the smallest deviations from the predicted values. These deviations from the straight line represent response variability plus the discrepancy from additivity.

Table 1 gives a condensed statistical analysis. The correlations in column 2 are between observed response and response predicted on the basis of the additive hypothesis. The mean correlation is .967, so it appears that the additive model accounts for the behavior quite well. These correlations are descriptive indices whose significance is best tested by the F ratios in column 3, all of which are highly significant.

That the model has high predictive power indicates that it will be useful in several ways. However, predictive power is only one criterion of the goodness of a model, and it is to be expected that any reasonable model will have rather high predictive power. In assessing the model, therefore, it is vital to test the deviations from prediction.

The discrepancies between predicted and observed values were tested by the F ratios for nonadditivity. The results are given in column 4 of Table 1. These are significant only for the last three subjects, and even for them the discrepancies are relatively small.

The square root of one-third the error mean square given in column 5 of Table 1 is the standard deviation of a single data point for the corresponding subject of Fig. 1.

Breakdown of the nonadditive component into the several interactions of the 3^3 design gave no new information. In general, however, study of the separate interactions will be useful in searching for regularity in whatever nonadditivity may obtain.

The statistical analysis has shown that the greater part of the subject's response behavior can be accounted for by the simple additive model. In other words, it was as though the subject assigned a value to each single adjective and, when presented with a set of adjectives, gave the mean of the corresponding values as his response.

This may seem more reasonable if the psychological values of the adjectives are considered to be points of equal weight on a line. The mean is then that point at which the algebraic sum of the distances from the mean to the several points representing the adjectives is zero.

Whether a similar degree of additivity will obtain with other stimuli and other judgment tasks remains to be seen. No doubt additivity cannot be generally expected, and even if the nonadditivities are small, they may still be of major interest in many situations. In any event, these techniques should be a useful tool in helping to bring impression-formation processes within the domain of experimental analysis (*4*).

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3. See, for example, G. W. Snedecor, *Statistical Methods* (Iowa State College Press, Ames, ed. 5, 1956); B. J. Winer, *Statistical Principles in Experimental Design* (McGraw-Hill, New York, 1962).
4. I am greatly indebted to Ann Norman for her assistance in this experiment. The work was supported by grant M-6087 from the U.S. Public Health Service.

22 June 1962

Pooled Estimates of Parent-Child Correlations in Stature from Birth to Maturity

Abstract. Correlations between the heights of children throughout their development and their parents' mature heights differ widely in different growth studies. The range of values, however, is shown to be within the limits of sampling error, making it possible to estimate correlations for each age from birth to maturity. Mother-child correlations are generally higher than father-child correlations, and for both comparisons the correlation increases when the child reaches early adolescence. There is no relation between the heights of the parents and the timing of the child's growth spurt, but there probably is a relation between the heights of the parents and the amount the child grows during early adolescence.

Stature is among the more heritable of human morphological characteristics. However, quantitative estimates, from different investigations, of the degree of correlation between the heights of the parents and the height of the child throughout the period of the child's growth show little agreement (*1*). The individual entries in Fig. 1 summarize the results we have been able to gather on this problem. The data are drawn from studies in which the children's heights were determined through standard anthropometric procedures and estimates of the heights of both parents were available (*2*). These correlations illustrate the wide discrepancies in the results obtained in different samples. Thus, in one investigation (*3*) there was higher correlation for parent and child of the same sex than for parent and child of opposite sexes, while in another (*4*), a higher correlation for father and child of either sex than for mother and child is suggested. Our own data from the Guidance Study of the University of California Institute of Human Development yield yet another pattern: the correlation for mother and child is generally the higher, regardless of the sex of the offspring (*5*).

The purpose of this study was to determine whether these disagreements are attributable to sampling error and, if they are, to combine the separate sample values for each of the four parent-child combinations into pooled best estimates of parent-child correlations for stature at each age level. Resolution of the disparity would seem to be a sufficient good in itself, but there were other reasons for making the study. The availability of more reliable parent-child correlations could increase the accuracy of multiple-regression predictions of children's mature height, whether the prediction was made from the parents' heights alone or from the parents' heights and the child's current height. Also, the matter of the inheritance of stature seems an open question, and such data may serve as reliable landmarks for theoretical speculation concerning the mechanisms involved.

In order to establish the legitimacy of pooling the correlation coefficients for each parent-child combination, the variation among the values available from different studies for a given parent-child combination at each age of the child was evaluated by the Snedecor chi-square test (*6*). In only five of the 84 [21 (ages) $\times$ 2 (parents) $\times$ 2 (sexes of child)] comparisons made was this variation statistically significant (*7*). Since these instances were so few, since they were scattered among the comparisons, and since the pooled values in these instances were in each case well in line with the adjacent pooled values, the values were permitted to contribute to the smoothed curves.

The pooled values of Fig. 1 were derived through a modification of the method of "moving averages," in which the correlation at any particular age is the weighted mean of coefficients

from that age and the two ages immediately adjacent to it. (Values from the central age are given double weighting.) All the coefficients were transformed to a normalizing Fisher's z-metric before they were averaged. The method has been elaborated to reflect differences in numbers of observations by weighting each original coefficient also by its degrees of freedom ($N=3$). Some slight shifts in inflection points, resulting in a small underestimation of the rates of change, are inevitable with this method.

The resultant family of curves show a generally upward drift in the correlation with age when they are considered over the entire growth span. This is most marked for correlation between mother and child. An increasingly high correlation is compatible with the view that genetic influences are gradually expressed throughout the growth period. The contributions of the mother and of the father to a son's height never differ significantly, but for a daughter the correlation with the father's height is significantly lower than that with the mother's from age 8 onward (t-test). This outcome, if true (*8*), is puzzling. So far as we know there is no mediating mechanism for such a between-parent differential; moreover, there are no significant differences in correlation when the child is mature (*9*).

There is an upswing in correlation between the height of mother or father and the height of a child of either sex at about the onset of the child's adolescent growth spurt. If we take ages 10 and 12 as the average age of onset of the growth spurt in girls and boys, respectively, and, by partial correlation, hold the height already attained at this age constant, we find a significant relation between the mother's height and the child's height 4 years later when the spurt is largely spent. The relation to the father's height after partial correlation is also positive, but not significant. From this we may infer that children of tall parents either mature earlier or grow more during their growth spurt, or both. For children investigated in the Guidance Study (the correlations for the period of adolescent growth are based almost wholly on data from that study), we find no correlation between the height of either parent and the rate of maturation of the child, whether *rate* is defined by any of a number of specific indices (age at attaining various percentages of mature height, skeletal age, age at

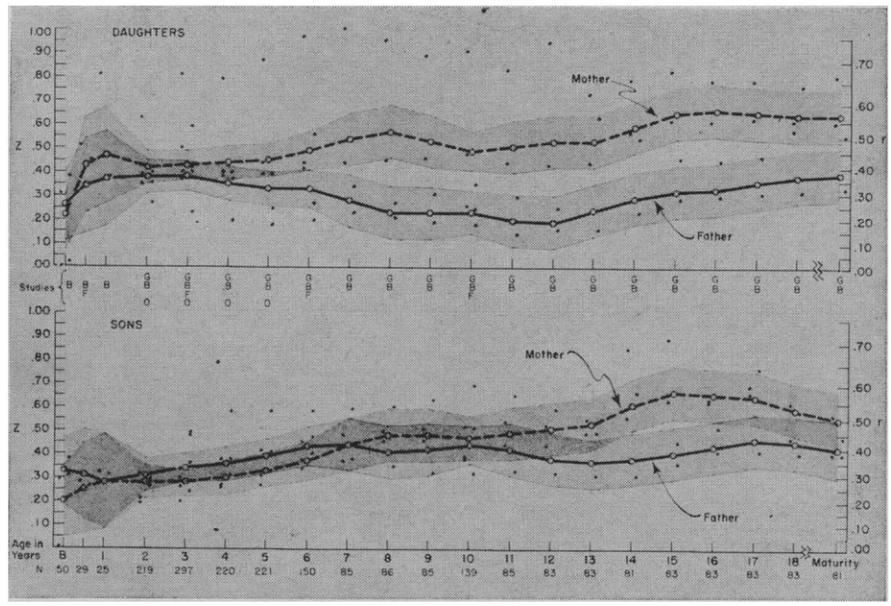

Fig. 1. Parent-child height correlations as a function of chronological age. The shaded regions represent a standard error of 1.0 about the means of the pooled z-coefficients. Very roughly, nonoverlap of the shaded regions indicates a difference in parent-child correlations significant at the 10-percent level. The studies from which data were taken for each age are noted between the two graphs, as follows: *G*, Guidance Study; *B*, Berkeley Growth Study; *F*, Fels Institute; *O*, Oxford Child Health Survey. The original correlations from these studies are plotted. For each age, the dots to the left of the ordinate are mother-child correlations; those to the right, father-child correlations. Since the numbers of observations underlying the four pooled parent-child correlations vary somewhat at each age of the child, the reported number N is the average for four subgroups. However, the range of sample sizes is never more than 10 percent of the average value.

emergence of various secondary sexual characteristics) or by a general factor score derived from these indices (*10*). It is probable, therefore, that the amount of growth during the adolescent growth spurt, quite apart from its timing, is to some degree genetically determined. Here again, no mechanism for the effect is evident.

Correlations of the heights of parents with annual increments in the height of the child would provide precise determinations of the relationships of which the pooled values of our study give only a rough estimate. The separate genetic contribution of either parent to the yearly increment of growth of the child would emerge from a partialing out of the other parent's height from these correlations. Hewitt's demonstration (*11*) of the attenuation of parent-child height correlations attributable to the variability in maturity level among children of the same chronological age suggests a further refinement. Height increments should be computed over intervals defined by skeletal ages or by percentiles of mature height. This procedure would not only remove extraneous variance but would also permit merging of results across samples differing in distributions of maturation rates. If the pure longitudinal series from birth to maturity which this ideal design requires were to include direct measurements of the heights of both parents, the precision of such data would make possible valid comparisons of parent-child height correlations between populations which differ in genetic composition or degree of homogeneity.

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5. The new correlations reported here are based on a core sample of 131 individuals (63 boys, 68 girls) from whom complete longtitudinal growth data were available in the Guidance Study of the University of California Institute of Human Development. The anthropometric procedures and raw data are presented 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]. The Berkeley Growth Study sample was chosen from the same community, and at the same time (1928–29).

6. Initially, values were smoothed within the study if sample sizes at adjacent ages differed. For "mature height" values, Bayley's age-21 results are combined with values based on our own criterion of near-completion of skeletal growth (skeletal age 17.25 for boys, 16.25 for girls, as determined from application of Todd's standards to x-rays of the hand). The "birth" points include Bayley's 1-month data.
7. Any assertions of statistical significance in the text are based on designated tests and denote $P < .05$.
8. Since the father-child correlations are on the whole somewhat lower than the mother-child correlations, it should be noted that the reported heights of fathers may be somewhat more suspect than those of mothers. In addition to the possibility that paternity may have extended beyond the immediate family, the fact that, for a good portion of these data, the mothers reported both their own and their husband's heights is a source of possible inaccuracy. This is particularly true for the data beyond about age 6, and it is in these years, especially when the child reaches early adolescence, that between-parent differences in parent-child correlation are most marked. If mothers report their own heights more accurately than they report their husbands' heights, it may be that the father-child correlations are lower because of greater error in reported measurements. We have one datum which is somewhat reassuring. For 21 cases where fathers' heights were reported independently by both man and wife, the correlation between the two sets is .92, the distributions do not differ, and the correlations with children's heights are the same. Of course, fathers' heights may be less accurately reported by both parents; the likelihood that height is a more salient characteristic for males in our culture suggests such a possibility. However, reporting errors can hardly account for the sex difference in between-parent differential.
9. K. Pearson and A. Lee [*Biometrika* **2**, 237 (1903)] find correlations ranging from .49 to .51 for the four parent-child comparisons at approximate maturity. Their data are based on measurements for the oldest children over 18 years of age living at home in over 1000 English families. These results, which show higher correlations between father and child than our pooled data show, were obtained by correspondence and are based on measurements made by family members themselves in accordance with detailed written instructions. However, these correlations may somewhat underestimate the true values for that population. The Pearson and Lee sample, having reached adolescence at about the turn of the century, would, in many cases, not have attained their mature stature at the time of measurement. The authors themselves state that full height in this sample was not achieved until age 28 for men, age 25 for women. The parent-son correlations would therefore be more attenuated by this factor than the parent-daughter correlations; thus the equal correlations for the two sexes which Pearson and Lee report may be taken to suggest slightly higher correlations in height between sons and their mothers and fathers than between daughters and their parents.
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2 September 1962

X-Ray-Induced Chromosome Aberrations in the Corneal Epithelium of the Chinese Hamster

Abstract. When the corneal epithelium of the Chinese hamster is irradiated with ionizing radiation in vivo, a rectilinear relationship is observed between the yield of chromosome aberrations and the dose of x-rays within a dose range of 10 to 150 r. There is no threshold effect for chromosome breakage down to the 10-r level. The mean breakage rate of 0.00352 breaks per cell per roentgen is in very close agreement with the data obtained on other mammalian tissues, both in vitro and in vivo.

The study of chromosome aberrations is, at present, one of the most reliable methods of assaying radiation-induced cellular damage. The number of chromosome breaks constitutes a reasonably accurate biological dosimeter (*1*) and also serves as a fairly reliable indication of radiation-induced genetic damage in cases where other genetic assay systems are either impracticable or too cumbersome.

Since the initial findings of Muller (*2*) on the mutagenic effects of x-rays in *Drosophila* and parallel findings in plants by Stadler (*3*) and by Goodspeed and Olsen (*4*), a vast literature has been compiled concerning the production of radiation-induced chromosome aberrations. Sax (*5, 6*) made the first critically quantitative evaluation of radiation-induced chromosome aberrations. He found that in x-irradiated *Tradescantia* microspores one-hit aberrations increased approximately linearly with increasing dose and two-hit aberrations increased as the square of the dose if the duration of exposure was held constant. Carlson (*7*), working with the grasshopper *Chortophaga viridifasciata* and scoring only acentric fragments at late anaphase and telophase, observed that the number of single breaks in neuroblasts was proportional to the dose within a range of 7.8 to 125 r. Several workers (*8*), using genetic techniques to determine breakage in *Drosophila*, have demonstrated that one-hit aberrations increased linearly with dose and that two-hit aberrations increased exponentially and as the square of the dose at high dosage levels.

Recently, attention has turned to the study of the production of aberrations in mammalian cells. Several authors (*1, 9–11*) have studied metaphase aberrations in tissue culture. The frequencies of radiation-induced chromosome aberrations in vivo have been observed in the human (*1, 10, 12*), the monkey (*10*), and the Chinese hamster (*10, 13*).

Bender and Wolff (*14*), for example, have shown that x-ray induced chromosome aberrations in mammalian cells follow the same kinetics as aberrations studied in plants, and they feel that there is no reason to believe that there is a threshold at which aberration production begins. The purpose of the study reported here was to determine whether the linear relationship between aberration yield and dose holds good at low doses of x-rays in cells irradiated *in situ*. The studies in the work cited either were based on cells growing outside the body or did not conclusively establish a direct linear proportionality between x-ray dosage and chromosome breakage at low doses in mammalian tissue.

The experiments reported here were performed on the Chinese hamster, *Cricetulus griseus* (*15*), a rodent especially suitable for the purpose because of its low chromosome number ($2n = 22$) and the ease with which individual chromosomes can be distinguished.

All the hamsters used in the experiments were adult males. They were lightly anesthetized with ether and immobilized by a clamp-like device which fits over the nose and has two small plugs that are inserted into the ear sockets. All of the animals were given whole-body irradiation with x-rays at a dose rate of 10 r/sec, measured by a Victoreen dosimeter. The x-rays were generated by a General Electric x-ray unit (Maxitron 250) operated at 250 kv (peak) and 30 ma with a 1-mm aluminum filter.

The cytological method used in making the corneal epithelium preparations was a modification of that of Fechheimer (*16*). One hour and 40 minutes before the animals were sacrificed they received an intraperitoneal injection of 0.3 ml of a 1-percent colchicine solution. They were sacrificed, and the eyes were dissected out. The eyes were placed in a hypotonic saline solution (Hanks' basal saline solution without NaCl, 95 percent; Hanks' basal saline solution, 5 percent) and incubated at 37°C for 20 minutes. Cold, fresh alcohol–acetic acid (3:1) fixative was added, and the tissues were kept in the cold for an 18- to 24-hour