mals and those with cortical lesions nor the difference between the caudate operates and cortex operates reached significance ($p = .13$ and $.27$, respectively).

Thus both acquisition and retention of the alternation habit were severely impaired by lesions of the caudate nucleus but not by lesions of posterior cortex. In order to exclude the possibility that the alternation deficit reflected either a general learning impairment or a motivational change, the present animals and others with similar lesions and experience were tested in a Hebb-Williams maze according to the procedure outlined by Rabinovich and Rosvold (*8*). On the 12 test problems the caudate group ($N = 13$) made a mean of 190.5 errors, the unoperated group ($N = 9$) made a mean of 152.7 errors, and the cortex group ($N = 9$) made a mean of 320.9 errors. The performance of the normal and caudate groups did not differ significantly, whereas both made significantly fewer errors than the cortex group ($p < .002$ in both cases). Since animals with caudate lesions were unimpaired on the maze task but failed the alternation test, it seems reasonable to assert that they showed neither a generalized learning deficit nor a marked disruption of motivation to work for food.

In conclusion, the caudate nucleus in the rat, as in the monkey, is necessary for adequate alternation performance. Further experiments are required to determine whether caudate lesions in rat and monkey impair alternation performance by equivalent mechanisms (*9*; *10*).

STEPHAN L. CHOROVER
CHARLES G. GROSS
Psychology Section, Massachusetts Institute of Technology, Cambridge

References and Notes

1. C. F. Jacobsen, *Comp. Psychol. Monogr.* **13**, 3 (1936); ——— and H. W. Nissen, *J. Comp. Psychol.* **23**, 101 (1939); H. E. Rosvold and J. M. R. Delgado, *J. Comp. Physiol. Psychol.* **49**, 356 (1956); W. H. Dean and G. D. Davis, *J. Neurophysiol.* **22**, 524 (1959).
2. J. M. Warren, H. B. Warren, K. Akert, *J. Comp. Neurol.* **118**, 17 (1960); S. Brutkowski, *Acta Biol. Exptl. Polish Acad. Sci.* **19**, 301 (1959).
3. H. E. Rosvold, M. K. Szwarcbart, A. F. Mirsky, M. Mishkin, *J. Comp. Physiol. Psychol.* **54**, 368 (1961).
4. S. L. Chorover, M. Cole, G. Ettlinger, unpublished data.
5. Grason-Stadler Co., Inc., model E3125B, with the addition of a light within the food well which operated for one-half second when food was presented.
6. A Grass model LM-2 lesion maker was used to produce the caudate lesions through a formvar-coated 10-mil wire with 1.0 mm of the insulation removed at the tip. The unit scale was set at 85 and current was passed for 30 seconds between the wire and a large electrode plug inserted in the anus. Location of the electrode in the brain was determined in accordance with stereotaxic coordinates from J. De Groot, *The Rat Forebrain in Stereotaxic Coordinates* (Noord-Hollandsche Uitgevers Maatschappij, Amsterdam, 1959) (Verhandelingen der koninklijke nederlandse akademie van wetenschappen, afd. natuurkunde, Tweede Reeks, Deel LII, No. 4), pp. 1–40. The caudate placement was aimed at L 2.5; A 8.6; V + 1.5.
7. The statistical values presented in this report are based upon comparisons made by means of two-tailed Mann-Whitney *U* tests [S. Seigel, *Non-Parametric Statistics for the Behavioral Sciences* (McGraw-Hill, New York, 1956)].
8. M. S. Rabinovich and H. E. Rosvold, *Can. J. Psychol.* **5**, 122 (1951).
9. It should be noted that our apparatus did not include a screen interposed between the animal and the levers. When monkeys with anterior forebrain lesions are tested in lever-pressing apparatus without such screens, their deficiency on delayed alternation may be reduced (J. Stamm, in K. Akert and J. M. Warren, *Frontal Granular Cortex and Behavior*, in press) or even absent [K. Battig, H. E. Rosvold, M. Mishkin, *J. Comp. Physiol. Psychol.* **53**, 400 (1960)].
10. This work was supported, in part, by the Rockefeller Foundation, the Air Force Office of Scientific Research, and the National Institutes of Mental Health (NIMH program grant M-5673 to H.-L. Teuber). We are indebted to Shoel Cohen and Ann Cooper for assistance in various phases of the experiment and to Marilyn Mohnkern for histological preparation of the brain sections.

28 June 1963

Varying Sensitivity of C57BL/Crg1 Mice to Grouping

Abstract. *Mice responded differentially to increases in population size, depending on their prior experience. Compared to animals grouped prior to an increase in population size, animals housed as isolates responded to grouping with more fighting, a greater diminuation of testis weight, and a higher level of locomotor activity. They also had lower testis weight and higher locomotor activity than individually housed, nongrouped controls.*

Many mammalian species are reported to respond to increases in population density with heightened adrenocortical activity, diminished testicular function, and heightened behavioral arousal (*1*). Often, however, even within the same species, the endocrine changes are variable and unrelated in any systematic way to increases in behavioral reactivity or to population number (*2*). Differential levels of aggression, as a result of varying genotype or past experience, may account for these inconsistencies. Several studies indicate, for example, that genetic predisposition toward aggression is related to adrenal enlargement in dense populations (*3*). Similarly, isolation prior to grouping will increase aggression and other responses, such as amphetamine toxicity and pseudopregnancy (*4*). This report offers evidence that isolation prior to grouping increases aggressive activity, endocrine response, and, to a lesser degree, behavioral reactivity, thus suggesting an interaction among these factors.

Twenty male C57BL/Crg1 mice were housed in individual cages about 25.4 by 12.7 by 10 cm high for 21 days beginning at 82 days of age, while ten others of the same age were housed as two groups of five for an equal period of time. All animals were ear-punched for identification. Prior to this time all animals had been housed since weaning in groups of five. Clean cages were routinely provided once a week. At the end of this 21-day period, ten of the individually housed animals were grouped in a single cage measuring about 31.8 by 21.6 by 7.6 cm high, while the two separate groups of five were housed as a single group of ten in a cage of similar dimensions. Ten other animals remained individually housed. Purina lab chow and water were freely available to the animals. Fighting behavior was recorded for the first half hour after grouping, while within-cage activity (measured as a composite of eating, drinking, moving from one area to another, and fighting) was recorded each hour for the next 7 hours. After 5 days of differential housing, all animals were tested individually for locomotor activity in a modified open field. This apparatus consisted of a 20-by-20-inch (50.8-by-50.8-cm) floor lined into 25 4-inch (10.16-cm) squares. Twelve barriers about 10.16 cm wide by 12.7 cm high were erected

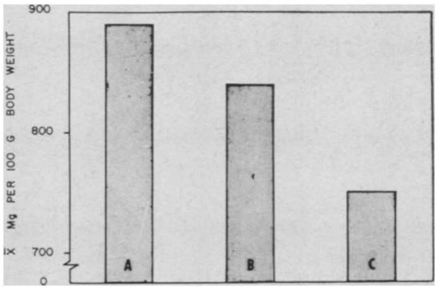

Fig. 1. Effect of grouping and prior housing conditions on testis weight. *A*, isolates; *B*, density 10 (prior grouping); *C*, density 10 (prior isolation).

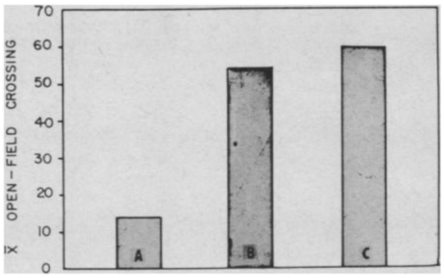

Fig. 2. Effect of grouping and prior housing conditions on locomotor activity. *A*, isolates; *B*, density 10 (prior grouping); *C*, density 10 (prior isolation).

at regular intervals on the floor area. The entire arena was enclosed by walls about 12.7 cm high. The floor and barriers were painted gray and the smaller squares were marked off in black. The animal was placed in one corner, a timer started, and the number of squares crossed during a 3-minute period recorded. Crossing a square was defined as placing both forepaws over a line. Twenty-four hours later the mice were killed, and their adrenals and testes were removed and weighed to the nearest 0.1 mg.

Animals previously living in groups of five showed no overt fighting during the half-hour observation when grouped in a single cage. Animals previously isolated prior to grouping began to fight after the first 10 minutes. Seventy-eight individual encounters were noted and nearly one-third of the half-hour period was spent in fighting. One hour later all fighting had ended but all animals of both groups were active. Although animals previously isolated remained more active during the second hour after grouping, by the third hour the groups could not be distinguished on the basis of activity, and by the sixth hour and thereafter animals of both conditions huddled inactive in one section of the cage.

Adrenal and body weight did not vary with conditions of housing, but testis weight showed a decline with grouping ($F = 3.88$; df = 2 and 27; $p < .05$), the greatest decline occurring with those animals having been isolated prior to grouping ($p < .01$). These values are set out in Fig. 1. The greater sensitivity of the testes to grouping is in accord with previous data (*2*).

Open field activity also varied with condition of housing and in the predicted direction. Animals from both populations were more active than animals living individually ($F = 8.43$; df = 2 and 27; $p < .01$) and grouped animals previously isolated were somewhat higher than the other two conditions. These relations are seen in Fig. 2.

A consistent pattern of results is evident from these data; isolation prior to grouping leads to intense fighting, testicular atrophy, and high levels of locomotor activity. It is possible that inattention to similar differences in living conditions preceding increases in density can account for varying results reported in the literature. The possibility exists that the level of aggression is the mediating factor regulating associated changes. Moreover, strict control over the experience of the animals can provide greater predictability for studying relations between population density, endocrine function, and behavior (*5*).

D. D. THIESSEN

Section of Medical Psychology,
Scripps Clinic and Research
Foundation, La Jolla, California

References and Notes

1. D. D. Thiessen and D. A. Rodgers, *Psychol. Bull.* **58**, 441 (1961).
2. D. Chitty, *Can. J. Zool.* **38**, 99 (1960); C. H. Southwick and V. P. Bland, *Am. J. Physiol.* **197**, 114 (1959); D. D. Thiessen, unpublished Ph.D. dissertation, University of California (1963); D. D. Thiessen, J. F. Zolman, D. A. Rodgers, *J. Comp. Physiol. Psychol.* **55**, 186 (1962).
3. F. H. Bronson and B. E. Eleftheriou, *Physiol. Zool.* **36**, 161 (1963); J. J. Christian, *Comparative Endocrinology* (Wiley, New York, 1959), p. 71.
4. P. F. D'Arcy, *J. Pharm. Pharmacol.* **14**, 397 (1962); D. R. Lamond, *J. Endocrinol.* **18**, 343 (1959); J. P. Scott and E. Fredericson, *Physiol. Zool.* **24**, 273–309 (1951).
5. Supported by grant M-6139 from the National Institute of Mental Health.

9 July 1963

Timing Behavior after Lesions of Zona Incerta and Mammillary Body

Abstract. *Rats with mammillary body lesions produced with an angled approach do not show a "scalloping" in their behavioral records during fixed-interval reinforcement. Control experiments suggest that this effect was due to the electrode piercing the zona incerta, implicating the participation of extrapyramidal structures in timing behavior.*

In previously published studies (*1*) we have shown that lesions in the septum and hippocampus did not prevent the appearance of the "scallop" in the behaviorial record when rats pressed a bar for food or water under a fixed-interval reinforcement contingency. In fixed-interval reinforcement, reward is delivered only if the animal makes a bar press at a fixed period of time after the previous reward. The "scallop" which reflects the distribution of responding during the fixed interval has been used as evidence for temporal discrimination (*2*, *3*) and has been shown to be unaffected by drive or motivational conditions, with only the total output of bar-pressing behavior being affected by such variables (*1*, *3*). The present report is an extension of these investigations and is concerned with the effects of lesions in the mammillary body and adjacent structures on timing behavior.

After preliminary training in bar pressing for food reward eight rats were prepared with electrolytic lesions 1 mm in diameter in the mammillary body. Two stereotaxic approaches were used in making these lesions. For half the animals a vertical approach through the midline was used, while for the remainder an angled approach aimed at the same focus as the vertical was used and served as a control for damage to midline structures, thereby preserving effects common to the mammillary body lesion. After the experiment the animals were killed, serial frozen sections were made and stained with cresyl violet, and the lesion location and extent were determined.

Seven days postoperatively all animals were started on a training schedule in which the delivery of a 45-mg food pellet was dependent on the animal making a bar press after a minimum interval of 1 minute after the delivery of the previous reward. This training on the 1-minute fixed-interval schedule lasted for 9 days.

Figure 1 consists of typical examples of the cumulative bar-pressing records of the animals with mammillary body lesions. The slope of these curves reflects the response rate, while the vertical deflection indicates the occurrence of reinforcement. The high rates of response shown on these records indicate that regardless of the approach, whether vertical or angled, the mammillary body lesion did not interfere with or prevent bar-pressing behavior. Specifically, on day 1 of the training, the animals with the vertically oriented lesions made approximately 36 responses per reinforcement, while the animals with the angled lesion made, on the average, 40 responses per reinforcement. These behavioral outputs compare favorably with what is obtained