

Fig. 3. Fluorescence spectra of 48-hour culture of *Rhodospirillum rubrum*, at room temperature (solid line) and liquid nitrogen temperature (broken line). Excitation, 365 mμ.

increase in aggregate concentration due to reversible association, or competing deactivation processes may be prevented. Another possibility is that low temperature may enhance energy-transfer processes into the aggregate from other pigments, by virtue of changes in relative molecular configuration, an increase in lifetime of the monomer excited state, or the shift to longer wavelength of the monomer luminescence. However, the most likely explanation seems to us to be a decrease in competing deactivation processes as the temperature is lowered.

In contrast to the situation in green plants, the various bacteria examined here show only one band in the low-temperature fluorescence spectrum, within the range of our fluorometer. This band is shifted about 20 mμ to longer wavelength, from the position of the room-temperature emission. The spectral sensitivity of the fluorometer was such that fluorescence bands as far out as 1020 mμ would have been detected, if their intensity were of the same order of magnitude as the single band observed at 910 mμ. We conclude, therefore, that only one type of fluorescent chlorophyll is present in the bacteria studied. This result is in agreement with the conclusions of Dyu-sens, based on room temperature fluorescence data (11). Whether the single emitting state in bacteria corresponds to monomeric or aggregate bacteriochlorophyll is still uncertain. Experiments in vitro and lifetime measurements are in progress to establish this.

The differences noted here between the behavior of green plant and bacterial chlorophyll systems are especially intriguing, in view of the differences in the photosynthetic processes found in these two types of organism. Thus, one may suspect that the participation of two distinct fluorescent levels is

needed for evolution of molecular oxygen, whereas the availability of only one such state can carry photosynthesis only to the level of an oxidizing agent intermediate between water and oxygen.

We draw attention (Figs. 1 and 2) to the fluorescence band near 515 mμ, which we believe is due to carotenoid emission. Excitation and emission spectra of *Euglena* in this region are similar to those of EPA solutions of antheroxanthin (12).

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Use of a Computer to Evaluate Alternative Insecticidal Programs

Abstract. A simple numerical routine which mimics the effects of density-dependent factors and weather has been used to simulate insect pest population trends without control, and with a variety of insecticidal procedures. Results of the analysis suggest that apparent benefits from spraying are illusory, since spraying elicits homeostatic response by the pest population.

In the last few years there has been great interest in novel techniques for insect pest control: species-specific sex attractants to lure adult male insects to a demise which precludes copulation (1), and manipulation of genetic composition of natural populations by releasing radiation-sterilized (2) or foreign (3) males. Two reasons for this concern with novel control methods are the failure of insecticides and the spectacular success of the program to control screwworms by mass release

of radiation-sterilized males (2). In view of the variety of insect pest control procedures available and the amount of money at stake in crops and forests, it would seem worthwhile to attempt construction of a mathematical theory of pest control. Such a theory should be designed to indicate the best type of control, or combination of types of control, for any situation, how to time control, and how intensive control should be for optimum effects.

There are a few well-documented cases showing what happens to a pest that has in fact been effectively controlled: it becomes extinct (2) or it persists at a very low level (4). With these cases as the standard of ideal control, it is clear that insecticides do not constitute ideal control. Failure of insecticide control has been attributed generally to selection of resistant strains (5), physiological stimulation by sublethal doses (6), and elimination of entomophagous species (7). However, perhaps there is a more basic explanation than any of these: use of insecticides reduces intraspecific competition pressure to such an extent that increased fecundity, fertility, and survival rates compensate, or more than compensate, for the drop in population (8).

As a first step in devising a mathematical model to compare the long-term effects of various control procedures, I have developed a simple numerical routine with which a computer can simulate pest population trends. N_i represents the number (per unit area) of adults in the i th generation; $N_{i+1/2}$ represents the number of larvae present at the end of the third instar (that is, after half the larval growing stage has been completed); D_i represents the average number of third-instar larvae surviving per adult under average weather conditions; D_i^1 represents the average number of adults surviving per third-instar larva under average weather conditions; S represents the proportion of third-instar larvae surviving spray treatment; and W_i is a factor by which the D_i and D_i^1 values must be multiplied to express the effect of weather in the i th generation. Then

$$N_{i+1/2} = N_i D_i W_i \quad (1)$$

$$N_{i+1} = N_{i+1/2} D_i^1 W_i S \quad (2)$$

D_i is computed from the empirical formula

$$D_i = \frac{C_i}{N_i} \left[1.0 - \exp \left\{ -N_i \times \frac{(C_1 + \exp [C_2 - C_3 N_i])}{C_4 + \exp (C_5 - C_6 N)} \right\} \right] \quad (3)$$

where the C 's are constants. Arguments in support of this general form for D_i are given elsewhere (8). D_i^1 has the same form, but a different set of

parameters, and N_i is replaced by $N_i + \frac{1}{2}S$. Values for the parameters in D_i and D_i^1 were chosen so as to obtain biologically realistic curves. Empirical studies show that, in plots of D_i^1 against $N_i + \frac{1}{2}D_i^1$, falls off above a maximum because of intraspecific competition and below the maximum because predators remove an increasing proportion of the pests as numbers of the pest decrease to very low levels (9).

The computer simulates population behavior for 100 generations, each i th generation taking the W_i from a table of 100 W values which has been constructed from Canadian weather records and survival versus temperature data in the literature. For each set of 14 parameters in D_i and D_i^1 , several series of data are obtained. In one series, data are obtained for 100 generations in which the population is unsprayed (that is, $S = 1.00$). In other series $S = S_i$ whenever $N_i + \frac{1}{2} \geq B$, some predetermined value at which the insect density has risen to a "pest" level. Data from 100 generations are obtained for each of the various combinations of S_i and B values.

The general conclusion from these simulation studies is that insecticides do not, according to this model, depress pest population level as much as one would expect, and for some combinations of parameters, sprayed populations exceed unsprayed controls, due to the homeostatic response of the population. For example, in one run for which $S_i = .01$ and $B = 2048$, the average N_i value over the "century" was 57 percent of the control N_i average, and N_i for the sprayed population exceeded N_i in the control population in 34 out of 100 generations. In no case examined did 99 percent spray kill result in a population equilibrium level as low as 1.0 percent of the control equilibrium level. The lowest population level produced by any spray program was 37 percent of the corresponding control population equilibrium level. It should be noted that the capacity for population recovery after spraying as assumed in this model is less than that in nature because the effect of selection for spray resistance is not included in the model.

In view of the importance of the indications from this primitive model, it would seem worthwhile to collect field data in order to check these findings (10).

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Diffusion-Precipitin Index to Antibody Avidity

Abstract. Antigen, diffusing into converging edges of an antibody depot, inhibits the precipitation unless the reaction is completed before an inhibiting antigen excess is established. Since the rate of the increase of antigen concentration depends on the size of the angle, relative avidities of similar preparations of antibody may be estimated from the angle just producing observable inhibition.

An analogy is sometimes drawn (1) between the densitometric distribution of precipitate along the axis of an Oudin tube and the precipitin curve of the system producing a zone of precipitate in that tube. The gradient of antigen concentrations ranging from a maximum in the fluid layer to a minimum at the momentary site of the plane, in advance of the zone, where antigen encounters free antibody, may be compared to the stepwise increments of antigen in successive tubes in a typical quantitative precipitin study.

The experiment shown in Fig. 1 demonstrates that the analogy is not a perfect one. Two small vessels, the shape of which has been found to be irrelevant to this experiment, were filled with agar containing 0.01 percent hen ovalbumin (H). Approximately equal areas were cut from the center of each vessel, and the agar removed was replaced with agar containing rabbit anti-hen ovalbumin precipitin sufficiently diluted to allow the zone which formed to migrate toward the center of the plate.

In the upper triangular vessel, antigen diffusing into the circular antibody depot advanced as an expanding gradient evenly distributed about the periphery. The deposited precipitate, which was not readily redissolved in antigen excess with this particular serum, was observed as a correspondingly uniform, broadening zone.

In the lower vessel, the same concentrations of antigen must have passed through each point in the region marked by precipitate as have passed through corresponding points in the circular antibody depot. Yet, the precipitate was not evenly distributed

when the antibody depot had the triangular shape. The difference between these two arrangements lies in the rate at which the antigen concentration increased at certain points within the antibody depot, rather than in the actual antigen-antibody ratios established.

With fluid precipitin tests, when one uses a stepwise increment of antigen concentrations from tube to tube, no such time factor is involved. Inhibition of precipitation in antigen excess is due to failure to produce a precipitate, rather than to the erosion of precipitate previously formed as in the trailing edge of a zone in the Oudin tube. While the densitometric patterns produced by the Sami (2) scanning device resemble precipitin curves and have similar significance, they result from the solubility of precipitate in excess antigen, rather than from the prevention of precipitate deposition. Only in instances where precipitate formation was perfectly reversible would the suggested analogy be entirely valid.

In the lower plate in Fig. 1, antigen arriving in certain areas simultaneously from two interfaces has led to the establishment of inhibiting concentrations. The precipitate distribution in the upper plate shows that precipitate, once formed with this particular precipitin, is not readily redissolved by these same concentrations. The wedge-shaped areas in which precipitate is less dense or absent must therefore result from the effect of antigen arriving in the interval between the initial antigen-antibody combination and the

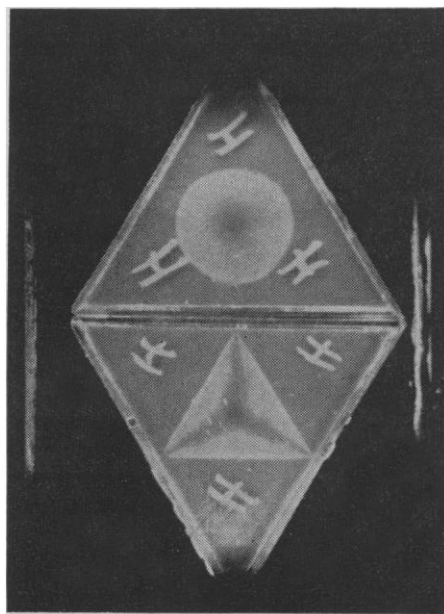


Fig 1. Agar plates containing hen ovalbumin diffusing from regions marked H into approximately equal volumes of rabbit anti-hen ovalbumin precipitin in agar in centrally located depots.