growth in saline soil patches, or both. There is independent evidence for the involvement of mechanism (ii) in the preferential colonization of nonsaline soil observed in this experiment. In a field transplant experiment where ramets of these clones were confined to uniform conditions of high (0.8 to 1.4 percent) or low (<0.2 percent) salinity (*6*), all nine genotypes proved capable of vegetative proliferation in both soil types. For every clone the difference in shoot production per surviving ramet in saline compared to nonsaline soil was smaller than the difference between the two ends of each pot in the greenhouse "choice" experiment (sign test, $n = 9, P < 0.002$). Thus, the colonization rate of two contrasting habitats differed more strongly when individual plants were offered a "choice" (Table 1) than when they were not.

Opportunities for habitat selection in natural populations depend on the rate of rhizome growth into new territory. In the field transplant experiment, rhizome growth in nonsaline soil was extremely limited; only 3 percent of daughter ramets occurred at 20 cm or farther from their parent plants; (range, 0 to 60 cm; $n = 1130$). Dispersal in saline soil was dramatically higher, with 28 percent of all ramets appearing at 20 cm or farther from their parents (range, 0 to 80 cm; $n = 982$). There was striking genotypic variation in shoot dispersal in saline soil: clonal mean dispersal distances ranged from 5.0 to 31.6 cm, and clonal differences accounted for 30 percent of the total variance in shoot dispersal distance (*10*). The greater dispersal achieved by plants in saline soil was unexpected, since salt severely depresses plant biomass accumulation. However, this commitment to extensive rhizomatous spread increases the rate at which plants in saline soil encounter new territory, thereby increasing the probability of locating favorable microsites. Clonal differences in dispersal result in large differences in the rate of sampling new habitats and ultimately determine each genotype's opportunity to exercise adaptive habitat choice.

The often striking associations between plant genotypes and local environments are usually thought to depend for their maintenance on high mortality rates among nonadapted immigrant genotypes (*11*). Habitat selection may permit clonal plants to become genetically segregated among habitats without the severe energetic and demographic costs of selective mortality. The capacity of western ragweed (*4*) and many other clonal species (*12*) to share resources among ramets through rhizome connections further increases the efficiency of colonization and habitat exploration in these species. The results show that active habitat choice could be a significant mechanism producing genotype-microenvironment correlations in natural populations of clonal plants. It is not yet possible to distinguish between this process and selective mortality.

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5. Salt tolerance index was calculated as the mean biomass accumulation after 6 weeks' growth in 1 percent NaCl saline soil of four cloned replicates per genotype in a common greenhouse environment. Assays of over 100 *A. psilostachya* genotypes indicate a fourfold range in salt tolerance index.
6. There were 13 to 16 replicates of 13 genotypes transplanted into saline (0.8 to 1.4 percent salinity) and nonsaline soil (<0.2 percent salinity) in Lancaster County, Nebraska. Overall survivorship through two growing seasons in saline soil was 0.42 ($n = 205$), significantly lower than 0.83 in nonsaline soil ($n = 162$; $\chi^2 = 62.63$; $P < 0.0001$). Clonal mean survivorship showed a significant positive regression on salt tolerance in saline soil (range 0.25 to 0.60; analysis of variance on arcsin square-root transformed data: $F = 14.95$; d.f. = 1,11; $P < 0.003$; $r^2 = 0.58$) but not in nonsaline soil (range 0.54 to 1.00; $F = 0.87$; d.f. = 1,11; $P > 0.35$; $r^2 = 0.07$). Soil salinities were determined as described by L. A. Richards [*Diagnosis and Improvement of Saline and Alkaline Soils: Agricultural Handbook 60* (Government Printing Office, Washington, D.C., 1954)].
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Amygdalectomy Impairs Crossmodal Association in Monkeys

Abstract. *Monkeys trained on both visual and tactual versions of an object memory task (delayed nonmatching-to-sample) received bilateral ablations of either the amygdaloid complex or the hippocampal formation of the brain. Although both groups performed well on the two intramodal versions (visual-to-visual and tactual-to-tactual), the amygdalectomized monkeys were severely impaired relative to the hippocampectomized monkeys on a crossmodal version (tactual-to-visual). The findings suggest that the amygdala is critical for certain forms of crossmodal association and that the loss of such associations underlies many of the bizarre behaviors that make up the Klüver-Bucy syndrome.*

The amygdaloid complex has long been considered essential for the sensory arousal of affective responses, that is, for the association of sensory stimuli with emotions (*1, 2*). We now report that the amygdala is also essential for associations that have no obvious emotional content. Amygdalectomized monkeys, like their controls, accurately recognized objects both visually and tactually; yet, unlike their controls, they failed to recognize by vision an object they had just examined by touch. This crossmodal impairment may help explain some of the dramatic and puzzling effects of amygdalectomy on naturally occurring behavior (*1*).

Six cynomolgus monkeys (*Macaca fascicularis*) were first trained on a one-trial visual memory task—delayed nonmatching-to-sample—with a set of 40 visually and tactually distinctive objects that differed in color, size, shape, texture, and compressibility (*3*). The objects

were randomly paired each day to provide 20 daily trials without object repetition. On each trial, the monkey found a banana pellet by displacing the sample object overlying the central well of a three-well test tray and then, 10 seconds later, found another pellet if it avoided the sample and displaced the paired alternative, each object now overlying a lateral well (*4*). The objects were mounted on corks that fit snugly into the wells, forcing the animals to grasp and lift, and, thereby, palpate the objects in order to obtain the reward (*5*). After reaching the criterion of 90 correct responses in 100 trials, the monkeys received training in a light-dimming phase on a titrated schedule (*6*), until they could perform the task in total darkness (*7*). After further testing (*8*), three of the monkeys received bilateral ablations of the amygdaloid complex and ventromedially adjacent cortex, and the three others, bilateral ablations of the hippocampal formation and parahippocampal gyrus. Details of surgery and histological verification of the lesions have been presented elsewhere (*3*). After surgery, the animals were retrained on the two intramodal versions of the task in reverse order, that is, tactile followed by visual (*9*). Although the amygdalectomized monkeys relearned the tactile task more slowly than the hippocampectomized monkeys (*3*), both groups reattained criterion on both intramodal versions. At this stage in the experiment, the scores of the two groups were nearly equal (Table 1).

The monkeys were then confronted for the first time with the crossmodal task, which had the same basic design as the intramodal versions, except for a within-trial change of lighting: the sample was presented for familiarization in the dark, but the sample and alternative were presented for the choice test in the light. A response was scored the moment the monkey touched either object, ensuring that choice was based on visual cues only. Hippocampectomized monkeys averaged about 90 percent correct responses over the 500 trials (Fig. 1). In contrast, the amygdalectomized monkeys averaged only about 55 percent correct responses ($U = 0$, $P = 0.05$) (*10*). Neither group's performance improved appreciably over the course of testing.

The amygdalectomized monkeys' impairment cannot be attributed to a perceptual, attentional, or motivational loss, since they performed well on the intramodal versions of the task. For the same reason, their impairment cannot be ascribed to a short-term memory loss in either sensory modality. By elimination, their deficit appears to be in long-term, crossmodal, associative memory.

Table 1. Mean percent correct responses (and ± standard deviation) in 100 trials obtained by amygdalectomized (A) and hippocampectomized (H) monkeys on the two intramodal versions of delayed nonmatching-to-sample. The scores of the two groups do not differ significantly on either version (Mann-Whitney U test, $P > 0.10$ for each comparison, $n = 3$ for each group).

Group	Touch	Vision
A	90.3 (0.6)	96.3 (3.5)
H	92.0 (1.7)	99.3 (0.6)

For many years, investigators had great difficulty demonstrating crossmodal associations in normal monkeys (*11*). The first clearly successful experiments used matching-to-sample procedures, though with only two different stimuli per session (*12*). In our study we used 40 different stimuli per session and consequently required the storage and retrieval of a large number of specific crossmodal associations (*5, 13*). Experience with the objects in the light gave the animals the opportunity to acquire these visuotactile associations (if they were not already available by transfer from everyday experience), and the control monkeys did acquire them, as indicated by their immediate application across modalities of the well-learned nonmatching rule.

It has repeatedly been proposed that crossmodal associations might be mediated by polysensory cortical areas (*14*). Tests of this proposal have yielded mixed results (*15*), however, and studies reporting impairment in crossmodal matching after cortical lesions did not provide evidence that both forms of intramodal matching were intact. By fulfilling this requirement, our study indicates that the amygdala, a subcortical structure receiving polymodal inputs, does participate in crossmodal associations. The magnitude of the impairment, which seems to be greater than any problem-solving deficit reported previously in amygdalectomized monkeys (*3, 16*), suggests that crossmodal association could be one of the amygdala's major functions. If so, it would help explain some of the bizarre behaviors that monkeys display after amygdalectomy, such as indiscriminate and repeated examination of inedible objects by touch, by smell, and by taste. This phenomenon, which Klüver and Bucy interpreted as psychic blindness and hypermetamorphosis, may be due in part to the loss of crossmodal associations. That is, the amygdalectomized monkey, seeing an object, may be unable to recall how that object feels, and even after feeling and smelling it may still not recall its taste.

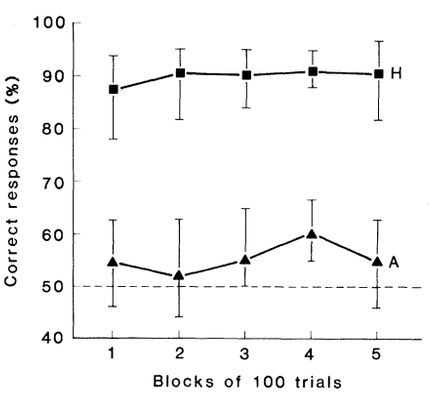

Fig. 1. Performance of amygdalectomized (A) and hippocampectomized (H) monkeys on crossmodal delayed nonmatching-to-sample. The dashed line indicates chance level of performance. Vertical bars indicate the range of scores for the three animals in each group.

How might the amygdala mediate crossmodal associations? Anatomical evidence indicates that each sensory system projects to the amygdaloid complex either directly (olfaction) or indirectly (vision, taction, audition, and gustation) through a series of cortical fields running from the primary sensory area to a final station in the anterior temporo-insular region (*17*). Amygdaloid neurons, in turn, project back to these cortical sensory systems (*18*). It has been proposed that the representation of a stimulus in a given sensory modality is stored in the cortical areas serving that modality (*19*). If this were the case, the cortico-amygdalo-cortical pathways may allow the formation of connections between representations stored in different sensory systems. In this way, the direct activation of a representation in one modality through perception of an object could indirectly activate connected representations in other modalities, thereby evoking recall of that object's unperceived sensory qualities.

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7. The animals were observed on a video monitor linked to an infrared-sensitive camera. The camera, together with the infrared light source, was mounted directly over the test area. To aid the animals in locating the objects in the dark, three light-emitting diodes were recessed in the testing board, one 4.5 cm in front of each well. During the sample presentation, only the central diode was lit, whereas during the test part of the trial, both lateral diodes were lit. That the animals could not see the objects under these conditions is indicated by the following: (i) near the end of the light-dimming phase all animals' scores dropped from criterion to chance levels; (ii) the monkeys did not subsequently reattain criterion levels of performance until they were observed on the video monitor to be deliberately comparing the objects by touch; and (iii) neither of two dark-adapted human observers tested in the same situation could detect the presence of the objects visually.
8. The animals were given a tactual memory performance test after they attained the criterion in the dark. In this performance test the delays between sample presentation and choice were increased from 10 seconds to 30, 60, and finally 120 seconds, in blocks of 100 trials each. In this and all other stages of the experiment, the monkeys were trained at the rate of 20 trials per day, 5 or 6 days per week.
9. The animals were retrained first in the dark to obtain a measure of tactile memory uncontaminated by any postoperative visual experience with the objects. After being retrained in each modality, animals were given the performance test described in (*8*) in that modality. By the end of this training, the animals had received an average of about 2000 trials with the same set of objects on the two intramodal tasks. Mean trials for amygdalectomized and hippocampectomized monkeys, respectively: Before surgery—light, 513 and 540; dark, 433 and 413. After surgery—dark, 920 and 427; light, 400 and 400.
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Methionine and Leucine Enkephalin in Rat Neurohypophysis: Different Responses to Osmotic Stimuli and T_2 Toxin

Abstract. *Specific radioimmunoassays were used to measure the effects of hypertonic saline (salt loading), water deprivation, and trichothecene mycotoxin (T_2 toxin) on the content of methionine enkephalin (ME), leucine enkephalin (LE), α-neoendorphin, dynorphin A, dynorphin B, vasopressin, and oxytocin in the rat posterior pituitary. Concentrations of vasopressin and oxytocin decreased in response to both osmotic stimuli and treatment with T_2 toxin, but the decrease was greater with osmotic stimulations. Similarly, concentrations of LE and dynorphin-related peptides declined after salt loading and water deprivation; LE concentrations also decreased after treatment with T_2 toxin. The concentration of ME decreased after water deprivation, did not change after salt loading, and increased after T_2 toxin treatment. The differentiating effects of these stimuli on the content of immunoreactive LE and ME are consistent with the hypothesis that LE and ME may be localized in separate populations of nerve endings with different roles in the posterior pituitary.*

Some investigators have reported that enkephalin-containing peptides inhibit the release of vasopressin and oxytocin from the posterior pituitary (*1*); others have reported that these peptides facilitate their release (*2*). At least two precursors provide enkephalin-containing peptides in the rat brain: proenkephalin A and proenkephalin B (prodynorphin). Proenkephalin A contains four copies of methionine enkephalin (ME) and one copy each of leucine enkephalin (LE), ME-Arg-Phe, and ME-Arg-Gly-Leu (*3*). Some of these peptides in turn are part of longer peptide sequences (for example, peptides I, F, E, and B) (*4*). Proenkephalin B contains three LE sequences, each of which represents the amino terminus of α-neoendorphin, dynorphin A, or dynorphin B (*5*). Different cells seem to process the enkephalin precursors in different ways. For example, chromaffin cells of the adrenal medulla make and store substantial amounts of high molecular weight enkephalin precursors as well as enkephalin octa- and heptapeptides (*4*). In the brain, however, most of the enkephalin measured in radioimmunoassays is present as ME, LE, ME-Arg-Phe, and Me-Arg-Gly-Leu. There is little or no high molecular weight material reacting with antibodies to enkephalin (*4*). In the posterior pituitary there are substantial amounts of ME and LE (*6–8*) but very little or no ME-Arg-Phe and ME-Arg-Gly-Leu (*8*). Thus, the proenkephalin A molecule can give rise to a variety of cellular secretory products in different tissues depending on the extent and pattern of its intracellular processing.

Proenkephalin B–derived peptides are present in high amounts in the posterior pituitary and throughout the brain but in low amounts in the adrenal gland (*9, 10*). Although proenkephalin B contains three LE sequences, it has been argued that LE derives exclusively from the single copy of LE in the proenkephalin A precursor (*4*). This is because the molar ratios of ME to LE in various areas of the brain (~4:1) correspond roughly to the ratio in the proenkephalin A precursor and because the regional distributions of dynorphin and enkephalin (LE or ME) in the brain differ (*11*). However, we have suggested that LE in the rat substantia nigra is derived mainly from proenkephalin B–containing neurons (*12*). To examine the functional relations among LE, ME, and dynorphin in the posterior pituitary, we used specific radioimmunoassays to measure amounts of ME, LE, and dynorphin-related peptides as well as vasopressin and oxytocin in the posterior pituitary of male Sprague-Dawley rats (200 to 250 g in body weight) under conditions of enhanced secretory activity. We found differentiating alterations in concentrations of LE and ME in the neural lobes of rats after hypertonic saline (salt loading) and treatment with