ACTH-induced reactivation of prior active avoidance training after intermediate intervals in hypophysectomized, adrenalectomized, and sham-operated rats

STEPHEN B. KLEIN

Old Dominion University, Norfolk, Virginia 23508

Negative transfer from active to passive avoidance training was evident 24 h after active avoidance acquisition in hypox, adx, and sham-operated rats injected with ACTH, corticosterone, or saline prior to 24-h test. No transfer was observed in control rats or in hypox, adx, or sham rats given corticosterone or saline 2½ h after active avoidance training. However, hypox, adx, or sham-operated rats given ACTH at 2½-h test exhibited negative transfer from prior active avoidance training. ACTH presented prior to 2½-h test eliminated the Kamin-like effect. These results suggested that ACTH reactivated at 2½ h processes related to prior active avoidance responding. A possible theory of the Kamin effect is offered.

Kamin (1957) reported that rats were unable to perform a previously acquired two-way active avoidance response on an intermediate interval retention test, while avoidance performance was excellent both immediately and 24 h after initial avoidance training experience. Interpretations of the Kamin effect have been based upon changes in fear occurring during the first 24 h following original avoidance training (Denny & Ditchman, 1962; Pinel & Cooper, 1966a, b), attributed to an inability to cope with stress during the intermediate interval following avoidance training as a consequence of the decrease in plasma concentration of corticosterone (Brush & Levine, 1966; Levine & Brush, 1967) or due to cholinergic inhibition of responding during intermediate interval following avoidance acquisition (Anisman, 1973). However, the results of several recent articles (Baum, 1968; Bintz, 1970; Klein, 1972; Klein & Kopish, 1974; Klein & Spear, 1969, 1970a, b; Spear, Klein, & Riley, 1971) clearly indicate that retrieval failure after an intermediate retention interval may be responsible for the appearance of the Kamin effect.

The importance of the adrenal-pituitary system in the Kamin effect has been well demonstrated (Bintz, 1972; Brush & Levine, 1966; Klein, 1972; Levine & Brush, 1967). Both Bintz (1972) and Brush and Levine (1966) found that detention of subjects in the avoidance apparatus for 1 h after avoidance training

eliminated the performance deficit normally observed after an intermediate retention interval. Brush and Levine suggested that the absence of the Kamin effect produced by detention was attributed to the maintenance of adrenal-pituitary responsiveness during the retention interval. Long-lasting ACTH administered after avoidance training eliminated the retention deficit seen in the Kamin effect after an intermediate interval (Levine & Brush, 1967), while a resumption of avoidance responding occurred when water stress, lateral anterior hypothalamic stimulation, or direct implantation of the adrenocorticotrophic hormone (ACTH) into the lateral anterior hypothalamus preceded the 2½-h test (Klein, 1972).

The absence of ACTH on intermediate interval retention tests due to central corticoid inhibition (e.g., McEwen, Weiss, & Schwartz, 1969, 1970; Sawyer, Kawakami, Meyerson, Whitmoyer, & Lilley, 1968; Smelik, 1963) may be responsible for the retrieval failure observed after intermediate intervals (Klein, 1972). Thus, it would appear that the organism is unable to respond according to its previous training on an intermediate-interval test as the result of the generalization decrement or stimulus dissociation caused by the presence of corticosterone and/or the absence of ACTH (e.g., Overton, 1966). The results of Spear, Klein, and Riley (1971) support a state-dependent interpretation of the Kamin effect by demonstrating that a passive-avoidance response acquired under the intermediate interval is not retained when tested at the 24-h retention interval.

However, the results of several experiments indicated that the Kamin effect occurred in rats deprived of normal adrenal-pituitary function (Barrett, Leith, & Ray, 1971; Marquis & Suboski, 1969; Snider, Marquis, Black, & Suboski, 1971; Suboski, Marquis, Black, & Platenius, 1970). Barrett

This research was supported by Grant MH 21525-01 from the National Institute of Mental Health and by a grant from the Old Dominion University Research Foundation to the author. Recognition is extended to Peter Mikulka, Fred Freeman, and Perry Duncan for editorial comments. Requests for reprints should be sent to Stephen B. Klein, Department of Psychology, Old Dominion University, Norfolk, Virginia 23508.

et al., Suboski et al., and Snider et al. demonstrated that adrenalectomy had no effect on the retention deficit observed after an intermediate interval. Their results suggest that central corticoid inhibition may not be essential for forgetting of prior avoidance training to occur after intermediate retention intervals. Furthermore, hypophysectomy does not seem to eliminate the retention differences which are noted during the first 24 h after avoidance acquisition (Marquis & Suboski, 1969).

Two apparently contradictory impressions are conveyed by the results of prior research. ACTH appears to be a memory attribute of avoidance responding, capable of facilitating the retrieval of the memory of avoidance training when presented prior to an intermediate interval test (Klein, 1972). Yet, the Kamin effect is present in hypophysectomized rats who possess no ACTH (Klein & Kopish, 1975; Marquis & Suboski, 1969).

In addition, central corticoid inhibition of ACTH release may produce the retrieval failure noted after intermediate retention intervals. However, subjects who are adrenalectomized prior to avoidance training and therefore possess no adrenal steroid hormones also exhibit retention deficits after intermediate retention intervals (Barrett et al., 1971; Klein & Kopish, 1975; Snider et al., 1971; Suboski et al., 1970).

EXPERIMENT I

The purpose of this experiment was to evaluate the effect of ACTH at intermediate intervals in hypophysectomized rats. Reactivation by ACTH at intermediate intervals in hypophysectomized rats, occurring as a consequence of association of the arousing action of hormones available in hypophysectomized rats and avoidance behavior, would support the hypothesis that ACTH functions as a memory attribute of avoidance behavior.

Similarly, the influence of corticosterone at the 24-h retention interval in adrenalectomized rats will be assessed in this experiment. Retrieval failure would implicate further central corticoid inhibition as contributing to the retention deficit noted after intermediate intervals. The continued availability of other arousing hormones at the 24-h test may negate a corticosterone effect and suggest multiple inhibition as the source of retrieval failure after intermediate intervals.

Weiss, McEwen, Silva, and Kalkuk (1969) found that ACTH increased active avoidance responding in hypophysectomized rats and that corticosterone reduced the number of active avoidance responses in adrenalectomized rats. The drug doses employed in this experiment were similar to those used in Weiss et al. (1969). Similarly, the dose levels used in Experiment I approximate the dosages found to

influence hippocampal function in Pfaff, Silva, and Weiss (1971). Any influence of ACTH and corticosterone on retention of avoidance behavior in this study would expand the behavioral findings of Weiss et al. and suggest a possible central neural mediation of hormonal influence.

Method

Subjects and Apparatus. The subjects were 207 experimentally naive female hypophysectomized, adrenalectomized, and sham-operated rats purchased from Charles River Laboratories, Inc. (Wilmington, Massachusetts). An equal number of sham animals received hypophysectomy and adrenalectomy surgical treatment. All rats were 75-90 days old at the time of training. The hypophysectomized rats weighed between 150 and 175 g, while the other rats weighed between 200 and 250 g before avoidance acquisition. Hypophysectomized rats were individually caged with ad-lib food and water, while the adrenalectomized rats were maintained on 0.9% saline solution. Adrenalectomized rats that did not die after 2 weeks as a consequence of a salt-free diet were eliminated from the study. Ninety-five percent of the adrenalectomized rats died in this 2-week period, while none of the sham-operated rats died as a consequence of being on a salt-free diet. Surgical effectiveness was assessed in hypophysectomized rats by adrenal weights. Adrenal weights in hypophysectomized rats ($\bar{X}$ = .0103 g; SD = .0032) were significantly different ($p < .05$) from those of sham-operated rats ($\bar{X}$ = .0273 g; SD = .0071), indicating success of hypophysectomy.

The apparatus was a two-chamber black and white avoidance apparatus (previously described in Klein & Spear, 1969). A 1.6-mA footshock was employed for active-avoidance training and a .3-mA shock of 1 sec duration was used for passive-avoidance acquisition. These particular shock parameters were employed because identical values were found to produce negative transfer (Klein, 1972).

Procedure and Design. The basic design was a 2 by 2 by 3 factorial, including retention interval, pretest drug treatment, and preacquisition surgical treatment.

Fifty-four hypophysectomized (hypox, $N = 9$), 42 adrenalectomized (adx, $N = 7$), and 54 sham-operated (sham, $N = 9$) rats were given one-way active avoidance training to a criterion of five consecutive avoidances using a procedure identical to that used in Klein and Spear (1970a). Either 2½ h or 24 h after the fifth consecutive avoidance, the rats from each surgical treatment group were given a passive avoidance acquisition retention test utilizing the procedure used in Klein and Spear (1970a).

Rats received adrenocorticotrophic hormone (ACTH, 30 I.U. dissolves in .5 cc saline), corticosterone (CORT, .5 mg dissolved in .5 cc saline), or .5 cc saline intraperitoneally prior to receiving passive avoidance acquisition training. Both ACTH and saline were administered 10 min prior to transfer test, while a 1-h interval separated corticosterone injections and passive avoidance training. A 1-h interval between corticosterone injection and testing was employed to allow drug to inhibit ACTH release, as Pfaff, Silva, and Weiss (1971) observed corticosterone influence on the hippocampus began 10 to 40 min after injection.

Nine additional control groups ($N = 6$) were also included in this experiment. These subjects did not receive active avoidance acquisition training in order to establish baseline performance in the absence of transfer from prior avoidance training. The nine control groups consisted of the factorial combinations of drug and surgical treatments. ACTH, corticosterone, or saline was administered prior to passive avoidance training in hypox, adx, and sham rats.

Results

Kruskal-Wallis one-way analyses of variance were used to evaluate acquisition and retention differences

throughout this paper, while Mann-Whitney U tests were conducted to evaluate specific differences between individual groups in terms of number of trials to acquire the competing aversive-conditioning experience (Siegel, 1956). Statistical results of critical group comparisons are presented in Table 1 with the observed U and probability values.

Active-avoidance acquisition. There were no significant differences between hypophysectomized (Mdn = 5.6), adrenalectomized (Mdn = 5.7), or sham-operated (Mdn = 5.4) rats in terms of number of trials to acquire active avoidance response ($H = 1.31$, $df = 2$, $p > .05$). In addition, there were no active avoidance acquisition differences between hypophysectomy groups ($H = 7.335$, $df = 5$, $p > .05$), adrenalectomy groups ($H = 6.30$, $df = 5$, $p > .05$), and sham-operated groups ($H = 5.42$, $df = 5$, $p > .05$). These results suggest that all groups were equivalent at the beginning of the transfer interval.

Transfer test in hypophysectomized rats. Analysis of the trials to acquire the passive avoidance response in hypox rats yielded a significant groups effect ($H = 23.69$, $df = 8$, $p < .01$). At 2½-h test, saline and cort rats exhibited the same rate of passive-avoidance learning as was exhibited by nontransfer control animals (see Figure 1). Slower learning in ACTH rats at 2½-h test and in all three 24-h groups (ACTH, cort, and saline) was observed compared with the acquisition of passive avoidance in both cort and saline 2½-h hypox rats and in nontransfer control animals. Therefore, negative transfer from active to passive avoidance was exhibited in 2½-h ACTH animals and all 24-h drug treatment rats, while an absence of the transfer was evident in 2½-h rats given cort or saline.

Table 1
Mann-Whitney U Comparisons

Comparison Groups	Drug Injection		
	ACTH U =	CORT U =	Saline U =
Hypox Treatment			
2½ vs 24	27	16.5**	13.5**
2½ vs Control	7†	23.5	26
24 vs Control	5†	9.5**	10.5*
Adx Treatment			
2½ vs 24	16	11*	0††
2½ vs Control	4†	10.5	10
24 vs Control	0††	4.5†	0††
Sham Treatment			
2½ vs 24	15**	16.5**	7.5†
2½ vs Control	18.5	26	29
24 vs Control	3.5††	11.5*	7.5**
*.05	** .025	†.01	††.001

Transfer test in adrenalectomized rats. A significant groups effect was observed in passive avoidance acquisition in adx rats ($H = 18.93$, $df = 8$, $p < .05$). Negative transfer was absent in 2½-h cort and saline rats, as indicated by equal rate of passive avoidance response in control animals and 2½-h cort and saline rats. ACTH animals tested 2½ h after original active avoidance acquisition learned the passive avoidance at the same rate as rats receiving ACTH prior to 24-h test and more slowly than nontransfer control animals. Thus, ACTH presented prior to 2½-h test produced negative transfer from prior active avoidance training.

Transfer test in sham-operated rats. Analysis of passive avoidance acquisition data in sham rats yielded a significant groups effect ($H = 16.31$, $df = 8$, $p < .05$). Sham rats receiving ACTH, CORT, or saline prior to 2½-h test learned the passive avoidance as rapidly as nontransfer control rats and more rapidly than corresponding sham rats tested 24 h after active avoidance acquisition. Passive avoidance learning was faster in control rats than was observed in sham rats receiving ACTH, CORT, or saline prior to 24-h test.

The appearance of no negative transfer in sham rats at the 2½-h test was at variance with the negative transfer observed in hypox-ACTH and adx-ACTH rats at 2½ h, and also at variance with other literature. Although the majority of sham rats given ACTH at 2½-h test acquired the passive avoidance response as rapidly as did naive control rats, the poor acquisition seen in three of nine subjects may indicate that impaired passive avoidance acquisition occurs after ACTH administration at 2½-h test under appropriate conditions.

Passive-avoidance acquisition in control rats. Analysis of acquisition of passive avoidance in control, hypox, adx, and sham rats receiving saline prior to training showed a nonsignificant groups effect ($H = 1.2$, $df = 2$, $p > .05$). As was observed with active avoidance training, neither hypox nor adx

Figure 1. Median number of trials to acquire passive avoidance 10 min following ACTH, corticosterone, or saline treatment are presented for hypophysectomized, adrenalectomized, and sham-operated rats as a function of retention interval after active-avoidance acquisition training. Bottom dotted lines refer to ACTH control groups, bottom dashed lines indicate corticosterone control groups, and bottom solid lines show saline control group rats. Open and closed circles refer to subjects given ACTH or saline 5 min prior to 2½-h test in Experiment II.

surgical treatment influenced number of trials to acquire passive-avoidance response. Furthermore, there were no passive avoidance acquisition differences in control hypox ($H = 5.9$, $df = 2$, $p > .05$), control adx ($H = 1.7$, $df = 2$, $p > .05$), or control sham ($H = 1.3$, $df = 2$, $p > .05$) rats receiving ACTH, CORT, or saline prior to passive avoidance training.

EXPERIMENT II

The absence of negative transfer in some sham-operated subjects at 2½-h test in Experiment I may have occurred because 10 min is too long an interval between drug treatment and testing for some subjects. The purpose of Experiment II was to determine if a shorter drug-test interval would produce reactivation in sham rats. Therefore, sham-operated rats received ACTH or saline 5 min prior to being tested for retention of original active avoidance training.

Method

Subjects and Apparatus. The subjects were 14 experimentally naive female sham-operated albino rats. All other aspects of the subjects and apparatus used in this experiment were identical to those employed in Experiment I.

Procedure and Design. The only change in the procedure and design from Experiment I was the administration of saline or ACTH 5 min prior to passive avoidance acquisition instead of the 10-min ACTH test interval used in Experiment I. Only the 2½-h conditions described in Experiment I were run in this study.

Results

Active-avoidance acquisition. There were no differences between the two experimental groups in terms of active avoidance acquisition ($Mdn = 6.7$ for ACTH and 7.4 for saline rats). These data indicate that both groups were equivalent before the retention interval.

Retention test (passive avoidance acquisition). The data indicate that sham-operated rats receiving ACTH 5 min prior to the 2½-h test acquired the antagonistic avoidance response significantly more slowly ($Mdn = 4.0$ trials) than the rats receiving saline under similar conditions (1.6 trials, $U = 7$, $p < .025$; see Figure 1). The results demonstrated that reactivation of some processes deriving from the active avoidance training occurred as a consequence of ACTH presentation 5 min prior to passive avoidance acquisition in sham-operated subjects.

GENERAL DISCUSSION

Absence of negative transfer from prior active avoidance training was consistently observed for all subjects receiving either corticosterone or saline prior to 2½-h test. Acquisition of the new antagonistic, passive avoidance response in hypophysectomized, adrenalectomized, and sham-operated rats proceeded as rapidly in these experiments at the intermediate

retention interval test as was observed in naive rats. These results suggest that rats are unable to recall original avoidance training on intermediate interval retention tests.

In contrast to the retention deficits observed after intermediate retention intervals, subjects tested 24 h after original active avoidance training appear to remember their prior avoidance experience. Negative transfer from initial avoidance training was found in hypophysectomized, adrenalectomized, and sham-operated rats at 24 h retention tests. The changes in avoidance performance noted during the initial 24 h following avoidance training may reflect differences in the ability of the rat to retrieve the memory of original avoidance acquisition.

The observed reactivation effects of ACTH in this study may be pharmacological rather than physiological. ACTH effects on retention in adx rats already possessing high ACTH levels may indicate a pharmacologic effect of ACTH. Similarly, the greater reactivation produced in sham rats when a 5-min rather than a 10-min interval between injection and testing was employed suggests that higher levels of ACTH are necessary to induce reactivation in sham rats.

Although it is difficult to draw conclusions from data derived using pharmacological drug levels concerning processes in the normal animal, these results may indicate what processes lead to forgetting after intermediate intervals. It is hypothesized that during avoidance training the animal attends to the physiological arousal produced by shock. The absence of arousal occurring at intermediate interval leads to retrieval failure. Treatments which reactivate arousal (shock, stress, adrenalin, ACTH) reproduce the arousal state, thereby enabling the rat to recall previous avoidance training.

The results presented in Experiment I may demonstrate that the influence of ACTH on retention is due to the general arousal action of ACTH. Improved retention following ACTH administration at intermediate interval in hypox rats may be attributed to the similar arousing action of ACTH and hormones present in hypox animals. Reactivation by adrenalin (Hablitz & Braud, 1972) supports the notion that arousal is the critical attribute of all reactivating agents. Pharmacological doses may merely represent a higher level of arousal and therefore can also act to reactivate avoidance memories.

The absence of negative transfer in 2½-h adx rats receiving saline prior to 2½-h transfer test would seem to argue against an absence of arousal interpretation of the Kamin effect. The high ACTH level present in adx rats should prevent the occurrence of the Kamin effect. Yet the Kamin effect has been consistently reported in adx rats (Barret, Leith, & Ray; Klein & Kopish, 1975; Snider et al., 1971; Suboski et al., 1970). However, reduced arousal may have been produced by other hormones not affected

by adrenalectomy. Parasympathetic rebound should still inhibit arousal in adx rats. Furthermore, even though resting ACTH (and arousal) level is high in adx rats, shock presented during active avoidance training would induce further arousal above that normally present. Although adx animals have a higher baseline level of ACTH, the changes induced by avoidance training may be the same in adx and sham rats, and therefore the process producing the Kamin effect would be identical in adx and sham rats. Perhaps ACTH induces reactivation at 2½ h in adx rats by reinstating the level of arousal present during original training.

Thus, one potential explanation of the Kamin effect involves the concept of arousal becoming associated with avoiding during initial avoidance acquisition. Multiple inhibitory systems act to produce retrieval failure observed after intermediate retention intervals by preventing the activation of arousal (produced by several systems, i.e., adrenal-pituitary, adrenergic-cholinergic, etc.). To produce forgetting of an avoidance response, all arousal systems must be inhibited. The fact that neither corticosterone (Experiment I; Klein, 1972) nor physostigmine (Anisman, 1973) can produce retention deficits on a 24-h test supports the assertion that multiple inhibitory systems mediate retrieval failure after intermediate intervals. The presence of more inhibitory systems in sham rats may cause greater inhibition. Thus, the 5-min injection-test interval produced greater reactivation in sham rats as a consequence of higher levels of arousal induced with 5-min interval. Future research should attempt to measure the effect of multiple inhibition on memory retrieval on a 24-h retention test.

REFERENCES

- ANISMAN, H. Cholinergic mechanisms and alterations in behavioral suppression as factors producing time-dependent changes in avoidance performance. *Journal of Comparative and Physiological Psychology*, 1973, 831, 465-477.
- BARRETT, R. J., LEITH, N. J., & RAY, O. S. The effects of pituitary-adrenal manipulations on time-dependent processes in avoidance learning. *Physiology and Behavior*, 1971, 7, 663-665.
- BAUM, M. Reversal learning of an avoidance response and the Kamin effect. *Journal of Comparative and Physiological Psychology*, 1968, 66, 495-497.
- BINTZ, J. Time-dependent memory deficits of aversively motivated behavior. *Learning and Motivation*, 1970, 1, 405-406.
- BINTZ, J. Detention effects on memory function. *Learning and Motivation*, 1972, 3, 44-50.
- BRUSH, R. R., & LEVINE, S. Adrenocortical activity and avoidance learning as a function of time after fear conditioning. *Physiology and Behavior*, 1966, 1, 309-311.
- DENNY, M. R., & DITCHMAN, R. E. The locus of maximal "Kamin effect" in rats. *Journal of Comparative and Physiological Psychology*, 1962, 55, 1060-1070.
- HABLITZ, J. J., & BRAUD, W. G. Adrenalin, sodium amobarbital, and the Kamin effect in the albino rat. *Learning and Motivation*, 1972, 3, 51-56.
- KAMIN, L. J. Retention of an incompletely learned avoidance response. *Journal of Comparative and Physiological Psychology*, 1957, 50, 457-460.
- KLEIN, S. B. Adrenal-pituitary influence in reactivation of avoidance-learning memory in the rat after intermediate intervals. *Journal of Comparative and Physiological Psychology*, 1972, 79, 341-359.
- KLEIN, S. B., & KOPISH, R. M. Memory-retrieval failure hypophysectomized and adrenalectomized rats after intermediate intervals. *Behavioral Biology*, 1975, 13, 377-381.
- KLEIN, S. B., & SPEAR, N. E. Influence of age on short-term retention of active-avoidance learning in rats. *Journal of Comparative and Physiological Psychology*, 1969, 69, 583-589.
- KLEIN, S. B., & SPEAR, N. E. Forgetting by the rat after intermediate intervals ("Kamin effect") as retrieval failure. *Journal of Comparative and Physiological Psychology*, 1970, 71, 165-170. (a)
- KLEIN, S. B., & SPEAR, N. E. Reactivation of avoidance learning memory in the rat after intermediate retention intervals. *Journal of Comparative and Physiological Psychology*, 1970, 72, 498-504. (b)
- LEVINE, S., & BRUSH, F. R. Adrenocortical activity and avoidance learning as a function of time after avoidance training. *Physiology and Behavior*, 1967, 2, 358-388.
- MARQUIS, H. A., & SUBOSKI, M. D. Hypophysectomy and ACTH replacement in the incubation of passive and shuttle box avoidance responses. *Proceedings of the 77th Annual Convention of the APA*, 1969, 4, 207-208.
- MC EWEN, B. S., WEISS, J. M., & SCHWARTZ, L. S. Uptake of corticosterone by rat brain and its concentration by certain limbic structures. *Brain Research*, 1969, 16, 227-241.
- MC EWEN, B. S., WEISS, J. M., & SCHWARTZ, L. S. Retention of corticosterone by cell nuclei from brain regions of adrenalectomized rats. *Brain Research*, 1970, 17, 471-482.
- OVERTON, D. A. State-dependent learning produced by depressant and atropinelike drugs. *Psychopharmacologia* (Berlin), 1966, 10, 6-31.
- PINEL, J. P. J., & COOPER, R. M. Demonstration of the Kamin effect after one-trial avoidance learning. *Psychonomic Science*, 1966, 4, 17-18. (a)
- PINEL, J. P. J., & COOPER, R. M. Incubation and its implications for the interpretation of the ECS gradient effect. *Psychonomic Science*, 1966, 6, 123-124. (b)
- PEAFF, D. W., SILVA, M. T. A., & WEISS, J. M. Telemeter recording of hormone effects on hippocampal neurons. *Science*, 1971, 172, 394-395.
- SAWYER, C. H., KAWAKAWI, M., MEYERSON, P., WHITMOYER, D. I., & LILLEY, J. J. Effects of ACTH, dexamethasone, and asphyxia on electrical activity of the rat. *Brain Research*, 1968, 10, 213-226.
- SIEGEL, S. *Nonparametric statistics for the behavioral sciences*. New York: McGraw-Hill, 1956.
- SMELIK, P. G. Relation between blood level of corticoids and their inhibiting effect on the hypophyseal stress response. *Proceedings for the Society for Experimental Biology and Medicine*, 1963, 113, 616-619.
- SNIDER, N., MARQUIS, M. A., BLACK, M., & SUBOSKI, M. D. Adrenal corticosteroids and the Kamin effect. *Psychonomic Science*, 1971, 22, 309-310.
- SPEAR, N. E., KLEIN, S. B., & RILEY, E. P. The Kamin effect as "state-dependent learning": Memory-retrieval failure in the rat. *Journal of Comparative and Physiological Psychology*, 1971, 74, 416-425.
- SUBOSKI, M. D., MARQUIS, H. A., BLACK, M., & PLATENIUS, P. Adrenal and amygdala function in the incubation of aversively conditioned responses. *Physiology and Behavior*, 1970, 5, 283-289.
- WEISS, J. M., MC EWEN, B. S., SILVA, M. T. A., & KALKUT, M. F. Pituitary-adrenal influences on fear responding. *Science*, 1969, 163, 197-199.

(Received for publication June 4, 1974;
revision accepted June 10, 1975.)