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# Norm-of-Reaction: Definition and Misinterpretation of Animal Research

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The development of a phenotype is due to an interaction of the genotype with the environment. Two terms have been used to describe the outcome of this interaction, the *norm-of-reaction* and the *reaction range*. The first represents the theoretically limitless distribution of the phenotypes that may be expressed by a given genotype. The reaction range implies an upper and lower limit for phenotype expression possible from a given genotype. A critical distinction between the reaction range and the norm-of-reaction is that the norm-of-reaction is a statement of the conceivable interactions found but does not imply any predictability other than that within the conditions previously tested experimentally, that is, the tails of a normal distribution are infinitely variable, whereas the concept of reaction range implies a limitation inherent in the genotype, that is, a finite range. Empirical support for the reaction-range concept is questionable. Animal studies cited in support of the reaction range have been inappropriately and incorrectly interpreted.

The emergence of a phenotype is due to a developmental interaction of a genotype with an environment. The term *interaction* does not refer to one or more components in a statistical analysis susceptible to elimination through some transformation of scale applied to group scores and thus to be dismissed as a mere measurement problem. *Interaction*, as used herein, is the intricate and unique interplay of the numerous causal processes of development from the micro-level of gene interactions and genetic-cellular chemistry to the macrolevel of individual-environment feedback loops. Two terms, *norm-of-reaction* and *reaction range*, have been used to describe the outcome of this interaction. The norm-of-reaction refers to all phenotypic outcomes of a single genotype exposed to all possible environments. It recognizes both the theoretically possible and experimentally measured outcomes and presupposes no practical limits on phenotypic variability. The reaction-range concept presumes that the genotype imposes a priori limits (a range) on the expression of a phenotype. This is a subtle but important distinction between the two concepts. Neither the complete norm-of-reaction of a genotype nor the limits of a reaction range can ever be determined experimentally. Because environments to which genotypes are exposed may vary along many dimensions, it follows that phenotypic expression is also multidimensional. We argue that animal experiments have been inappropriately used to infer limits on the expression of complex human phenotypes.

## Norm-of-Reaction

The contribution of genotype-environment interactions to phenotypic variability was first defined as the "norm-of-reac-

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tion" (*Reaktionsnorm*) by Richard Woltereck (1909, 1928), professor of zoology at Leipzig. Replicates of a specific genotype (clones) may develop differently in different environments. Different genotypes do not necessarily respond similarly in the same environment. Using isolated pure lines of *Daphnia* reproduced parthenogenetically, Woltereck raised a succession of generations in carefully controlled environments differing in temperature and nutritive levels. Looking mainly at head form, he obtained for each population (genotype) under each condition a frequency distribution of phenotypes, a "phenotype curve."

"Die in all diesen Kurven dargestellte Gesamtheit der Relationszahlen Können wir als die spezifisch-relative *Reaktionsnorm* des analysierten Quantitativmerkmals bezeichnen. . . ." (Woltereck, 1909, p. 135) ("The numerical relations as a whole represented in all of these curves can be designated as the specific and relative *norm of reaction* of the quantitative characters being analyzed. . . ." [Translated by Dunn, 1965, p. 96])

Thus the norm-of-reaction was defined as an array of phenotypes expressed under a controlled set of environments. Neither phenotype nor phenotypic variability is encoded in the genotype per se. Rather the range of phenotypes produced represents samples from a distribution of possible phenotypes.

Theodosius Dobzhansky (1955) clarified this concept of the norm-of-reaction.

The norm of reaction of a genotype is at best only incompletely known. Complete knowledge of a norm of reaction would require placing the carriers of a given genotype in all possible environments, and observing the phenotypes that develop. This is a practical impossibility. The existing variety of environments is immense, and new environments are constantly produced. Invention of a new drug, a new diet, a new type of housing, a new educational system, a new political regime introduces new environments. (pp. 74-75)

And under the subheading "Superior and Inferior Norms of Reaction," Dobzhansky (1955) provided the following caution.

[I]t may be misleading to say that the carriers of a certain genotype must reach certain "intrinsic" height, or weight, or skin color, or intelligence level. Any height or weight or intelligence a person may have is "intrinsic," in the sense that the phenotype observed is the necessary outcome of the development brought about by a certain genotype in a certain succession of environments. We can never be sure that any of these traits have reached the maximal development possible with a given genotype. The performance of a genotype cannot be tested in all possible environments, because the latter are infinitely variable. (p. 77)

### Reaction Range

The reaction-range position can be characterized as "genes set the limits, but the environment determines where within those limits the phenotype will fall." Hirsch (1967) noted how easy this is to misinterpret.

Paradoxically that statement is at once true and misleading. Its truth lies in its expression of the norm-of-reaction concept: The phenotypic development of each genotype *is* determined by its ontogenetic environment. . . . The misleading aspects of this statement are due to typological thinking. Because there is no place for individual differences in the typological frame (uniformity is axiomatic), a true statement has been misconstrued as justifying the impossible, that is, the study of environmental influences *per se*. What I call impossible (theoretically) might have been practically feasible (loosely speaking) if the variation pattern for responding to the limitless set of conceivable environmental conditions were exactly the same for all possible genotypes. Since genotypic diversity and genotype-environment interaction are apparently ubiquitous, attempts to study the laws of environmental influence have been grasping at shadows. (pp. 420-421)

The origin of the reaction-range concept is traced to I. I. Gottesman (1963a, 1963b). Gottesman proposed the reaction range to explain the simultaneous contributions of genotype and environment to variations in phenotype (Gottesman, 1963a, 1968, 1974; Gottesman & Heston, 1974).

For our purposes the best way to conceptualize the contribution of heredity to intelligence is to think of heredity as determining a norm of reaction (Dobzhansky, 1955) or as fixing a reaction range. Within this framework a genotype determines an indefinite but circumscribed assortment of phenotypes, each of which corresponds to one of the possible environments to which the genotype may be exposed. [Figure 1] illustrates schematically the concept of reaction range as applied either to four different individuals or to four classes of individuals. For each of the four curves to apply to individuals, it would require the carrier of a given genotype to be exposed to as wide a range of environments as appeared to lead to a change in the phenotypic expression of intelligence. This is a practical impossibility with humans but may be approached with highly inbred strains of mammals (Thompson, 1954). (Gottesman, 1963a, pp. 254-255)

Gottesman's graphic illustration and concept of the reaction range has received wide acceptance and citation (e.g., Eckland, 1967, p. 179; Gottesman, 1966, p. 200; Henderson, 1970, p. 510; Jensen, 1969, p. 64; Keating, 1975, p. 45; Pettigrew, 1964, p. 107), in several developmental textbooks (e.g., Clarke-Stewart, Friedman, & Koch, 1985; Clarke-Stewart & Koch, 1983; Hetherington & Parke, 1979; Shaffer, 1985), and inclusion in many introductory psychology textbooks (e.g., Atkinson, Atkinson, Smith, & Hilgard, 1987, p. 409; Bernstein, Roy, Srull, & Wickens, 1988, p. 384; Crider, Goethals, Kavanaugh, & Solomon, 1986, p. 285; Krebs & Blackman,

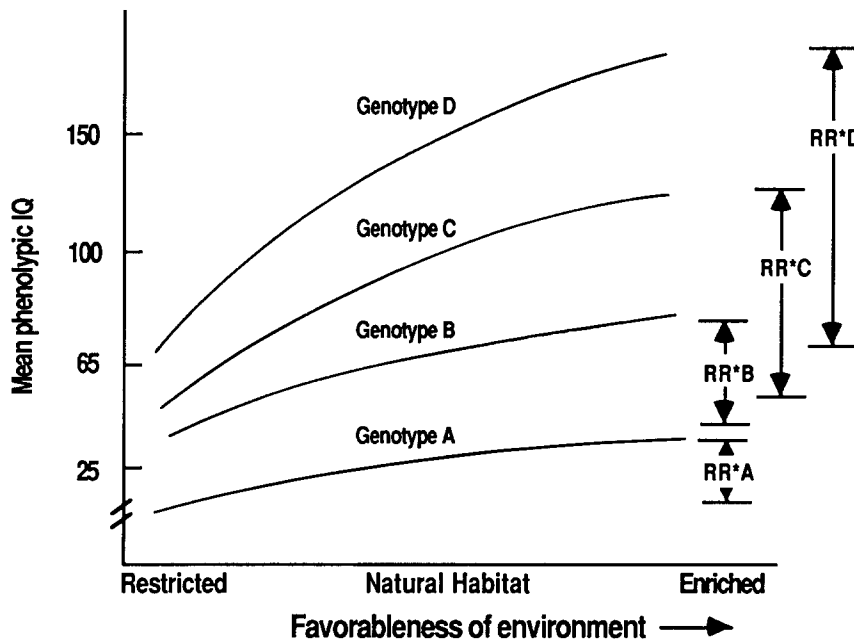


Figure 1. Scheme of the reaction-range concept for four genotypes. (Marked deviation from the natural habitat has a low probability of occurrence. RR indicates reaction range in phenotypic IQ. From "Genetic Aspects of Intelligent Behavior" by I. I. Gottesman, 1963, p. 255, in N. Ellis [Ed.], *The Handbook of Mental Deficiency* [pp. 253-296], New York: McGraw-Hill. Copyright 1963 by N. R. Ellis. Reprinted by permission.)"

1988, p. 74; McGee & Wilson, 1984, p. 318; Ornstein, 1988, p. 79; Papalia & Olds, 1988, p. 305; Ruch, 1984, pp. 438–439; Wortman, Loftus, & Marshall, 1988, p. 386). Gottesman’s reaction-range concept has also been applied to other phenotypic variables such as height and schizophrenic susceptibility (e.g., Garnezy, 1974; Ornstein, 1985; Ruch, 1984).

There are several peculiarities with the graph shown in Figure 1. The ordinate scaling is irregular. If the same data points are plotted on an interval scale on the ordinate, the reader will discover that the curves approach linearity and appear to continue upward leading the viewer to query, what would be the result of further environmental enrichment? In essence, there would appear to be no limits. The lower limit of each genotype appears to be arbitrarily set. Surely one could conceptualize any number of environments wherein Genotypes A through D would be so restricted that a zero or unmeasurable IQ might be noted. Thus this graph actually represents a single measure of proposed upper IQ limits for each of four genotypes given a conceptually limited range of environments.

The ranking of genotypes is an obvious implication inherent in the term *reaction range* and is easily illustrated by Figure 1. Although all acknowledge the interactive effects of genotypes with environments, introductory psychology textbooks vary in their interpretation of the parameters of the genetic contribution. The discussion usually takes place in the context of nature–nurture and IQ issues.

We can think of a person’s genes as imposing a top and a bottom limit on intelligence, or establishing a range of intellectual ability.

Environmental influences . . . will determine where the person’s IQ will fall within that range. In other words, genes do not specify behavior; rather, they establish a range of probable responses to the environment, which is called the *reaction range*. (Atkinson et al., 1987, p. 409).

This quote from the ninth edition of the very popular *Introduction to Psychology* is followed by a slightly modified reproduction of the Gottesman schematic and is included herein as Figure 2. Note that genotype is replaced with “Type” and an inappropriate phenotype label is added (“superior intelligence, average intelligence, retarded, and mentally defective”). This further strengthens the misconception that certain “types” are superior in any situation, that is, superiority is built in rather than being the result of a fortuitous genotype–environment interaction.

One merely has to ask a class of introductory psychology students “Which genotype would you want to be?” to see the persuasive power of this misleading graph. Thus where the term *reaction range* is used we can see a subtle emphasis not present in the original delineating of the term *norm-of-reaction*, an emphasis on the concept of limits built in by the genes and the implication of genotypic ranking by potential. Limits are circumscribed by current knowledge and technological understanding and are not really reflective of a genotype per se. In this context genotypic limits, as implied by the reaction range, make little or no sense. Purely through development of new technologies or educational programs, current perceived limits may become obsolete (e.g., the understanding of phenylketonuria or the development of synthetic human growth-hormone; see also Lovaas, 1987, in which through

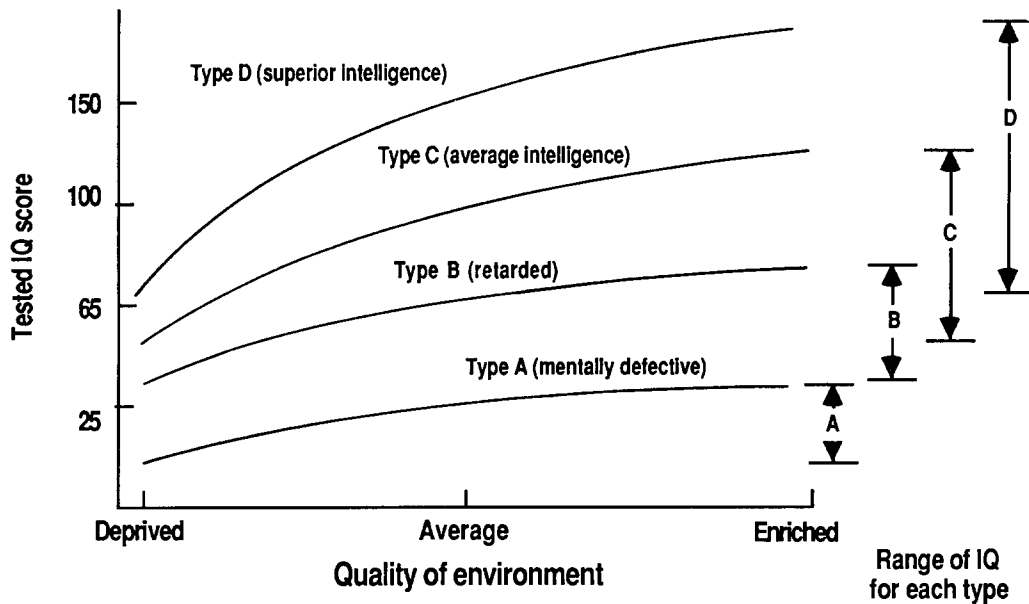


Figure 2. Effects of different environments on IQ. (The curves represent hypothetical reaction ranges for four individuals who vary in inherited intellectual potential. For example, the individual labeled Type D has an IQ of about 65 when raised in a deprived environment but an IQ of over 180 when raised in a maximally enriched environment. The vertical arrows to the right indicate the range of possible IQ scores for each type. Adapted from “Genetic Aspects of Intelligent Behavior” by I. I. Gottesman, 1963, in *Introduction to Psychology* [9th ed., p. 409] by R. L. Atkinson, R. C. Atkinson, E. E. Smith, and E. R. Hilgard, 1987, New York: Harcourt Brace Jovanovich. Copyright 1987 by Ernest R. Hilgard. Reprinted by permission.)

intensive behavioral intervention autistic children were able to achieve normal intellectual and educational functioning).

### Support From Animal Studies

The data base for Gottesman's "scheme of the reaction range concept for four hypothesized genotypes" comes from animal studies "with highly inbred strains of mammals" (Gottesman, 1963a, p. 255). Two studies are cited in particular, Thompson (1954) and Cooper and Zubek (1958).

In a cautiously worded review, Thompson (1954) makes no claims that would justify a reaction-range interpretation, that is, a finite range inherent in a particular genotype. After illustrating the selective breeding results of the first six generations of the McGill *bright* and *dull* strains (in a graphic representation that has received some considerable citation and manipulation; see, in particular, Hirsch, 1975/1976, pp. 75: E 2703-2705; Jensen, 1969, p. 31; Lefton, 1985, p. 305), Thompson states: "The second requirement of homozygosity . . . has not yet been satisfactorily met due to the infertility of many of the brother-sister matings, particularly in the last generation [Generation 6]. In fact, in order to insure survival of the strains, random matings were necessary" (p. 218). It cannot be claimed, therefore, that these were highly inbred strains of mammals.

Thompson's data are contained in Table 1. When these same data are included in a 1955 publication an interesting phrase change occurs.

In fact, as shown in [Table 1], the amount of variance that can be produced in rat intelligence by altering environment *is almost as much as* [italics added] can be obtained by selective breeding for brightness and dullness. (Thompson, 1954, p. 221)

It is interesting, as I have pointed out previously (1954), that the extent of change that can be induced in rat intelligence by manipulating the early environment *is as great as* [italics added] that obtained by selectively breeding for brightness and dullness in a maze. (Thompson, 1955, p. 127)

The essence of this, albeit very slight, phrase change is to reduce the implicit domination of the genotype in determining the phenotype.

More important, Thompson's data (see Table 1) come from three different experiments. Not knowing the conditions, sex, or strains used for collection of the hereditary dull or bright scores seriously limits our interpretation of the data. The score for the environmental free condition actually came from an earlier study by Hymovitch (1952) of a group of 20 male rats (Group 1, Experiment 2, p. 317) reared in a 6 foot × 4 foot × 6 inch (183 cm × 122 cm × 15 cm) box (wire mesh top) with "a number of blind alleys, inclined runways, small enclosed areas, apertures, etc." Although Forgays and Forgays (1952) is referenced as the source of the score of 238.2 from the environmentally restricted condition, the restricted animals reported in Forgays and Forgays actually had an error score of 241.25. Despite these shortcomings we can infer variability of phenotypes from these data, but a reaction range built in by the genotype is an unwarranted inference, because there are many alternate environments. Thompson's (1955)

methodology can in no way be considered as exhaustive of all environments.

Frequently Cooper and Zubek (1958) have been cited in support of the concept of a reaction range of genotype-environment interactions. In 1958 in the *Canadian Journal of Psychology*, Cooper and Zubek reported their findings of the learning performance of maze *bright* and *dull* rats reared under conditions of environmental restriction and enrichment. This study has become something of a classic in the nature-nurture controversy. It is generally claimed to be an example of the interaction of heredity and environment (e.g., Carlson, 1987; Eysenck, 1971; Fernald & Fernald, 1985; Fessler & Beatty, 1976; Finger, 1978; Gottesman, 1963a, 1968; Heaton & Klein, 1981; Henderson, 1970; Isaacson & Hartesveldt, 1978; Jensen, 1969; Kimble, Garmezy, & Zigler, 1984; Li, 1978; Montagu, 1972; Pettigrew, 1964; Risch, 1979; Roediger, Rushton, Capaldi, & Paris, 1987). However, not all concur that Cooper & Zubek (1958) illustrate a genotype-environment interaction (e.g., Ferchmin, Eterovic, & Levin, 1980; Myslivecek & Stipek, 1979; Rajalakshmi & Jeeves, 1968; and, in particular, Throne, 1975). Some cite the study as an example of a learning experiment (Huck & Price, 1976) or as an example of environmental intervention (Miller, 1980). Given such wide citation, the manner in which the Cooper and Zubek (1958) results are interpreted is central to an understanding of the interaction of genotypes and environments in determining variability among phenotypes.

For purposes of the present discussion, Cooper and Zubek (1958) was interpreted by Gottesman (1963a) as an example of the differential effects three rearing environments ("restricted, natural, and enriched") have upon two genotypes—*maze-bright* and *maze-dull* rats.

Cooper and Zubek (1958) used the [13th generation] of both the bright and dull lines of Thompson's McGill rats as their subjects. [Table 2] gives the mean error scores for the brights and dulls on the first 12 problems of the Hebb-Williams maze under three environmental conditions. . . . An enriched early environment led to a considerable improvement in the performance of the dulls but had little or no effect upon the brights. The dulls reduced their errors by about 27 per cent. A restricted early environment increased the errors of the brights by about 44 per cent but had little or no effect upon the dulls. Notice that with an enriched environment the dulls were equal to the brights under the latter's normal or natural habitat and that the restricted

Table 1  
*A Comparison of Hereditary and Environmental Effects on the Intelligence of Rats*

Strain	Error score on Hebb-Williams maze
Hereditary dull	279.5
Environmental restriction	238.2 <sup>a</sup>
Hereditary bright	142.8
No environmental restriction	137.3 <sup>a</sup>

Note. From "The Inheritance and Development of Intelligence" by W. R. Thompson, 1954, p. 221, in D. Hooker and C. C. Hare (Eds.), *Genetics and the Inheritance of Integrated Neurological and Psychiatric Patterns* (pp. 209-231), Baltimore: Williams & Wilkins. Copyright 1954 by Williams & Wilkins. Reprinted by permission.

<sup>a</sup> Scores based on data of Forgays and Forgays (1952) and Hymovitch (1952).

Table 2  
Maze Error Scores for Bright and Dull Rats Reared Under Three Different Conditions

Strain	Environment					
	Enriched		Natural		Restricted	
	n	Score	n	Score	n	Score
Brights	12	111.2	11	117.0	13	169.7
Dulls	9	119.7	11	164.0	9	169.5

Note. From "Genetic Aspects of Intelligent Behavior" by I. I. Gottesman, 1963, p. 272, in N. Ellis (Ed.), *The Handbook of Mental Deficiency* (pp. 253–296), New York: McGraw-Hill. Copyright 1963 by N. R. Ellis. Reprinted by permission.

brights equaled the natural dulls. These data fit in very nicely with the earlier exposition of the reaction range. . . . Extrapolating to the human case, it may be that eventual manipulation of the intelligence phenotype will be only a question of economics. Within the range of intelligence accounted for by a polygenic system, a great expenditure of effort may replace the effects of intelligence-enhancing genes. (Gottesman, 1963a, pp. 272–273)

In 1968, when Gottesman again discussed the Cooper and Zubek study, he displayed the data in the form shown in Figure 3 and attributed the bar graph to Pettigrew (1964). Fernald and Fernald (1985, p. 77) also credited their bar graph of Cooper and Zubek's data to Pettigrew (1964).

Pettigrew (1964), in his book titled *A Profile of the Negro American*, attributed much of his discussion of genotype–environment interactions to Gottesman's (1963) review and discussed the Cooper and Zubek (1958) study as follows:

An ingenious animal experiment by Cooper and Zubek illustrates this genetic–environmental interaction. These investigators em-

ployed two genetically distinct strains of rats, carefully bred for 13 generations as either "bright" or "dull." Separate groups of the two strains grew up after weaning in three contrasting environments: a restricted environment, . . . a natural environment, . . . and an enriched environment. . . . [Figure 3] shows the maze learning performances of the six groups of rats (the fewer the errors, the more "intelligent" the behavior). Note that the two genetically diverse groups did almost equally well in the enriched and restricted environments, sharply differing only in the natural situation. In fact, the environment masks genetic potential to the point where it is impossible to distinguish the enriched dulls from the natural brights or the natural dulls from the restricted brights. (Pettigrew, 1964, pp. 105–106).

In the second edition of his introductory psychology textbook, Carlson (1987) discusses the Cooper and Zubek research in the following manner.

[E]vidence for an interaction effect in human intelligence is circumstantial, not direct. However, Cooper and Zubek (1958) gathered evidence in the laboratory that illustrates the interaction between heredity and environment. [They] raised groups of both strains of rats in three different environments . . . then tested . . . their ability to learn a maze.

The results, shown in [Figure 4], reveal a very strong interaction between heredity and environment. . . .

From these results we can conclude that (1) heredity has a strong effect on a rat's ability to learn a maze, (2) a rat's early environment has a strong effect on its ability to learn a maze, and (3) the effect of a rat's heredity depends on the environment in which it is raised; the two factors interact. Although we have no comparable experimental data from humans, the graph suggests that it is important to provide people with an optimal environment, whatever their heredity may be. (Carlson, 1987, p. 388).

The graph of Cooper and Zubek's data shown in Figure 4 is first presented in this graphic form by Jensen (1969, p. 40).

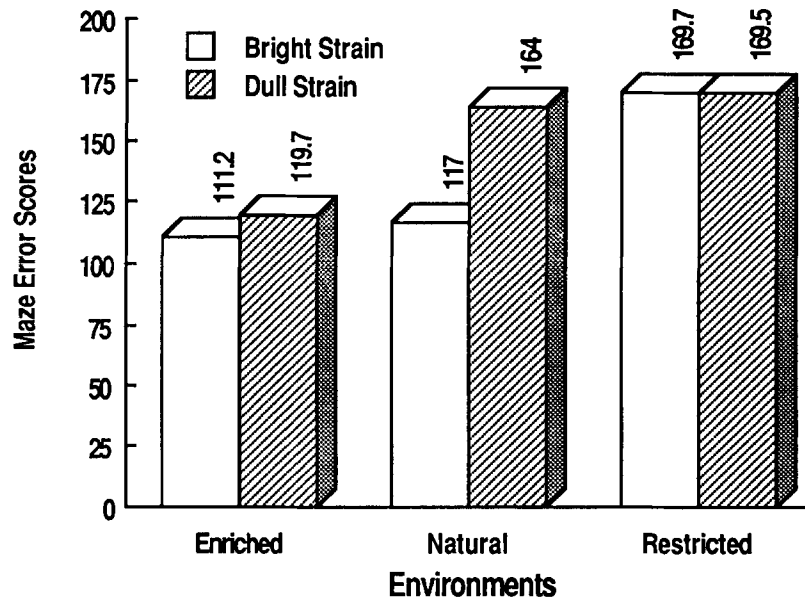


Figure 3. Maze error scores of genetically bright and dull rats reared in three different environments. (After *A Profile of the Negro American* by T. F. Pettigrew, 1964, Princeton, NJ: Van Nostrand in "Biogenetics of Race and Class" by I. I. Gottesman, 1968, p. 31, in M. Deutsch, I. Katz, and A. R. Jensen [Eds.], *Social Class, Race, and Psychological Development* [pp. 11–51], New York: Holt, Rinehart & Winston. Copyright 1964 by I. I. Gottesman. Reprinted by permission.)

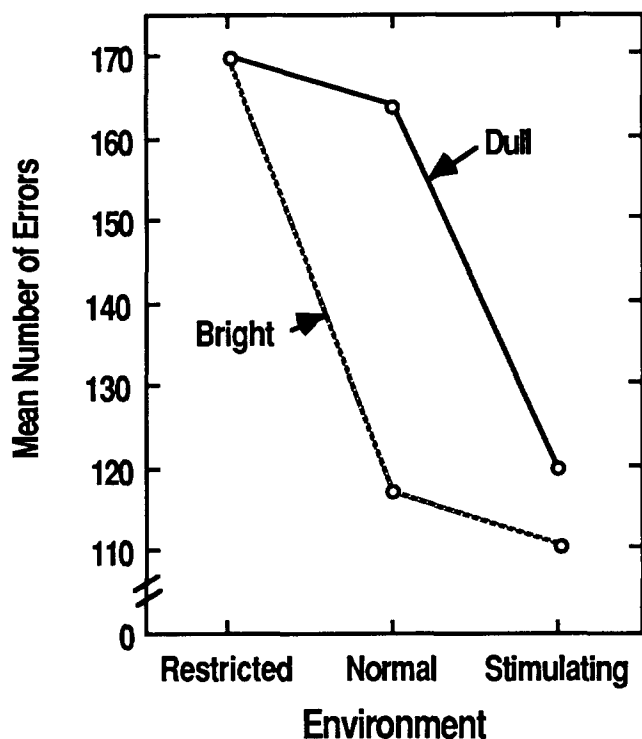


Figure 4. Illustration of a true Genotype  $\times$  Environment interaction for error scores in maze learning by bright and dull strains of rats raised in restricted, normal, and stimulating environments. (After "Effects of Enriched and Restricted Early Environments on the Learning Ability of Bright and Dull Rats" by R. M. Cooper and J. P. Zubek, 1958, *Canadian Journal of Psychology*, 12, pp. 159–164 in "How Much Can We Boost IQ and Scholastic Achievement?" by A. R. Jensen, 1969, *Harvard Educational Review*, 39, p. 40. Copyright 1969 by Harvard Educational Review. Reprinted by permission.)

#### Reanalysis of Cooper and Zubek (1958)

Cooper and Zubek (1958) reported data from 43 rats of the McGill bright and dull strains of the 13th generation of selection raised in only two environments. The subjects were placed in either a restricted or enriched environment for 40 postweaning days (Days 25–65).

The two restricted cages were placed on one side of [a] partition, the two enriched cages on the other side. The side of the partition facing the restricted cages was grey, matching the colour of the room. The side of the partition facing the enriched cages was white with "modernistic" designs painted upon it in black and luminous paint. (pp. 159–160).

The restricted cages contained a food box and a water pan. The enriched cages had the addition of various objects to manipulate. These experimental rearing environments are hardly indicative of the range of possible environments to which young rats could be exposed. Many introductory psychology students have probably discovered how innovative their "pet" rat can be after special handling and manipulation.

The subjects reared in a "normal" environment, upon which all claims of genotype-environment interactions are made above, did not come from this study.

For purposes of statistical analysis and interpretation of the data the performances of the enriched and restricted animals were compared with the performances of 11 bright and 11 dull animals raised in a "normal" laboratory environment. These were the animals that formed two control groups in an experiment by Hughes and Zubek [1956]. (Cooper & Zubek, 1958, p. 160).

These control group animals came from the 10th generation of selection as reported in a study published two years earlier. Thus Cooper and Zubek (1958) only demonstrated that both maze-bright and dull rats performed equally poorly in a restricted condition and equally well in a slightly enriched condition.

Although the dull-enriched group averaged 8.5 more errors than did the bright-enriched, this difference is not significant ( $t = .819, p > .5$ ). . . . The difference between the bright- and dull-restricted groups . . . is also obviously insignificant [bright = 169.7 errors; dull = 169.5 errors]. (p. 161)

Cooper and Zubek did not demonstrate genotype-environment interactions and certainly did not provide any data indicating that a hypothetical reaction range had been ascertained. The line graph representation of the Cooper and Zubek data (Jensen, 1969, p. 40) is inappropriate for two reasons. The implication of the graph is that animals were raised under three conditions. Remove the middle data points, and the message is entirely different. The exploded difference between the enriched dull and bright groups is a function of the graphic representation and not of the data. The difference is not statistically significant.

The message frequently implied and often stated in the reporting of Cooper and Zubek is that the dull's genetic inferiority might be masked by environmental enrichment and can only be improved by especially strenuous effort with radical environmental intervention. It is Hughes and Zubek (1956) that could be cited as an example of genotype-environment interaction, but the message would not imply any genotypic or variability limitations. Hughes and Zubek reported that the performance of maze dull rats vastly improved when monosodium glutamate (MSG) was added to their diet. The performance of MSG-treated maze-bright rats did not improve.

#### Conclusion

Besides the previously mentioned graphic limitations and restricted data base, the reaction-range concept presents environmental change on a single linear dimension. However, genotypes and environments are interactive. Genotype-environment interactions take place at multiple developmental levels and across and along many dimensions. Current data on phenotypic variation merely indicate the range of relevant environments to which the genotypes in question have been exposed. It needs to be reiterated: Just as the basic tenet of behavior-genetic research is that different genotypes exhibit different norms-of-reaction for variation along a given environmental dimension, so the same genotype may develop

different phenotypes in different environments. In the analysis of genotype–environment interactions, to assign proportions of responsibility is akin to asking of the equation for the area of a rectangle, how important is the length?

Thus, the limits of phenotypic expression are determined by the limits of current technology and our imagination to apply it to the discovery of the environmental complex necessary to facilitate the production of the phenotype we desire given the genotype with which we are working.

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