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 $S = S_1$ whenever $N_i + \frac{1}{2} \geqq B$, 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_1 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 $S_1 = .01$ 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

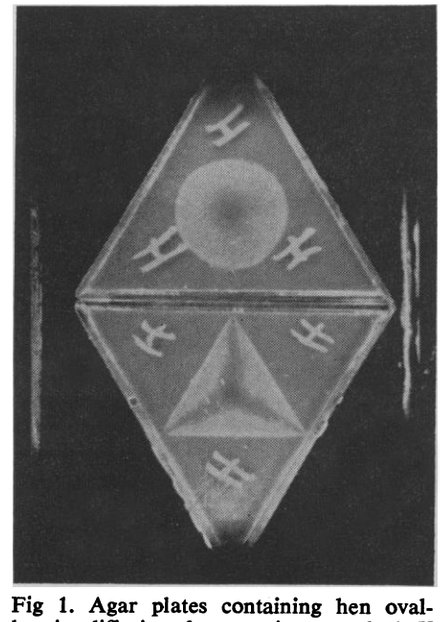

Fig 1. Agar plates containing hen ovalbumin diffusing from regions marked *H* into approximately equal volumes of rabbit anti-hen ovalbumin precipitin in agar in centrally located depots.

completion of the second stage of the precipitin reaction.

The following device may therefore be used as a means of carrying out rapid, rough estimates of the avidities of antisera which are identical to each other in specificity. A small plate of convenient shape may be filled with agar containing a standard antigen preparation. A polygonal depot may be cut or cast in the center of this agar, and refilled with agar containing the serum which is to be examined. We have found it convenient to form the antigen agar about a plastic die of the shape shown in Fig. 2, having the following angles. At the bottom of the heart-shaped figure, the angle is 90°. In ascending order along one side, the angles are 170°, 140°, 130°, and 100°. In the same order along the other side, the angles are 160°, 150°, 120°, and 110°. When the antigen-agar has hardened, this template is carefully withdrawn and the depression is filled with the serum agar. Tests should be run to determine a suitable dilution of serum, which permits the formation of an easily visible zone migrating rapidly into the antibody depot.

No precipitin system examined thus far has failed to show a wedge of clearing within the 90° angle; none has shown such a wedge with the 170° angle adjacent to it. Seemingly identical sera, in other respects, do, however, differ with respect to the angle in which clearing can just be seen, within this range.

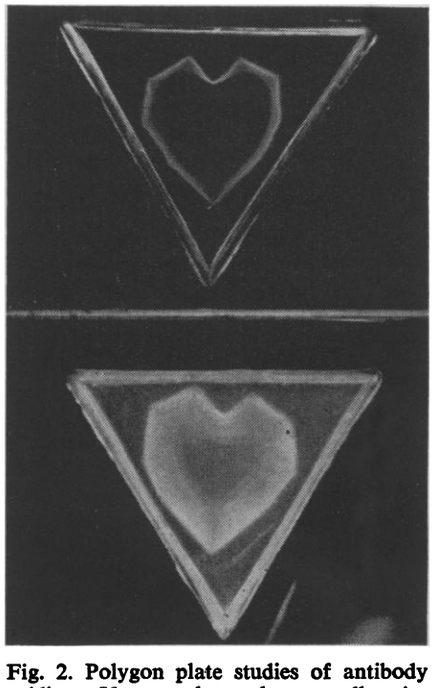

Fig. 2. Polygon plate studies of antibody avidity. Upper plate, hen ovalbumin, rabbit anti-hen ovalbumin system, after 18 hours of diffusion. Maximum angle in which inhibition could be observed, 130°. Lower plate, complex precipitating system illustrating unsuitability of method for comparison of heterospecific precipitins.

In the plate at the top in Fig. 2, it is demonstrated that wedges of inhibition can be observed after diffusion of 18 hours or less in favorable instances. The prolonged diffusion, giving rise to zones as wide as those shown in Fig. 1, is never necessary.

The lower plate in Fig. 2 has been included to emphasize the fallacy of attempting to make comparisons from one precipitin system to another by this method. A complex precipitating system (*3*) composed of 10 parts of hen ovalbumin to 1 of duck, diffusing into rabbit anti-hen-ovalbumin serum, was used to produce two zones.

The angle of inhibition of the homologous system, producing the zone closer to the peripheral antigen source, was found to be 140°. The wedge of inhibition could only be seen within the 90° angle in the case of the heterologous, cross-reacting system producing the inner band of precipitate. It would not be justifiable to conclude that the cross-reacting portion of the precipitin entered into a firmer union with the heterologous antigen than did the total precipitin with the antigen responsible for its production. Homologous, as well as heterologous, antigen is present in the area occupied by the proximal zone, but the leading edge of this zone marks the greatest excursion of the homologous antigen into the agar at the time of observation. The forces tending to reverse the two reactions differ, and no comparison is possible.

The method thus describes avidity of the antibody only in a relative sense. In a broader sense, avidity is the result of forces exerted between antigen and antibody and is not a characteristic of the antibody alone. The angle of inhibition will not indicate the absolute avidity and cannot be used, for example, to compare the avidity of an albumin-anti-albumin system with that of a globulin-anti-globulin system.

On the other hand, the method offers a simple, rapid means for establishing the relative avidity of each of a series of precipitin preparations specific for a given standard antigen preparation examined under uniform conditions (*4*).

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Variability in Male Stature as Function of Adolescent Maturation Rate

Abstract. Boys who mature very early and, to a lesser degree, those who mature later than average show less variation in stature than boys who are somewhat early in adolescent development. These variability differences are paralleled in the heights of the mothers and fathers except in the case of boys who mature very early; there is far less variability in height among these boys than among their parents.

Variability in height among boys increases gradually from birth to the onset of adolescence, shows a relatively abrupt increase during the adolescent growth spurt, and returns almost to preadolescent levels at maturity (*1*). The peaking during adolescence is clearly attributable to wide variation in the ages at which boys enter this growth phase and to the rates at which they proceed toward physical maturity during this period. At physical maturity, however, residual differences in stature are presumably of genetic origin (disregarding the minimal contribution of nutritional differences), and therefore no systematic relationship between dispersion in mature height and maturation rate is to be expected.

A sample of 78 boys, born in 1928–29, was drawn from the University of California Guidance Study ($N = 64$) and from the Berkeley Growth Study ($N = 14$), the sole criterion being availability of complete records of physical development from birth to age 18 years (*2*). Age at reaching 90 percent of mature height ($M = 13.6$ years, $\sigma = 0.96$) was taken as the measure of maturation rate during adolescence (*3*), mature height itself ($M = 180.1$ cm, $\sigma = 6.6$) being defined as height at approximate skeletal maturity (*4*).

Figure 1 presents the relationship of variability in mature height to maturation rate. The probability that this degree of variation in σ among maturational groups is due to sampling fluctuations from a common population is .04 (by the Bartlett test for homogeneity of variance), and if we consider that this test does not take into account the rather regular trend of the data, even greater confidence may be placed in the reliability of this relationship. This result is not a product of development *during* adolescence, since a replication of this analysis with height data for age 9 (well before there is any evidence of pubescence in even the earliest-maturing group) provides a closely similar result. Here the relationship is even more clearly a non-chance outcome ($P < .001$). The two results are, of course, not independent (about 75 percent of the total-group variance in height at maturity can be predicted from the age 9