

was put in the chamber during the final 30 minutes.

No major change in avoidance behavior took place when the dummy was put in the chamber (Fig. 3). Furthermore, no attack movements were made toward the dummy. In contrast, when a naive animal was placed with the trained animal, there occurred a sharp drop-off in the number of responses (145 to 24). Observation indicated that in the social situation with the naive animal, bar pressing had been replaced by aggressive behavior. In fact, during the first two sessions the subjects fought after almost every shock. In the subsequent sessions, however, the actual fighting fell off rapidly to an average of ten fighting responses per session. On the other hand, during the same period, the escape-avoidance responding did not return to the single-subject rate. It appeared that the reason for this lack of recovery in bar pressing was related again to the fighting phenomenon. In the single-subject situation the trained animals tended to remain near the bar and only occasionally after a response did they move away. However, after a bar press in the presence of another live subject, the trained animal would frequently return to the naive animal and reassume the stereotyped fighting posture. This posture was maintained until the pre-shock stimulus appeared, whereupon the animal would return to the bar, press it, and return again to the naive animal, thus apparently lowering the probability of bar pressing. This preoccupation with the naive animal appeared to be one of the factors responsible for the low frequency.

R. E. ULRICH
W. H. CRAINE

Illinois Wesleyan University,
Bloomington

References and Notes

1. N. H. Azrin, W. C. Holz, D. F. Hake, *J. Exptl. Anal. Behav.* **6**, 141 (1963); W. C. Holz, N. H. Azrin, R. E. Ulrich, *ibid.* **6**, 115 (1963).
2. N. H. Azrin, *ibid.* **1**, 183 (1958).
3. W. N. Schoenfeld, unpublished study (1947).
4. O. H. Mowrer, *J. Abnormal (Soc.) Psychol.* **35**, 56 (1940).
5. F. S. Keller, *Psychol. Rec.* **4**, 235 (1941).
6. N. E. Miller, *J. Abnormal (Soc.) Psychol.* **43**, 155 (1948).
7. R. E. Ulrich and N. H. Azrin, *J. Exptl. Anal. Behav.* **5**, 511 (1962).
8. R. E. Ulrich, P. C. Wolff, N. H. Azrin, *Animal Behav.*, in press.
9. Supported by grants from the National Institute of Mental Health and the Research Fund of the Illinois Department of Mental Health. The assistance of T. J. Stachnik, P. C. Wolff, and Miss M. Mack is gratefully acknowledged.

28 October 1963

28 FEBRUARY 1964

Hormonal Control of Egg Development in *Calliphora*

In "Tanning in the adult fly: A new function of neurosecretion in the brain" G. Fraenkel and Catherine Hsiao mention a "gonadotropic" hormone from the brain of the fly *Calliphora erythrocephala*, and state that the term "gonadotropic" hormone is used in the sense given it in a paper by A. O. Lea and E. Thomsen (2), "according to which ovarian development in the fly is activated by the corpus allatum by means of a secretion from the median neurosecretory cells, and not, as was formerly assumed [here referring to E. Thomsen (3)] by the cells through the corpus allatum."

I want to point out that we have not used the word "gonadotropic" hormone for the hormone produced by the medial neurosecretory cells, (m.n.c.), and do not regard it as such. The m.n.c. have a profound influence on the development of the ovaries (3), but they also regulate the production of

proteolytic enzymes by the midgut cells, which must be regarded as the protein synthesis of these cells (4). That the m.n.c. might exert their effect on the growth of the ovaries through an influence on the protein metabolism and not solely through the corpus allatum was already suggested in the paper by E. Thomsen in 1952 (3).

ELLEN THOMSEN

Zoological Laboratory of the Royal
Veterinary and Agricultural College,
Copenhagen, Denmark

References

1. G. Fraenkel and C. Hsiao, *Science* **141**, 1057 (1963).
2. A. O. Lea and E. Thomsen, *Mem. Soc. Endocrinol. No. 12* (1962), pp. 345-347.
3. E. Thomsen, *J. Exptl. Biol.* **29**, 137 (1952).
4. ——— and I. Møller, *Nature* **183**, 1401 (1959); "The Ontogeny of Insects," *Acta Symposii de Evolutione Insectorum* (Prague, 1960), pp. 121-126 (English-language edition, I. Hrdy, Ed., Academic Press, New York, 1959); *J. Exptl. Biol.* **40**, 301 (1963).

3 December 1963

Retrograde Amnesia from Electroconvulsive Shock

Lewis and Adams contend in their report (1) that the retrograde amnesia which occurs if electroconvulsive shock is given immediately after a learning trial results from the convulsive response itself, which competes with and replaces the previously learned response. This, they suggest, gives "the appearance of amnesia" but is actually a conditioned prepotent response.

Thus this report emphasizes, as does much of the critical research on perseveration theory, the effects of the interference technique rather than the effects of interference upon learning. This is understandable, since most studies on perseveration theory use electroconvulsive shock as the interference technique. However, ether anesthetization can also be used and is as effective as shock in producing retrograde amnesia. (2). It is difficult to conceive of the complete loss of response which occurs with anesthetization as conditioned competing responses. A better explanation would seem to be that both the shock and anesthetization somehow interfere with the consolidation of perseverative neural activity and thereby produce amnesia.

In addition, it is difficult to fit the competition-of-response explanation to

the data of studies indicating a time relationship in retention. These studies have shown that as length of time between end of learning trials and administration of interference increases, amount retained increases also. Again, it seems that there must be a neurological mechanism involved.

Further, evidence for the competition-of-response theory is apparently based on the assumption that, in previous studies on perseveration theory, animals were given the electroconvulsive shock in the stimulus situation. This is not the case. Examination of at least several studies indicates that animals were removed from the stimulus situation before shock was administered (3).

CAROL J. DYE

Jefferson Barracks,
Veterans Administration Hospital,
St. Louis, Missouri

References

1. D. J. Lewis and H. E. Adams, *Science* **141**, 516 (1963).
2. C. J. Dye, thesis, Washington Univ., St. Louis, Mo., unpublished; W. B. Essman and M. E. Jarvik, *Psychopharmacologia* **2**, 172 (1960).
3. C. P. Duncan, *J. Comp. Physiol. Psychol.* **42**, 32 (1949); K. J. Hayes, *ibid.* **46**, 216 (1953); R. Thompson, *ibid.* **50**, 644 (1958).

19 November 1963