and lymphocytes treated with medium only gave biphasic distribution profiles (Fig. 1, a and c). The neuraminidase-treated population gave a monophasic profile with a mean mobilty lower than that observed in either of the peaks of untreated cells (Fig. 1b). This experiment was repeated four times, with similar results.

A decrease in electrophoretic mobility may have been due to removal of charged groups, to adsorption of substances which thus covered charged groups, or to a rearrangement of the surface. The reaction mixtures were therefore also incubated in the presence of 10 m*M* ethylenediaminetetraacetic acid (EDTA) at 4°C, under which conditions *V. cholera* neuraminidase is enzymatically ineffective. This treatment did not disturb the diphasic distribution profile (Fig. 1d). Thus adsorption of neuraminidase cannot be the reason for the observed changes. These cells migrated somewhat faster than the untreated controls, perhaps because of the chelating action of EDTA, which alters the electrophoretic mobility of cultured cells (*13*).

There could have been selective loss of either B or T cells during neuraminidase treatment and the electrophoresis procedure. Therefore, samples from the peak fraction of the neuraminidase-treated and electrophoretically separated cells were tested with antiserum to MBLA and antiserum to θ (*3, 4*). In the presence of complement, 28 percent of the cells were killed by the antiserum to MBLA, as judged by the trypan blue dye exclusion test, and 58 percent were killed by antiserum to θ (Table 1). This roughly corresponds to the amounts of B and T cells in the untreated material and proves that selective loss has not taken place.

Thus, the difference in electrophoretic mobility and hence in the density of charged groups on the surfaces of B and T lymphocytes seems to be due to different amounts of sialic acid exposed at the electrokinetic plane of shear. The θ antigen cannot be responsible for the difference, since thymocytes on which the density of θ antigen is higher than on mature T cells (*14*) have the same electrophoretic mobility as B cells (*2*), and since both the MBLA and the θ antigen are still demonstrable on neuraminidase-treated lymphocytes. This indicates that sialic acid is not an essential part of these surface antigens.

Our findings, however, do not necessarily mean that the total amount of sialic acid on B and T cells is different. A definite possibility is that some of the sialic acid groups on the B cells are covered. Lymphocyte surface receptors are considered to be entirely immunoglobulins. They exist on the surface of both B and T cells, but are more abundant on B cells (*15*). The hypothesis that the low electrophoretic mobility of B cells is due to covering of some of the charged groups by receptor antibody molecules is supported by the fact that treatment of various types of cells with specific antibodies causes measurable reduction of the surface charge density, provided that the density of bound immunoglobulins is high enough (*16*). This does not explain why thymocytes have a low electrophoretic mobility (*2*). To obtain convincing evidence about whether or not the low electrophoretic mobility of B lymphocyte is due to the covering effect of surface immunoglobulins, it would be necessary to remove these immunoglobulins specifically.

STIG NORDLING, LEIF C. ANDERSSON
PEKKA HÄYRY
Third Department of Pathology,
University of Helsinki,
SF-00290 Helsinki, Finland

References and Notes

1. K. Zeiller, E. Holzberg, G. Pascher, K. Hannig, *Hoppe-Seyler's Z. Physiol. Chem.* **353**, 105 (1972).
2. S. Nordling, L. Andersson, P. Häyry, *Eur. J. Immunol.*, in press.
3. M. C. Raff, *Nature* **224**, 378 (1969).
4. ———, S. Nase, N. A. Mitchinson, *ibid.* **230**, 50 (1971).
5. G. Bert, J. A. Forrester, A. J. S. Davies, *Nature New Biol.* **234**, 86 (1971).
6. K. Zeiller, G. Pascher, K. Hannig, *Hoppe-Seyler's Z. Physiol. Chem.* **351**, 435 (1970); K. Zeiller and K. Hannig, *ibid.* **352**, 1162 (1971).
7. L. Weiss, *The Cell Periphery, Metastasis and Other Contact Phenomena* (North-Holland, Amsterdam, 1967); L. Weiss, *In Vitro* **5**, 48 (1970).
8. B. C. W. Hummel, *Can. J. Biochem. Physiol.* **37**, 1393 (1959).
9. G. Kalnitsky, J. P. Hummel, C. Dierks, *J. Biol. Chem.* **234**, 1512 (1959).
10. P. Häyry, K. Penttinen, E. Saxén, *Ann. Med. Exp. Biol. Fenn.* **43**, 91 (1965); E. Mayhew, *J. Cell. Physiol.* **69**, 305 (1967).
11. K. Hannig, in *Methods in Microbiology*, J. R. Norris and D. W. Ribbons, Eds. (Academic Press, New York, 1971), vol. 5B, p. 513.
12. C. G. Bhattacharya, *Biometrics* **23**, 115 (1967).
13. L. Weiss, *J. Cell Biol.* **35**, 347 (1967).
14. T. Aoki, U. Hämmerling, E. de Harven, E. A. Boyse, L. J. Old, *J. Exp. Med.* **130**, 979 (1969).
15. J. M. Dwyer and I. R. Mackay, *Rev. Eur. Etud. Clin. Biol.* **16**, 743 (1971).
16. G. Bert, A. L. Massaro, D. Lajolo di Cossano, M. Maja, *Immunology* **17**, 1 (1969); P. Sachtleben, in *Cell Electrophoresis*, E. J. Ambrose, Ed. (Churchill, London, 1965), p. 100.
17. We thank Miss M. Schoultz and Mrs. Hilkka Sokura for technical assistance. Supported by grants from the Sigrid Jusélius Foundation and the National Research Council for Medical Sciences, Finland.

5 July 1972

Acquisition of Key-Pecking via Autoshaping as a Function of Prior Experience: "Learned Laziness"?

Abstract. *A group of pigeons that had previously received noncontingent food delivery acquired the key-peck response (in autoshape training) more slowly than did a naive control group; key-peck acquisition was most rapid for a group given operant treadle-press training in the initial phase.*

Consider the following experiment reported by Seligman and Maier (*1*). In the initial stage of training, dogs were strapped in a Pavlovian harness and administered inescapable, unavoidable shocks on a random schedule. Twenty-four hours later they were placed in a shuttle box and given training during which the animal could escape or avoid the shock by jumping over a hurdle that separated the two compartments of the apparatus. About 65 percent of the subjects given the inescapable shocks failed to learn the escape response in the shuttle box, whereas only 6 percent of a naive control group failed to escape. The authors' interpretation was that during the first stage, the subject learns that instrumental behavior has no effect on either the onset of the aversive stimulus or its termination. When the subject is placed in the shuttle box with the same aversive stimulus present, he responds with the "expectation of helplessness" derived from previous experience with a problem that lacked an instrumental solution. After this and other demonstrations of "learned helplessness," this phenomenon was studied in an attempt to establish the necessary and sufficient conditions for its development (*2, 3*).

Maier *et al.* speculated (*2*) that a parallel phenomenon might exist in an appetitive situation; that is, a subject given repeated exposure to noncontingent reinforcement might be retarded in acquiring a response that now procures reinforcement. Most appetitive responses are acquired, however, through a shaping procedure involving reinforcement of successive approximations to the desired response. Since this is typically done manually, it is difficult

to assess the course of acquisition of the response. Brown and Jenkins (*4*) developed a method that allows for the mechanization of the shaping process. Their "autoshaping" procedure involves projecting a light stimulus on a pigeon's response key for a few seconds before a noncontingent delivery of food. Brown and Jenkins reported that all pigeons began spontaneously pecking the illuminated key after a few presentations. The behavioral control generated by this situation, which strongly resembles Pavlovian delay conditioning (*5*), is so powerful that subjects persist in responding even when the contingencies are rearranged so that key-pecking prevents the presentation of the food (*6*).

In the present experiment, we assessed the effects of initial treatments by measuring resistance to the powerful autoshaping procedure. If noncontingent food delivery produces a helplessness-like effect, then subjects so treated should autoshape slower than controls not given this treatment. Our procedures differ conceptually from the conditioned helplessness experiments. In the latter, classical fear conditioning interferes with later instrumental learning (*2*); whereas our study involves transfer from a quasi-operant situation to what has been called a classical conditioning paradigm (*5*). This difference should not be important if, as Maier *et al.* (*2*) have suggested, the interference arises from some kind of an expectation or conceptual set to not respond.

Twenty-seven naive adult domestic pigeons, maintained at 75 percent of their body weight when free access to food had been given, were trained in a standard single key operant conditioning chamber. The subjects' compartment was modified by the insertion of a removable treadle (6 cm long by 9 cm wide) which protruded from a piece of plastic (18 cm wide) that ran from floor to ceiling and was placed diagonally across the left corner of the chamber. This treadle was present only for the subjects in the treadle group during the initial training phase of the experiment.

Subjects were randomly assigned to one of three groups. The treadle group was trained to approach and eat from the hopper for 1 day. On the next day they were trained by the method of successive approximations to press the treadle with their feet and were allowed to earn 30 reinforcers on a continuous reinforcement schedule. Reinforcement on day 3 was at variable intervals averaging 12 seconds (a variable-interval, 12-second schedule); a variable-interval, 20-second schedule was in effect on day 4. Starting on day 5 and continuing for the next 30 days, subjects were allowed to earn 30 reinforcers per day for treadle-pressing, with reinforcement on a variable-interval, 30-second schedule. The reinforcer used throughout the experiment consisted of 3 seconds of access to a hopper of mixed grain. The hopper group was given the same number and patterning of food presentations as the treadle group, but food delivery was independent of the subjects' behavior. The control group was weighed and handled each day but was not introduced into the chamber until the last day of initial training. During this session the subjects were given 30 hopper presentations.

Table 1. Trials to criterion in autoshape key-peck training (S, subject).

Treadle group		Hopper group		Control group	
S	Trials	S	Trials	S	Trials
7	17	12	61	26	30
10	30	9	77	8	41
1	31	30	98	5	60
28	39	15	121	2	66
19	60	18	121	20	92
4	60	24	165	23	103
13	60	3	273	11	164
16	129	27	360	14	762
22	360	21	516		
Median					
	60		121		79

After initial training all subjects were exposed to an autoshape procedure in which the treadle was removed from the box and the response key was illuminated with a 555-nm light for 8 seconds immediately preceding a noncontingent food delivery. Thirty such food presentations were delivered during each session on a variable-interval, 30-second schedule with a geometric distribution of intertrial intervals ranging from 5 to 55 seconds. The autoshape schedule was followed until subjects reached a criterion of responding on at least eight out of ten consecutive stimulus presentations.

The results are shown in Table 1. The treadle group acquired the response in a median of 60 trials (excluding the criterion trials themselves), the control group required 79 trials, and the hopper group required 121 trials to criterion (*7*). Whereas only two of the nine subjects in the treadle group needed more than 60 trials to criterion, five of the subjects in the control group and all nine of the subjects in the hopper group needed more than 60 trials. A Kruskal-Wallis analysis of variance revealed a significant group effect ($H = 6.92$, d.f. $= 25$, $P < .05$).

A comparison can be made between the present results and those of the conditioned helplessness experiments. The concept of helplessness has been applied (*1–3*) to a situation in which the subjects' failure to respond is maladaptive, because responding is now reinforced. In the present study, the failure to respond is not maladaptive, because no response is required to receive food. The hopper subjects appear to have learned that they will be fed regardless of their behavior and therefore the probability of their responding is low. The notion of "learned laziness" better connotes such behavior than that of learned helplessness. The effect of initial training on later autoshaping performance is bidirectional, in that the treadle group—trained to make a response to obtain food—acquired the key-pecking response faster than did the control group. This suggests that not only is laziness conditionable, but so is "industriousness." A comparable effect was not found in the conditioned helplessness experiments; subjects given prior escape training did no better on the shuttle box task than did naive control subjects (*8*).

It is appropriate to consider alternative interpretations of the present results. One posibility is that the hopper group acquired some sort of (adventitiously reinforced) superstitious behavior pattern that was incompatible with key-pecking. Our experiment was designed with this possibility in mind. The treadle group was explicitly trained to make a response that could compete with key-pecking. Any tendency to press a now-absent treadle or even to orient toward that corner of the chamber in which the treadle had been placed would remove the subject from the immediate vicinity of the key and perhaps even lessen the likelihood that the key light would be seen. Nevertheless, the treadle group autoshaped the best, rather than the worst. Furthermore, the hopper group received their food deliveries on a variable interval schedule, making it unlikely that they would develop any particular superstitious response pattern. On the contrary, such subjects would probably be engaging in a variety of different responses at the time of food delivery, and they might therefore learn that food presentation is independent of their behavior.

The results for both the treadle and hopper groups can be explained by a single underlying mechanism, that is, the development of a set to respond (industriousness) or of a set to not respond (laziness). Presumably the treadle group learned that reinforcement is contingent on some response and they entered the autoshaping situation with a set or expectation that a similar contingency still applied. The hopper birds responded less because they anticipated a continuation of the noncontingency that existed in their first stage of training.

Our interpretation is entirely parallel to that offered by Maier *et al.* (*2*) for the learned helplessness phenomenon. If that interpretation is valid, then the significance of subjects' ability to control their environment is not restricted to commerce with aversive stimuli or to any single motivational or reward system. Thus, speculations involving presumed physiological or biochemical correlates of helplessness in terms of stress reactions (*3*) are likely to be of limited use. Further research will be necessary to validate our interpretation as well as to explore the limits of its applicability.

LARRY A. ENGBERG
GARY HANSEN
ROBERT L. WELKER
DAVID R. THOMAS
Department of Psychology, University of Colorado, Boulder 80302

References and Notes

1. M. E. P. Seligman and S. F. Maier, *J. Exp. Psychol.* **74**, 1 (1967).
2. S. F. Maier, M. E. P. Seligman, R. L. Solomon, in *Punishment and Aversive Behavior*, B. A. Campbell and R. M. Church, Eds. (Appleton-Century-Crofts, New York, 1969), pp. 299–342.
3. M. E. P. Seligman, S. F. Maier, R. L. Solomon, in *Aversive Conditioning and Learning*, F. R. Brush, Ed. (Academic Press, New York, 1971), pp. 347–400.
4. P. L. Brown and H. M. Jenkins, *J. Exp. Anal. Behav.* **11**, 1 (1968).
5. E. Gamzu and D. R. Williams, *Science* **171**, 923 (1971).
6. D. R. Williams and H. Williams, *J. Exp. Anal. Behav.* **12**, 511 (1969).
7. One subject in the control group died during the experiment and was not replaced.
8. J. B. Overmier and M. E. P. Seligman, *J. Comp. Physiol. Psychol.* **63**, 28 (1967).
9. Supported by NIH research grant HD-03486 and training grant MH-10427.

2 August 1972

Similarity in Developmental Profile among Related Pairs of Human Infants

Wilson (*1*) presented evidence that he interprets as demonstrating that the similarity in overall level and developmental profile contour of scores on the Bayley infant test is greater for monozygotic (MZ) than for dizygotic (DZ) twins for assessments made during the first year (3, 6, 9, and 12 months) and the second year (that is, 12, 18, and 24 months) of life. The intraclass correlations expressing similarity within pairs were very high, approaching and in some cases equaling the reliability of the test.

Wilson cites the fact that the intraclass correlations for DZ twins were high for both overall level ($r = .75$ and .79, for the first and second years, respectively) and profile contour ($r = .52$ and .50), and that the size of these concordances signifies "that the differences within DZ pairs produced by gene segregation and different life experiences are comparatively small in relation to the sizable differences between pairs" (*1*, p. 917). He concludes that genetic factors are paramount in such development. However, circumstances surrounding the analysis of these data may modify this interpretation.

The degree of heritability for a trait depends on the difference between the correlations for MZ and DZ twins, not on the absolute size of the DZ correlation. For example, "broadsense heritabilities" can be calculated by taking twice the difference between r_{MZ} and r_{DZ} (*2*). Mating is assumed to be random in this heritability estimate, an assumption that is probably tenable since infant test scores do not relate very strongly to adult mental or personality characteristics that might influence mate selection (*3*). Wilson's data treated in this manner give heritabilities of .30 and .20 for overall level (first and second years, respectively) and .50 and .30 for profile contour. These values are lower than those reported for IQ in later childhood (*4*), and do not seem to warrant the conclusion that "infant mental development was primarily determined by the twins' genetic blueprint and that . . . other factors served mainly a supportive function" (*1*, p. 914).

The high correlations for DZ twins may derive from common nongenetic as well as genetic circumstances. In fact, since the genetic correlation for DZ twins averages .50 (they have half their genes in common), correlations between DZ twins substantially higher than .50 must reflect common environmental circumstances or assortive mating, or both these factors.

When these same analyses were performed on 142 sibling pairs from the Fels Longitudinal Study, the intraclass correlations for Gesell developmental scores were .24 and .44 for overall level (at 6 and 12 months and at 12, 18, and 24 months, respectively) and .09 and .14 for profile contour. The twin correlations reported by Wilson are two to six times these values, despite the fact that the degree of genetic overlap is the same for DZ and sibling pairs. Twins may be more similar than siblings because they share environmental circumstances (prenatal environment, stimulating familial experiences, and so forth) and because those environmental factors have their effects at the same age for twins but at different ages for siblings.

The method of analysis used by Wilson also raises some issues of interpretation. His intraclass correlations express within-pair variability relative to the appropriate total variability separately for MZ's and DZ's, before these groups are compared. Such a procedure may be justified when the variability between individuals in one group is different than that in another but this is reportedly not the case for the data presented.

A more direct approach is to contrast the within-pair variability for MZ's and DZ's by calculating the ratio of the mean square within pairs for DZ's divided by the mean square within pairs for MZ's. For Wilson's data, this procedure yields significant differences between zygosity groups for overall level during the first year ($F = 3.44$, d.f. $= 51/45$, $P < .0001$) and second year ($F = 1.74$, d.f. $= 46/51$, $P < .03$) and for profile contour during the first year ($F = 1.94$, d.f. $= 153/135$, $P < .0001$) but not during the second year ($F = 1.18$, d.f. $= 92/102$, $P \cong .25$). Thus, when differences in similarity within pairs are considered directly, MZ's are more similar than DZ's in profile contour only during the first year of life.

The entire univariate analysis-of-variance model involving repeated measures, which was used by Wilson to estimate profile similarity (as well as the above-mentioned analyses), tends to have a positive bias toward significant effects (in this case favoring within-pair similarity) if the covariances (that is, correlations) between all pairs of scores measured on the same individuals are not equal (*5*). Such heterogeneity is almost always present in