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 When the antigen-agar has hardened, this template is carefully withdrawn and the depression is filled with the serum agar.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.

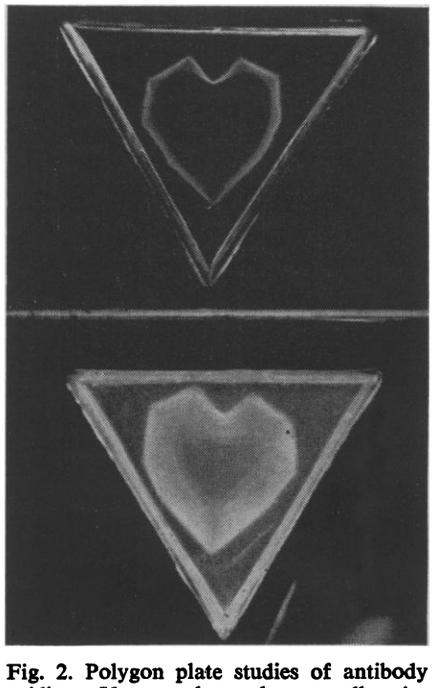

Fig. 2. Polygon plate studies of antibody avidity. Upper plate, hen ovalbumin, rabbit anti-hen ovalbumin system, after 18 hours of diffusion. Maximum angle in which inhibition could be observed, 130°. Lower plate, complex precipitating system illustrating unsuitability of method for comparison of heterospecific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

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4. This report was supported by the Asthmatic Children's Aid Society.

14 November 1960

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

data), but it is apparent that variability differences among maturational groups predate adolescence.

One hypothesis to account for these results is that they stem from corresponding variability differences in the heights of one or both parents. This expectation is largely supported. Working with the heights *reported* by 63 sets of parents from the Guidance Study subgroup (for fathers, $M = 176.3$ cm, $\sigma = 8.3$; for mothers, $M = 163.1$ cm, $\sigma = 7.4$), we find that, for both parents, the plots of the σ-values against the sons' maturational rates closely resemble the boys' curves except for boys in the earliest-maturing group; the striking homogeneity for this group has no counterpart in the corresponding parental heights (Fig. 1). This finding, of course, does nothing to clarify the mechanism responsible for the relationship between maturation rate and variability, but the focus of speculation is shifted.

A possible, though unlikely, explanation of the low variability in stature of boys who mature early would be that, in this group alone, parental heights are *negatively* correlated, the usual homogamy with respect to stature being reversed. If this were so, and since predicted heights of offspring are essentially an average of the heights of the two parents (relative to their sex) (*5*), there would be less variability in height among the sons of these parents than among the parents. This is not the case: the between-parents height correlation is positive for this as for the other maturational groups. With this possibility ruled out, we infer that there is some attenuation, for the boys who mature earliest, of the hereditary factor. Thus, multiple-regression predictions, from parental stature, of mature heights of sons would be more in error for boys who mature early than for the remainder of the sample (*6*). Groups 1 and 2 in Fig. 1 represent boys who matured early. Data for these groups ($N = 20$) were combined, and predicted mature heights and squared deviations of the predicted heights from the true heights were computed. Similar calculations were made for a combined average-late maturational group ($N = 43$). The standard error of estimate for the group that matured early is 6.1 cm (yielding an estimated R of .53); for the others, the corresponding values are 4.8 cm and .75, respectively, indicating considerably greater predictive accuracy for the latter group. This result leads us to speculate that a common mechanism may underlie both maturational acceleration and this partial breakdown in the usual pattern of inheritance of stature.

Many more observations are of course necessary before it can be concluded that any of the several results point reliably to a population difference. Since even the relationships of maturation rate with boys' variability in stature, while most firmly based, require replication, we hope that evaluation of these preliminary findings will be undertaken in other growth studies.

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Group	1	2	3	4	5	6	7
Maturity	2.7	7.7	7.0	6.6	6.5	3.5	4.5
Age 9	1.4	5.9	5.7	3.8	4.5	2 3	5.4
Father	10.7	9.6	7.6	9.6	7.4	5.2	6.5
Mother	9.4	7.9	4.6	9.4	7.2	3.7	4.3

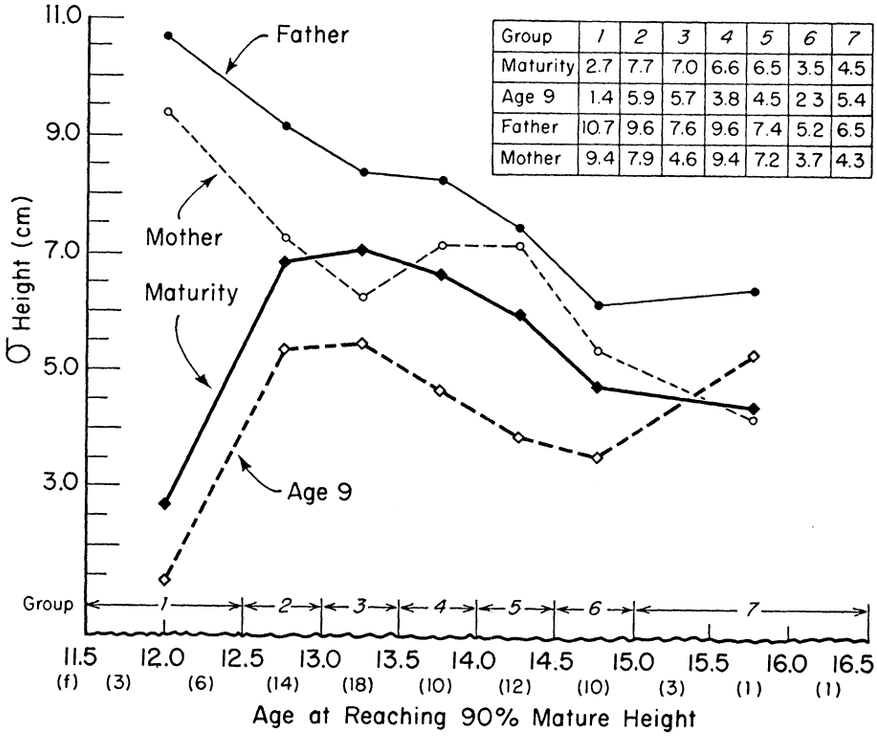

Fig. 1. Variability in height as a function of adolescent maturation rate. Extreme class intervals have been combined, as indicated. The plotted points have been derived from an algebraic (moving-mean) smoothing of the tabulated original values.

References and Notes

1. This is a well-established generalization, most recently supported → N. Bayley and S. Pinneau [*J. Pediat.* **40**, 423 (1952)] and by R. D. Tuddenham and M. M. Snyder [*Physical Growth of California Boys and Girls from Birth to Eighteen Years* (Univ. of California Press, Berkeley, 1954), vol. 1, No. 2]; these are reports, respectively, from the Berkeley Growth Study and the Guidance Study—longitudinal investigations from which the data reported here, are drawn. We are indebted to the staffs of these studies whose assistance made this report possible.
2. For the Guidance Study subgroup, raw data and descriptions of the measurement schedule and procedures are presented in Tuddenham and Snyder [*Physical Growth of California Boys and Girls from Birth to Eighteen Years* (Univ. of California Press, Berkeley, 1954), vol. 1, No. 2]; for details on the other subgroup → H. E. Jones and N. Bayley, *Child Develop.* **12**, 167 (1941).
3. The choice of this measure was recommended by the findings → A. B. Nicolson and C. Hanley [*Child Develop.* **24**, 3, (1953)], whose factor analysis of 11 indicators of adolescent maturation rate in these males demonstrates that a single general factor can account for all reliable covariation and is best estimated ($r = .99$) by age at 90 percent of mature height.
4. Skeletal maturity was taken to be age 17.25 (Todd standards), as stringent a criterion of epiphyseal closure as considerations of adequate sample size would allow. If any growth in stature occurred beyond this point, the mean of fluctuating annual heights or the maximum of regularly increasing heights was taken as mature height. For 11 cases where measurement at maturity was missing but where height had been measured through late adolescence, mature height was predicted from the skeletal age tables → N. Bayley [*J. Pediat.* **28**, 49 (1946)]. The standard error of estimate of predictions of mature height made from this age range is less than 1 cm.
5. K. Pearson and A. Lee, *Biometrika* **2**, 357 (190→ N. Bayley, *J. Educ. Psychol.* **45**, 1 (1954).
6. The relevant height correlations for this prediction are as follows: father × mother, .30; father × son, .47; mother × son, .62; the multiple correlation (R) is .69, and the resultant standard error of estimate for the total group is 5.2 cm.

18 July 1960