Table 1. Average distance that the chicks moved toward a stationary sound source. In the experimental group one chick did not respond under either stimulus; in the control group, four did not respond to the 200-hz stimulus and four (two of them nonresponders under the first stimulus) did not respond to the 2000-hz stimulus. The averages, however, were computed for the total number of chicks tested.

Group	Number tested	Distance (cm) moved when stimulus:	
		200 hz	2000 hz
Experimental	15	25.22	11.84
Control	20	13.72	13.46

were painted on the table beginning at the center and extending for 60 cm; these circles facilitated measurement of the chicks' movement from the center. On the remaining 15-cm periphery two small speakers were mounted at angles of 0° and 180°. Shortly after it hatched, each chick was placed in the center of the test board, and either the experimental or novel sound was turned on for 45 seconds. Only one speaker was active at a time. At the end of 45 seconds the distance the chick had moved toward the speaker was measured, the chick was returned to center, and the other tone was given. The order of presentation of the two sounds and the order of use of the two speakers were counterbalanced. The sound was approximately 65 db at the center of the table. The results are given in Table 1.

The difference between the distances moved in response to the two test tones in the imprinted group is highly significant ($t = 3.37$, 14 df, $P < .01$). There is no appreciable difference between the responses of the control group to the two frequencies. Although both sounds were clearly attractive, the chicks responded differentially to a sound presented during the prenatal period.

In a second experiment chicks in the experimental group were tested for following behavior. At the end of the discrimination test each chick was returned to the center of the table and a child's pull-toy model chicken was moved in front of it by hand from one edge of the table to the other at a rate of about 1.2 m/min. Every chick was tested under each of three conditions: two passes with the model quiet, two passes with a small speaker on its back emitting the novel sound, and two passes emitting the experimental sound. The order of presentation of the conditions was counterbalanced from chick to chick. When the model reached the edge of the table, the trial was terminated. The chick was considered to be following as long as it was within 10 cm of the model. The average amount of time that the chick spent following the model out of a maximum of about 30 seconds was: no sound, 5.35 seconds, novel sound, 10.07 seconds, and experimental sound, 15.21 seconds. An analysis of variance of these scores showed a significant difference ($F=10.15$, 2/26 df, $P <.005$). The 5 percent least significant difference between the means is 4.71, indicating that each of the three conditions differs significantly from the others.

Newly hatched chicks seem to find any sound attractive, but a sound heard during the prenatal period proved more attractive than a novel one in two tests. In the second test the imprinted chicks even occasionally tried to jump on the toy model to get to the speaker. The results of these tests do not seem attributable to a natural preference for lower-frequency stimulation, since chicks in the control group found both the 200-hz and 2000-hz patterns equally attractive. Thus, young chicks are able to respond differentially to a sound heard prenatally. One possible explanation of the results of the following test could be that the sound merely called attention to the model, so that the following was primarily a response to a visual form. But since the model was passed directly in front of the chick several times it is unlikely that the chick could not see it. These studies indicate that the auditory system functions considerably prior to hatching, and perhaps more important, that auditory events during the prenatal period can influence immediate postnatal preferences and behavior. To the extent that the term "imprinting" implies the ability to use this earlier exposure to stimuli as a basis for later behavior such as recognition, attraction, or following, we believe that the experiment demonstrated prenatal auditory imprinting.

J. BROWN GRIER
Department of Psychology,
Northern Illinois University, DeKalb
S. ALLEN COUNTER
WILLIAM M. SHEARER
Department of Speech,
Northern Illinois University, DeKalb

References and Notes

1. G. Gottlieb, *Science* **140**, 399 (1963).
2. ———, *ibid.* **147**, 1596 (1965); ———, *Animal Behav.* **14**, 282 (1966).
3. E. Witschi, *Development in Vertebrates* (Saunders, Philadelphia, (1956), pp. 281-285.
4. Research was facilitated by the cooperation of DeKalb Agricultural Association, Inc.

31 January 1967

Brain Monoamine Oxidase in Mice after Exposure to Aggression and Defeat

Abstract. *Effects on the monoamine oxidase activity of the hypothalamus, amygdala, and frontal cortex of untrained mice exposed to repeated defeat by trained fighters for two 5-minute periods a day for 0, 1, 2, 4, 8, 14, or 20 days were studied. Activity in the hypothalamus increased significantly during the first 2 days of fighting, while the activity in the amygdala and frontal cortex remained essentially unchanged. After 8 days, activity in all three brain areas declined. After 14 days of fighting the monoamine oxidase activity returned to normal, but another decrease was observed in the three areas after 20 days of fighting.*

Changes in the concentration of serotonin in the brain directly affect behavior (*1*). Norepinephrine is also directly involved with behavior (*2*). In addition, norepinephrine is related to the behavioral alterations observed when an animal is attacking or being attacked (*3*). Both serotonin and norepinephrine are involved in the learning ability of mice (*4*). These observations were made after the concentrations of amine in the brain were changed by the administration of drugs or by electrical stimulation of the brain. No experiments, however, have been reported which show the effects of the manipulation of behavior on concentrations of amine in the brain.

Before the role of serotonin and norepinephrine in behavior can be clarified, it is necessary to study the enzyme systems involved in their metabolism. We studied monoamine oxidase, the enzyme that is responsible for the eventual oxidative deamination of monoamines, to determine the effect on this enzyme of repeated exposure to fighting aggression in mice. Mono-

Table 1. Effect of exposure to a trained fighter for two 5-minute periods a day, for indicated days, on monoamine oxidase activity in the hypothalamus, amygdala, and frontal cortex of defeated mice (± S.E.). Monoamine oxidase activity expressed in units in which 1 unit is equal to the amount of enzyme contained in 1 g of tissue which converts 1 μmole of 5-hydroxytryptamine to 1 μmole of 5-hydroxyindoleacetic acid in 1 hour.

Days	Monoamine activity		
	Hypothalamus	Amygdala	Frontal cortex
0	6.46 ± 0.31	5.65 ± 0.24	5.97 ± 0.94
1	8.75 ± .26	5.92 ± .78	5.91 ± .83
2	9.86 ± .70	6.07 ± .40	5.09 ± .18
4	5.68 ± .84	3.97 ± .70	6.53 ± .54
8	3.67 ± .92	4.16 ± .36	4.05 ± .46
14	7.28 ± .15	6.27 ± .93	5.05 ± .76
20	4.24 ± .24	3.79 ± .28	2.50 ± 1.13

amine oxidase deaminates only the physiologically important intracellular free monoamines.

We used *Mus musculus*, strain C57BL/6J. Before experimental data were gathered, a colony of mice was trained to fight according to the procedure of Scott (*5*); these mice were used in fighting encounters with experimental mice.

The experimental mice were weaned and isolated at 21 days of age. After 40 days of isolation, the mice which were not trained to fight were exposed to the trained fighters for two 5-minute periods each day for 1, 2, 4, 8, 14, or 20 days; controls were taken from the same group but they were not exposed to the trained mice. Twelve mice were each exposed at each of these periods for a total of 84 experimental mice. In addition, six mice were transferred from their isolation cage to the fighters' home cage but the fighter was not present at each of these periods. Twenty minutes after the last exposure to a fighter or after cage transfer, the mice were killed by decapitation and their brains were removed quickly; the hypothalamus, amygdala, and frontal cortex were dissected out, frozen in acetone dry ice, and weighed to the nearest 0.1 mg. These three brain areas were kept frozen for a maximum of 2 weeks, then they were analyzed for monoamine oxidase activity. The enzyme was assayed by the method previously devised in our laboratory (*6*). Data were analyzed statistically by means of a standard *t*-test.

Results indicate that monoamine oxidase increased significantly ($P < .005$) in the hypothalamus from 6.46 enzyme units (one unit is equal to the amount of enzyme contained in 1 g of tissue which converts 1 μmole of 5-hydroxytryptamine to 1 μmole of 5-hydroxyindoleacetic acid in 1 hour) to 8.75 and 9.86 at 1 and 2 days of exposure to fighters, respectively (Table 1). During the same period, however, no significant changes were observed in the amygdala and frontal cortex. These latter areas and the hypothalamus exhibited a significant ($P < .01$) decline in monoamine oxidase at 8 days; this decline was followed by a rise at 14 days with an additional significant decline by day 20 of continuous exposure to defeat. Generally, the data show that exposure to aggression profoundly affects the monoamine oxidase activity of the hypothalamus, amygdala, and frontal cortex of the defeated mice. Mice that were transferred to the fighters' cage did not exhibit any changes in brain monoamine oxidase.

The changes in monoamine oxidase activity may reflect the sum total of heightened neural activity associated with various brain areas during exposure to stress. Or changes in monoamine oxidase activity may reflect differential demand and synthesis of serotonin. It is also possible that during aggression stress there is a shift in serotonin from "bound" to "free" which must be oxidatively deaminated by monoamine oxidase.

Several investigators (*7*) have suggested that there are different monoamine oxidases from different amines or that there may be more than one active site involved in this enzyme. We used serotonin as the substrate in the monoamine oxidase analysis, and therefore, the monoamine oxidase activity measured was that for serotonin deamination.

BASIL E. ELEFTHERIOU
KENNETH W. BOEHLKE
Department of Zoology, Kansas State University, Manhattan 66504

References and Notes

1. M. H. Aprison, M. A. Wolf, G. J. Poulos, T. L. Folkerth, *J. Neurochem.* **9**, 575 (1962); ———, *Prog. Brain Res.* **16**, 48 (1965); S. Udenfriend, H. Weissbach, D. F. Bogdanski, *J. Biol. Chem.* **224**, 803 (1957); ———, *J. Pharmacol. Exp. Therap.* **120**, 255 (1957); — →, *Ann. N.Y. Acad. Sci.* **66**, 602 (1957); M. H. Aprison and C. B. Ferster, *J. Neurochem.* **6**, 350 (1961); ———, *Recent Advan. Biol. Psychiat.* **3**, 151 (1961); J. N. Hingtgen and M. H. Aprison, *Science* **141**, 169 (1963); B. B. Brodie and P. A. Shore, *Ann. N.Y. Acad. Sci.* **66**, 631 (1957); D. F. Bogdanski, H. Weissbach, S. Udenfriend, *J. Pharmacol. Exp. Therap.* **122**, 182 (1958); E. Costa and F. Rinaldi, *Am. J. Physiol.* **194**, 214 (1958); P. A. Shore, A. Pletscher, E. G. Tonich, A. Carlsson, R. Kuntzman, B. B. Brodie, *Ann. N.Y. Acad. Sci.* **66**, 609 (1957).
2. M. H. Aprison and J. N. Hingtgen, *J. Neurochem.* **12**, 959 (19(→ B. B. Brodie and P. A. Shore, *Ann. N.Y. Acad. Sci.* **66**, 631 (1957).
3. L. M. Gunne and D. J. Reis, *Life Sci.* **2**, 804 (1963).
4. D. W. Woolley, *Science* **136**, 330 (1962); ———, *Biochem. Pharmacol.* **12** suppl., 233 (1963); ——— and van der Hoeven, *Science* **139**, 610 (1963).
5. J. P. Scott, *J. Comp. Psychol.* **39**, 379 (1946).
6. K. W. Boehlke and B. E. Eleftheriou, *Nature*, in press.
7. M. H. Van Woert and C. C. Cotzias, *Biochem. Pharmacol.* **15**, 275 (1966); S. R. Guha, and C. R. K. Murti, *Biochem. Biophys. Res. Commun.* **18**, 350 (1965); V. Z. Gorkin, N. V. Komisarova, M. I. Lerman, I. V. Veryovkina, *ibid.* **15**, 383 (1964).
8. Contribution No. 387, Department of Zoology, Kansas Agricultural Experiment Station, Manhattan 66504.

23 January 1967

Recommendations on Nomenclature of the Order Mycoplasmatales:

The need of international agreement on the nomenclature of mycoplasmas (pleuropneumonia-like organism, PPLO) has been felt for a long time and has been further accentuated by the rapidly increasing interest in this group of microorganisms. More particularly, there have recently been examples of new species being named without adequate descriptions and there have been other departures from the accepted Code of Nomenclature of Bacteria. Realizing that a chaotic situation was starting, the initiative was taken by a small group particularly interested in *Mycoplasma* taxonomy to take advantage of the New York Academy of Sciences Second Conference on the Biology of Mycoplasmas, held in May 1966, to establish a provisional Subcommittee on the Taxonomy of Mycoplasmatales. Subsequently, at its meeting on 23 July 1966, in Moscow, U.S.S.R., the International Committee on Nomenclature of Bacteria authorized this subcommittee and approved its recommendations.

The minutes of the meeting of the Subcommittee on the Taxonomy of Mycoplasmatales in May 1966 have been published in the *International Journal of Systematic Bacteriology* (*1*). The recommendations of the Subcommittee are summarized here together with further comments and advice, so that they may be brought to the immediate attention of as wide a circle of microbiologists as possible.

1) The Subcommittee noted that the system of classification and nomenclature of the mycoplasmas, based on the binomial system, that was proposed in 1956 by Edward and Freundt (*2*) has