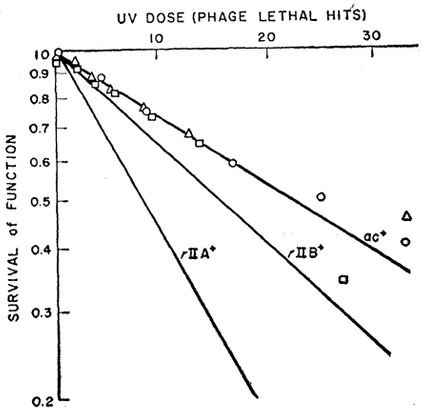

Fig. 2. Ultraviolet-light inactivation curve of the ac^+ function. Survival of ac^+ function is calculated as described in the text. Data from experiments illustrated in Fig. 1 (circles and triangles) and from an experiment with irradiated *r*IIA*r*IIB$^+$*ac*$^+$ and unirradiated *r*IIA$^+$*r*IIB*ac* (rectangles). Survival curves for the *r*IIA$^+$ and the *r*IIB$^+$ functions (obtained in these experiments) are shown for reference. For the ac^+ function, zero-dose values are less than 1, due to the small fraction of mixedly infected bacteria which form plaques in the presence of acriflavine (at zero dose).

aged *r*II$^+$ and an undamaged ac^+ gene. This number, divided by the number on unsupplemented plates, gives the fraction of complexes at each dose in which the ac^+ function survives *among* those in which the *r*IIA$^+$ function survives as well. Data from two experiments of this type are given in Fig. 1. The calculation of the data to give the survival of the ac^+ gene is presented in Fig. 2. Also given in Fig. 2 are comparable results from an experiment with unirradiated *r*IIA$^+$*r*IIB*ac* and irradiated *r*IIA*r*IIB$^+$*ac*$^+$. In this experiment the inactivation of the ac^+ function, together with the survival of the *r*IIB$^+$ rather than the *r*IIA$^+$ gene function, is measured.

Our results on the target sizes of the *r*IIA$^+$ and *r*IIB$^+$ functions are in good agreement with the previous measurements by Krieg (*1*). The survival of the ac^+ function, like that of the *r*IIA$^+$ and *r*IIB$^+$ functions, decreases in a simple exponential manner with increasing irradiation dose. The slope of the survival curve indicates that the ac^+ function is 0.03 of the total ultraviolet-sensitive target of the phage. No data are available at the present time which will permit a comparison between the ultraviolet cross section of the ac^+ function and an estimate of the length of the ac^+ gene obtained from mapping experiments.

In the experiments described here we measure only the damage to the ac^+ genes among those phage in which the *r*II$^+$ function survives. Any class of damages which simultaneously inactivate both the ac^+ and one of the *r*II$^+$ genes is not included in our estimate of the *ac* target size. One class of damage which might be expected to occur is that which renders the phage totally inoperative (for example, that which prevents injection). However, Barricelli (*2*) has suggested that irradiation damages, although discretely localized in the genome, may have a finite size. If this were true one would expect that some ultraviolet hits of this type could inactivate two neighboring genes at once. Indeed, Krieg (*1*) found a correlation between the inactivation of the *r*IIA$^+$ and the *r*IIB$^+$ genes which could be explained on this basis. If such extended damages contributed significantly to our data, the apparent target size of the ac^+ gene would be smaller in experiments requiring survival of the *r*IIB$^+$ gene than in experiments requiring survival of the *r*IIA$^+$ gene. As can be seen from the data presented in Fig. 2, no such effect is detectable. This only means that, if ultraviolet-induced lesions have a finite size, this size must be smaller than the distance between the *r*IIB$^+$ gene and the ac^+ gene (less than six map units).

Features of this system may make it useful in other studies on the genetic effects of ultraviolet irradiation. Since the cells which yield phage are those in which the irradiated parent has received at least one ultraviolet-induced lesion, there exists a selective system for examining the genetic consequences of very low doses of ultraviolet light. Since only that fraction of the irradiated phage which has a surviving *r*II$^+$ function yields phage, experiments are not influenced by damages which render the page totally inoperative (*3*).

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Nonreinforced Trial Procedure for Probability Learning

Abstract. For rats in a T-maze, nonreinforced trials were interspersed with reinforced trials to insure that .67 of all reinforcements would be dispensed on the more frequently reinforced side. At asymptote, the proportion of responses to the more frequently reinforced side was .67.

Probability learning (as defined here) refers to experiments in which each trial ends with one of two responses. One response (called M in this report) is more frequently reinforced than the other response (called L). The independent variable is the ratio of reinforcements for M to all reinforcements; this ratio is called π. The dependent variable is the proportion of trials during which M is emitted; this proportion is called P. Functional relationships between π and P have been derived from theories about the effect of reinforcements on response probability, and much probability learning experimentation has been devoted to the development of these theories (*1*, *2*). Unfortunately, all the procedures used to control π in animal learning situations involve the introduction of factors not considered in these theories. In the light of generally applicable behavior principles, these factors should affect P as follows.

1) In the *forced trial procedure*, forced trials are interspersed with free trials in order to control π, and P is reckoned as the proportion of free trials during which M is emitted. An extraneous factor is a discrimination between forced trials and free trials which develops in the course of the experiment, so that reinforcement of forced trials affects P less than does reinforcement of free trials (*3*).

2) In the *correction procedure*, one response is selected for reinforcement on each trial, M being selected with probability π. If the initial response of the trial is not the response to be reinforced, the trial does not end until the reinforced response is emitted (that is, "correction" is permitted). P is reckoned as the proportion of trials during which M is the initial response of the trial. One possible extraneous factor in this procedure is that an initial response not scheduled for reinforcement may receive delayed reinforcement because it is followed by reinforcement of a correction. In addition, a correction is topographically different from an initial response, so that a discrimination between corrections and initial responses

Table 1. P as a function of trials.

Trials	Mean of P	.05 Fiducial limits of mean
1–30	.593	.562–.624
31–60	.660	.625–.694
61–90	.672	.636–.708

may develop. For both of these reasons, reinforcement of corrections should not change P as much as reinforcement of initial responses. By the rules of the correction procedure, P of all reinforcements for M will follow initial responses; the corresponding proportion for L is $(1-P)$, since L is the initial response on $(1-P)$ of the trials. It is known (*2*) that after a few trials P becomes larger than .5, so that most of the M reinforcements follow initial responses, while most of the L reinforcements follow corrections.

For the above reasons, a new procedure, called the *nonreinforced trial procedure,* was devised. Only one response is allowed to occur on any one trial. Nonreinforced trials are interspersed with reinforced trials, so that π is controlled.

In the present experiment, 19 rats, deprived of food for approximately 22.5 hr, were subjected to one trial per day for 90 days in a slightly modified version of a T-maze described by Nunis (*4*). The nonreinforced trial procedure was used to reinforce a position preference with $\pi = .67$. Reinforcements were administered in blocks of three, consisting of two M reinforcements and one L reinforcement. At the beginning of a block, whichever response was made was reinforced. When all of the assigned reinforcements had been received for one of the two responses, that response was not reinforced until the assigned number of reinforcements had been obtained for the other response. It so happened that .608 of all trials were reinforced. (Reinforcement consisted of allowing the rat to enter a white goal box from a black alley to eat ground chow for 20 sec. When nonreinforcements were scheduled, a swinging door leading to the nonreinforced goal box was locked. Five seconds after the rat interrupted a photobeam 1.5 in. in front of this door, it was removed from the apparatus.)

Table 1 shows the mean of P during each 30-trial portion of the experiment and .05 fiducial limits for this mean (t-test). Toward the end of the experiment, P is approximately equal to π. Estes (*2*) has shown that asymptotic equality of P and π is implied by certain assumptions about the effect of reinforcement on response probability provided that all trials are reinforced. If our result is to confirm Estes' views, it must be shown that the nonreinforced trials do not affect P. Under the present use of the nonreinforced trial procedure, nonreinforcements of M can occur only during a block of reinforcements in which the two reinforcements of M have already occurred, but L has not yet occurred. The value of P on the trial following the second reinforcement of M during such a block is called *Prr*. If the trial following these two successive reinforcements of M also results in M, this last trial is not reinforced; the value of P after such a nonreinforcement is called *Prrn*. Similarly, the value of P following two such nonreinforcements is called *Prrnn*. These statistics were obtained for each rat and their means were: $Prr = .839$; $Prrn = .788$; and $Prrnn = .664$. This decrease in P as a function of nonreinforcement of M is significant at the .05 level (Kendall $W = .161$, 18 degrees of freedom), indicating that without special assumptions about the interaction of reinforcement and nonreinforcement, the present experiment, like previous probability learning experiments with animals, cannot decisively confirm or reject theories about the effect of reinforcement on response probability.

Important information about the role of the nonreinforcements in the nonreinforced trial procedure is supplied by two proportions, $F(M)$ and $F(L)$. $F(M)$ is the proportion of M occurrences which are reinforced:

$$F(M) = x_\pi / P$$

where x is the proportion of all trials (both M and L) which are reinforced (so that x_π is the proportion of all trials containing reinforced occurrences of M). Similarly, $F(L)$ is the proportion of L occurrences which are reinforced:

$$F(L) = x(1-\pi)/(1-P)$$

It may be verified that when $P < \pi$, $F(M) > F(L)$; that when $P = \pi$, $F(M) = F(L)$; and that when $P > \pi$, $F(M) < F(L)$. I have not been able to relate these mathematically derived facts to the above-mentioned theories (*1, 2*) in any rigorous way. However, if it is assumed that when $F(M) > F(L)$, P increases, and that when $F(M) < F(L)$, P decreases, these facts imply that once P reaches π, it will oscillate around π. (The possibility that P will remain constant at π is ignored because it was empirically shown that $Prr > \pi$.) Presumably, then, the mean of P will approximate π (*5*).

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Hemorrhage in a Coccinellid Beetle and Its Repellent Effect on Ants

Abstract. Special refinements of the bleeding mechanism of the Mexican bean beetle, *Epilachna varivestis* Mulsant, are described, and the defensive effectiveness of the mechanism against ants is demonstrated. Ants may have provided a major selective force in the evolution of the mechanism.

A variety of insects and other terrestrial arthropods have the peculiar habit of discharging small droplets of blood from one or more points on their body surface when they are handled or otherwise molested. This autohemorrhage or "reflex bleeding" as it is often called, is generally acknowledged to be a mechanism of defense against predators. The purpose of this report (*1*) is to describe some hitherto unnoticed adaptive features of this mechanism as it occurs in adults and larvae of the Mexican bean beetle, *Epilachna varivestis* Mulsant.

In adult *Epilachna*, as in apparently all coccinellid beetles that show reflex bleeding, the release of blood is exclusively from the tibio-femoral joints of the legs (*2*). In order to facilitate observation of the bleeding response, individual beetles were affixed to rods [by a technique used previously with other insects and described elsewhere (*3*)], and were subjected to localized traumatic stimuli, applied either by pinching individual appendages with forceps, or by touching different body