

Reports

Intellectual Potential and Heredity

Abstract. When infant developmental quotients are compared with children's intelligence quotients, it appears that most subnormality is manifested only at the later age. This phenomenon has been cited as evidence that environment chiefly determines intelligence, but the argument is circular. A helpful approach to the nature-nurture problem is afforded by the geneticists' concept of reaction norms or "reaction repertoires."

In the AAAS Symposium of 1956, Knobloch and Pasamanick (1) reported studies on the distribution of developmental quotients in Baltimore infants. They found only 1.8 percent with quotients below 85, while approximately 14 percent of older children are known to have intelligence quotients in this range. They cite this alleged increase in subnormality as evidence for the operation of psychosocial as opposed to genetic factors. Finally, on the basis of their study "as well as others," they conclude that, "So heavily do these psychosocial factors seem to outweigh any genetic behavior variation that it seems extremely difficult to find any evidence for the importance or even the very existence of the latter" (1, p. 263).

My intention is not to challenge the conclusion of Knobloch and Pasamanick, though I shall disagree with it, but to show that the findings they report provide no more support for their conclusion than for an opposite one. Their conclusion remains a hypothesis for which their study yields no new evidence.

The essential difficulty is one of circular reasoning. While the circle appears at several points in the article, it

is best illustrated in the following two quotations, preceding and following what they regard as the crucial synthesis of evidence (*italics mine*):

"Let us turn now to a comparison of the present findings with the distributions of intelligence quotients of older children and adolescents as reported in the literature. While the intelligence quotient is influenced by more environmental factors than affect the intellectual potential as diagnosed in infancy, the IQ is obviously related to neural integration. *A lowered score may be the result of many environmental factors, physical, psychological and social*, but a higher score can only be the result of learning. A comparison of these two estimates, therefore, may help elucidate the question of how large is the influence of environmental factors on intelligence test scores" (1, p. 259).

"The observations which we have presented in this report would appear to us to lend support to the hypothesis that *the measures of intelligence used in later life are greatly influenced by learning and affected by life experiences which tend to limit opportunities of acquiring the kinds of information that the tests seek to evaluate*" (1, p. 261).

Note that what appears in the first quotation as an assumption upon which the argument is built, reappears in the second as a conclusion. Clearly, if one considers development to be controlled only by environmental influences, any developmental differences among individuals must be attributed to the environment. One could as well start with an alternate assumption, that intellectual development, except for its content, depends upon learning rate, learning capacity, and maturation of thought and behavior patterns. Then the increase in subnormality at later ages would appear to support a hypothesis that measures of intelligence used in later life reflect relevant genetic potentials more adequately than does the infant developmental quotient.

I will not dispute the plausibility of the authors' initial assumptions or the validity of their data, although comparability of developmental quotient and intelligence quotient scales is open to question (2). The data by themselves illustrate individual differences and suggest that infant behavior does not ex-

press all the determining factors for final intellectual functions. The point is that the findings contain no internal evidence as to the nature of these determining factors. It can be argued similarly, I think, that most of the other evidence referred to by the authors is as ambiguous as their own. When analyzed on the basis of restrictive assumptions, such data will yield restricted conclusions.

Any extreme view in the nature-nurture controversy appears to be unjustified at the present time. The most useful hypothesis is neither of those hypotheses of a generation ago that considered one or the other influence alone, but a hypothesis that assumes undetermined contributions from both.

The well-established genetic concept of the reaction norm (3), better described by the term "reaction repertoire" (4), is a useful framework in which to consider heredity-environment interactions. In the application of this concept to quantitative variates, a genotype determines not a limiting phenotype but an indefinite assortment of phenotypes each of which corresponds to certain possible environments. The relative probabilities depend on relative frequencies of different environments; the assortment of phenotypes or the repertoire of reactions and responses is characteristic of the genotype. Since the most probable phenotype of some genotypes may be an extreme or pathological character, these genotypes will produce normal individuals only in unusual environments, if at all. Thus, persons homozygous for phenylketonuria will have normal mentality only in the very rare environments that lack dietary phenylalanine. If a genotype determines an intelligence quotient around 120 in the commonest environments, some rare environments may restrict that individual's achievement to a score of 100 or lower, and others may raise it to 130 or 160. Another genotype in the same lifelong environments might respond entirely differently.

Under this view, the two traditionally conflicting approaches to heredity and environment can be restated in terms of separate and complementary problems:

(i) What are the differences between individual reaction repertoires?

(ii) How flexible is individual development and behavior, that is, how great is the phenotypic variance of individual genotypes?

These questions presuppose a rather well-defined array of environments, and with experimental organisms the approach to both problems is straightforward. For man and particularly for human mental traits, the array of environments cannot be adequately speci-

Instructions for preparing reports. Begin the report with an abstract of from 45 to 55 words. The abstract should *not* repeat phrases employed in the title. It should work with the title to give the reader a summary of the results presented in the report proper.

Type manuscripts double-spaced and submit one ribbon copy and one carbon copy.

Limit the report proper to the equivalent of 1200 words. This space includes that occupied by illustrative material as well as by the references and notes.

Limit illustrative material to *one* 2-column figure (that is, a figure whose width equals two columns of text) or to *one* 2-column table or to *two* 1-column illustrations, which may consist of two figures or two tables or one of each.

For further details see "Suggestions to Contributors" [*Science* 125, 16 (1957)].

fied; the two questions cannot be separately investigated, but they must nevertheless receive separate answers. Solutions will be reached only by a difficult and slow process of successive approximations.

GORDON ALLEN

National Institute of
Mental Health, Bethesda, Maryland

References

1. H. Knobloch and B. Pasamanick, in "Epidemiology of mental disorder," *Publ. Am. Assoc. Advance. Sci. No. 60*, 249 (1959).
2. A. Anastasi, in *ibid.*, p. 264.
3. T. Dobzhansky, *Evolution, Genetics, and Man* (Wiley, New York, 1955).
4. See J. L. Fuller and W. R. Thompson, *Behavior Genetics* (Wiley, New York, 1960), p. 91.

10 October 1960

In the paper (1) criticized by Gordon Allen, we used data from a longitudinal study of a thousand Baltimore infants to speculate upon the major sources of variation in intelligence to be anticipated in them in later life. Only 1.8 percent of this stratified sample, when adjusted for the stratification variables to the Baltimore population, had quotients below 85, in contrast to the 14 percent which might be expected during school age. We also indicated further environmental sources of variability as a result of the largely socio-economically associated prenatal damage following complications of pregnancy as well as prematurity. Allen does not point out that a 30 percent sample of this cohort was reexamined at 3 years of age. The average score for the white infants rose to above 110, while that for the Negroes fell to below 100. In the 4 years since the reading of this paper we have published the findings of additional studies in this area (2). One point pertinent to the matter under discussion is that at 3 years of age there was an increased percentage of low scores; it lay between the 1.8 percent found in the infants and the 14 percent found in school-age children. As was predicted, it occurred almost wholly in the Negro and lower-class white portions of the population.

Comparisons of distributions were possible because of the demonstration of a highly significant correlation between performances in infants and 3-year-olds, reaching 0.75 in those children with the lower scores under discussion. Anne Anastasi, one of the foremost experts in child development and differential psychology, did not question the comparability of developmental quotient and intelligence quotient scales, as Allen implies. In her discussion (3) of the paper she asked for some points of information which we felt were adequately supplied in the succeeding response [(1), p. 269].

We prefer to avoid debate of Allen's

contention that our findings present "no new evidence." Dr. Anastasi opened her discussion [(1), p. 264] by stating, "As a psychologist, I cannot help but be impressed with the importance of the research we have just heard reported and with its far-reaching implications. With regard to the heredity-environment problem, the chief value of such a study lies in its bringing us a step closer to answering the question, 'How?' By tracing relationships between socio-economic factors, prematurity, neurological damage, and subsequent behavioral development, such an investigation helps to disentangle the chain of events leading up to individual differences in intellectual functioning."

Our paper was presented to a sophisticated audience and it was unnecessary, except bibliographically, to refer them to the enormous collection of data on the nature and nurture controversy of this century (4). The English canal boat children findings, the Isle of Man study, Klineberg's work on racial differences, and the Army experiences with both white and Negro illiterates on intelligence tests are not "ambiguous." These studies are not definitive, but they all point in one direction and indicate the importance of environmental factors in determining intelligence test responses. New bits of information which are crucial signposts along the same path appear constantly. One of the most recent of these which is pertinent to the paper under discussion is the observation that within a few years' time prematurity rates in some of the Scandinavian countries have been reduced to 3 percent. This was effected largely by changes in prenatal care. This rapid reduction would seem to indicate that environmental rather than genetic factors are etiologically in prematurity, which is so highly associated with mental defect and poor intellectual performance.

We would like to point out that in the process of comparing infant examination findings with the performance on tests of intelligence later in life by the same individuals, we have in large measure removed the environmentally determined "content" as a contaminating and confounding variable which occurs even in so-called "culture-free" tests of intelligence. It is this point which Allen apparently fails to comprehend when he suggests "that measures of intelligence used in later life reflect relevant genetic potentials more adequately."

A common misunderstanding of scientific strategy is the concept that, if an alternative explanation is possible, it is necessarily equally good. The choice of the most acceptable hypothesis obviously must rest upon such considerations as scientific parsimony,

the weight of the evidence, and fruitfulness for further investigation. This is particularly true in the area of variations in human intelligence where the definitive studies are obviously impossible at this time. Elucidation of the specific enzymatic nature of the genes involved, breeding studies, or even control of the crucial environmental variables do not appear likely in the immediate future. This does not mean that hypotheses cannot be advanced and even partially tested by means of longitudinal investigations. Such studies comprise the process of successive approximations required by the very nature of epidemiologic investigations. Elsewhere we have proposed studies which can more definitively test the hypotheses we advanced (5).

We have no quarrel with Dobzhansky's concept of the "reaction repertoire." Indeed, it is implicit in our theoretical substrate when we stated "the genetic constitution gives man his distinctly human character, and neurologic integrity is basic to the realization of his full developmental potential. In the human organism with an undamaged central nervous system, however, it is life experiences rather than hereditary influences which seem more important in molding intellectual functioning" [(1), p. 250]. It was by studies similar to ours that the first narrowly rigid genetic conceptualizations were altered. We believe that our data have in fact shed some light on the question raised by Allen of "how flexible is individual development and behavior." Parenthetically, the example chosen by him as an illustration of the "reaction repertoire" is rather unfortunate, since the reported number of apparently homozygous phenylketonurics with normal mentality increases yearly (6).

It merely remains to discuss Allen's opening comments on some alleged circular reasoning. Both paragraphs he used were taken out of context from the discussion section at the end of the paper. The second paragraph refers to a hypothesis and not a "conclusion"; it is quite subsidiary to the chief hypothesis of the study. It is also somewhat irrelevant, since we were not primarily concerned with testing variables affecting "measures of intelligence." We merely indicated that the data lent some support to the concept that those measures used in later life are greatly influenced by learning and, in Allen's terms, help determine the "content" of "intellectual development." In the first paragraph Allen chose to look only at the generalization and did not relate the statements on "the lowered score" to the children we reexamined at 3 years of age.

We would like to reiterate the statement made at the conclusion of our

paper. "Even though there are lacunae in the evidence, the patterning of almost all the recent studies, ours as well as others, points the total picture overwhelmingly in one direction. The geneticists will need to give more than *post hoc* data and will require experimental or better controlled epidemiological studies than have previously been offered to support their views. Otherwise, scientific parsimony seems to lead one to the conclusion that at the present time the most useful theory is that while man's fundamental structure and consequently his basic functioning is genetically determined, it is his socio-cultural milieu affecting biological and psychological variables which modifies his behavior and, in the absence of organic brain damage, makes one individual significantly different from the next" [(1), p. 263].

HILDA KNOBLOCH

Ohio State University and
Clinic of Child Development,
Children's Hospital, Columbus

BENJAMIN PASAMANICK

Ohio State University and
Columbus Psychiatric Institute
and Hospital, Columbus

References

1. H. Knobloch and B. Pasamanick, in "Epidemiology of mental disorder," *Publ. Am. Assoc. Advance. Sci.* No. 60, 249 (1959).
2. ———, *Pediatrics* 26, 210 (1960).
3. A. Anastasi, see (1), p. 266.
4. *Yearbook Natl. Soc. Stud. Educ.* 27 (1928); 39 (1940).
5. B. Pasamanick, *Am. J. Mental Deficiency* 64, 316 (1959).
6. B. S. Sutherland, H. K. Berry, H. C. Shirkey, *J. Pediatr.* 57, 521 (1960).

27 December 1960

Calcite in *Lesquerella ovalifolia* Trichomes

Abstract. By chemical analysis, trichomes of *Lesquerella ovalifolia* Rydb. have been shown to contain a high percentage of calcium carbonate. X-ray diffraction patterns showed that it was in the form of calcite. The calcite was inside the trichomes, and its depositional pattern conformed to the shape of the trichomes. A small amount of opal was present in the trichomes.

Recently a photograph of trichomes of *Lesquerella ovalifolia* Rydb. appeared on the cover of *Science* (1); they were reported to be highly refractive. The present investigation was begun to see whether or not the refractivity might be due to the presence of silica.

The plants used in these experiments grew in Scott County, Kansas, on steep rocky slopes with limestone outcroppings. The trichomes were scraped from the leaves with a razor blade and dried in an oven at 110°C. Part of the tri-



Fig. 1. Spodogram showing calcite deposition in *Lesquerella ovalifolia* Rydb. trichomes (about $\times 53$).

chomes were ashed at 500° to 600° and silica was determined by standard gravimetric techniques. The silicon dioxide content was determined by difference of weights before and after treatment with hydrofluoric acid.

Calcium was determined on another ashed sample by the standard A.O.A.C. method with a Beckman model DU flame spectrophotometer and a Sargent recorder. Carbon dioxide was determined by using the standard gas-evolution method directly on oven-dried, powdered trichomes. X-ray diffraction patterns were made for both powdered trichomes and the ash of trichomes on a North American Phillips diffractometer with nickel-filtered copper radiation obtained with a current setting of 20 ma at 40 kv.

Petrographic microscope studies were made on the silica obtained by ashing trichomes and treating the ash with hydrochloric acid. Trichomes were also examined directly with the petrographic microscope.

The depositional pattern of the carbonate was determined by making a spodogram. The spodogram process was developed by Uber (2), modified by Ponnaiya (3), and used by Lanning *et al.* (4) for determining silica depositional patterns in plants.

The results showed that the trichomes made up 51.7 percent of the leaves and that the trichomes were 30.8 percent ash. Chemical analysis showed that the trichomes contain 0.492 percent silicon dioxide, 10.8 percent calcium, and 12.23 percent carbon dioxide. Petrographic microscope examination of the silica showed it to be part plant opal and part detrital quartz. The calcium and carbon dioxide values indicated that the trichomes were 27.0 percent calcium carbonate and that the ash was 87.7 percent calcium carbonate. Trichomes of plants from Sheridan County State Park in Kansas contained 11.1 percent calcium.

High values for plants from two different areas indicate that high deposition of calcium carbonate in the trichomes is a characteristic of the species. The values also indicate an exceptional differential accumulation of calcium, for the leaves without trichomes contained only 2.25 percent calcium. The latter value is about average for leaves of many of the Cruciferae (5).

Calcium compounds are commonly deposited in phloem tissue and veins of plants (6), often in the form of the oxalate, pectate, or carbonate. In 1932 hackberry seed was reported to contain a very high percentage of calcium (7). In 1959 Swineford and Franks (8) found hackberry seed to contain 45.51 percent calcium carbonate in the form of aragonite.

The x-ray diffraction pattern of the ash showed the sharp peaks characteristic of calcite (9). No other sharp peaks were observed, indicating that the ash was largely calcium carbonate in the form of calcite. The x-ray diffraction pattern of the powdered trichomes also showed the calcite peaks. In addition it showed x-ray peaks of cellulose and small quartz peaks (detrital).

A spodogram of the trichomes (Fig. 1) shows that the depositional pattern of calcite is just like that of the trichomes. Microscopic examination showed that the calcium carbonate was inside the trichome and surrounded by organic matter of the cell wall. Some plant opal was also observed (10).

As far as I am aware, this is a first report of calcite in *Lesquerella ovalifolia* and in the genus. It also appears to be a first report of high calcium carbonate deposition in trichomes.

F. C. LANNING

Department of Chemistry, Kansas
State University, Manhattan

References and Notes

1. R. C. Rollins, *Science* 131, 683, 687 (1960).
2. F. M. Uber, *Botan. Rev.* 6, 204 (1940).
3. B. W. X. Ponnaiya, "Studies in the genus *Sorghum*: the cause of resistance in sorghum to the insect pest *Antherigona indica* M.," *J. Madras Univ.* B21, No. 2 (1951).
4. F. C. Lanning, B. W. X. Ponnaiya, C. F. Crumpton, *Plant Physiol.* 33, 339 (1958).
5. K. C. Beeson, "The mineral composition of crops with particular reference to the soils in which they were grown," *U.S. Dept. of Agriculture Misc. Publ.* No. 369 (1941).
6. B. S. Meyer and D. B. Anderson, *Plant Physiology* (Van Nostrand, New York, ed. 2, 1952), p. 480.
7. E. Yanovsky, E. K. Nelson, R. M. Kingsbury, *Science* 75, 564 (1932).
8. A. Swineford and P. C. Franks, "Opal in the Ogallala formation in Kansas. Silica in sediments," *Soc. Econ. Paleontologists and Mineralogists Special Publ.* No. 7 (1959).
9. H. E. Swanson and R. K. Fuyat, *Natl. Bur. Standards (U.S.) Circ.* No. 539 (1953), p. 51.
10. The investigation was supported by National Science Foundation grant No. NSF 7101. I wish to express my gratitude to Carl F. Crumpton for making the x-ray diffraction patterns and to Dr. L. C. Hulbert for furnishing the *Lesquerella ovalifolia* plants.

12 December 1960