Table 1. Complement fixation by heat-aggregated normal human IgG. Eleven samples were studied.

IgG (mg/ml)	Total $C'H_{50}$ units	$C'H_{50}$ units fixed	Mean $C'H_{50}$ units fixed (%)
0.5	16.0 ± 0.5	13.4 ± 1.0	83.6
0.25	16.0 ± 0.5	8.3 ± 1.3	51.8

or the presence of an IgG population which lacks large numbers of these sites, or both. It is also possible that the defect in complement fixation is due to incomplete aggregation of the IgG of the patients. However, this is also consistent with the interpretation that there is a qualitative difference in their IgG which results in the biological variance. Under the conditions used there was no visible evidence of aggregation either in normal samples or in those of the patients. The difficulty of obtaining adequate amounts of isolated IgG from the patients precluded testing for aggregation by other methods.

An interesting and significant parallel for such electrophoretic and functional differences in IgG subgroups is present in the guinea pig. In this species, two distinct types of IgG, one "fast" (γ1) and one "slow" (γ2), were shown by electrophoresis. Each differs in several biologic functions (*6*); for example, the γ1 has been shown not to fix complement, while the γ2 subclass does (*6*). In addition, γ1 antibodies are capable of sensitizing guinea pig lung for antigen-induced histamine release; γ2 antibodies are not capable of this (*6*). Furthermore, populations of IgG molecules differing in heavy-chain antigenic groups vary in biological activity. Terry (*7*) has shown that the γ2a (Ne) subgroup of myeloma proteins do not fix to skin. Müller-Eberhard (*8*) found deficient interaction between myeloma proteins of the γ2d (Ge) and γ2a (Ne) subgroups with C'1q.

Apparently, individual γ-globulin molecules are not biologically equivalent, and the total biological capacity of the humoral antibody system is probably a function of normal population distribution.

Alterations in normal population ratios might result in qualitative defects not explainable on a quantitative basis. While this may be the result of faulty genetic information, the point at which the disturbance in synthesis occurs cannot be defined from these data.

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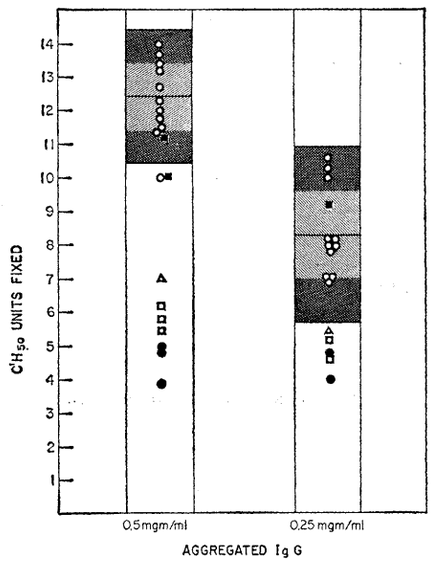

Fig. 1. Number of $C'H_{50}$ units of guinea pig complement fixed from a total of 16 $C'H_{50}$ units by heat-aggregated human IgG. The shaded area indicates a range of two standard deviations from the normal mean based on studies of 11 normals. S.W., S, H.X., and H are four patients with hypogammaglobulinemia. Normal, ○; S, ●; H.X., □; S.W., ■; H, △.

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26 May 1967

Reward and Learning in the Goldfish

Abstract. *An experiment with goldfish showed the effects of change in amount of reward that are predicted from reinforcement theory. The performance of animals shifted from small to large reward improved gradually to the level of unshifted large-reward controls, while the performance of animals shifted from large to small reward remained at the large-reward level. The difference between these results and those obtained in analogous experiments with the rat suggests that reward functions differently in the instrumental learning of the two animals.*

The central fact of instrumental learning is that behavior can be controlled by manipulation of its consequences. If a hungry rat is rewarded with a pellet of food for pressing a retractable bar each time the bar is introduced into its cage, it comes to press the bar more and more readily as training proceeds. Large rewards are more effective than small rewards; the animal responds more promptly when, say, ten pellets are given for each press than when only one pellet is given. How does reward work?

One early answer to this question was that reward acts directly to strengthen or "reinforce" connections between contiguously active sensory and motor centers in the brain (as between the centers activated by the introduction of a bar and the motor center for pressing the bar). Performance is better with large rewards than with small rewards because large rewards produce stronger connections. Another early answer was that the animal learns *about* reward—that its increasing readiness to respond as training continues reflects a growing anticipation of reward. Differences in level of performance produced by differences in amount of reward reflect, in this view, differences in the attractiveness of the rewards that are anticipated, the requisite neural connections being assumed to develop as a function of contiguity alone. Although the reinforcement interpretation was widely accepted for a time, contemporary learning theorists are virtually unanimous in preference for the contiguity interpretation (*1*). Of the various experiments whose results dictate this preference, one of the most important is Crespi's (*2*).

Suppose that two groups of rats are

trained to make some simple instrumental response, one group with small reward and the other with large reward, until their performance stabilizes (the second group responding more rapidly than the first). Then half the small-reward animals are shifted to large reward, and half the large-reward animals are shifted to small reward. What should be the effects of these changes? The reinforcement principle suggests that the speed of the animals shifted from small to large reward should increase gradually to the large-reward level, but that the speed of the animals shifted from large to small reward should remain at the large-reward level (since connections of the strength required for performance at that level already have been established in them by large reward).

Crespi's results in just such an experiment were quite different: both shifted groups showed dramatic changes in performance. The speed of the animals shifted from small to large reward increased rapidly to a level well *above* that of the unshifted large-reward controls (the "elation" effect), while the speed of the animals shifted from large to small reward fell well *below* that of the unshifted low-reward controls (the "depression" effect). These so-called contrast effects suggested that the shifted animals were reacting to perceived differences between preshift and postshift amounts of reward, in which case the animals must have learned and remembered something about the preshift amounts of reward as such. The bidirectionality of the changes in performance produced by the changes in reward, their suddenness, and their magnitude were difficult to understand in terms of the reinforcement principle. It was largely on the basis of Crespi's results that Hull, the leading exponent of the reinforcement principle (*3*), found it necessary to abandon the idea that "habit strength" varies with amount of reward (*4*).

Our experiment with goldfish, patterned after Crespi's experiment with the rat, was prompted by the results of some recent comparative studies of resistance to extinction in fish and rat as a function of frequency and amount of reward in training (*5*); the results suggested that reward plays different roles in the learning of the two animals. Consider, for example, the fact that resistance to extinction in the rat is *less* after training with large reward than after training with small reward, while resistance to extinction in the fish increases with amount of reward. The rat result seems to be another contrast effect, but the fish result follows from the reinforcement principle and leads us to look for other evidence of the operation of a reinforcement mechanism in the learning of the fish.

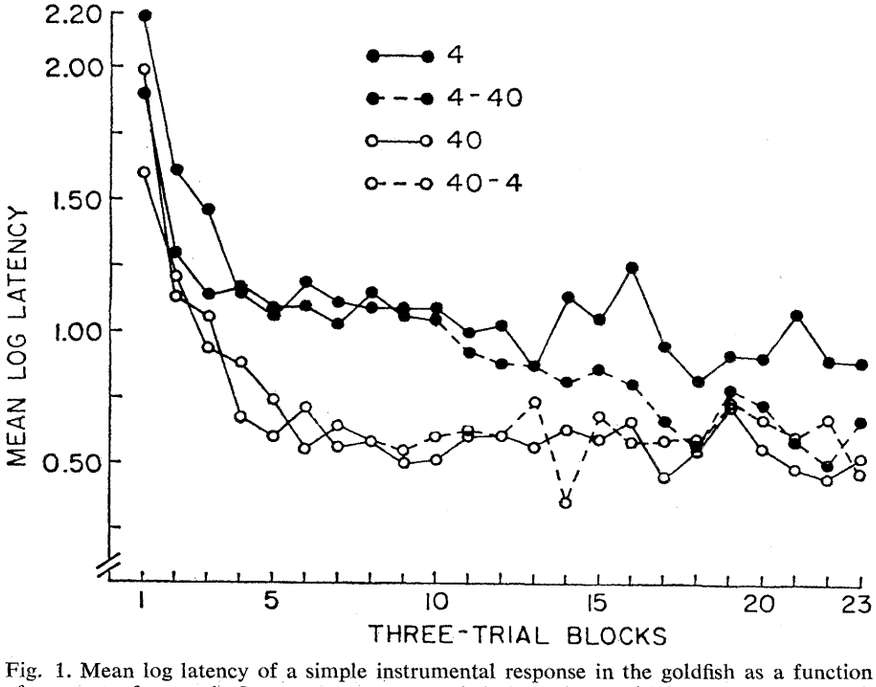

Fig. 1. Mean log latency of a simple instrumental response in the goldfish as a function of amount of reward. One group was rewarded throughout with 4 worms; a second, with 40 worms; a third was shifted from 4 to 40 worms; and a fourth, from 40 to 4 worms. Postshift performance is shown by broken lines.

Our subjects were 48 10-cm goldfish, housed in individual 8-liter tanks of conditioned water which was filtered, aerated, and regulated as to temperature. Each animal was trained in its own tank, which was carried once daily to a black plexiglass enclosure diffusely illuminated by a white house light; the apparatus has been described (*6*). In the preliminary (feeder) training, a worm-feeder was operated once shortly after the animal had been brought to the apparatus, and the animal was removed after it had eaten all the worms (a cluster of 40 *Tubifex* worms for half the animals, and a cluster of four worms for the others). When the animals were taking the worms immediately (after four to six trials), instrumental (target) training was begun, with one trial daily. A wire-mesh target, suspended from the lid of the enclosure, was illuminated with red light (and the house light was extinguished) 2 seconds after the lid had been lowered.

For the first six trials, the target was baited with dry food to attract the animal; on all subsequent trials it was unbaited. As soon as the animal made contact with the target, the red target light was turned off, the white house light was turned on again, and the feeder was operated (delivering the same number of worms as had been given in feeder training); the animal was then removed from the apparatus. The latency of response (the time between onset of the target light and the animal's contact with the target) was read from a timer (*7*). All events of each trial (changes in illumination, detection of response, and operation of the feeder and of the latency timer) were automated.

On trial 23 and thereafter, half the 40-worm animals (group 40-4; *N*, 13) were given four worms, while the rest (group 40; *N*, 12) continued to receive 40 worms. On trial 30 and thereafter, half the four-worm animals (group 4-40; *N*, 12) were given 40 worms, while the rest (group 4; *N*, 11) continued to receive four worms. In all, there were 63 training trials (one daily for 63 days). Throughout the experiment total daily intake of food was the same for all groups; 1 hour after the experimental session each fish was fed a basic ration of dry food plus either 4 or 40 worms (depending on the number that had been given on that day's trial) to make a total of 44 worms for each day.

In Fig. 1 the performance of the four groups is plotted in terms of mean log latency of response over three-trial blocks. (The logarithmic transformation was used to normalize the distributions for statistical purposes.) It seems clear from the curves that the two amounts of reward produced different asymptotic latencies, that a shift from small to large reward produced a gradual improvement in performance, and that a shift from large to small reward produced no deterioration of performance —precisely the pattern of results indicated by the reinforcement principle. A repeated-measures factorial analysis of variance for the postshift data (blocks 11 to 23) shows significant effects of (postshift) amount of reward (F, 11.11; df, 1/44; $P < .01$) and of change in amount of reward (F, 4.76; $P < .05$), as well as a significant interaction of amount with change (F, 30.24; $P < .01$). There are significant differences between groups 4 and 40 (F, 39.01; $P < .01$) and between groups 4 and 4-40 (F, 14.01; $P < .01$), but not between groups 40 and 40-4 ($F < 1$).

These results with goldfish differ from Crespi's results with rats in two respects: One of the differences—the absence of an elation effect in the data for the fish—cannot be given much weight because the effect does not appear dependably even in data for the rat; the conditions necessary to produce it have not yet been clearly defined. The second difference does, however, seem to be of considerable importance. Although the depression effect in the rat is a highly dependable one, the fish not only fails to show it, but also fails to show any decrement in performance whatsoever with a discriminable downward shift in the amount of reward. (The discriminability of the change in amount of reward is shown by the significant difference in the asymptotic latencies of groups 4 and 40, as well as by the postshift decrease in the latency of group 4-40.)

One should consider the possibility, of course, that the results for the fish are the product simply of a rather special set of experimental conditions that happen to differ markedly from those under which the depression effect appeared in the rat (*8*), but that is not very likely. The conditions certainly were similar enough to produce the same preshift pattern. Furthermore, the results for the fish were anticipated on the basis of the results of a series of related extinction experiments.

The simplest interpretation of our results, perhaps, is that the reinforcement principle holds for the fish but not for the rat. Another possibility is that the reinforcement principle holds for both animals, but that its operation in the rat is masked by a contrast mechanism (based on learning about, and anticipation of, reward) which is not present in the fish. Already there is evidence from surgical studies indicating that qualitative differences in the adjustment of the two animals may be due to operation in the rat of second-order processes masking lower-order functional communalities. For example, fish-like behavior is found in adult rats that have been extensively decorticated in infancy (*9*). It will be interesting to see the effects of decortication on performance in a Crespi experiment and in experiments on resistance to extinction as a function of amount of reward.

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15 May 1967

Evoked Potentials: Three-Dimensional Display

Abstract. *Procedures are described for displaying large numbers of evoked potentials. A photographic superposition of average evoked responses, with the concurrent modulation of the brightness of each trace, yields a display having the appearance of a three-dimensional surface formed from hundreds of average responses.*

The study of evoked potentials in the nervous system is a very active field in brain research throughout the world. (We use "evoked potential" to mean the time-locked changes in potential that are recorded with macroelectrodes after presentation of a sensory stimulus.) To a large extent this widespread interest in evoked potentials has been spurred by the advent of computers and the development of averaging techniques (*1*). The summation of potentials evoked by successive stimuli, and the appropriate scaling of these sums to obtain the average evoked response, can now be achieved with a number of special-purpose averaging devices as well as with larger programmable machines.

In many instances average evoked responses have exceedingly complex wave forms; description of these wave forms is seldom simple, and even the variability of average responses may be distressingly large. Summarizing data of this kind has frequently proved difficult, especially for experiments of relatively long duration in which hundreds or even thousands of average responses are obtained from individual subjects. Perhaps the most common way of dealing with these problems has been to present the "typical" average response for some experimental condition; other, more satisfactory, procedures also have been employed. Quantitative measurements of amplitudes or latencies may provide a satisfactory description of some changes in evoked potentials, especially when accompanied by an estimate of variability.

Changes in complex wave forms do not always lend themselves, however, to descriptions by a few specific meas-