placed in the arenas. Occasionally the first or second female was upon a host when she was removed from the arena. These hosts are not included in Fig. 1b since the female may not have completed oviposition.

19. Other theories predict that circumstances can favor small (or young) individuals to bias investment to male function. This is favored, for example, when reproductive success increases with size in females, but does not increase with size in males [R. R. Warner, *Am. Nat.* **99**, 419 (1975); E. L. Charnov, *ibid.* **113**, 715 (1979)]. The relative LMC model presented in this paper shows that a size effect results from LMC itself. E. L. Charnov has found that similar arguments apply to barnacles (personal communication).
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Reactivation of Infant Memory

Abstract. *Three-month-old infants learned to activate a crib mobile by means of operant footkicks. Retention of the conditioned response was assessed during a cued recall test with the nonmoving mobile. Although forgetting is typically complete after an 8-day retention interval, infants who received a reactivation treatment—a brief exposure to the reinforcer 24 hours before retention testing—showed no forgetting after retention intervals of either 2 or 4 weeks. Further, the forgetting function after a reactivation treatment did not differ from the original forgetting function. These experiments demonstrate that (i) "reactivation" or "reinstatement" is an effective mechanism by which early experiences can continue to influence behavior over lengthy intervals and (ii) memory deficits in young infants are best viewed as retrieval deficits.*

The pervasive influences of early experiences on later behavior have been extensively documented, as have early memory deficits or "infantile amnesia" (*1*). Considered jointly, these phenomena pose a major paradox for students of development: How can the effects of early experiences persist into adolescence and adulthood if they are forgotten during infancy and early childhood? Campbell and Jaynes (*2*) proposed a resolution to this paradox in terms of reinstatement, a mechanism that maintains a memory which would otherwise be forgotten through occasional reencounters with the original training conditions over the period of development. Any given reencounter, however, would be insufficient to promote new learning in organisms lacking the early experience. Spear (*3*) attributed the efficacy of reinstatement procedures to improved retrieval produced by the reactivation of a sufficient number (or kind) of existing but otherwise inaccessible attributes of the target memory. He hypothesized that reexposure to stimuli from the original training context, which had been stored as attributes of the memory, could prime or arouse other attributes that represented the original experience, increasing their accessibility and, thus, the probability of their retrieval.

"Reinstatement" or "reactivation" has been demonstrated in young and adult rats (*2*, *4*, *5*) and in grade-school children (*6*). We now report that a reactivation treatment can alleviate forgetting in 3-month-old infants after a retention interval as long as 4 weeks and that the forgetting function after a reactivation treatment is similar to the function after original training.

Our procedures were modeled after those of animal memory studies in which the experimenter trains a specific response in a distinctive context and later returns the subject to that context to see if the response is still produced. Because the retrieval cues are contextual and response production is assessed before reinforcement is reintroduced, the procedure is analogous to a test of cued recall (*3*).

In our studies, footkicks of 3-month-olds were reinforced by movement of an overhead crib mobile. The infant controlled both the intensity and frequency of the mobile movement by means of a ribbon connecting the ankle (Fig. 1A) with the hook from which the mobile hung. This procedure, "mobile conjugate reinforcement," produces rapid acquisition and high, stable response rates attributable to the contingency and not to behavioral arousal (*7*). During nonreinforcement phases (baseline, retention tests, extinction), the mobile remained in view but was hung from a second mobile stand with no ribbon attachment and could not be activated by kicks.

Infants received three procedurally identical sessions in their home cribs. The first two were training sessions, spaced by 24 hours; the third followed a lengthy retention interval. Each session consisted of a 9-minute reinforcement phase preceded and followed by a 3-minute nonreinforcement period. In session

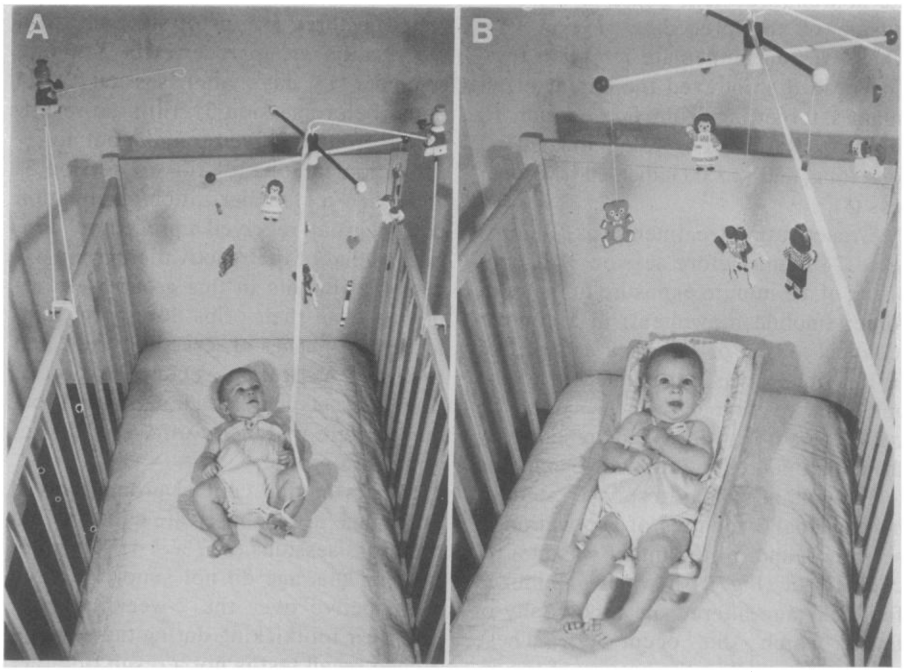

Fig. 1. (A) An infant during a reinforcement phase with the ankle ribbon attached to the same suspension bar as that from which the mobile hangs. The empty mobile stand, clamped to the crib rail at the left, will hold the mobile during periods of nonreinforcement. (B) The same infant during a reactivation treatment. The mobile and ribbon are attached to the same suspension hook, but the ribbon is drawn and released by the experimenter (not shown), concealed from the infant's view at the side of the crib. Also not shown is the empty stand, positioned as before. The infant will be exposed to the reinforcer (the moving mobile) for only 3 minutes 24 hours before retention testing. [Photograph by Breck P. Kent]

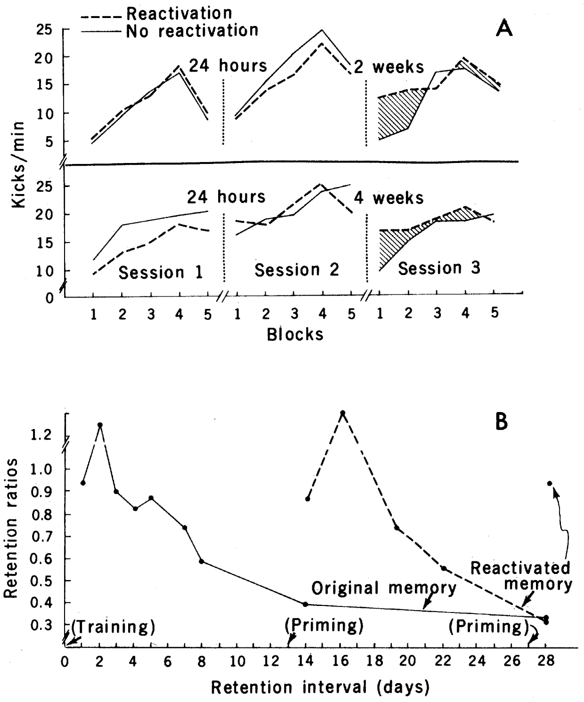

Fig. 2. (A) Mean kicks per minute during training (sessions 1 and 2) and an identical session (session 3) occurring either 2 or 4 weeks after the completion of session 2. Blocks 1 and 5 are nonreinforcement phases; performance during long-term retention tests (block 1, session 2 or 3) is expressed as a fraction of the infant's performance during immediate retention tests (block 5, session 1 or 2, respectively). The reactivation group received a reminder 24 hours before the 2- or 4-week session; the facilitating or priming effect of the reactivation treatment is indicated by the hatched area, session 3. (B) Retention ratios after 2 days of training (solid line) or 2 days of training plus a reactivation treatment (broken line); priming occurred 13 days after training for all points connected by broken lines or 27 days after training for the single data point at the 28-day retention interval. Each data point represents at least five infants.

1, the initial 3-minute period defined the baseline; in sessions 2 and 3, it was a long-term retention test of the effects of prior training. Total footkicks during this test (*B*) were expressed as a fraction of the infant's total kicks during the 3-minute nonreinforcement phase at the conclusion of the preceding session (*A*), which was an immediate retention test. The ratio *B*/*A* indexed the extent of an infant's forgetting from one session to the next. Ratios of ≥ 1.00 indicated no forgetting, and < 1.00 indicated fractional loss (*8*).

A reactivation treatment was administered 24 hours before session 3. It consisted of a 3-minute exposure to the reinforcer (mobile movement) in a context identical to that of session 2 except that (i) the ribbon was not connected to the ankle but was draped over the side of the crib, where it was drawn and released by the experimenter at a rate corresponding to each infant's mean response rate during the final 3 minutes of acquisition in session 2; and (ii) the infant was in a reclining seat (Fig. 1B), which minimized footkicks and altered the topography of those which did occur (*9*). These changes, as well as the brevity of the reminder, precluded the opportunity for new learning or practice during a reactivation treatment. Footkicks were recorded by the experimenter and, independently, by a second observer present for at least a third of the sessions and naive with respect to group assignment and session number. Pearson product-moment reliability coefficients were > .95 for all studies reported here.

In study 1, retention of conditioned footkicks was assessed 2 weeks after training. Infants [mean ($\bar{X}$) age = 88.4 days, standard error (S.E.) = 3.3] were tested in three groups of six each: (i) a reactivation group received a 3-minute reminder 13 days after session 2 (24 hours before session 3); (ii) a no-reactivation group received training but no reactivation treatment prior to session 3; and (iii) a familiarization/reactivation control group received a procedure identical to that of the reactivation group except that infants in this group were removed from their cribs during the reinforcement phases of sessions 1 and 2 and thus had no training before session 3. The rates at which their reminders occurred were matched to those of the reactivation group.

Infants in this control group showed no change in response rate either within or across sessions (all t's < 1). Thus, infants of this age do not simply become more active over the 2-week interval, and their footkicking during the session-3 cued recall test is not a result of either elicited familiarity reactions or the reactivation treatment per se. The acquisition curve of this group in session 3, when reinforcement was introduced for the first time, was indistinguishable from the session-1 learning curves of the other two groups. An analysis of variance with repeated measures over sessions and blocks confirmed that response rates of the reactivation and no-reactivation groups did not differ during training (Fig. 2A). A 2 by 2 analysis of variance over retention ratios yielded a significant group-by-sessions interaction: Although 24-hour retention ratios did not differ, the 14-day retention ratio of the reactivation group significantly exceeded that of the no-reactivation group ($P < .01$), whose ratio reflected a return to baseline performance of session 1 (Fig. 2B). The retention ratio of the reactivation group was as high as in the 24-hour measure. Thus, both prior training and a reminder are prerequisite for reactivation.

In study 2, we repeated the procedure with 18 infants ($\bar{X}$ age = 76.9 days, S.E. = 2.0) but doubled the length of the retention interval. The reactivation group ($N = 9$) received a reminder 27 days after training, and retention was assessed the next day. A significant group-by-sessions interaction ($P < .03$) again confirmed the superior retention of the reactivation group in session 3 relative to that of the no-reactivation group ($N = 9$), which received no reminder during the retention interval (Fig. 2B). As before, the groups had not differed during training (Fig. 2A) or in 24-hour retention performance. The 28-day ratio of the no-reactivation group reflected performance equivalent to their session-1 baseline level. The retention ratio (.96) of the reactivation group is remarkable in view of the relatively young age of the infants during training and the relatively large portion of their lives that 4 weeks constitutes.

In study 3, we determined the course of forgetting following a reactivation treatment. Twenty infants ($\bar{X}$ age = 90.0 days, S.E. = 1.3) received a reactivation treatment 13 days after training as described for study 1; however, session 3 now occurred 3, 6, 9, or 15 days ($N = 5$ per interval) after the reminder. This corresponded to 16, 19, 22, or 28 days, respectively, after the completion of training. The session-3 retention ratios, along with those of the six infants tested 1 day after a reactivation treatment in study 1, were compared with retention ratios describing the original forgetting function. [We had previously obtained this function from 69 infants in a number of different experiments (*10*) carried out according to the same procedure as that used with the no-reactivation groups of this report.] At least five infants per retention interval contributed data 1, 2, 3, 4, 5, 7, 8, or 14 days after training (Fig. 2B). The no-reactivation group of study 2, tested after a 28-day retention interval, was also a control group for the reactivation group tested 15 days after the reminder (28 days after training).

Figure 2B is a composite of retention ratios of all groups tested after 2 days of training only ("original memory" function) or after 2 days of training plus a reactivation treatment ("priming") given either 13 (studies 1 and 3) or 27 (study 2) days after training ("reactivated memory" function). A one-way analysis of variance over all data points except that of the study-2 reactivation group indicated that ratios differed reliably as a function of retention interval ($P < .025$) and provided the error term for individual comparisons between means (Duncan's multiple range test). The latter indicated that the apparent increase above 1.00 in retention ratios in each function (Fig. 2B) was reliable; also, ratios of groups tested 8 (original memory function) and 19 (reactivated memory function) days after training did not differ from ratios of no-reactivation groups tested after retention intervals of 14 and 28 days, respectively. Regression analyses indicated that retention was a linear decreasing function of time since either training ($P < .005$) or priming ($P < .005$). Although the linear model provided a relatively poor fit in each instance, the intercepts and slopes of the two functions did not differ (t's < 1). Thus, forgetting of a reactivated memory followed the same temporal course as forgetting of the original experience.

Our findings confirm Campbell and Jaynes' (*2*) proposition that reinstatement is a potent mechanism through which experiences of early infancy can continue to influence behavior. An infant's reencounters with contextual aspects of prior training or an earlier experience can prime or recycle the remaining memory attributes and enhance access to them, alleviating forgetting which otherwise appeared complete weeks earlier. Moreover, a reencounter with the original context can maintain access to the target memory with the same efficacy as original training. Our findings also implicate reinstatement as the mechanism which, during infancy, facilitates the acquisition of the vast amount of learning characteristic of that period of development.

More generally, our findings support a distinction between availability and accessibility of information in memory and imply that failures to observe retention in infants should be discussed in terms of retrieval failures rather than memory deficits (*3, 4*). We think that procedures that improve accessibility to important retrieval cues will radically alter current views of infant memory (*11*) and that conditioning procedures, which permit a direct assessment of retention in infants, offer a promising means by which to bridge the gap between human and animal memory research.

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8. Because operant levels are typically doubled or tripled during acquisition, retention ratios of .30 to .40 usually indicate performance at operant level. A 3-minute period of nonreinforcement at the conclusion of initial training sessions does not typically extinguish responding in infants 11 to 13 weeks of age.
9. During the reactivation treatment, infants produced responses at a rate of 0 to 2 kicks per minute; operant levels are typically 8 to 11 kicks per minute. In the infant seat, infants rarely exhibit the vertical leg thrusts characteristic of conditioned responding; rather, their movements seem to be postural adjustments or horizontal squirming.
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Vertical Transmission of Acquired Ulcer Susceptibility in the Rat

Abstract. *Premature separation of rat pups from their dams greatly increases their susceptibility to restraint-induced gastric erosions. When prematurely separated female rats grow to adulthood and mate with stock males, their normally reared* F_1 *progeny also have increased susceptibility to restraint-induced erosions. Cross-fostering studies show that prenatal rather than postnatal factors transmit this susceptibility to the* F_1 *progeny.*

Experimental interventions during an animal's early development can result in modified behavior patterns, physiologic response characteristics, and susceptibility to disease that persist for long periods. For example, it was found (*1, 2*) that premature separation of rat pups from their dams increases their subsequent susceptibility to restraint-induced gastric erosion (RGE) (*3*). We now report that this increased susceptibility to RGE is transmitted to the F_1 progeny of female rats who are prematurely separated from their mothers in their own infancy. We also report that the increased RGE susceptibility of prematurely separated rats is transmitted to their progeny prenatally. To our knowledge, this is the first report of an altered susceptibility to a particular disease, acquired by an environmental manipulation during postnatal development in one generation, that is transmitted to offspring in the next.

Gastric erosions can be induced in rats by a combination of food deprivation and restraint. If rats are separated from their mothers at the customary time (postnatal day 21), approximately 10 to 20 percent develop gastric erosions during restraint on day 30. However, after premature separation on postnatal day 14, approximately 80 to 90 percent develop gastric erosions during restraint on day 30 (*2*).

To evaluate the RGE susceptibility of the offspring of female rats, we compared four groups of F_1 progeny (Fig. 1A). In the parent generation, ten litters were separated from their mothers on postnatal day 14 and ten on day 21. The females from each litter were then allowed to grow undisturbed to maturity. At about day 100, one female from each litter was randomly selected and bred to a stock Wistar male. Their offspring were separated from the mothers either prematurely (day 14) or at the usual time (day 21).

On postnatal day 27 all four groups of F_1 progeny were deprived of food for 26 hours and then restrained for 28 hours at an ambient temperature of 22°C. Afterwards the animals were killed and their stomachs examined for gastric erosions under a light microscope (×30). The experimenter was unaware of the origin of the stomachs.

The group of special interest was the normally separated progeny of mothers who had been prematurely separated in their own infancy. Sixty-four percent of

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