaud et al., 2014; Raper et al., 2014). Clearly, further research on the development of social behavior following early amygdala damage is warranted.

Subjects in the current study received bilateral, neurotoxic lesions of the amygdala or hippocampus or control operations at two weeks of age and subsequently participated in several years of affective processing and social behavior testing while being socially housed in various configurations (Bauman et al., 2004a, 2004b, 2006, 2008; Bliss-Moreau et al., 2010, 2013; Bliss-Moreau, Toscano, et al., 2011; Bliss-Moreau, Bauman, et al., 2011; Moadab et al., 2015). Animals with early amygdala damage from this cohort, in contrast to their hippocampus-lesioned and neurologically intact peers, demonstrated consistent blunting of affective responsivity to novel and threatening objects (Bliss-Moreau et al., 2010; Bliss-Moreau, Toscano, et al., 2011). Patterns of social behavior have varied over development. Early in development (9 and 12 months), amygdala-lesioned animals were more expressive compared with controls in social contexts with both familiar and unfamiliar partners—generating more affiliative and submissive signals (Bauman et al., 2004a). Yet, at 18 months of age, they were less expressive with familiar partners (Bliss-Moreau et al., 2013). Early in development, hippocampus-lesioned animals, as compared with controls, solicit grooming behavior more frequently during social interactions (Bauman et al., 2004a). As juveniles, hippocampus-lesioned animals, spent less time with familiar peers, but exhibited similar frequencies of communicative or affiliative signaling (Bliss-Moreau et al., 2013).

One particularly important developmental milestone for young animals, and especially group living animals, is the transition to adulthood. This is a particularly important time in the lives of female mammals who typically begin to reproduce and raise young. Rhesus macaque females become sexually mature around 3 to 4 years of age and typically begin to become pregnant and give birth soon thereafter (Anderson & Simpson, 1979; Gagliardi, Liukkonen, Phillippi-Falkenstein, Harrison, & Kubisch, 2007; Kaufmann, 1965; Smuts, Cheney, Seyfarth, Wrangham, & Struhsaker, 1987; Stephens & Wallen, 2013; Wilson, Gordon, Blank, & Collins, 1984). To meet these developmental milestones, young females are tasked with securing a mating partner by forming temporary, but strong, social bonds with sexually reproductive males. These reciprocal relationships with males, referred to as "consortships," typically include increased durations of contact, proximity, and reciprocal grooming (Bernstein, 1963; Drickamer, 1974; Fedigan, 1982; Manson, 1997; Small, 1990; Smuts et al., 1987). Given that these sort of close social interactions appear to be altered as the result of amygdala damage (Bauman et al., 2004a; Bliss-Moreau et al., 2013; Brown & Schafer, 1888; Emery et al., 2001; Kling, 1968; Kling et al., 1970; Kling & Cornell, 1971; Kling, 1974; Klüver & Bucy, 1939; Machado, Emery, et al., 2008; Mirsky, 1960; Moadab et al., 2015; Schreiner & Kling, 1956), the social effects of early damage to the amygdala may be particularly apparent during the transition to adulthood. This is underscored by the observation that damage to the amygdala has also been associated with alterations to sexual behavior (Kling, 1968; Kling, 1974; Klüver & Bucy, 1939; Schreiner & Kling, 1956). Limited evidence suggests that amygdala damage may influence pubertal timing (Stephens et al., 2015; but see Norman & Spies, 1981). Early cycling attributable to amygdala lesions could be one mechanism responsible for variation in social behavior (e.g., as in Bliss-Moreau et al., 2013) although Stephens and colleagues (2015) did not evaluate social behavior. The present report extends the observations of Stephens et al. (2015) by evaluating the possibility that early damage to the amygdala might alter social behavior of female rhesus macaques during the transition to adulthood.

Method

All experimental procedures were developed in consultation with the research and veterinary staff at the California National Primate Research Center (*CNPRC*). All protocols were approved by the University of California, Davis Institutional Animal Care and Use Committee—the research ethics committee overseeing nonhuman animal research (protocol numbers 11059 and 12655).

Animals and Living Conditions

A more detailed description of subject selection and rearing history has been previously published (Bauman et al., 2004a, 2004b; Bliss-Moreau, Toscano, et al., 2010, 2011; Bliss-Moreau, Bauman, et al., 2011; Bliss-Moreau et al., 2013). The subjects were part of a larger cohort that includes both males and females. Only the female subjects ($N = 14$) were included in the experimental procedures of this report. Surgical procedures were conducted when the subjects were approximately two weeks of age. Subjects received either sham control operations (4 females) or bilateral ibotenic acid lesions of either the amygdala (5 females) or the hippocampus (5 females).

Surgical procedures. Surgical procedures will be summarized briefly here, as they have been described in detail in previous publications (Bauman et al., 2004a, 2004b). The morning of surgery, each animal was anesthetized with ketamine hydrochloride (15 mg/kg i.m.) and medatomidine (30 μg/kg) and placed in an MRI-compatible stereotaxic apparatus (Crist Instruments, Damascus, MD) to determine the stereotaxic coordinates of the amygdala or hippocampus. The following parameters were used on a General Electric 1.5 T Gyroscan magnet for brain imaging: slice thickness = 1.0 mm, T1-weighted Inversion Recovery Pulse sequence, TR = 21, TE = 7.9, NEX 3, FOV = 8 cm, Matrix 256 × 256.

Animals were intubated after MRI for ventilation during surgery and were anesthetized with a combination of isoflurane (1.0% -

varied as needed to maintain a surgical level of anesthesia) and intravenous infusion of fentanyl (7–10 μg/kg/hour). Operated subjects received two craniotomies over the right and left amygdala or hippocampus. Using 10 μl Hamilton syringes with 26 gauge beveled needles, Ibotenic acid (*IBO*, Biosearch Technologies Inc., 10 mg/ml in 0.1 M phosphate buffered saline) was injected concurrently bilaterally into the hippocampus or amygdala at a rate of 0.2 μl/min. Sham-operated control animals experienced equivalent presurgical preparations, then received a midline incision to expose the skull, and were maintained under anesthesia for the average duration of the lesion surgeries. Following surgery, all animals were monitored closely by a veterinarian. Once fully alert, they were returned to their mothers.

Lesion analysis. Lesion placement has been confirmed at various time points using MRI and more recently by direct histological analysis (Bliss-Moreau, Moadab, Santistevan, & Amaral, in press). Edema associated with brain lesions was measured 10 days postsurgery, using T2-weighted magnetic resonance images. Animals were scanned on a General Electric 1.5 T Gyroscan magnet (slice thickness = 1.5 mm thick; T2 weighted Inversion Recovery Pulse sequence: TR = 4000, TE = 102, NEX 3, FOV = 8 cm, Matrix, 256 × 256). The T2-weighted signal for each of the animals was evaluated to confirm the general target and extent of the lesions. The T2-weighted images of coronal sections through the mid portion of the amygdala have been illustrated in previous publications (Bauman et al., 2004a, 2004b; Bliss-Moreau, Toscano, et al., 2011), confirming that ibotenic acid was injected and caused damage to either the hippocampal formation or the amygdaloid complex. Lesion extent was also investigated in T1-weight MRI images collected when animals were approximately four years of age (Machado, Snyder, Cherry, Lavenex, & Amaral, 2008). Volumes of amygdalae and hippocampi were calculated for the sham operated (control), the amygdala-lesioned, and the hippocampus-lesioned animals. The extent of atrophy was calculated by comparing the structural volumes in the lesioned animals to the structural volumes of the control animals. On average, amygdala-lesioned animals demonstrated a 72.56% (*SD* = 4.57%) volumetric reduction of the amygdala and a 23.71% (*SD* = 12.17%) volumetric reduction in the adjoining hippocampus. Hippocampus-lesioned animals demonstrated a 76.65% average (*SD* = 7.46%) volumetric reduction of the hippocampus and a 15.17% average (*SD* = 13.93) volumetric reduction of the amygdala.

Socialization history. Following surgery, animals were returned to their mothers and housed in standard primate caging (61 cm W × 66 cm D × 81 cm H) until fully recovered. Subjects and their mothers were subsequently socialized in large, chain-link enclosures (2.13m W × 3.35m D × 2.44m H) for three hours a day, five days a week. Each social group consisted of 13 animals: two subjects from each of the three experimental conditions (males and females), their mothers, and an adult male. All animals were fed standardized monkey chow twice a day, foraging enrichment daily, and produce biweekly. They were provided water ad-libidum, and maintained on an automatically regulated 12-hr light cycle with temperatures ranging from 17°C to 28°C.

Consistent with standard CNPRC protocols, subjects were weaned from their mothers at 6 months of age and continued to be socialized in their familiar groups without their mothers, but with the addition of an unfamiliar adult female, for three hours a day, five days a week. At one year of age, subjects were housed permanently with these social groups in large enclosures.

At approximately three years of age, each social group moved from their indoor enclosures to large outdoor enclosures (6.10m W × 4.27m D × 2.44m H) and remained there for one year. At that time, animals moved back indoors into standard primate caging. While housed indoors, all animals had access to a compatible social partner (opposite sex or same sex) for at least 6 hours/day five days per week. Animals lived indoors in these pairs for an average of 2.143 months (*SD* = 1.099). Animals were then relocated outdoors into the groups described below when they were approximately 4 years of age.

During group formations, one additional age-matched neurologically intact female was added to the original cohort to ensure that each social group had a control female. This animal had previously been a social partner for one of the control animals and was known to be a social and tolerant animal. She was born and reared in the large field cage enclosures at CNPRC (30.5m W × 61m D × 2.44m H; ~50–120 animals per cage) for two years. She was then relocated into a standard indoor cage where she was socially housed with a colony female. She joined the study as a social partner when she was approximately three years old and remained in this role until her involvement with the current experiment.

Adult Males to Serve as Social and Mating Partners

Adult males were selected based on their social rearing history, prior reproductive success, and age. All males were born in the large CNPRC field enclosures and lived in large social groups for at least 5 years. They were all known to be social animals with no abnormal behaviors who were successfully integrated into the matriline of their social groups (indicating that they were capable of forming social bonds with females) but had since been relocated indoors for social management reasons. Prior to the group formations detailed here, each male had fathered at least 5 infants in previous social groups (number of conceptions ranged from 5–40), ensuring successful socialization history with female animals. At the time of formation, the average age of the males was 11.42 years (*SD* = 1.17). Males had been living in standard indoor cages (61 cm W × 91.44 cm D × 81 cm H) with a compatible social partner for at least three months prior to the start of the experiment. One male was permanently removed from his social group nine days after formation due to excessive aggression toward one of the females in his group. He was replaced with a different male two days later, at which time focal observations resumed.

Experimental Design and Procedures

The present experiment began when the females were an average 4.11 (*SD* = 0.11) years of age, just at the point at which females are typically reproductively viable (at 3–4 years; Anderson & Simpson, 1979; Gagliardi et al., 2007; Kaufmann, 1965; Smuts et al., 1987; Wilson et al., 1984). Prior to the group formation, 14 of 16 animals were observed to have experienced menarche. Two amygdala-lesioned animals were not observed to have experienced menarche. One of those two did become pregnant while living in the social group, and the other was noted to menstruate subsequently to the experiment. Given the available data from this cohort and another report which details earlier

menarche and ovulation following early amygdala damage (Stephens et al., 2015), it is clear that the neonatal amygdalectomy did not eliminate cycling. With the available data, it is not possible, however, to attest to whether any of the females were ovulating during the experiment or whether subjects had species-typical normal levels of circulating gonadal steroids.

To index female social behavior, both with female peers and with males, observers recorded behaviors that are typical of social and sexual interest (see behavioral ethogram in Table 1). Sociability measures included both affiliative and agonistic behaviors, whereas sexual behaviors included sexual presentations and behaviors related to the establishment of sexual consortships (coordinated relationships with males consisting of heightened durations of contact, proximity, and reciprocal grooming; Bernstein, 1963; Fedigan, 1982; Manson, 1997; Small, 1990; Smuts et al., 1987). Previous research has documented brain-based variation in these behaviors early in the formation of relationships with possible mating partners (Emery et al., 2001). Given that, groups were closely monitored for one month after social group formation.

Five social groups were formed. Each group consisted of one male, one amygdala-lesioned female, one hippocampus-lesioned female, and one control female. All animals were novel to each other at the time of group formation. Groups were formed in July and August prior to the start of the breeding season. Each group was monitored closely for the first month during which formal data collection occurred, although social groups were maintained for an average of 390.20 days ($SD = 11.55$).

Each female was formally observed daily for 30 days using focal sampling technique (Altmann, 1974). Two 10-min samples were collected per day for the first 14 days, at which time observations decreased to one 10-min sample per day for the next 16 days. This yielded 44 observations per subject. Animals were observed between the hours of 8-12AM or 12-4PM. Observers were two laboratory members with an interrater reliability of greater than 90%. Frequency and duration of behaviors were collected using The Observer 5.0 (Noldus, 1991). In addition to specific behaviors, observers also recorded the direction of the behavior (whether it was initiated by the subject or directed to the subject from another animal), as well as the recipient of the behavior. At least one observer was blind to the lesion condition of the animals. Observation order was pseudorandomized, and the observers scoring the behaviors were balanced across days and animals.

Data Analysis Strategy

Statistical analyses were conducted in SPSS (IBM Corp., IBM SPSS Statistics for Windows, Version 21.0 and 22.0 Armonk, NY). Behaviors were grouped into broad theoretical categories as indicated in Table 1 and as previously used to evaluate social behavior of these animals (Bliss-Moreau et al., 2013). Frequency and durations were summed across behavioral categories to generate total values for behavioral interactions with peers (the total values for all behaviors directed to the females in the group), and behavioral interactions with the male. Behavioral categories were then averaged across the number of observations to create a mean value per observation. Analyses of variance (ANOVAs) were performed on each behavioral category using focal lesion group as the between-subjects factor, with post hoc or follow-up t tests where appropriate. p values associated with LSD post hoc tests are indicated in the text where appropriate. In cases where data were not normally distributed, data were $\log_{10}(x + 1)$ transformed as indicated. Raw data are presented here for the purposes of interpretation; log transformed data are available upon request. For the sake of analytic continuity with previous reports on these subjects (Bauman et al., 2004a; Bauman et al., 2008) and to establish an understanding of the developmental trajectory of specific behaviors, ANOVAs were conducted on individual behaviors. In some cases, the omnibus test did not reach conventional levels of significances, but visual inspection of each group's marginal means suggested that there were significant differences between two of the three groups. In those cases, data were further evaluated using t tests so as not to miss important group variation that may have been masked in the omnibus tests. Cohen's d effect size is reported for t tests. This strategy was adopted for the sake of completeness, despite the fact that this approach is nontraditional (rather than only probing between group differences when the omnibus effect was significant at $p < .05$). Nonhuman primate studies of this sort utilize small samples, are rare, and are unlikely to be repeated using the exact same designs. The goal was to present all relevant analyses, thus creating a full scientific record, and allowing the reader ample evidence from which to draw conclusions. In cases where data violated Levene's test for equality of variance for t tests, corrected degrees of freedom are presented using the Welch-Satterthwaite method. For the sake of brevity, only significant results and those for which there were a priori hypotheses based on previous publications are presented here. All other analyses are available upon request.

Finally, multiple analysis of variance (MANOVA) and discriminant function analysis were used to investigate whether the organization of behaviors that occurred in the presence of the male varied by lesion condition. Behaviors occurring with the male were evaluated because those behaviors would be most indicative of lesion-based variation in the social behavior that might eventually subserve consortships and reproduction-related sexual behavior. Because MANOVA and discriminant function analysis require that only a small number of dependent variables are used when sample sizes are low (as is typical in nonhuman primate studies), behavioral variables were grouped across categories as indicated in Table 1. Traditionally, discriminant function analysis follows MANOVA. For the sake of brevity and clarity, only the discriminant function analyses are presented here. The MANOVAs are available upon request.

Results

Social Behaviors With the Adult Male

Duration of time spent in close social interactions. Amygdala-lesioned females initiated shorter periods of close social interactions (those occurring within arms' reach) with the males as compared with the other female members of their groups, $F(2, 12) = 3.607$, $p = .059$, $\eta_p^2 = 0.375$. Despite the omnibus test not reaching conventional levels of significance ($p < .05$), between-groups differences were evaluated with t tests because the η_p^2 value indicated that lesion condition accounted for a moderate level of variation and the confidence intervals of the marginal means suggested that the amygdala-lesioned animals might differ

Table 1
Social Behavior Ethogram

Behavior	Description
States	
Social states	
Extended contact	Any physical contact between focal animal and other animal.
Proximity	Animal is within arm's reach of another animal.
Extended groom	Examination, picking, or licking of another animal's fur or body.
Extended play	Rough and tumble play or chase play.
Extended mount	Any instance of mounting.
Extended negative	Any instance of aggression or chase.
Nonsocial states	
Nonsocial activity	Animal remains out of all social states with head up, actively engaged in the environment.
Nonsocial inactivity	Animal remains out of all social states with head down, not engaged in environment.
Sleep contact	Animal is asleep while in contact with another animal.
Sleep proximity	Animal is asleep while sitting within arm's reach of another animal.
Sleep solo	Animal is asleep, but out of contact or proximity with other animals.
Extended stereotypy	Repetitive motor or abnormal behavior.
Extended toy play	Manipulation of toy.
Events	
Communicative signaling	
Bark[a]	Low pitched, sharp, guttural sound.
Affiliative	
Anogenital exploration[c]	Oral, olfactory, or manual exploration of another animal's anogenital area.
Approach[b]	Intentional movement within arm's reach of another animal.
Coo[d]	Clear, soft sounds, moderate in pitch and intensity; usually sounds like "whoooooo."
Contact	Any physical contact between focal animal and other animal.
Follow[b]	Intentional follow of another animal.
Grunt[d]	Deep, muffled, low-intensity vocalization.
Groom	Examination, picking, or licking of another animal's fur or body.
Lipsmack[d]	Rapid lip movements with pursed or puckered lips, usually accompanied by smacking sounds.
Incomplete mount[c]	Mount that includes one or two, but not all three of the necessary components of a "Mount".
Inappropriate mount[c]	An attempt to mount an inappropriate part of the body—head, side, or shoulder instead of perineum.
Mount[c]	Mount that includes all of the following components: appropriate positioning of partner, hands on back, double foot clasp.
Huddle	Physical contact that involves one animal ventrally touching another animal.
Play threat	Relaxed open mouth threats, ear flaps, lunges or head bobs. Often occurs in context of Rough and Tumble Play.
Present groom	Intentional presentation of neck, belly, or other part of body to another animal.
Present rump	Rigid posture with rump and tail elevated and oriented toward another individual.
Rough and tumble play	Contact play consisting of mounting, tumbling, and wrestling.
Agonistic/"aggression"	
Aggressive bite	Animal aggressively bites another.
Aggressive slap	Animal aggressively slaps another.
Aggressive grab	Animal aggressively grabs another.
Aggressive grunt	Deep, muffled, low-intensity vocalization occurring in conjunction with a threat and/or aggression.
Chase	Rapid pursuit of another animal lasting more than three seconds.
Displacement	Physical movement in which an animal "takes the place" of another animal.
Redirect threat	A "Threat" is directed at a third party after the occurrence of an unrelated interaction.
Threat	Contains one or more of the following components: open mouth stare, head bobbing, ear flaps, bark vocalizations, or lunges.
Toy-steal	Deliberate and intentional taking of toy from another animal.
Submission/"fear"	
Avoid	Animal leaves the area due to the arrival of another animal.
Crooktail	Tail held in stiff "?" shape.
Grimace	Exaggerated movement of lips such that lips are pulled back with teeth showing.
Flee	Rapid, intentional movement away from another animal.
Flinch	Animal jerks, jumps, or flinches at approach of movement of another animal.
Freeze	Stiff body posture without any movement for more than three seconds.
Scream	High-pitched vocalization, with extreme high intensity; sounds like "eeeeeeeeee."
Exploration	
Manual	Exploration of the cage or environment with the hands.
Oral	Exploration of the cage or environment with the mouth.
Toy-play	Exploration of toy.
Stress	
Scratch	Scratches own body.
Self-groom	Examining, picking, or licking one's own fur or skin.
Tooth grind	Repetitive, audible rubbing of upper and lower teeth.
Yawn	Yawn.

Table 1 (*continued*)

Behavior	Description
Other events	
Cage shake	Vigorous shaking of cage bars or body slams against the cage.
Crouch	Animal is quadrupedal and bending down low but not exploring, eating, or drinking.
Mount refusal	Animal who is being mounted moves away or physically pushes partner away.
Self sex	Anogenital exploration of self.
Withdraw	Animal moves out of arm's reach of another animal after being in proximity or contact.
Stereotypies	
Self-directed	
Rocking	Repetitive swaying back and forth.
Salute	Animal covers hand over eye or eye pokes.
Self-clasp	Unusual holding of body part or limb.
Self-bite	Biting at oneself.
Whole body	
Backflip	Repetitive back flipping.
Bounce	Repetitive hopping.
Pace	Repetitive undirected movement with the same path repeated.
Spin	Repetitive twirling.
Swinging	Repetitive swinging.
Other	
Heat twist	Animal twists neck in a dramatic display.
Other stereotypy	Repetitive motor or abnormal behavior patterns not described by any of the above definitions.

Note. To be scored in a "state," behavior must occur for three seconds, with the exception of Extended Stereotypy and Extended Toy Play, which must occur for six seconds.
[a] In addition to the subordinate categories, Bark was included in the Communicative Signaling category. [b] These behaviors are included in the category "approach and follow" used in the MANOVA as described in the Data Analysis section of Method. [c] These behaviors are included in the category "anogenital exploration and mount" used in the MANOVA as described in the Data Analysis section of Method. [d] These behaviors are included in the category "vocal and facial signals" used in the MANOVA as described in the Data Analysis section of Method.

significantly from the other two groups. Amygdala-lesioned animals differed significantly from the two other groups: amygdala-lesioned animals spent less time in close social states as compared with control animals, $t(8) = 2.572$, $p = .033$, $d = 1.626$ and hippocampus-lesioned animals, $t(8) = 2.356$, $p = .046$, $d = 1.490$ (see Figure 1a). The large effect sizes indicate robust effects.

Lesion-based differences in specific types of close social interactions were assessed next. Those analyses revealed that amygdala-lesioned animals spent the least time grooming the males, $F(2, 12) = 13.309$, $p = .001$, $\eta_p^2 = 0.689$—significantly less time than control animals ($p = .0003$) and hippocampus-lesioned animals ($p = .004$) (analyses on log transformed data). Similarly, amygdala-lesioned animals spent the least amount of time in contact with the males, $F(2, 12) = 6.411$, $p = .013$, $\eta_p^2 = 0.517$—significantly less than control animals ($p = .004$) and hippocampus-lesioned animals ($p = .041$; analyses on log transformed data, see Figure 1a).

Male behavior with the subjects varied by lesion condition as well. The males initiated longer close social interactions with the control females as compared with the amygdala-lesioned and hippocampus-lesioned animals, $F(2, 12) = 5.100$, $p = .025$, $\eta_p^2 = 0.459$—significantly more than control animals ($p = .009$) and hippocampus-lesioned animals ($p = .049$) (see Figure 1b).

Frequency of close social interactions. Amygdala-lesioned animals not only spent less overall time socializing with the male, they also engaged in close social states less frequently than control and hippocampus-lesioned animals, $F(2, 12) = 3.073$, $p = .084$, $\eta_p^2 = 0.339$. Again, although the omnibus test did not reach conventional levels of significance, because the η_p^2 value and evaluation of the marginal means suggested possible lesion-based variation, t tests were used to compute pairwise comparisons between the amygdala-lesioned animals and the other subjects. Amygdala-lesioned animals initiated fewer close social states than control animals or hippocampus-lesioned animals, $t(4.025) = 2.441$, $p = .071$, $d = 1.544$ and $t(4.051) = 2.198$, $p = .092$, $d = 1.390$. Although these effects did not reach conventional levels of significance (i.e., $p < .05$), the very large effect sizes (indicated by $d > 0.80$) suggest that there might be meaningful differences between the groups.

Frequency of communicative signaling. Amygdala-lesioned animals generated fewer communicative signals such as facial behaviors and vocalziations toward the males than controls animals did, $F(2, 12) = 3.835$, $p = .052$, $\eta_p^2 = 0.390$. There was a significant difference in the frequency of communicative signals directed toward the male when the amygdala-lesioned animals were compared directly to the control animals, $t(4.251) = 2.801$, $p = .046$, $d = 1.771$. Amygdala-lesioned animals also generated the fewest affiliative signals of all of the females toward the male, $F(2, 12) = 8.006$, $p = .006$, $\eta_p^2 = 0.572$—significantly less than control animals ($p = .003$) and hippocampus-lesioned animals ($p = .011$; analyses on log transformed data). There were no lesion-based differences in submission related behaviors or agonistic behaviors directed toward the male: $F(2, 12) = 0.051$, $p = .950$, $\eta_p^2 = 0.008$ and $F(2, 12) = 1.000$, $p = .397$, $\eta_p^2 = 0.143$ (analyses on log transformed data), respectively (see Figure 1c).

The males' social behavior varied based on the lesion condition of the females. The males initiated close social states least frequently with the amygdala-lesioned animals and most frequently with the control animals, $F(2, 12) = 5.905$, $p = .016$, $\eta_p^2 = 0.496$; control > amygdala-lesioned: $p = .005$. Similarly, they generated fewer communicative signals directed to the amygdala-lesioned animals as compared with the other females, $F(2, 12) = 4.466$, $p =$

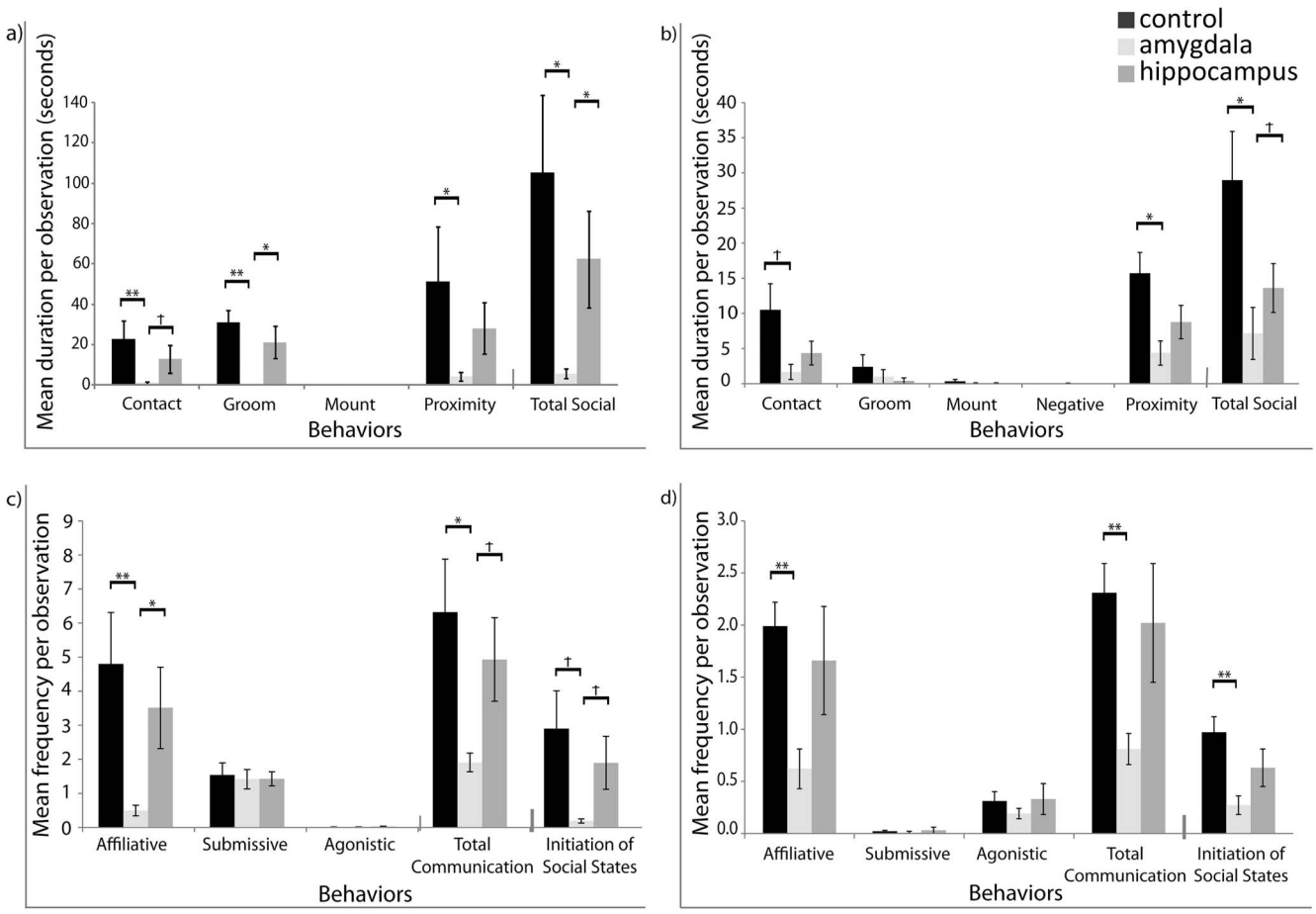

Figure 1. Social behavior with male. Significant differences between lesion conditions as per independent sample *t* tests are indicated using the following symbol key: † $p < .10$, * $p < .05$, ** $p < .01$. (a) Durations of close social states initiated with male. Raw means are presented in the figure although analyses for extended contact, groom, mount, and proximity were performed on log transformed data to account for non-normalcy. (b) Durations of close social states initiated by males to each lesion condition. Raw means are presented in the figure although analyses for groom, mount, and negative were performed on log transformed data to account for non-normalcy. (c) Frequency of behaviors directed to males. Raw means are presented in the figure although analyses for affiliative and agonistic categories were performed on log transformed data to account for non-normalcy. (d) Frequency of behaviors initiated by males to each lesion condition. Raw means are presented in the figure although analyses for submissive and agonistic categories were performed on log transformed data to account for non-normalcy.

.036, $\eta_p^2 = 0.427$; control > amygdala-lesioned: $p = .016$, hippocampus-lesioned > amygdala-lesioned: $p = .042$. The males also generated fewer affiliative behaviors (prosocial communicative signals, i.e., approaches, positive vocalizations and facial signals, and nonaggressive contact) directed toward the amygdala-lesioned animals as compared with the other females, $F(2, 12) = 4.233$, $p = .041$, $\eta_p^2 = 0.414$; control > amygdala-lesioned: $p = .017$, hippocampus-lesioned > amygdala-lesioned: $p = .055$ (see Figure 1d).

Given this pattern of findings, behaviors that are representative of relationships that typically lead to reproduction—consortship behaviors—were evaluated. This behavioral class comprises mounting, following, proximity, and grooming behaviors. Males consorted more frequently with control females than with amygdala-lesioned animals, $F(2, 12) = 6.475$, $p = .012$, $\eta_p^2 = 0.519$; control > amygdala-lesioned: $p = .004$ (see Figure 2).

Social Behaviors With Female Peers

Duration of time spent in close social interactions. The hippocampus-lesioned animals initiated longer close social interactions with their female peers than control and amygdala-lesioned animals, although the omnibus test did not reach conventional levels of significance, $F(2, 12) = 3.388$, $p = .068$, $\eta_p^2 = 0.361$. When the hippocampus-lesioned animals were compared directly with the control animals, there was a significant difference in the duration of time in close social states, $t(8) = 2.826$, $p = .022$, $d = 1.787$. As with the assessments of behaviors with the male, group

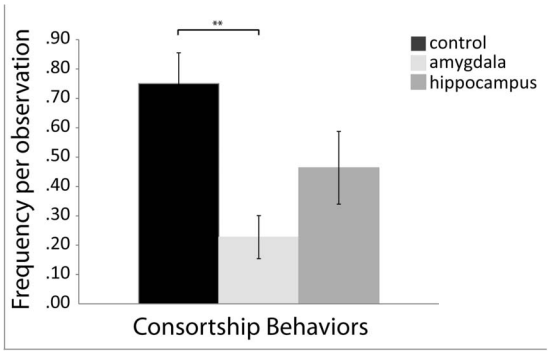

Figure 2. Consortship behaviors initiated by males to each lesion condition. Significant differences between lesion conditions as per ANOVA post hoc tests are indicated using the following symbol key: † $p < .10$, * $p < .05$, ** $p < .01$. Raw means are presented in the figure although analyses were performed on log transformed data to account for non-normalcy.

differences in the specific close social interactions were also evaluated. Hippocampus-lesioned animals initiated longer durations of physical contact with their peers than the other groups, $F(2, 12) = 3.995$, $p = .047$, $\eta_p^2 = 0.400$; hippocampus-lesioned > controls, $p = .022$; hippocampus-lesioned > amygdala-lesioned, $p = .047$. Similarly, hippocampus-lesioned subjects tended to initiate longer durations of grooming than their peers, $F(2, 12) = 3.772$, $p = .054$, $\eta_p^2 = 0.386$. Further evaluation of between-groups differences of grooming yielded the same results, hippocampus-lesioned animals groomed their female peers more than controls and amygdala-lesioned animals, $t(8) = 2.147$, $p = .064$, $d = 1.358$ and $t(8) = 2.113$, $p = .068$, $d = 1.336$, respectively. Although these effects did not reach conventional levels of significance (i.e., $p < .05$), the very large effect sizes (indicated by d>0.80) suggest that there were meaningful differences between the groups (see Figure 3a).

Frequency of close social interactions. Although the hippocampus-lesioned animals tended to spend longer durations in social states with their peers, there were no lesion-based differences in the frequency in which subjects initiated close social interactions, $F(2, 12) = 2.469$, $p = .126$, $\eta_p^2 = 0.292$.

Frequency of communicative signals. The hippocampus-lesioned animals had higher rates of communicative signaling than controls and amygdala-lesioned animals, $F(2, 12) = 10.730$, $p = .002$, $\eta_p^2 = 0.641$; hippocampus-lesioned > controls, $p = .004$, hippocampus-lesioned > amygdala-lesioned, $p = .001$ (see Figure 3c). They also had higher frequencies of affiliative behaviors than their peers $F(2, 12) = 10.668$, $p = .002$, $\eta_p^2 = 0.640$; hippocampus-lesioned > controls, $p = .006$, hippocampus-lesioned > amygdala-lesioned, $p = .001$ (see Figure 3c).

There were also lesion-based differences in submissive and agonistic behaviors generated in the presence of peers. Consistent with previous findings from this group (i.e., Bauman et al., 2004a; Bliss-Moreau et al., 2013) amygdala-lesioned animals produced fewer agonistic behaviors than their peers, $F(2, 12) = 7.250$, $p = .009$, $\eta_p^2 = 0.547$; controls > amygdala-lesioned, $p = .030$; hippocampus-lesioned > amygdala-lesioned, $p = .003$. A greater number of agonistic behaviors were generated by peers and directed toward amygdala-lesioned animals, $F(2, 12) = 5.714$, $p = .018$, $\eta_p^2 = 0.488$; amygdala-lesioned > controls, $p = .009$; amygdala-lesioned > hippocampus-lesioned, $p = .019$ (see Figure 3d). Despite being the target of more frequent agonistic behaviors, amygdala-lesioned animals were not more submissive to their peers—rates of submissive signaling did not vary across groups, $F(2, 12) = 0.423$, $p = .665$, $\eta_p^2 = 0.066$ (analyses on log transformed data; Figure 3c). Control and hippocampus-lesioned animals signaled submission less frequently to amygdala-lesioned animals than to each other, $F(2, 12) = 4.371$, $p = .037$, $\eta_p^2 = 0.421$; controls > amygdala-lesioned, $p = .072$; hippocampus-lesioned > amygdala-lesioned, $p = .013$ (analyses on log transformed data; Figure 3d).

Dominance Status

Lesion based differences in dominance-related displacements (i.e., taking over a resource, typically a physical space or a toy) and avoidance (i.e., when an animal moves away in response to another animal approaching) indicated that the amygdala-lesioned animals had the lowest status. Amygdala-lesioned animals were less likely to displace control ($p = .043$) and hippocampus-lesioned ($p = .003$) females, $F(2, 14) = 6.710$, $p = .011$, $\eta_p^2 = 0.528$ (analyses on log transformed data). Control and hippocampus-lesioned animals were equally likely to displace each other ($p = .199$). Similarly, amygdala-lesioned animals were less likely than control ($p = .010$) and hippocampus-lesioned animals ($p = .002$) to be avoided by their peers, $F(2, 14) = 8.231$, $p = .006$, $\eta_p^2 = 0.578$ (analyses on log transformed data). Control and hippocampus-lesioned animals were equally likely to avoid each other ($p = .436$). See Figure 4a for means.

Frequency of displacements and avoidance by group were evaluated to determine rank (high, mid, low) for each group. In each group, the female with the highest frequency of displacing other females and being avoided by them was ranked as "high" dominance status, whereas the female with the lowest frequencies of these behaviors was ranked as "low." The third female subject received the "mid" dominance status ranking. Control animals were most commonly highest ranking—3 of 5 control females were high ranking. Hippocampus animals were most commonly mid ranking—4 of 5 of the hippocampus-lesioned animals were midranking and amygdala-lesioned animals were most commonly lowest ranking—4 of 5 of the amygdala-lesioned animals were lowest ranked. One control animal was mid ranked and one was lowest ranked. One hippocampus-lesioned animal and one amygdala-lesioned animal were highest ranked in their group (see Figure 4b).

Nonsocial Behaviors

Amygdala-lesioned animals, compared with control animals, spent more time in nonsocial states, $F(2, 12) = 5.291$, $p = .023$, $\eta_p^2 = 0.469$; amygdala-lesioned > controls, $p = .008$. This overall difference was driven by variation in the duration of time moving about the cage $F(2, 12) = 4.836$, $p = .029$, $\eta_p^2 = 0.446$; amygdala-lesioned > controls, $p = .010$; and sitting quietly in the cage $F(2, 12) = 4.030$, $p = .046$, $\eta_p^2 = 0.402$; amygdala-lesioned > controls, $p = .015$ (analyses on log transformed data). There were no lesion-based differences in the

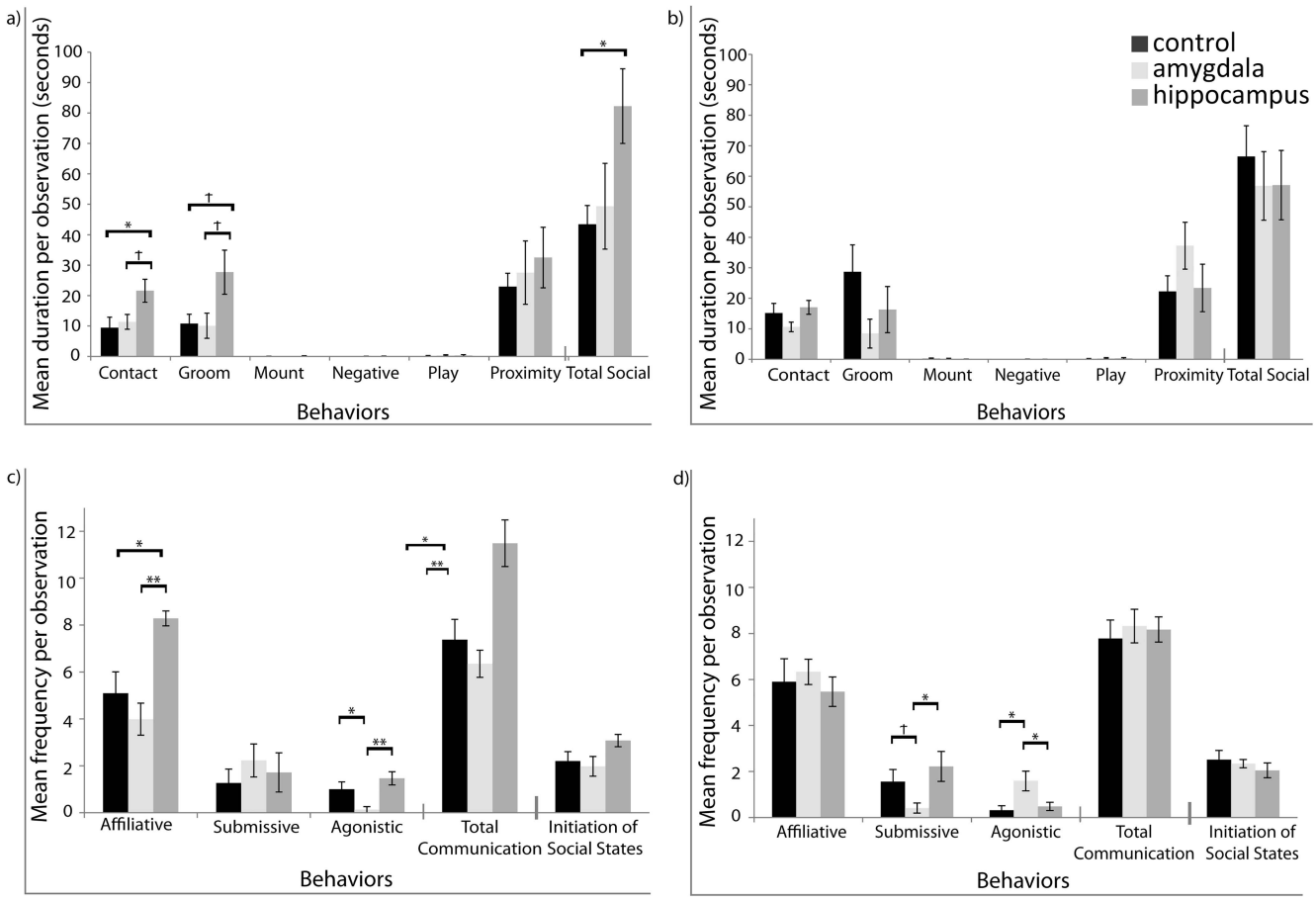

Figure 3. Social behavior with peers. Significant differences between lesion conditions as per independent sample *t* tests are indicated using the following symbol key: † $p < .10$, * $p < .05$, ** $p < .01$. (a) Durations of close social states initiated with female peers. Raw means are presented in the figure although analyses for mount, negative, play and proximity were performed on log transformed data to account for non-normalcy. (b) Durations of close social states initiated by peers to each lesion group. Raw means are presented in the figure although analyses for groom, mount, negative, and play were performed on log transformed data to account for non-normalcy. (c) Frequency of behaviors directed to female peers. Raw means are presented in the figure although analyses for the Submissive category were performed on log transformed data to account for non-normalcy. (d) Frequency of behaviors initiated by peers to each lesion group. Raw means are presented in the figure although analyses for the Submissive category were performed on log transformed data to account for non-normalcy.

durations of playing with toys, $F(2, 12) = 0.655$, $p = .537$, $\eta_p^2 = 0.098$ (analyses on log transformed data), engaging in stereotypies $F(2, 12) = 2.713$, $p = .107$, $\eta_p^2 = 0.311$ (analyses on log transformed data), or sleeping $F(2, 12) = 1.277$, $p = .314$, $\eta_p^2 = 0.176$ (Figure 5a). Despite lesion-based differences in the duration of time spent in nonsocial states, there were no group differences in the frequency of initiating these states, $F(2, 12) = 1.767$, $p = .213$, $\eta_p^2 = 0.227$.

Consistent with findings from this cohort when they were approximately 2 years old, (Bauman et al., 2008), amygdala-lesioned animals engaged in self-directed stereotypies more frequently than the other animals, $F(2, 12) = 4.009$, $p = .046$, $\eta_p^2 = 0.401$; amygdala-lesioned > controls, $p = .025$; amygdala-lesioned > hippocampus-lesioned, $p = 0.039$ (analyses of log transformed data). However, unlike the previous reports, there were no observed differences in the frequencies of whole body stereotypies, $F(2, 12) = 1.564$, $p = .249$, $\eta_p^2 = 0.207$ (analyses of log transformed data; see Figure 5b). Nor were there group differences in exploratory behaviors $F(2, 12) = 1.006$, $p = .394$, $\eta_p^2 = 0.144$; raw means: $M_{amygdala\text{-}lesioned} = 1.105$, $SE_{\text{amygdala-lesioned}} = 0.399$; $M_{hippocampus\text{-}lesioned} = 1.677$, $SE_{\text{hippocampus-lesioned}} = 0.212$; $M_{controls} = 1.950$, $SE_{\text{controls}} = 0.132$).

Lesion Group Classification Based on Patterns of Behaviors With Males

In addition to assessing global frequency and duration of social behavior differences related to early damage, the organization of behavior as it related to lesion group membership

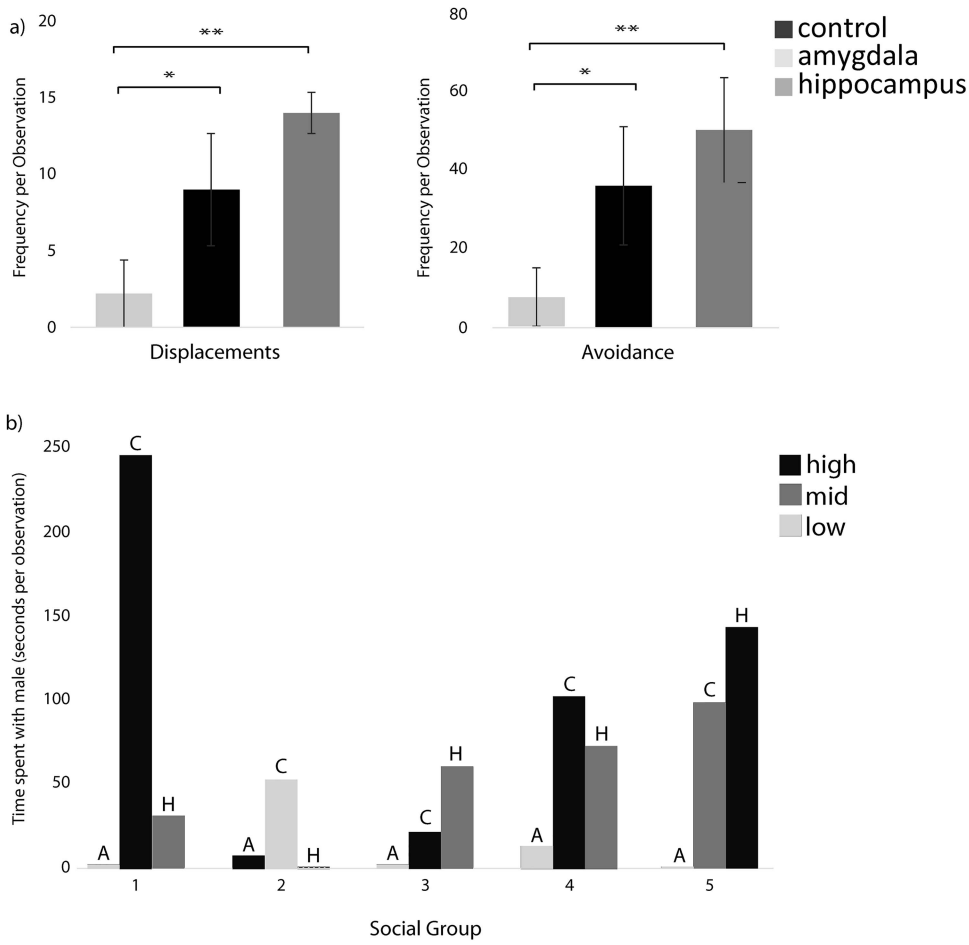

Figure 4. Dominance. Significant differences between lesion conditions as per ANOVA post hoc tests are indicated using the following symbol key: † $p < .10$, * $p < .05$, ** $p < .01$. (a) Frequency of displacements and being avoided. Raw means are presented in the figure although analyses for these behaviors were performed on log transformed data to account for non-normalcy. (b) Time spent with male and social rank. Social rank and lesion group are presented for each social group.

was also evaluated. Additional analyses were conducted using the social behavior data generated during affiliative interactions with the male (e.g., those used in prosocial contexts to establish and maintain social relationships). The organization of affiliative behavior was assessed both when behaviors were generated by the females (directed toward the male) and by the male (directed toward the females). These behaviors included presentation of rump, total contact, anogenital exploration and mount (although the male did not initiate anogenital exploration), huddle, approach and follow (although males never followed females), and vocal and facial signals. First, MANOVAs were evaluated on the dependent variables using lesion condition as a between subjects factor. Then, the same dependent variables were subjected to discriminant function analysis to determine how the relationship between these variables allowed for the classification of animals based on their lesion condition.

Affiliative behaviors generated by the female subjects directed toward the males. The discriminant analysis function revealed two discriminant functions; the first function explained 64.6% of the variance (canonical $R^2 = 0.854$) and the second explained 35.4% of the variance (canonical $R^2 = 0.762$). A combination of both functions differentiated the lesion conditions, $\Lambda = 0.035$, $\chi^2(16) = 28.526$, $p = .027$, but removing the first function revealed that the second function did not significantly differentiate the lesion groups, $\Lambda = 0.238$, $\chi^2(7) = 12.198$, $p = .094$. The following behaviors loaded more highly onto factor one: presentation for groom ($r = .314$), presentation of rump ($r = .285$), and contact ($r = .221$). Anogenital exploration and mount ($r = .385$), huddle ($r = .268$), approach and follow ($r = .196$), and vocalizations and facial signals ($r = .147$) loaded more highly onto the second factor. Groom loaded almost equally on both functions ($r = .266$ for the first function, and $r = .256$ for the second). These functions were able to correctly classify 93.3% of the animals into their lesion groups (5/5 controls, 5/5 amygdala-lesioned, 4/5 hippocampus-lesioned); Press's Q Statistic = 24.3, $p < .001$. The only misclassification was of one hippocampus-lesioned animal who was classified as an amygdala-lesioned animal. See Figure 6a for a visual depiction of the group classification.

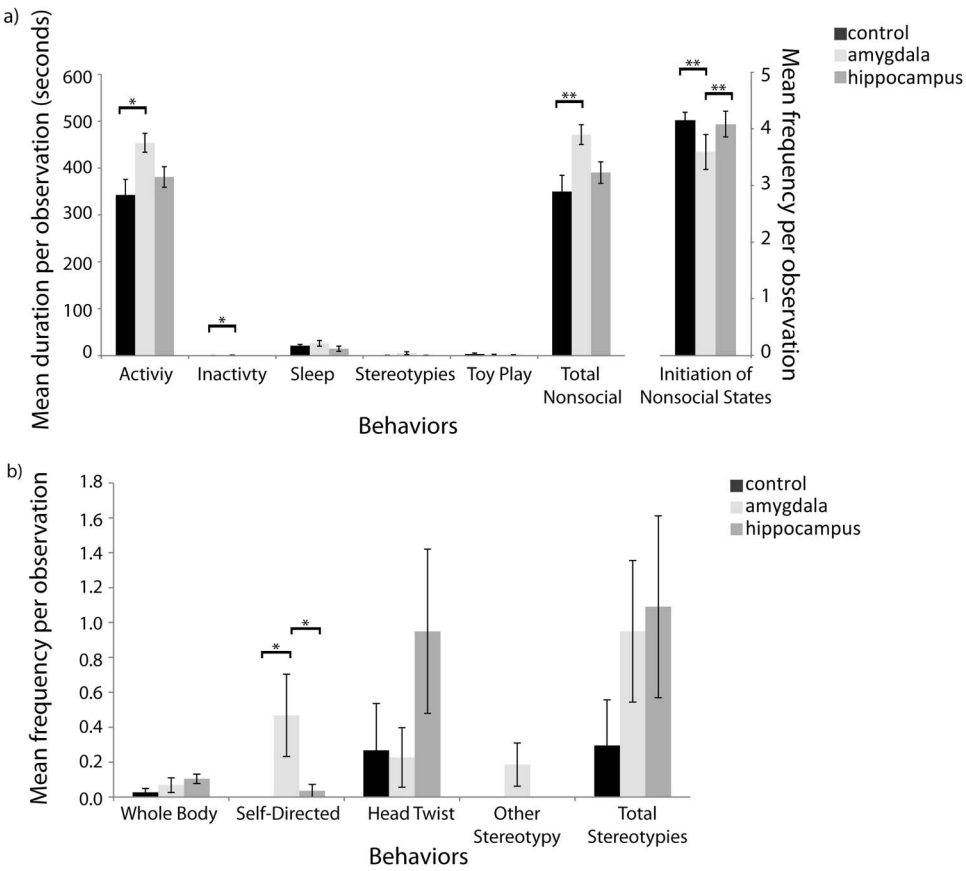

Figure 5. Nonsocial behaviors. Significant differences between lesion conditions as per ANOVA post hoc tests are indicated using the following symbol key: † $p < .10$, * $p < .05$, ** $p < .01$. (a) Duration and frequency of nonsocial states. Raw means are presented in the figure although analyses for inactivity, stereotypies, and toy play were performed on log transformed data to account for non-normalcy. (b) Stereotypies. Raw means are presented in the figure although analyses for all categories were performed on log transformed data to account for non-normalcy.

Affiliative behaviors generated by the males toward the subjects. Discriminant function analyses revealed that the relationship between the specific affiliative behaviors was captured by two functions, the first explaining 93.9% of the variance (canonical $R^2 = 0.929$) and the second explaining 6.1% of the variance (canonical $R^2 = 0.462$). A combination of these functions differentiated the lesion conditions, $\Lambda = 0.038$, $\chi^2(14) = 29.431$, $p = .009$, but removing the first function revealed that the second function did not significantly differentiate the lesion groups, $\Lambda = 0.537$, $\chi^2(6) = 5.589$, $p = .471$. The correlations between the affiliative behaviors and discriminant functions indicated that grooming behavior loaded fairly evenly on both functions ($r = .114$ on function one and $r = .117$ on function two), whereas the following behaviors loaded more highly on factor two: presentation of groom ($r = .738$), total contact ($r = .590$), vocal and facial signals ($r = .450$), approach (males did not follow; $r = .369$), huddle ($r = .357$), and anogenital exploration and mounting ($r = .238$). These functions were able to classify 86.7% of the animals correctly into their lesion groups (5/5 controls, 4/5 amygdala-lesioned, 4/5 hippocampus-lesioned); Press's Q Statistic = 19.2, $p < .001$. The only misclassifications occurred with lesioned animals—one hippocampus-lesioned animal was misclassified as an amygdala-lesioned animal, and one amygdala-lesioned animal was misclassified as a hippocampus-lesioned animal). See Figure 6b for a visual depiction of the group classification.

Lesion-Based Variation in the Timing of Conception

Although focal observations were conducted only over the first month that the groups were together, the groups were maintained for approximately one year with the hope that all animals would become pregnant (allowing for a study of maternal behavior). Of note, one hippocampus-lesioned animal had a tubal ligation prior to this study and so her data is not included here. The time required to become pregnant was evaluated to assess whether it varied by lesion-condition. Specifically, each animal was assigned a value that reflected the total number of days in her social group to the date of conception. The values for the amygdala-lesioned animals that did not conceive were set to the total number of days living in their respective social groups. Because this inherently skewed the data, the data were log transformed. All of the females in this study became pregnant except for two amygdala-lesioned animals.

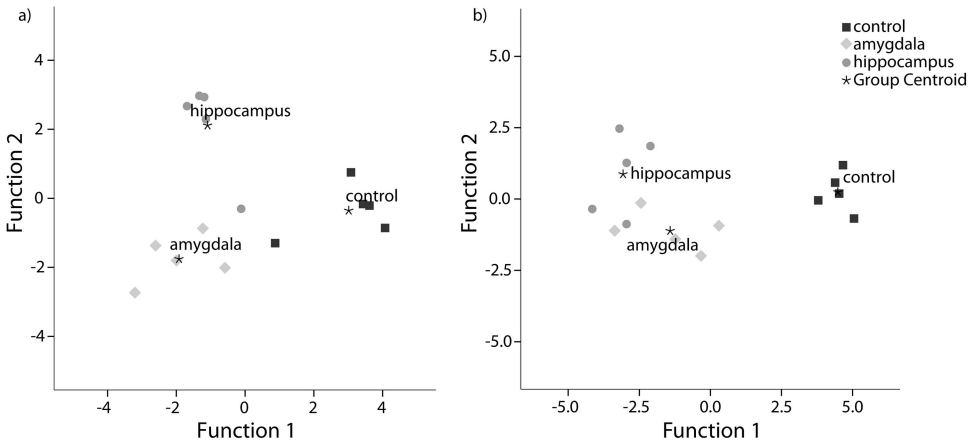

Figure 6. Classification of lesion groups based on (a) affiliative behaviors directed to the males and (b) affiliative behaviors generated by the males toward females from each lesion group. Each individual data point represents a single animal. In both cases, function 1 maximally separated control from lesion groups.

Moreover, the amygdala-lesioned animals conceived significantly later than the control and hippocampus-lesioned animals, $F(1, 12) = 6.435$, $p = .026$, $\eta_p^2 = 0.349$ (log transformed analyses; raw means: $M_{amygdala\text{-}lesioned} = 215.40$ days, $SE_{\text{amygdala-lesioned}} = 70.008$ days; $M_{other\ females} = 79.00$ days, $SE_{other\ females} = 13.689$ days).

Discussion

These data suggest that early amygdala damage, and the neural reorganization that follows early damage, reduces sociability of young adult females when they are given the opportunity to interact with adult males. The social effects of hippocampus-lesioned animals were observed with their female peers—early hippocampus damage *increased* sociability. Lesion-based alterations to social behavior competency were not evident; all animals were capable of generating social behaviors although differences emerged in the patterning of social behaviors in two specific contexts. First, animals with early amygdala damage, compared with neurologically intact controls, spent less time interacting with the male, initiated social interactions less frequently, and initiated communicative signals (e.g., facial signals, vocalizations) less frequently, suggesting a global blunting of sociality. Amygdala-lesioned animals were observed to spend more time alone during which they engaged in higher frequencies of self-directed behavior. Second, subjects with early hippocampal damage initiated longer and more frequent social interactions with their peers than control and amygdala-lesioned animals. Despite this variation, the frequency and duration of the vast majority of hippocampus-lesioned animals' behavior appeared to be equivalent to control animals. Understanding why early damage to hippocampus influences specific social behaviors in specific behavioral contexts is not immediately obvious and therefore is an important avenue for future research.

The results of this experiment are particularly striking given the context in which social behavior was assessed. Female macaques at the age of the subjects in this experiment (approximately 4 years old) are generally motivated to interact with reproductively viable available males (Fedigan, 1982; Kaufmann, 1965; Wilson et al., 1984) or at the very least to form strong social bonds with new social group members. Males were selected based on their robust reproductive histories to ensure that they would interact with the female subjects—all males had fathered at least 5 infants previously. Although the control and hippocampus-lesioned subjects interacted with the male as predicted, amygdala-lesioned animals did not. Animals with early amygdala damage had reduced frequencies and durations of social interactions with the male, and reduced frequencies of consortship behaviors (i.e., those behaviors that signal sexual or reproductive interest that occur prior to sexual interactions). In contrast to previous reports on behavior following amygdala-damage, there was no evidence of hypersexuality in the amygdala-lesioned animals as has been documented in the adult lesion literature (Emery et al., 2001; Kling, 1968; Kling et al., 1970; Kling & Cornell, 1971; Kling, 1974; Klüver & Bucy, 1939; Schreiner & Kling, 1956).

Although there were group differences in overall sociability with the male, there were no lesion-based differences in sexual behaviors per se (i.e., mounting, the presentation of rump for mounting, or self-directed sexual stimulation). The rates of these behaviors were very low for all animals. One possible explanation is that the sampling period ended before animals became sexually active (prior to the breeding season). Another possibility is that heightened periods of social interaction with the male were closely coupled to the females' hormonal cycling. Thus bouts of sexual behavior would occur only when the females were closest to ovulation, particularly for low ranking females (Wallen, 1990). In this case, lesion-based differences in sexual behavior may not have been observed (or may not have occurred during the two 10-min daily focal observations). These possibilities do not, however, explain the observed differences in social interactions with the male that were observed in our sampling of social behavior.

Although the present results indicate that early damage to the amygdala or hippocampus alters the patterning of social behavior in young adulthood in significant, yet subtle ways, they do not speak to the specific mechanisms that underlie this variation. There are a number of possible mechanisms to be considered and ex-

plored in future work. Given the unique number of sex hormone receptors in the amygdala and its regulation of magnocellular and parvicellular nuclei of the hypothalamus, early damage to the amygdala could potentially delay ovulation or appreciably alter the interplay between hormonal function and social behavior (for a review, Scherf, Smyth, & Delgado, 2013). The amygdala is heavily connected to the hypothalamus (Amaral, Veazey, & Cowan, 1982) and the ventromedial nucleus of the hypothalamus plays a critical role in the regulation of female sex hormones (Griffin & Flanagan-Cato, 2011). Alteration of cycle or hormone level would likely have important consequences for social behavior (Wallen & Winston, 1984). Studies of animals with early amygdala lesions suggest that amygdala damage can impact hormonal function but the timing of amygdala damage is critical to the hormonal outcomes. For example, studies of early medial amygdala nucleus damage in rats have shown variable alteration of puberty based on the timing of amygdala damage (e.g., lesions at 15 days shift puberty later, Döcke, Rohde, Lange, & Dörner, 1980; lesions at 21 days shift puberty earlier, Döcke, 1974; Döcke, Lemke, & Okrasa, 1976; Döcke et al., 1980; and lesions at 26 have no effect, Döcke et al., 1976; Döcke et al., 1980). Rhesus macaques who sustain amygdala damage at 10–13 months of age show no changes to pubertal timing (Norman & Spies, 1981) but when the damage occurs at 24 days of age, they have earlier menarche and ovulation (Stephens et al., 2015). Taken together, these studies suggest that, depending on the age at which amygdala damage is sustained, menarche and ovulation may occur earlier, later, or have no effect at all. Unfortunately, no hormonal data were collected at the time of our experiments—only observed menarche data, which is not a reliable indicator of whether animals had experienced first ovulation at the onset of the present experiment (Wilson & Gordon, 1989). Group differences in social behavior with the male, therefore, may have in fact been a result of changes to hormonal cycling resulting from early amygdala damage. In this view, social behavior with the male might have been altered in the amygdala-lesioned animals not because of amygdala damage per se but rather because of the downstream hormonal consequences of amygdala damage. To evaluate this possibility, future studies should monitor hormone levels throughout maturation so that they will be able to map variation in hormone levels to variation in social behavior. Regardless, the altered social behavior of these animals points to the importance of the amygdala in development.

Another possible mechanism that might account for the observed lesioned-based variation in behavior relates to the social structure of the groups. Generally, in single-male, multifemale groups, higher-ranking females have greater access to the male because they are not susceptible to repercussions of agonistic behavior from their peers (Wallen, 1990). It is possible that the group differences in social behaviors with the male were due to amygdala-lesioned animals' having limited access to him because of their low social ranks. A previous experiment with these subjects demonstrated that early amygdala damage altered dominance status such that all but one amygdala-lesioned animal was the lowest ranking (Bauman et al., 2008). This dominance structure persisted across development such that in this Experiment 4 of the 5 amygdala-lesioned animals were lowest ranked in their group (based on the frequency of displacements by peers), potentially contributing to their reduced sociability with males.

The present findings also demonstrate that early amygdala or hippocampus damage alters how animals are perceived by others. Specifically, the adult males in the groups did not behave toward amygdala-lesioned animals as they did toward the other females. Further, even though there were no remarkable differences between hippocampus-lesioned and control animals in their propensities to affiliate with the males, the males did distinguish between them (and the amygdala-lesioned animals as well). Overall, the males generated higher frequencies of affiliative signaling to the control females, and spent more time with the controls than they did with the hippocampus- or amygdala-lesioned animals. These differences were further elaborated in the classifier analyses. When evaluating the relationship between affiliative behaviors generated by the male toward the females, animals were correctly classified into their a priori lesion groups with 86.7% accuracy. This pattern of effects suggests that it is not simply the frequency of affiliative signals generated by the male that differed between lesion groups but also the pattern of execution of these behaviors. Notably, these analyses also suggest that the males' patterns of affiliative social behavior were able to distinguish not only between amygdala-lesioned animals and controls, but also between hippocampus-lesioned animals and controls, and amygdala-lesioned animals and hippocampus-lesioned animals. This is especially telling because control and hippocampus-lesioned animals looked essentially the same when only the frequency and duration of behaviors with the males was assessed. The classifier analyses capitalize on unique information that is able to differentiate the groups above and beyond analyses of frequency and duration. Again, further study of the impact of early brain damage on hormonal cycling will be critical to determine the mechanisms subserving these effects—it is possible that males were more motivated to interact with females experiencing a particular hormonal state.

One goal of the current study was to investigate how amygdala-lesioned females would perform as mothers given the important role of the amygdala in maternal behavior and interest in infants (Sheehan, Paul, Amaral, Numan, & Numan, 2001; Toscano, Bauman, Mason, & Amaral, 2009). Thus, the groups were left together for a full year with the hope that all females would become pregnant. All of the control and hippocampus-lesioned animals and three of the five amygdala-lesioned animals did become pregnant, although the amygdala-lesioned animals became pregnant significantly later than all others. It is possible that the two amygdala-lesioned animals did not become pregnant during the experiment because they had not begun cycling when the experiment began. Alternatively, they may have not become pregnant due to their altered, limited behavioral interaction with the males. Nevertheless, there were many complications with the pregnancies that did occur. As is common for primiparous females (Gagliardi et al., 2007), a number of the fetuses were not viable. Additionally, as is also common for first time mothers (Gagliardi et al., 2007), some of the animals were unable to mother their infants. Only four of the females (2 controls and 2 hippocampus-lesioned animals) were able to raise their infants in the social group, which unfortunately eliminated the possibility of a formal study of maternal behavior.

Despite not being able to conduct a formal study of maternal behavior, the present findings do point to important lesion-based differences in social behavior at a critical developmental time point. The overall blunted sociability and later onset of pregnancy of the amygdala-lesioned females suggests that early damage to the amygdala either impairs social behavior per se or impairs component processes necessary for execution of normal social behavior. These

findings also suggest that healthy, socially reared animals perceive lesion-based variations in social behavior even if those differences are extremely subtle (i.e., those of the control and hippocampus-lesioned animals). Thus, using alternative experimental and statistical methods for evaluating variation in nonhuman primate social behavior is incredibly important. By employing these quantitative tools to evaluate patterns of social behavior across the life span in this cohort, we hope to elucidate the mechanisms by which early brain damage, and in particular early amygdala damage, generate variation in social and affective behavior.

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