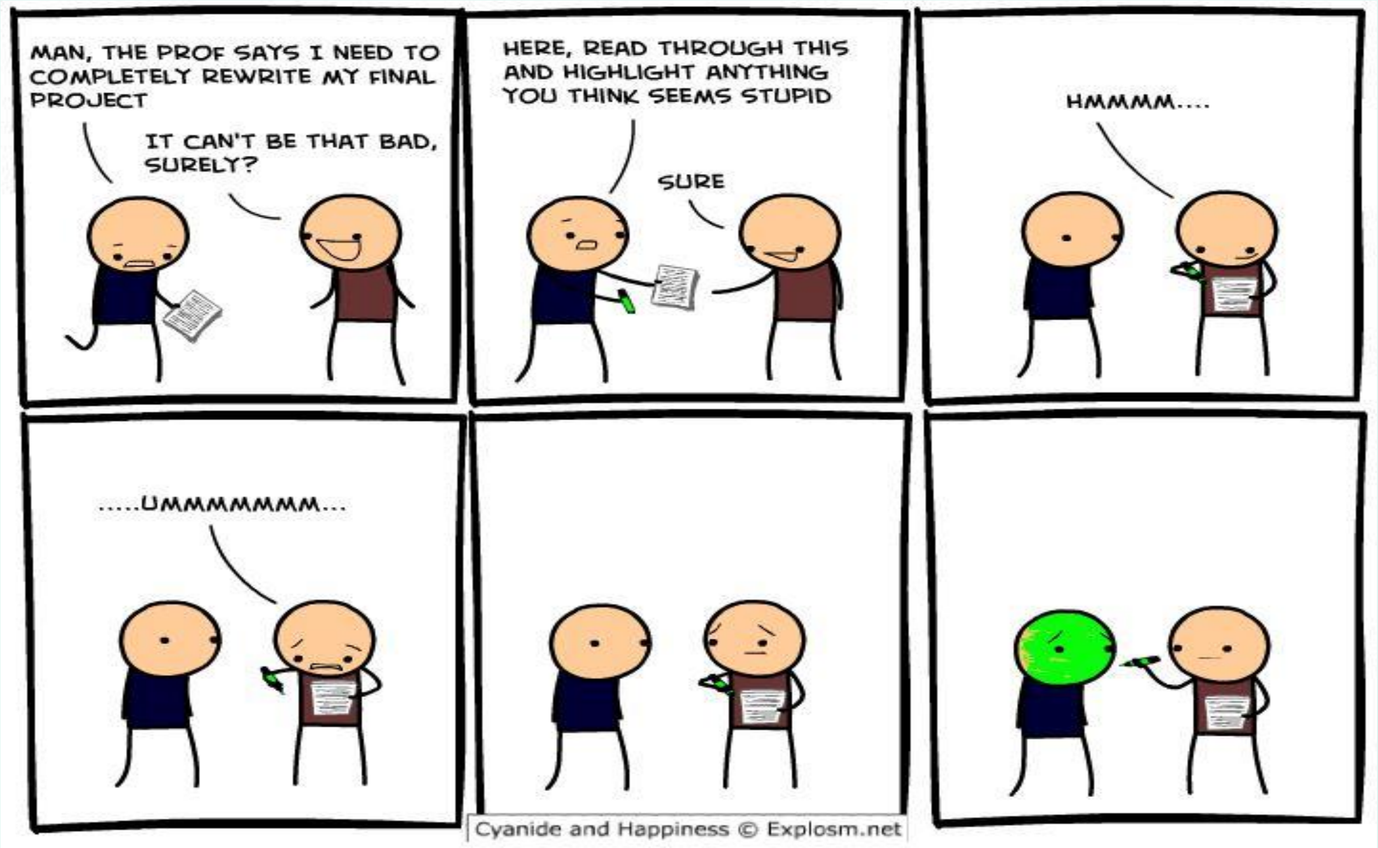
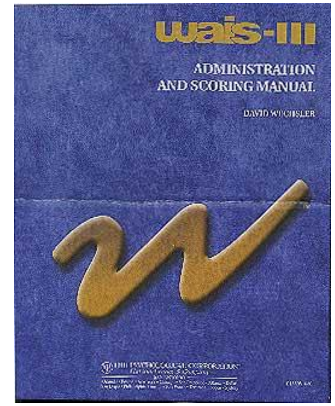
Psych 3102
Introduction to Behavior Genetics
Lecture 19
Genetics of cognitive abilities



Hierarchical, psychometric model of cognitive ability Spearman, 1904

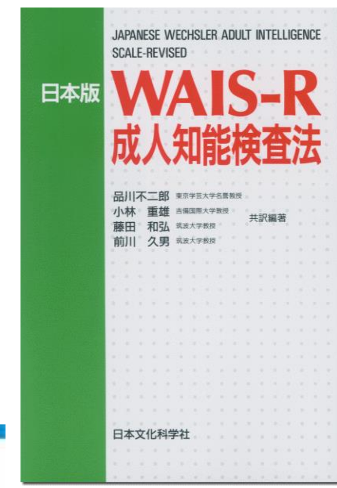
General cognitive ability (g)

- derived by factor analysis of scores from various weighted measures of more specific abilities



Specific cognitive abilities:

verbal spatial processing speed memory

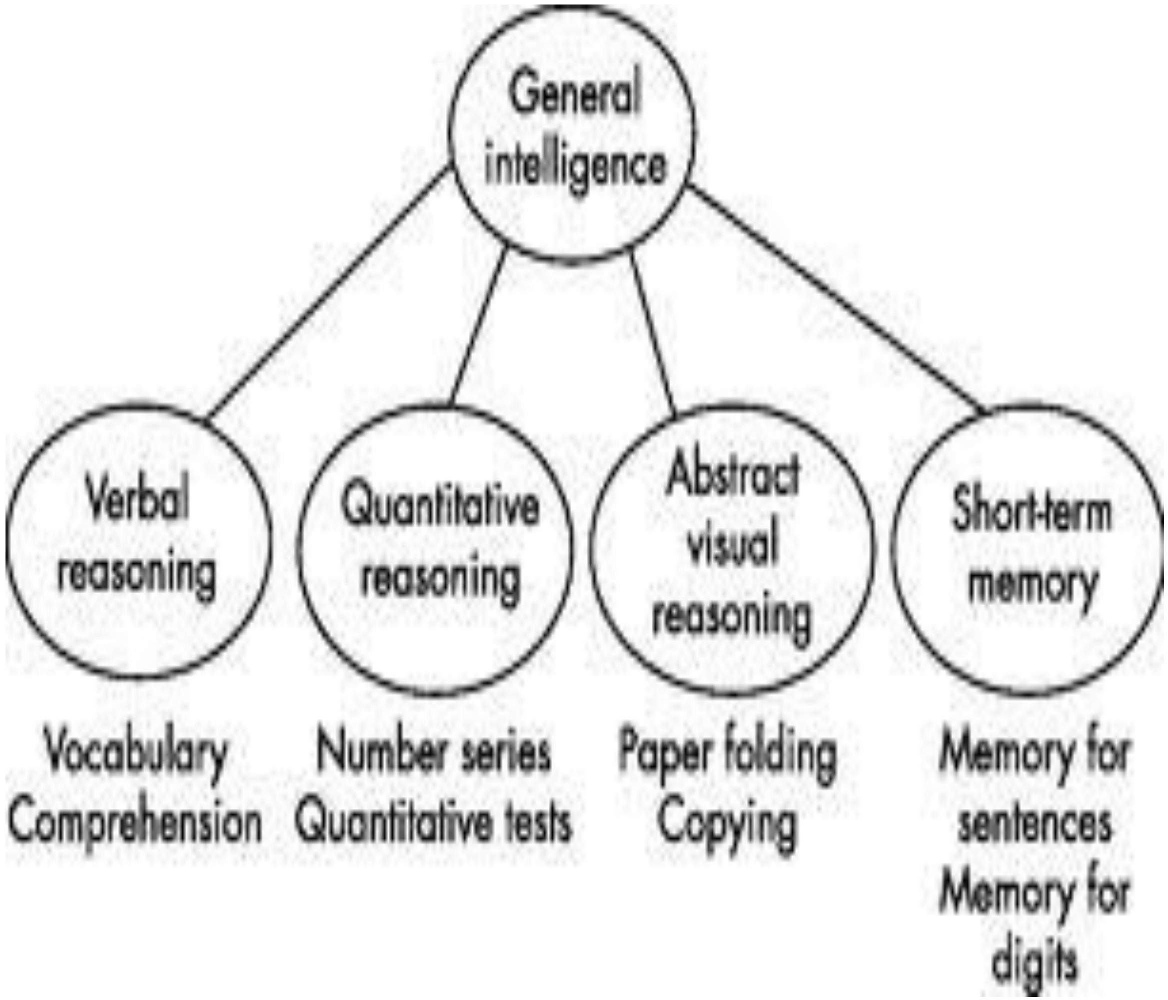


Measures (tests):

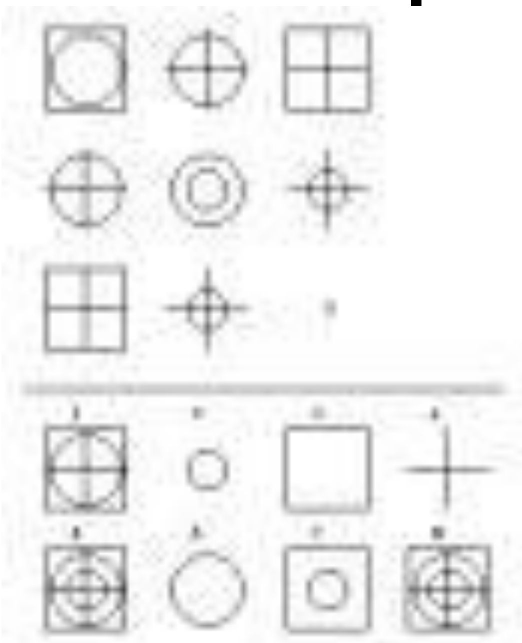
Wechsler Raven's matrices Stanford-Binet ..

- weight given to an item is determined by its correlation with other items
  - items that correlate highly and items that measure more complex tasks are weighted more (contribute more to g)

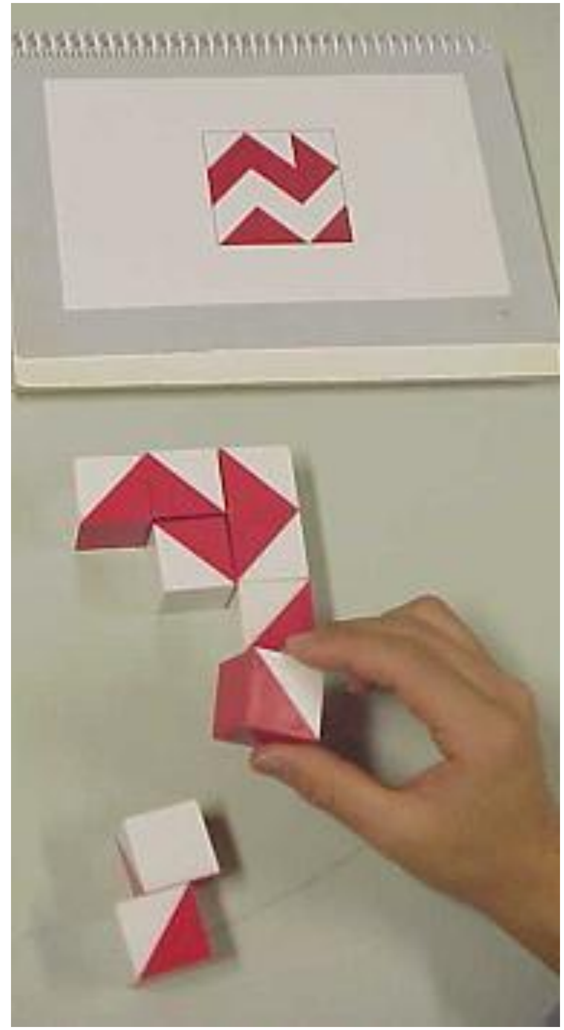
abstract reasoning > simple sensory discrimination


General intelligence
Verbal reasoning
Quantitative reasoning
Abstract visual reasoning
Short-term memory
Vocabulary
Comprehension
Number series
Quantitative tests
Paper folding
Copying
Memory for sentences
Memory for digits

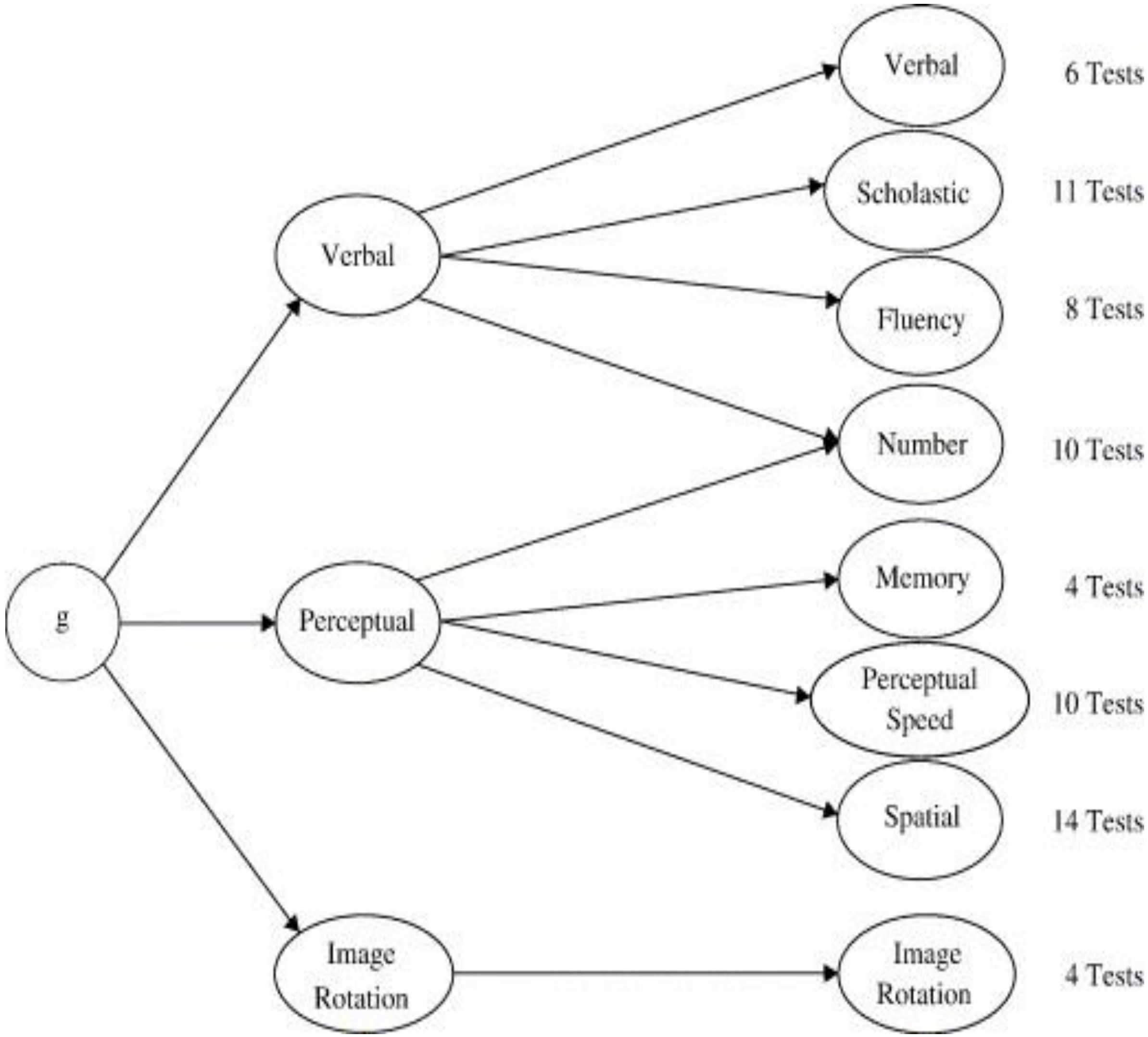
Examples of cognitive tests



Sample Item from Raven's Progressive Matrices



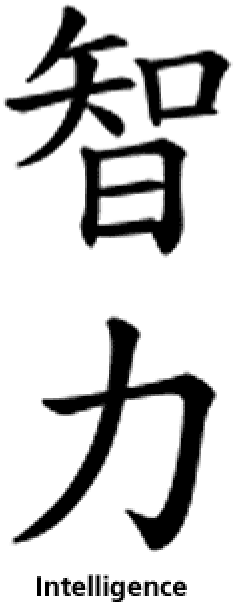
Wechsler block Design Task



Structural portion of verbal-perceptual-rotation (VPR) model of intelligence

Johnson et al(2006) Intelligence, 35, 542

Definitions of Intelligence:
Which one do we prefer?



- E. G. **Boring**, a well-known Harvard psychologist in the 1920's
  ..."whatever intelligence tests measure"

- Alfred **Binet** in The Individual
  ...the ability to "judge well, to comprehend well, to reason well."

- David **Wechsler** cited in Annual Editions
  ..."the global capacity of the individual to act purposefully, to think rationally, and to deal effectively with the environment."

- **Benjamin, Hopkins and Nation** in Psychology (a textbook)
  ..."the capacity to acquire and use knowledge, a capacity that is supported by a host of cognitive abilities such as perception, memory storage and retrieval, reasoning, problem solving and creativity."

- from the **Merriam-Webster Dictionary**
  (1) the ability to learn or understand or to deal with new or trying situations; also, the skilled use of reason
  (2) the ability to apply knowledge to manipulate one's environment or to think abstractly as measured by objective criteria (such as tests)

g – general intelligence

In the words of 52 experts in cognition:

g is a very general mental capacity that, among other things, involves the ability to reason, plan, solve problems, think abstractly, comprehend complex ideas, learn quickly, and learn from experience. It is not merely book learning, a narrow academic skill, or test-taking smarts. It reflects a broader and deeper capacity for comprehending our surroundings – ‘catching-on’, ‘making sense’ of things, or ‘figuring out’ what to do.

Cattell's fluid and crystallized intelligence

- fluid intelligence (G_F) - higher mental abilities eg reasoning

prefrontal cortex

correlates most with performance IQ (PIQ) perceptual, processing speed

Ravens progressive matrices WAIS Block design

- crystallized intelligence (G_C) – knowledge acquired from culture, education, experience

cortical networks

correlates most with verbal IQ (VIQ) WAIS Info subtests

Executive functions (EF)

- multidimensional construct
- higher order processes that control and regulate thought and action, operate on lower level processes

in everyday life – planning, organizing, decision-making, flexibility, judgement, regulation of everyday behavior - all hallmarks of intelligence

inhibiting prepotent responses (inhibiting) h^2 ~90%

shifting mental states (shifting) h^2 ~ 76%

updating working memory (updating) h^2 ~ 100%

Friedman et al

(pilot study data)

WAIS ~ 70%

- would expect EF scores to correlate with IQ scores
- whilst measures of EFs intercorrelate, only ‘updating’ correlates highly with IQ (both fluid and crystallized, and Wechsler)
- current IQ tests do not assess all abilities required for ‘intelligent’ behaviors?

What does an estimate of 'g' tell us?

- it is the best psychological predictor of school achievement across all levels of schooling
- it is the best predictor of occupational success in jobs that require complex cognitive tasks
- it predicts income and success in every profession
- it may not tell us about other talents – physical, artistic
- distrusted by general public?
- older tests were culturally, socially biased
- not true for newer alternative tests:
    - information-processing methods
    - direct assessment of brain functioning (eg ERPs, fMRI)

Table 1 The Validity of Various Predictors of Job Performance

TECHNIQUE	VALIDITY
ABILITY COMPOSITE (Cognitive Ability Test Battery)	.53
JOB TRYOUT (Probationary Period)	.44
SITUATIONAL INTERVIEW (Structured/job related interview)	.37
REFERENCE CHECKS (Check with past employers)	.26
CLASS RANK OR GRADE POINT AVERAGE (Self-explanatory)	.21
AMOUNT OF EXPERIENCE (Years on the job)	.18
UNSTRUCTURED INTERVIEW (General discussion with applicant)	.14
TRAINING AND EXPERIENCE (Time spent in job/training)	.13
AMOUNT OF EDUCATION (Years in school)	.10

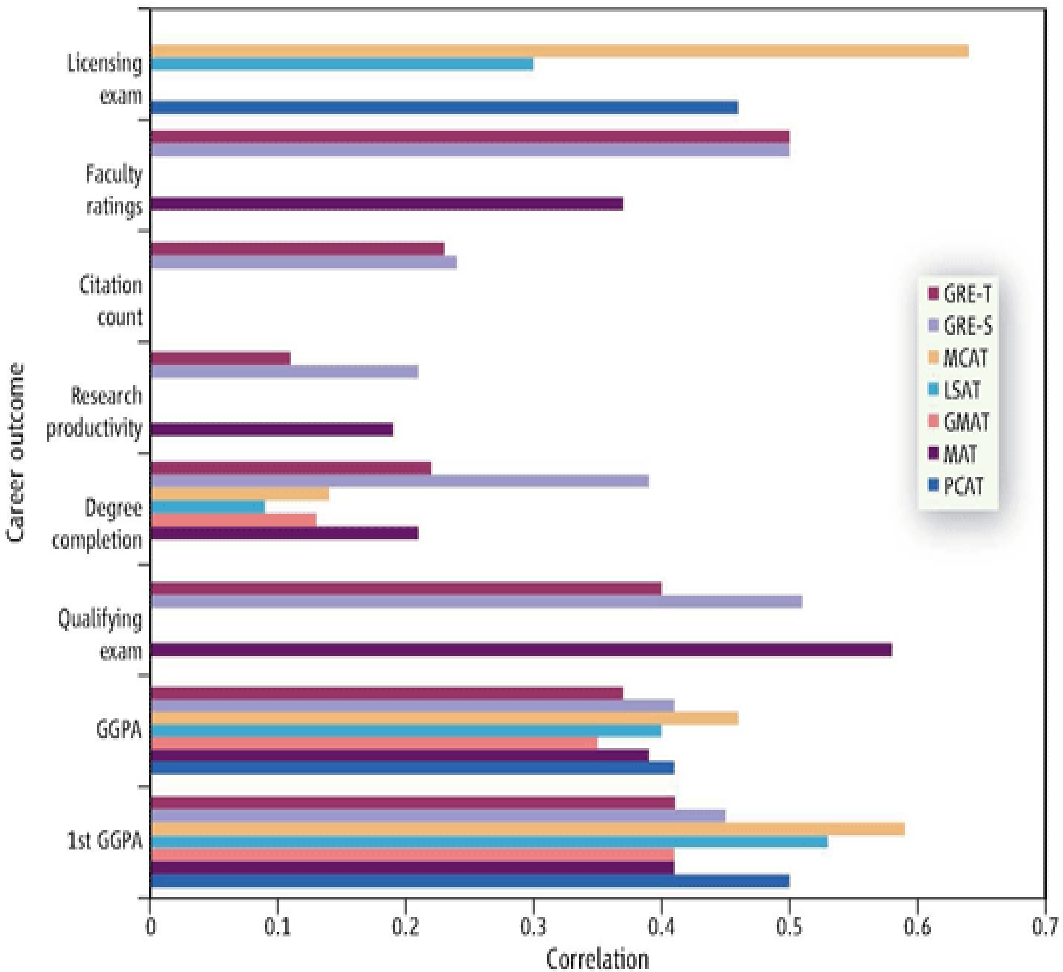
Hunter and Hunter, Michigan State University, (1984). American Psychological Association, 96 (1), 72-98.

- ***Economic and social correlates of IQ :***

Factors	Correlation
School grades and IQ	0.5
Total years of education and IQ	0.55
IQ and parental socioeconomic status	0.33
Job performance and IQ	0.54
Negative social outcomes and IQ	−0.2
IQs of identical twins	0.86
IQs of husband and wife	0.4
Heights of parent and child	0.47

- ***Economic and social correlates of IQ in the USA :***

IQ	<75	75–90	90–110	110–125	>125
US population distribution	5	20	50	20	5
Married by age 30	72	81	81	72	67
Out of labor force more than 1 month out of year (men)	22	19	15	14	10
Unemployed more than 1 month out of year (men)	12	10	7	7	2
Divorced in 5 years	21	22	23	15	9
% of children w/ IQ in bottom decile (mothers)	39	17	6	7	< 1
Had an illegitimate baby (mothers)	32	17	8	4	2
Lives in poverty	30	16	6	3	2
Ever incarcerated (men)	7	7	3	1	< 1
Chronic welfare recipient (mothers)	31	17	8	2	< 1
High school dropout	55	35	6	0.4	< 0.4
Values are the percentage of each IQ sub-population, among non-Hispanic whites only, fitting each descriptor. Compiled by Gottfredson (1997) from a US study by Herrnstein & Murray (1994) pp. 171, 158, 163, 174, 230, 180, 132, 194, 247–248, 194, 146 respectively.					



EDUCATIONFORUM

ASSESSMENT

Standardized Tests Predict Graduate Students' Success

Standardized admissions tests are valid predictors of many aspects of student success across academic and applied fields.

Nathan R. Kuncel[1] and Sarah A. Hezlett[2]

Accurately predicting which students are best suited for postbaccalaureate graduate school programs benefits the programs, the students, and society at large, because it allows education to be concentrated on those most likely to profit. Standardized tests are used to forecast which students will be the most successful and obtain the greatest benefit from graduate education in disciplines ranging from medicine to the humanities and from physics to law. However, controversy remains about whether such tests effectively predict performance in graduate school. Studies of standardized test scores and subsequent success in graduate school over the past 80 years have often suffered from limited sample size and present mixed conclusions of variable reliability.

Several meta-analyses have been conducted to extract more reliable conclusions about standardized tests from a variety of disciplines. To date, these review studies have been conducted on several tests commonly used in the United States: the Graduate Record Examination (GRE-T) (*1*), Graduate Record Examination Subject tests (GRE-S) (*1*), the Law School Admissions Test (LSAT) (*2–4*), the Pharmacy College Admissions Test (PCAT) (*5*), the Miller Analogies Test (MAT) (*6*), the Graduate Management Admissions Test (GMAT) (*7*), and the Medical College Admissions Test (MCAT) (*8*, *9*).

We collected and synthesized these studies. Four consistent findings emerged: (i) Standardized tests are effective predictors of performance in graduate school. (ii) Both tests and undergraduate grades predict important academic outcomes beyond grades earned in graduate school. (iii) Standardized admissions tests predict most measures of student success better than prior college academic records do (*1–5*, *7*, *8*). (iv) The combination of tests and grades yields the most accurate predictions of success (*1–4*, *7*, *8*).

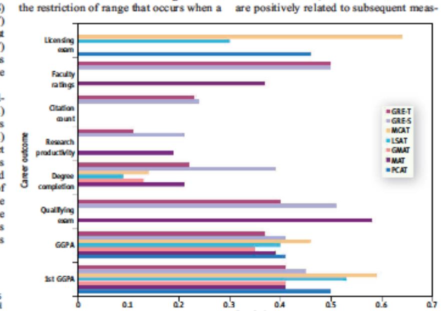
Structure of Admissions Tests

Most standardized tests assess some combination of verbal, quantitative, writing, and analytical reasoning skills or discipline-specific knowledge. This is no accident, as work in all fields requires some combination of the above. The tests aim to measure the most relevant skills and knowledge for mastering a particular discipline. Although the general verbal and quantitative scales are effective predictors of student success, the strongest predictors are tests with content specifically linked to the discipline (*1*, *5*).

Estimating Predictive Validity

The predictive validity of tests is typically evaluated with statistics that estimate the linear relationship between predictors and a measure of academic performance. Meta-analyses synthesizing primary studies of test validity aggregate Pearson correlations. In many primary studies, the correlations are weakened by statistical artifacts, thus contributing to misinterpretation of conclusions. The first attenuating factor is the restriction of range that occurs when a sample is selected on the basis of a predictor variable that has a nonzero correlation with an outcome measure (*10*). The second attenuating factor is unreliability in the success measure resulting from inconsistency in human judgment (*11*). Where possible, recognized corrections were used (*12*) to account for these artifacts.

Research has been conducted on the correlation between test scores and various measures of student success: first-year grade point average (GPA), graduate GPA, degree attainment, qualifying or comprehensive examination scores, research productivity, research citation counts, licensing examination performance, and faculty evaluations of students. These results are based on analyses of 3 to 1231 studies across 244 to 259,640 students. The programs represented include humanities, social sciences, biological sciences, physical sciences, mathematics, and professional graduate programs in management, law, pharmacy, and medicine. For all tests across all relevant success measures, standardized test scores are positively related to subsequent meas-



Tests as predictors. Standardized test scores correlate with student success in graduate school. See table S1 for detailed data.

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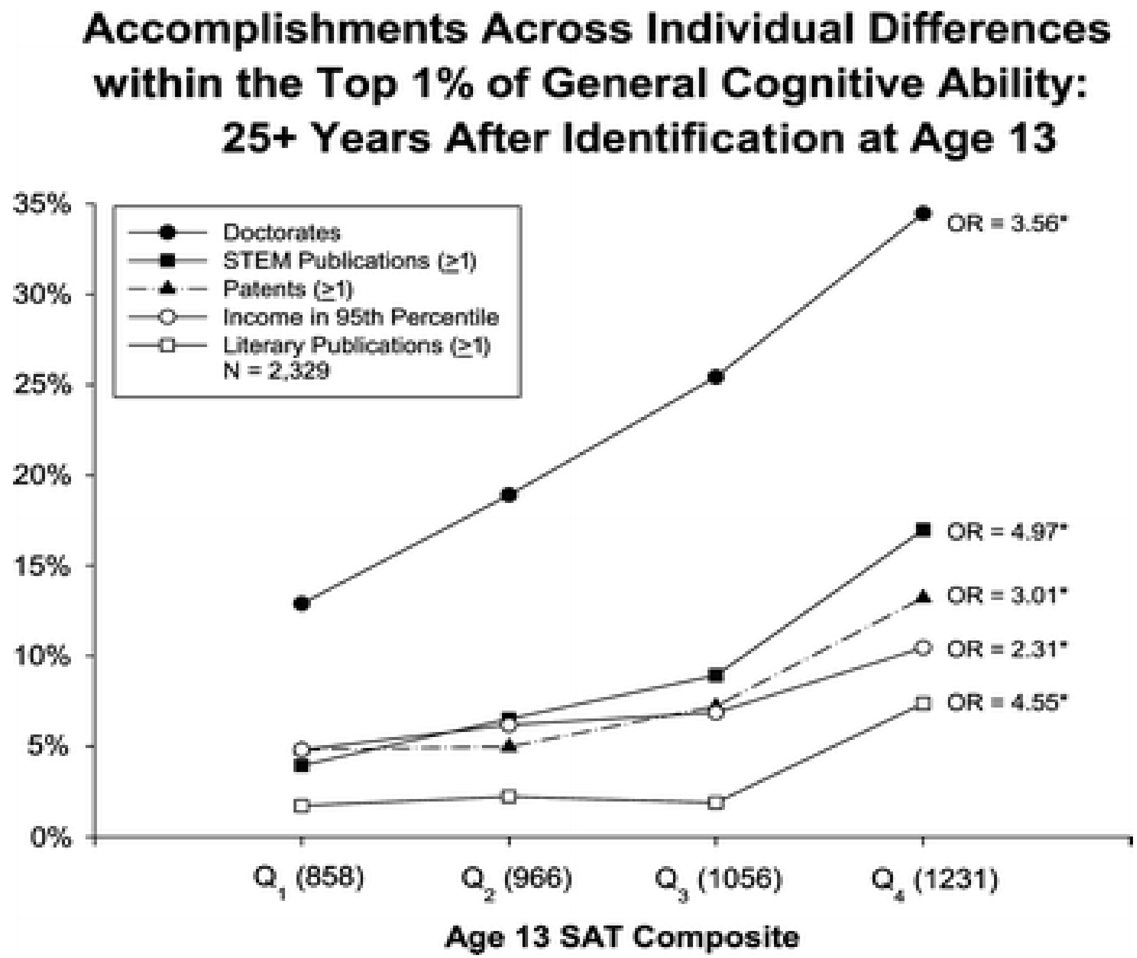

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Does Socioeconomic Status Explain the Relationship Between Admissions Tests and Post-Secondary Academic Performance?

Paul R. Sackett, Nathan R. Kuncel, Justin J. Arneson, Sara R. Cooper, and Shonna D. Waters
University of Minnesota, Twin Cities Campus

Critics of educational admissions tests assert that tests measure nothing more than socioeconomic status (SES) and that their apparent validity in predicting academic performance is an artifact of SES. The authors examined multiple large data sets containing data on admissions and related tests, SES, and grades showing that (a) SES is related to test scores ($r = .42$ among the population of SAT takers), (b) test scores are predictive of academic performance, and (c) statistically controlling for SES reduces the estimated test–grade correlation from $r = .47$ to $r = .44$. Thus, the vast majority of the test–academic performance relationship was independent of SES: The authors concluded that the test–grade relationship is not an artifact of common influences of SES on both test scores and grades.

Keywords: socioeconomic status, academic performance, admissions testing, test validity

Each year millions of individuals in the United States take post-secondary admissions tests (e.g., SAT [formerly the Scholastic Aptitude Test], the ACT [formerly, American College Testing], the Graduate Record Examination [GRE], the Law School Admission Test [LSAT], the Medical College Admission Test [MCAT], and the Graduate Management Admission Test [GMAT]. Given their prominent role in influencing educational opportunities, these tests are of great interest to the public and undergo considerable scrutiny. A common assertion among test critics is that test scores used for high-stakes decisions (e.g., college admission) measure nothing more than socioeconomic status (SES). Examples of this assertion, drawn from Zwick (2002), include the claim that "in the interest of truth in advertising, the SAT should simply be called a 'wealth test'" (Guiner, cited in Zwick, 2002), that "the SAT merely measures the size of students' houses" (Kohn, 2001), and that the "only thing the SAT predicts well now is socioeconomic status" (Colvin, 1997). Implicit in these criticisms is that socioeconomic status (SES) has an artificial and irrelevant effect on test scores: High SES leads to higher test scores (e.g., through knowledge of test-taking techniques) but not to higher true standing on the characteristic the test is intended to measure (i.e., developed abilities relevant to academic performance). This assertion can be paired with another one, namely, that SES has a similar artificial effect on academic performance measures (e.g., grading is biased in favor of high-SES students) and, thus, that the appearance of test validity (i.e., test–grade correlations) is also an artifact. If SES inflates both test scores and grades of high-SES students and deflates both test scores and grades of low-SES students, then a test that is, in fact, completely invalid as a predictor of academic performance will appear valid as a result of the common effects of SES on both test and grades.

Assertions that the appearance of test validity is an artifact of SES have also been prominently placed within the psychological literature. One claim is that "it has now been documented with massive data sets from the University of California that SAT I scores lose any ability to predict freshman year grades if the regression analyses control for socioeconomic status" (Crosby, Iyer, Clayton, & Downing, 2003). Similarly, "SAT scores used for college admission do not predict freshman year grades when socioeconomic status is controlled" (Biernat, 2003, p. 1023). The most visible critic of the SAT, former president of the University of California system Richard Atkinson (2005), stated that "after controlling for [SES]. . .the relationship between SAT I scores and UC [University of California] grades virtually disappears." Moving beyond the specific issue of SES and test validity, it is noteworthy that a task force commissioned by APA to examine SES and recommend directions for psychological research and practice has recently issued a report (Saegert et al., 2007). This task force affirmed the criticality of understanding the role of SES.

We concluded that a systematic exploration of the degree to which SES accounts for test–grade relationships was in order. Our goal was to summarize findings from data sets that permit the examination of three relationships: (a) the correlation between SES and scores on cognitively loaded tests, with primary focus on those

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The order among the latter three authors is alphabetical; all contributed equally to the project.

Justin J. Arneson is now at Target Corporation, Minneapolis, Minnesota, and Shonna D. Waters is now at the Human Resource Research Organization, Alexandria, Virginia.

This research was supported by a grant from The College Board to Paul R. Sackett and Nathan R. Kuncel. Paul R. Sackett serves on The College Board's SAT Psychometric Panel and the Educational Testing Service's Visiting Panel on Research. Nathan R. Kuncel serves on the Educational Testing Service's GRE (Graduate Record Exam) Technical Advisory Committee.

We thank Sarah Hezlett and Jana Rigdon for helpful comments on a draft of this article.

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IQ predicts many things but does belonging to a particular group predict IQ?

- evidence suggests some 40% of IQ differences in occupation & income in Western societies are associated with genetic differences

  Rowe et al (1998) Tambs et al (1989)

- based on WAIS, there is a 22 point difference between average IQs of persons in professional/technical jobs versus unskilled laborers

  Reynolds et al (1987)

BUT there was nearly as much variation in IQ within occupational groups as in US population as a whole

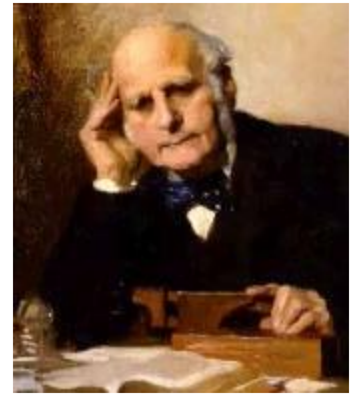
so, is membership of a particular group likely to predict IQ of an individual?

What else is important?

special talents motivation personality traits hard work privilege

Long history of research into cognitive ability:

Galton (1865) Sir Francis Galton (1865, 1869), Darwin's cousin, immediately recognized the implications for human variation. Galton carried out surveys and found that good and bad temperament, as well as intelligence, ran in families. He discovered the phenomenon of regression-to-the mean and the implication that family variation was heritable




NATURAL INHERITANCE

FRANCIS GALTON, F.R.S.

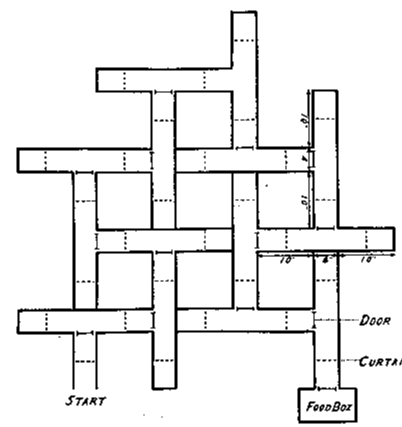
MACMILLAN AND CO.

Burks (1928) Barbara Stoddard Burks, “The Relative Influence of Nature and Nurture Upon Mental Development; A Comparative Study of Foster Parent-Foster child Resemblance and True Parent-True Child Resemblance,” *27th Yearbook of the NationalSocietyfor the Study of Education*, (1928)

Merriman (1924) twin methodology

Tolman (1924) selection for maze

Tryon learning in rats



Plan of maze
14-Unit T-Alley Maze

FIG. 1

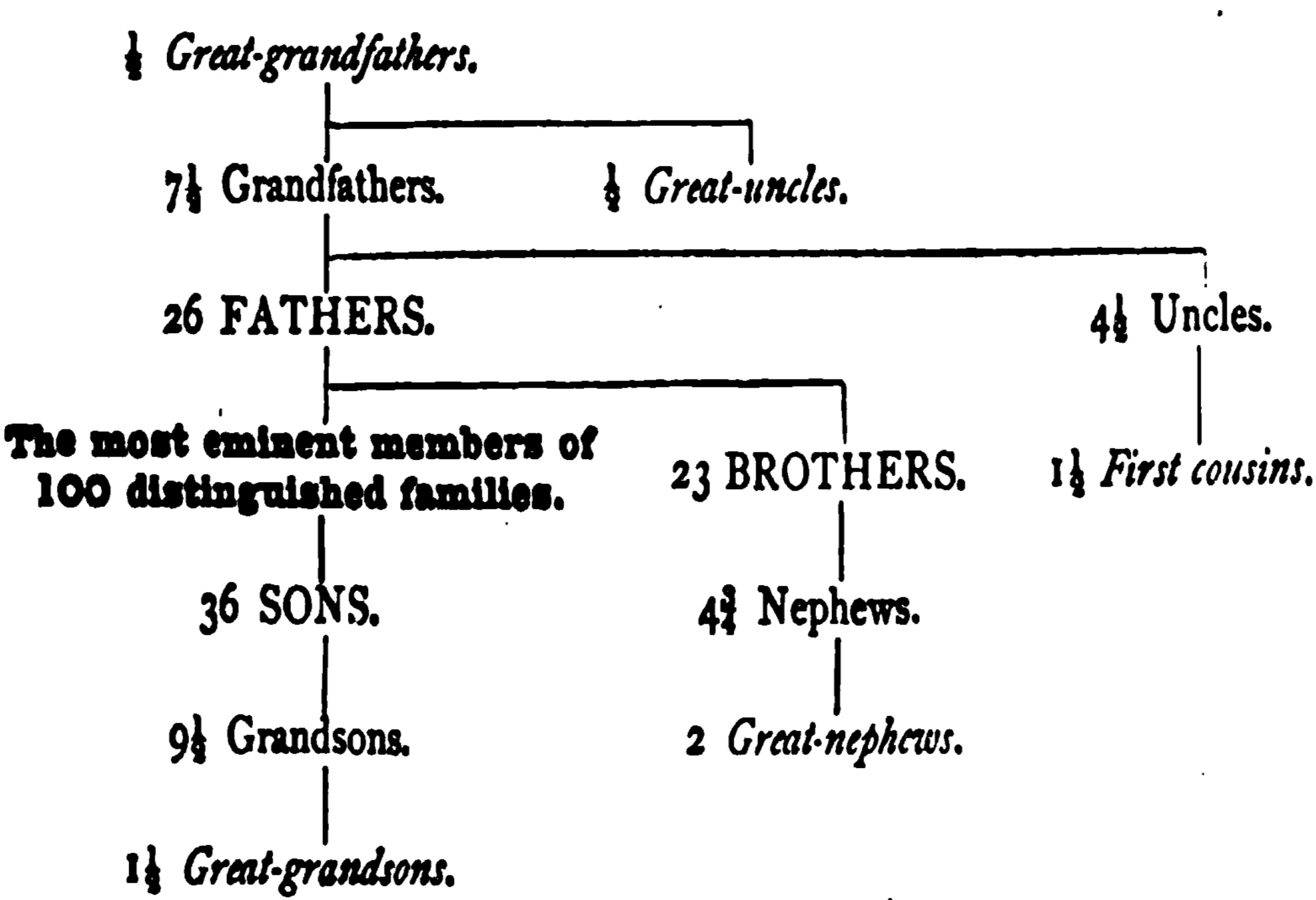
(From M. H. Elliott, The effect of change of reward on the maze performance of rats. *Univ. Calif. Publ. Psychol.*, 1928, 4, p. 20.)

Prevailing view, however , was that nurture was more important in human abilitites

see John B Watson 1925 “Give me a dozen healthy infants……… “

Galton (1869) Hereditary genius: An enquiry into its laws and consequences

PERCENTAGE OF EMINENT MEN IN EACH DEGREE OF KINSHIP TO THE MOST GIFTED MEMBER OF DISTINGUISHED FAMILIES.

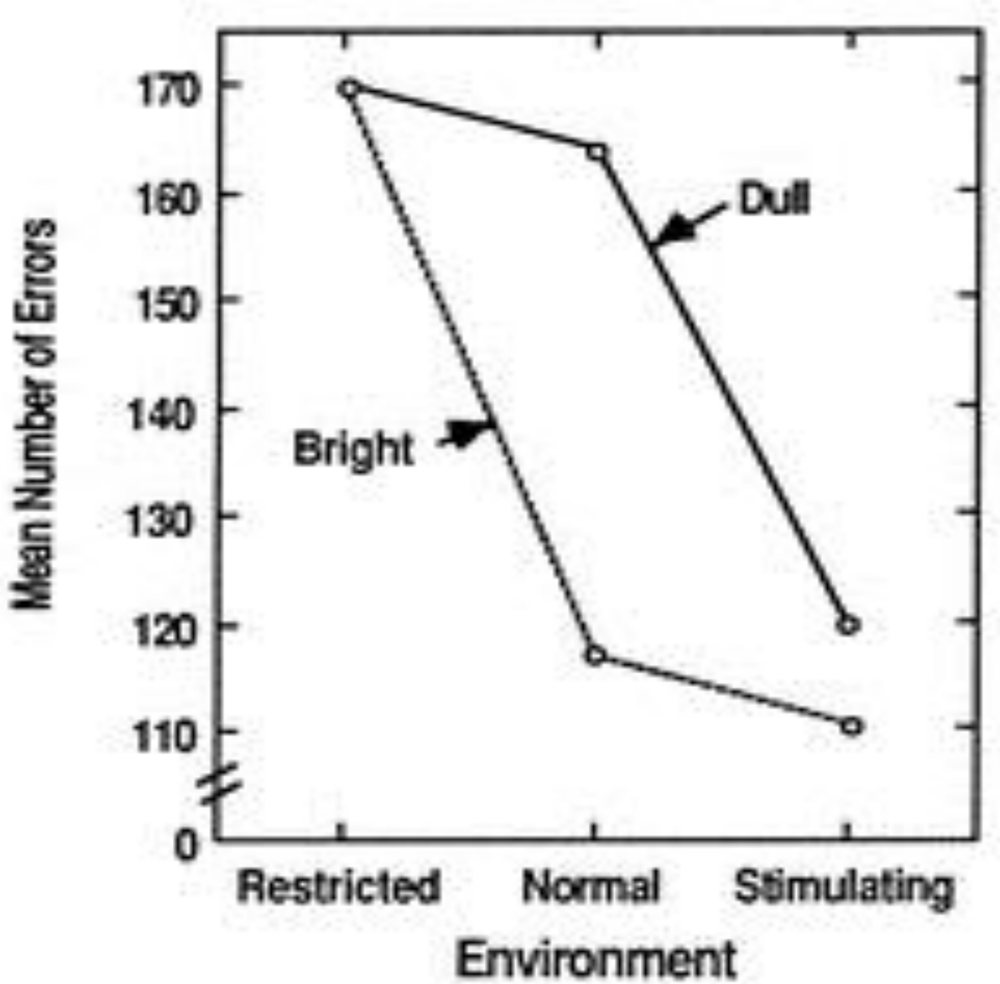


Recognition that genotype and environment can interact to determine phenotype

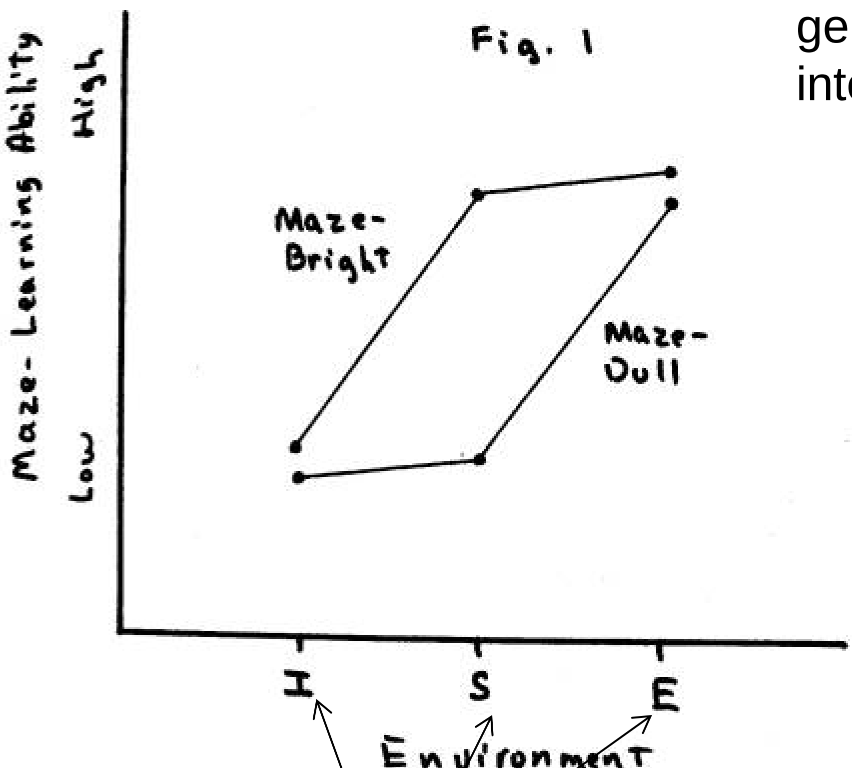
Cooper & Zubek (1958) tested maze dull and maze bright rats after rearing in different environments

genotype/environment interaction

– changes caused by environment depended on genotype of rats



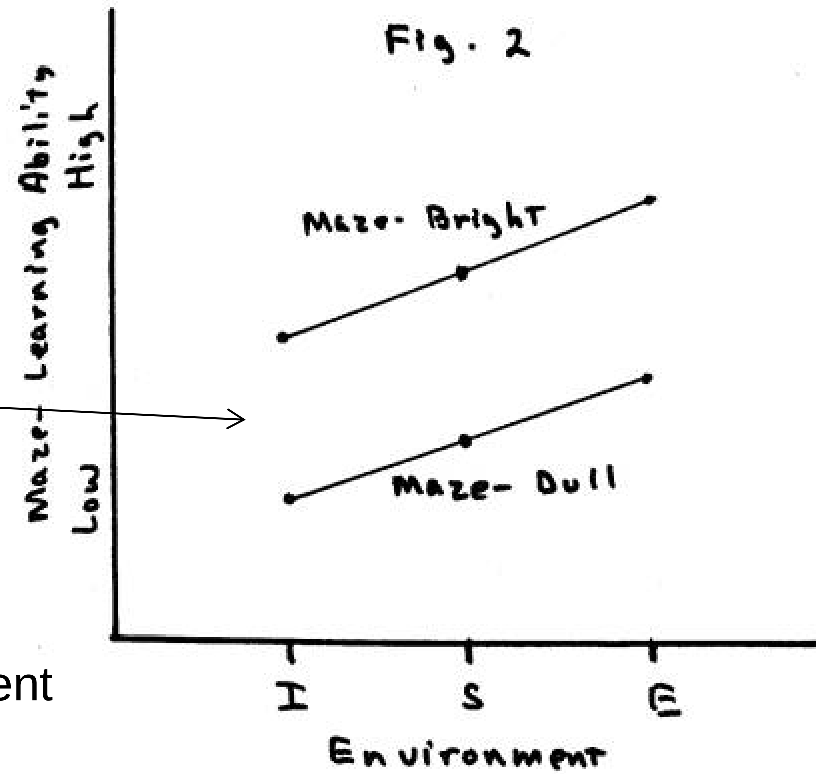
From Cooper and Zubek, 1958



genotype/environment
interaction

I = impoverished
S = standard
E = enriched

heritability?



no genotype/environment
interaction

Change in acceptance of genetic influence on cognitive ability in the 60's and 70's - the nature nurture debate

- to this time, some general acceptance of genetic influence on both animal and human cognition
- Infuriated those with strong belief in equality stemming from religious, political and philosophical roots

Typical psychology department in the 60's

reductionist theories – all behaviors could be traced to one basic single causative event **"intrapsychic conflicts of infancy"**

- all influences were entirely environmental

- individual differences were viewed as 'error'

Very unattractive connotations from the, then, recent political past

eugenics – idea that humanity can be improved by selective breeding

intelligence, aggression, antisocial behavior- all subject to eugenic practices in past

Bad science

Burt (UK) falsified data to enhance his results showing gene influence on g

Controversy

Jensen (US) published “How much can we boost IQ and scholastic achievement” (1969) in response to research showing poor results from compensatory education programs

Did lack of results reflect genetic influence ?

Also IQ is substantially heritable, different ethnic groups have different mean IQ levels.

Could the measurable differences between ethnic groups result from genetic differences?

- whole area of research thrown into acrimonious ‘debate’
- general conclusion in psychology departments was that a genetic influence on human cognition did not exist
- behavior geneticists said evidence showed otherwise

prevented when actinomycin D was added 4 days after the addition of T_2. (iii) Antibody formation was only partially suppressed when secondary immunization with T_2 was effected 4 days before the nodes were removed and cultivated with actinomycin D.

Ambrose and Coons (6) found that the secondary antibody response was also inhibited in vitro by low concentrations of actinomycin D; and Jerne has shown that actinomycin D injected into mice inhibits antibody formation in vivo (7).

The interpretation of our results depends upon the mechanism of action of actinomycin D on those cells involved in specific antibody formation to bacteriophage T_2. Since no information is available at present on this relatively small and possibly heterogeneous cell population (8) within the lymph node, a tentative explanation must rely on results obtained from studies of other systems. In this respect there has recently accumulated considerable evidence to indicate that, at the concentrations of actinomycin D used in this study ($5 \times 10^{-7}M$), cellular RNA synthesis is specifically inhibited, while DNA synthesis remains relatively unaffected in bacterial (9) liver (10) HeLa (11) and mouse L cells (12). The basis for this specificity has been elucidated recently by Kahan *et al.* (13) who have shown that actinomycin D binds specifically to the deoxyguanosine residue of native DNA, but has a poor affinity for denatured DNA. Actinomycin D also blocks the protein synthesis initiated in vitro by T_2 DNA and RNA polymerase (14). In this system, the effect of actinomycin is probably due to the prevention of messenger RNA (mRNA) formation, since protein synthesis is not inhibited if a messenger such as polyU, G (uridylate, guanilate) is added.

Our results suggest, therefore, that antibody formation depends upon a DNA-dependent RNA synthesis, and, in particular, upon mRNA formation. The prompt and complete inhibition of already established antibody synthesis by actinomycin D is also consistent with an effect on messenger RNA rather than on the other, more stable classes of cytoplasmic RNA, and suggests that this messenger has a half-life of less than several days. This explanation leaves unanswered, however, the crucial question of whether or not the messenger carries information for immunological specificity. In addition, our data do not exclude the possibility that actinomycin D has damaged antibody-producing cells, possibly by interfering with cell division (12). We are therefore trying to determine whether mRNA, synthesized in vitro by DNA obtained from lymphoid cells of hyperimmunized animals, and RNA polymerase, can stimulate specific antibody formation by unimmunized lymphoid cells (15).

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15. Research aided by U.S. Public Health Service grant AI-01821-07, conducted in part under the sponsorship of the Commission on Immunization of the Armed Forces Epidemiological Board, and supported in part by the Office of the Surgeon General, Department of the Army, Washington, D.C.

19 July 1963

Genetics and Intelligence: A Review

Abstract. *A survey of the literature of the past 50 years reveals remarkable consistency in the accumulated data relating mental functioning to genetic potentials. Intragroup resemblance in intellectual abilities increases in proportion to the degree of genetic relationship.*

Nomothetic psychological theories have been distinguished by the tendency to disregard the individual variability which is characteristic of all behavior. A parallel between genetic individuality and psychologic individuality has rarely been drawn because the usual assumption has been, as recently noted in these pages (1), that the organisms intervening between stimulus and response are equivalent "black boxes," which react in uniform ways to given stimuli.

While behavior theory and its analytic methods as yet make few provisions for modern genetic concepts, the literature contains more information than is generally realized about the relationship between genotypic similarity and similarity of performance on mental tests. In a search for order among the published data on intellectual ability, we have recently summarized the work of the past half century (2). By using the most commonly reported statistical measure, namely, the correlation coefficient, it has been possible to assemble comparative figures from the majority of the investigations.

Certain studies giving correlations had to be excluded from this compilation for one of the following reasons: (i) type of test used (for example, achievement tests, scholastic performance, or subjective rating of intelligence); (ii) type of subject used (for example, mental defectives); (iii) inadequate information about zygosity diagnosis in twin studies (3); (iv) reports on too few special twin pairs.

The 52 studies (2) remaining after these exclusions yield over 30,000 correlational pairings (4) for the genetic relationship categories shown in Fig. 1. The data, in aggregate, provide a broad basis for the comparison of genotypic and phenotypic correlations. Considering only *ranges* of the observed measures, a marked trend is seen toward an increasing degree of intellectual resemblance in direct proportion to an increasing degree of genetic relationship, regardless of environmental communality.

Furthermore, for most relationship categories, the *median* of the empirical correlations closely approaches the theoretical value predicted on the basis of genetic relationship alone. The average genetic correlation between parent and

Why did this view not last long?

good empirical studies – large sample sizes
quantitative measures

well-designed to separate genetic and environmental influences

From

Kamin (1974): "… little or no evidence that intelligence is a heritable trait."

To

Brody (1990) "… it is inconceivable.. that any responsible scholar could.. take this position"

Currently, g is

- one of the most reliable, valid measures in behavioral science
- stable - it's long-term stability after childhood is greater than the stability of any other behavioral trait
- widely accepted as a valuable concept
- substantially heritable

Molecular Psychiatry (2011), 1–10

www.nature.com/mp

IMMEDIATE COMMUNICATION

Genome-wide association studies establish that human intelligence is highly heritable and polygenic

G Davies[1], A Tenesa[2,3], A Payton[4], J Yang[5], SE Harris[6,7], D Liewald[1,7], X Ke[8], S Le Hellard[9], A Christoforou[9], M Luciano[1,7], K McGhee[1], L Lopez[1,7], AJ Gow[1,7], J Corley[1], P Redmond[1], HC Fox[10], P Haggarty[11], LJ Whalley[10], G McNeill[10], ME Goddard[12,13], T Espeseth[14], AJ Lundervold[15], I Reinvang[14], A Pickles[16], VM Steen[9,17], W Ollier[4], DJ Porteous[6,7], M Horan[18], JM Starr[7,19], N Pendleton[18], PM Visscher[5,7,20] and IJ Deary[1,7,20]

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General intelligence is an important human quantitative trait that accounts for much of the variation in diverse cognitive abilities. Individual differences in intelligence are strongly associated with many important life outcomes, including educational and occupational attainments, income, health and lifespan. Data from twin and family studies are consistent with a high heritability of intelligence, but this inference has been controversial. We conducted a genome-wide analysis of 3511 unrelated adults with data on 549 692 single nucleotide polymorphisms (SNPs) and detailed phenotypes on cognitive traits. We estimate that 40% of the variation in crystallized-type intelligence and 51% of the variation in fluid-type intelligence between individuals is accounted for by linkage disequilibrium between genotyped common SNP markers and unknown causal variants. These estimates provide lower bounds for the narrow-sense heritability of the traits. We partitioned genetic variation on individual chromosomes and found that, on average, longer chromosomes explain more variation. Finally, using just SNP data we predicted ~1% of the variance of crystallized and fluid cognitive phenotypes in an independent sample (*P*= 0.009 and 0.028, respectively). Our results unequivocally confirm that a substantial proportion of individual differences in human intelligence is due to genetic variation, and are consistent with many genes of small effects underlying the additive genetic influences on intelligence.
Molecular Psychiatry advance online publication, 9 August 2011; doi:10.1038/mp.2011.85

Keywords: genetics; GWAS; intelligence; quantitative trait

Introduction

People differ in their cognitive abilities, and the origins and impacts of these differences are sought after and much debated. The quantitative trait of general intelligence reflects the fact that diverse cognitive abilities show universally positive covariation; that is, no matter the cognitive task being undertaken, much of the human variation in any

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ORIGINAL ARTICLE

A genome-wide survey and functional brain imaging study identify *CTNNBL1* as a memory-related gene

A Papassotiropoulos[1,2,3], E Stefanova[4], C Vogler[1], L Gschwind[1], S Ackermann[1], K Spalek[5], B Rasch[1,5], A Heck[1], A Aerni[2,5], E Hanser[1,3], P Demougin[1,3], K-D Huynh[1,3], R Luechinger[6], M Klarhöfer[7], I Novakovic[8], V Kostic[4], P Boesiger[6], K Scheffler[9,10] and DJ-F de Quervain[2,5]

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Unbiased genome-wide screens combined with imaging data on brain function may identify novel molecular pathways related to human cognition. Here we performed a dense genome-wide screen to identify episodic memory-related gene variants. A genomic locus encoding the brain-expressed beta-catenin-like protein 1 (*CTNNBL1*) was significantly ($P = 7 \times 10^{-8}$) associated with verbal memory performance in a cognitively healthy cohort from Switzerland ($n = 1073$) and was replicated in a second cohort from Serbia ($n = 524$; $P = 0.003$). Gene expression studies showed *CTNNBL1* genotype-dependent differences in beta-catenin-like protein 1 mRNA levels in the human cortex. Functional magnetic resonance imaging in 322 subjects detected *CTNNBL1* genotype-dependent differences in memory-related brain activations. Converging evidence from independent experiments and different methodological approaches suggests a role for *CTNNBL1* in human memory.
Molecular Psychiatry (2013) 18, 255–263; doi:10.1038/mp.2011.148; published online 22 November 2011

Keywords: beta-catenin-like; fMRI; GWAS; memory

Introduction

Searching for molecules related to human cognition is instrumental for understanding the biological mechanisms related to such complex traits as memory capacity and for identifying pathways possibly amenable to pharmacological interventions. Recent advances in the development of high-density genotyping platforms allow for high-resolution genome-wide association studies (GWAS) of genetically complex traits and have already led to a substantial increase in knowledge of the genetic underpinnings of physiological and pathological conditions of human cognition.[1] Such studies can be performed using both pooled and individual DNA samples.[2] Pooled GWAS followed by individual genotyping of the most significant variants of the DNA pools are a cost-effective way to perform genome-wide surveys in large cohorts. Although pooled GWAS may be a worthwhile and fast approach as a preliminary screen, they lack the ability of retrospectively stratifying the genotyped pooled cohort by secondary phenotypic traits and control variables.[2]

Here we performed a GWAS in individual DNA samples in a homogenous cohort of Swiss healthy young adults ($n = 1198$) assessed for verbal episodic memory performance, as quantified by an unexpected delayed free-recall test of 30 previously learned words (see Materials and methods). Single-nucleotide polymorphisms (SNPs) surpassing genome-wide correction for multiple comparisons were analyzed in an independent sample of healthy young adults from Serbia ($n = 524$), who were also characterized for verbal episodic memory performance. To further validate the genetic findings of the behavioral studies we analyzed data from studies on gene expression in human post-mortem brain tissue and also used functional magnetic resonance imaging (fMRI), which can detect genotype-dependent differences in brain

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Hierarchical Genetic Organization of Human Cortical Surface Area

Chen et al
30 MARCH 2012 VOL 335 SCIENCE www.sciencemag.org

hierarchy demonstrated a biologically sensible organizational structure of the human brain.

We described a previously unidentified parcellation system for the human cortex that reflects shared genetic influences on cortical areal expansion. This system constitutes the first human brain atlas based solely on genetically informative data, which may provide presently undescribed phenotypes that will have greater statistical power for genome-wide genetic association studies in comparison with traditional cortical parcellations. We found evidence for a hierarchical, modular, and bilaterally symmetric genetic architecture. Genetically based lobar regions have been demonstrated across mammalian species (*7*, *8*), and our results are consistent with genetically based regions of human specialization being increasingly differentiated subdivisions of these lobar regions. Our findings may thus be useful for translating results from model organisms into functional and clinical insights about human specializations, so as to understand both order and disorder in the human brain.

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Supporting Online Material
www.sciencemag.org/cgi/content/full/335/6076/1634/DC1
Materials and Methods
Figs. S1 to S9
Table S1
References

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Ecological Context Influences Epidemic Size and Parasite-Driven Evolution

Meghan A. Duffy,[1]* Jessica Housley Ochs,[1] Rachel M. Penczykowski,[1] David J. Civitello,[2] Christopher A. Klausmeier,[3] Spencer R. Hall[2]

The occurrence and magnitude of disease outbreaks can strongly influence host evolution. In particular, when hosts face a resistance-fecundity trade-off, they might evolve increased resistance to infection during larger epidemics but increased susceptibility during smaller ones. We tested this theoretical prediction by using a zooplankton-yeast host-parasite system in which ecological factors determine epidemic size. Lakes with high productivity and low predation pressure had large yeast epidemics; during these outbreaks, hosts became more resistant to infection. However, with low productivity and high predation, epidemics remained small and hosts evolved increased susceptibility. Thus, by modulating disease outbreaks, ecological context (productivity and predation) shaped host evolution during epidemics. Consequently, anthropogenic alteration of productivity and predation might strongly influence both ecological and evolutionary outcomes of disease.

Parasites can impose strong evolutionary pressure on their hosts during epidemics (*1*, *2*). Parasites often virulently depress survival and/or birth rate of their hosts. As a result, if epidemics become large enough, host populations might evolve resistance to infection because of parasite-mediated directional selection (*1*). Alternatively, if the susceptibility of a host genotype depends on the parasite genotype to which it is exposed, negative frequency-dependent selection can drive cycling of host genotypes through time [that is, "Red Queen dynamics" (*3*, *4*)]. These two ideas about host (co-)evolution during epidemics, evolution of increased resistance and the Red Queen hypothesis, dominate research on evolutionary epidemiology (*1*). However, theory reveals other possibilities, including the evolution of higher susceptibility to infection (*1*, *5*–*8*). Why would hosts evolve greater susceptibility to their virulent parasites during epidemics? When would host populations evolve this way in nature?

The answers to these questions involve trade-offs and ecologically driven variation in disease prevalence. Resistance to virulent parasites can trade off with reproduction; some genotypes have higher fecundity but lower disease resistance, whereas others are less fecund but more resistant. The fittest strategy, then, depends on the net balance between resisting infection and enhancing fecundity. That balance, in turn, depends on ecologically determined disease prevalence. Environments with high resources for hosts (higher productivity) and lower mortality (lower predation) on hosts should fuel large epidemics (*9*–*12*). In these systems, theory predicts that hosts should evolve increased resistance to disease, even though resistant genotypes have lower fecundity. However, when low productivity and/or higher predation constrain epidemic size, populations should become more susceptible because more susceptible genotypes are more fecund.

We test these predictions in a host-parasite system that exhibits the requisite trade-offs and ecologically driven variation in epidemics. Clonal genotypes of the zooplankton grazer *Daphnia dentifera* face a trade-off between fecundity and resistance to infection by a virulent yeast parasite [*Metschnikowia bicuspidata* (*13*)]. Mechanistically, the resistance-fecundity trade-off is driven by variation in feeding rate: Slow feeders consume fewer free-living propagules (spores) of the yeast (conferring higher resistance) but assimilate energy less quickly (yielding fewer offspring). Neither host-parasite genotype specificity nor Red Queen dynamics appear in this system; host resistance does not depend on the parasite genotype to which it is exposed (*14*). This parasite reduces fecundity and survival (*15*). Epidemics erupt commonly in *Daphnia* populations, with maximal infection prevalence sometimes exceeding 60% (*16*, *17*).

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Current problems

– convincing people environment is still important, countering fatalistic views

-countering new forms of eugenics :
changing genes perceived as ‘bad’
preventing birth of those with ‘bad’ genes

-misuse of information and unfair discrimination
genetic testing, insurance, employment

-IQ and gender/race still cannot be researched or even discussed
ask Larry Summers (ex-president of Harvard)
James Watson (ex-chancellor of Cold Spring Harbor Labs)

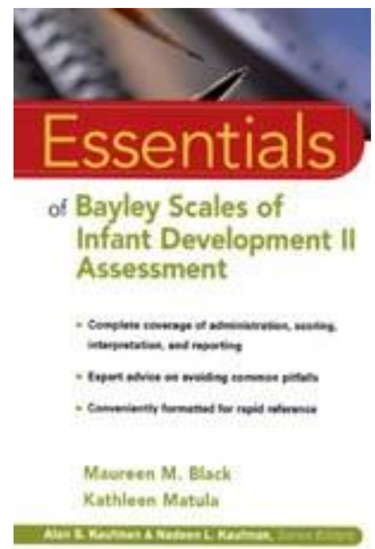
Commonly-used tests of cognitive ability

WISC – Wechsler intelligence scales
measurement error $\pm$ 5 points (score 70, range=65-75)

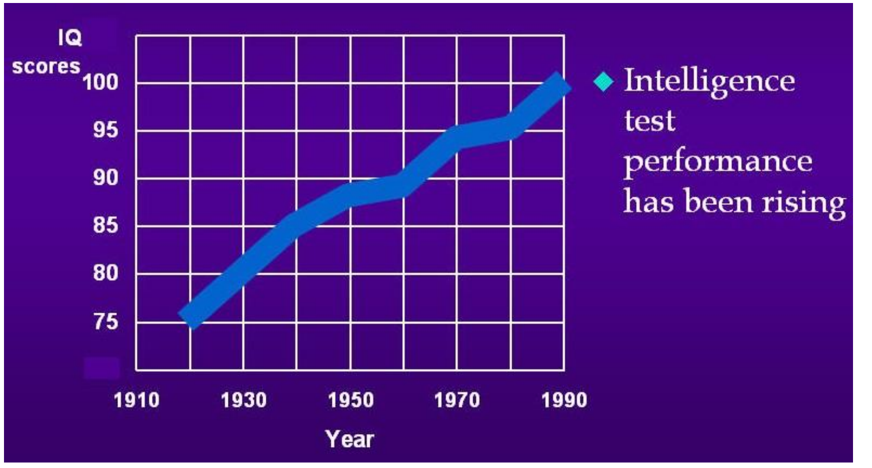
WAIS - Wechsler Adult intelligence scales

Stanford-Binet

Bayley Scales of Infant Development



Flynn effect



- average IQ has steadily been rising since measurement began

UK 27 point increase

US 24 point increase since WWII

- shown as overall increase in population mean
- due to environment that we all share (cultural environment)
- intelligence tests have to be re-normed periodically

Possible reasons for Flynn effect?

our genes have not changed

shows importance of environment

nutrition outbreeding on global scale (population admixture)

better test-taking skills better education for more people

widespread access to information via TV internet travel

huge increase in information processed (av word ‘consumption’ = 100,000 - a 350% increase from 1980’s)

Summary of evidence for influence of genes on cognitive ability

Bouchard & McGue (1981)

- summary of results from many studies

what is heritability here?

Adoption studies

Reared apart P/O, sibs r = 0.24

heritability = 0.48

- about half variation in scores is due to variation in genes

what is heritability here?

Twin studies

Reared together MZ r = 0.86

DZ r = 0.60

- test/retest reliability = 0.8-0.9 MZs are as similar as same person tested twice
- evidence for shared environment
- heritability = 2(MZr – DZr) = 0.52
- agrees with results from adoption studies

Note: most of the data for these studies came from samples where offspring were late adolescent or younger

Adopted apart twins

- MZ r = 0.67 - .79 = heritability
- much higher than estimates from family, twin , adoption studies in general

- assessed at later age

Similar data from other parts of world not included in Bouchard& McGue

- Russia
- E. Germany
- rural India urban India
- Japan

and from information-processing tests

Does ‘general intelligence’ exist ? - evidence for ‘g’

- meta-analysis of results from 322 studies of cognitive ability
- in spite of hundreds of different tests being used, average correlation among tests was 0.30

diverse cognitive processes do intercorrelate

- no-one has been able to devise a test where scores do NOT correlate with other test scores

- a common factor (‘g’) accounts for ~40% of total variance on cognitive tasks

g shows substantial heritability

- more studies on g than any other human characteristic

80,000 parent/offspring pairs

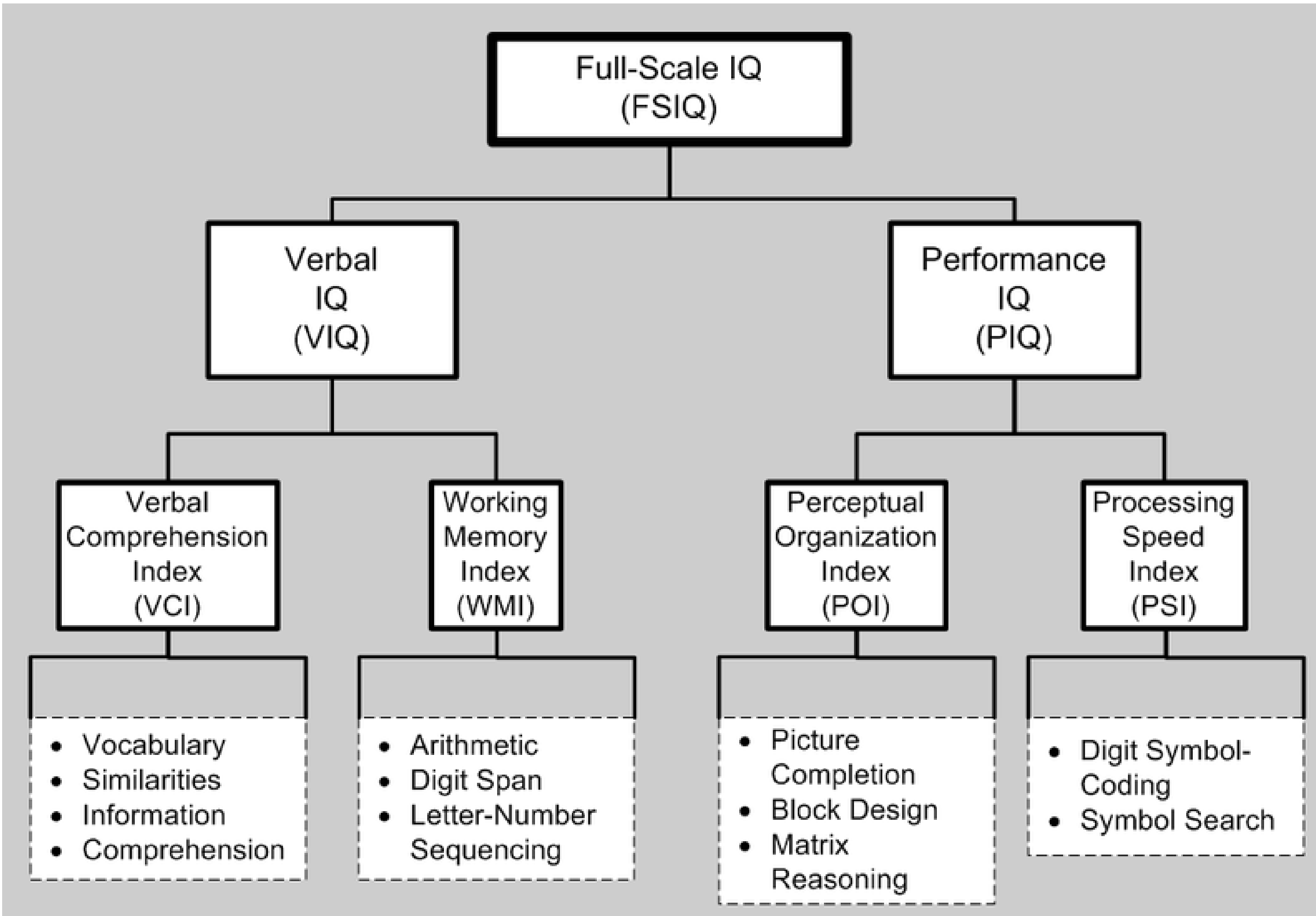
25,000 sib pairs

10,000 twin pairs + adoptive family data

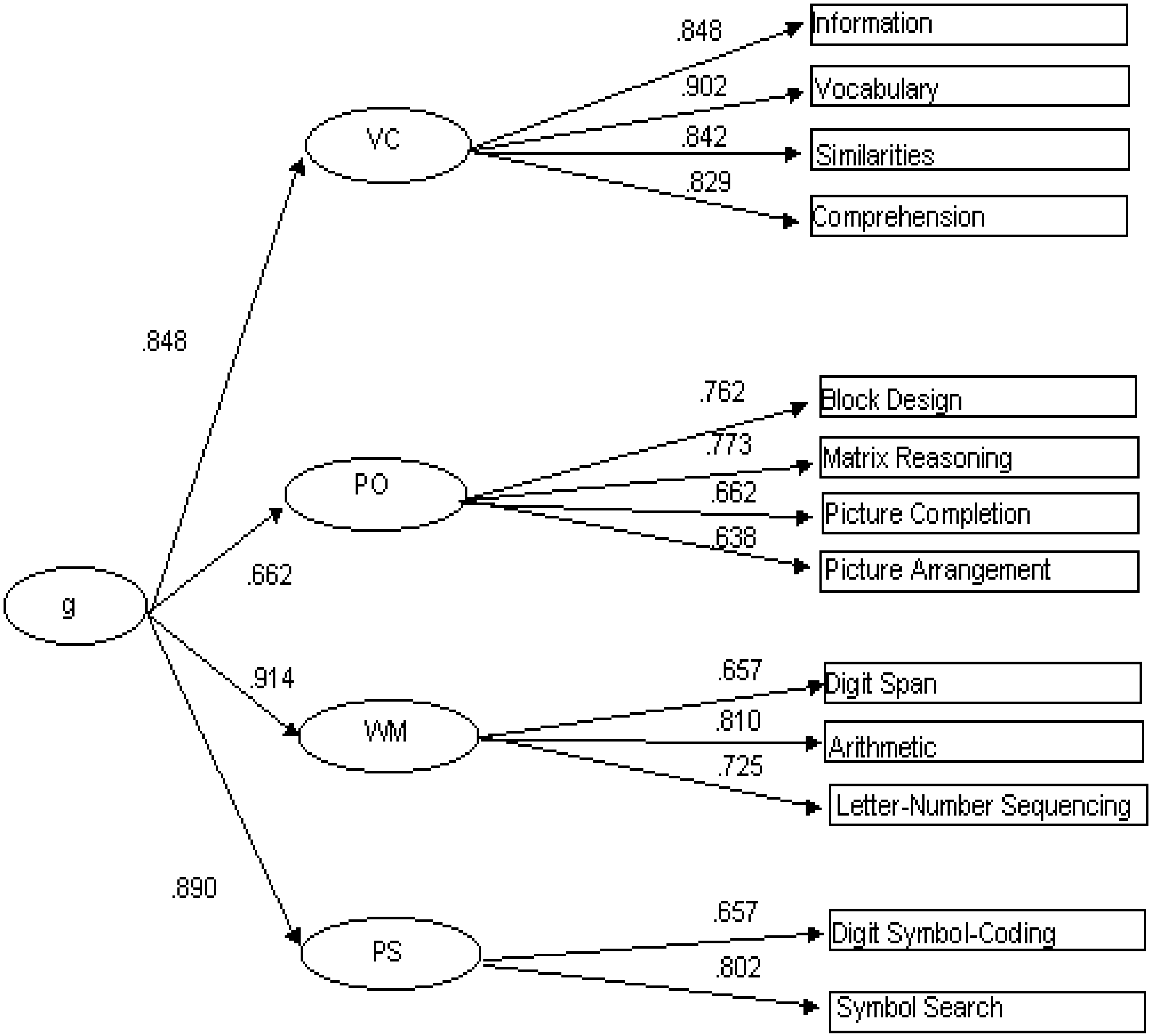
Lowest, most conservative estimate of **heritability = 50% for g**
mostly additive gene effects

genetic correlations across tests indicate extent to which same genes influence different specific abilities (evidence for pleiotropy)

Wechsler adult intelligence scale (WAIS)



g from the WAIS-III



non-standardized partial regression coefficients (path coefficients) shown

Examples of intercorrelation between specific abilities

Mathematics ability Plomin et al (2004)

- many studies indicate high heritability

phenotypic correlations with g score and other cognitive measures at age 7:

reading and math scores	r = 0.70
math and g scores	r = 0.43
reading and g scores	r = 0.47

'Generalist' genes:

genetic correlations of 0.62 to 0.76 found – indicating shared gene influences across these specific abilities

Genes for specific abilities:

evidence for specific genes for math and reading also since not all genetic variation for trait accounted for

Mathematics numeracy measures Plomin, 2012

- Age 12 ~3000 twin pairs, similar number of unrelateds
- twin analysis and GCTA DNA analysis used to estimate genetic correlations across different measures of numeracy

twin analysis: average genetic correlation across measures = .93

DNA analysis: average = .98 across measures

- substantial pleiotropy on behalf of genes influencing numeracy
- important theoretically in neuroscience

Average heritability = 0.46 from twin studies

Environmental influences

- heritability of 50% indicates the environment also accounts for 50% of the variation

- adoptive family data indicates that shared environment is important during development:

  P/adopted child r = 0.19

  Adoptive sibs r = 0.32

  both give estimates of c^2

- family and twin data indicate that non-shared environment is less important and accounts for less than 20% of variance

  MZ twins r = 0.86 14% of variance is e^2

Shared environment

- relationship is non-linear (not everyone is influenced by their environment in the same way), likely to be genotype x environment interaction
- interaction with socioeconomic status (SES):

Turkheimer et al (2001) 350 MZ and DZ twin pairs

middle-class environments – most variation is due to genes and e^2

poor environments – most variation is accounted for by c^2

Rowe et al (1999) ADD health study - a national longitudinal study of adolescent health

different heritabilities with different levels of education of parents

Genetic relatedness	Verbal IQ correlations by level of parental education: Low education	High education
High (MZ)	0.55	0.75
Moderate (DZ, sibs)	0.33	0.37
Low (half-sibs, cousins in SAME house)	0.32	0.10
average	h^2 = 26%	h^2 = 74%
	more c^2 e^2	no c^2 less e^2

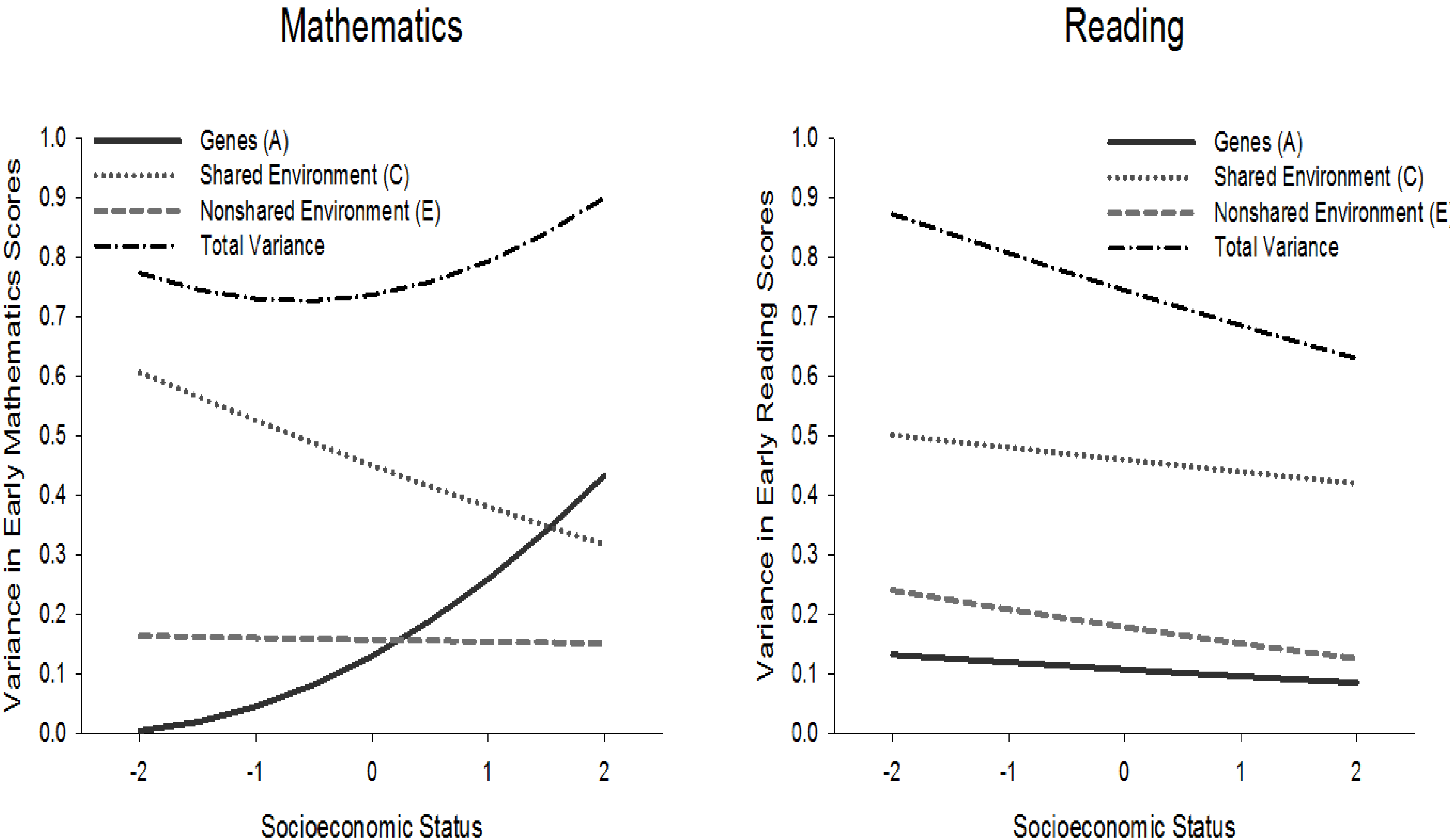
SES affects cognitive skills before entry into school

- school readiness (esp.math, reading skills) predicts achievement throughout school years
- child’s genes may help determine response to environment (gxe)

Rhemtulla, Tucker-Drob (2012) Behavior Genetics

longitudinal study of preschool children

assessed age 2 and age 4 so far



Amounts of unstandardized variance in early mathematics skill (left), and early reading skill (right) accounted for by genes (A), the shared environment (C), and the nonshared environment (E), as functions of SES. Total variance reflects the sum of the variance accounted for by A, C, and E, as a function of SES.

SES, Early Mathematics Scores, and Early Readings Scores were *z-transformed prior to analyses*

Sample of 4 year olds

Why? several theories put forward:

1. threshold effect (Scarr) - a ‘good enough’ environment is important in achieving genetic potential, rest doesn’t matter

2. more effective gene expression in good environments, poor environments ‘trap’ the individual (Bronfenbrenner & Ceci, Raine)

“proximal processes” – quality of reciprocal interactions between child and older individuals in environment

high quality - genetic potential reached, h^2 rises, better cognitive functioning

low quality – persisting disadvantage and/or recurring disadvantage leads to lower h^2, more shared e, lower cognitive functioning

3. environment is more variable in low SES groups and accounts for more variation (Turkheimer, Rowe)

What might be the effect of positive educational intervention on achievement?

Which simple statistics would be useful in measuring changes?

What would you expect to be the effects of the following on these statistics?

- no change in achievement

  no change in mean and variance or heritability

- everyone improves and scores become more similar

  higher mean lower variance lower heritability

- everyone improves, same spread of individual differences

  higher mean no change in variance or heritability

- everyone improves, those with higher abilities improve more, those with lower ability improve less

  higher mean higher variance higher heritability

"when the 'have nots' gain but the 'haves' gain even more" (Ceci & Papierno, 2005, Am Psychol 60:149-160)

However, eventual outcomes rely on : learning potential (ability)

+ learning achievement (knowledge) ambition commitment opportunity

Assortative mating

- non-random mating
- positive (assortative mating) – like chooses like
- negative (disassortative mating)– opposites attract
- effects are generally small, usually positive

Correlations between partners:

height r = 0.25 weight r = 0.20

personality measures r = 0.10 - 0.20

but, for g r = 0.40 between partners

- most mate selection is on basis of educational background

between partners r = 0.60 for educational background

r = 0.60 between g and educational background

Effects of assortative mating

- decreases variation within families

- if unaccounted for, could lead to overestimated h^2 and c^2 from family studies by

  increasing correlations within family

- leads to underestimated h^2 from twin studies because it does not effect MZ twins but increases DZ correlation
  - effects of assortative mating seen as shared e

- increases population variation
  effects accumulate over generations

Random mating (or disassortative mating)		Assortative mating
Parents		Parents
higher IQ x lower IQ		higher IQ x higher IQ
↓		↓
average IQ	children	higher IQ
lower IQ x higher IQ		lower IQ x lower IQ
↓		↓
average IQ	children	lower IQ

- effects of assortative mating should be factored out of data before estimates of variance components are obtained

Non-additive gene effects epistasis dominance

- in twin and family data, non-additive gene effects will be masked by effects of assortative mating and shared environment:

shared environment – increases all correlations

assortative mating – increases all correlations except MZ twin

non-additive gene effects – decrease all correlations except MZ twin

If higher cognitive ability was related to higher fitness, would expect to find dominance for alleles for higher IQ levels

If alleles for higher cognitive ability were dominant, would expect to find a depression of scores on inbreeding

inbreeding depression

- hence, can find indirect evidence for non-additive gene influence by looking for inbreeding depression

Inbreeding and IQ scores

- Bashi (1977) + several studies since

  Raven's matrices test

Degree of consanguinity	Grade 4		Grade 6	
	n	mean	n	mean
Children of unrelated	1054	8.8	1054	13.1
Children of first cousins	503	8.6	467	12.3
Children of double first cousins	71	7.9	54	10.6

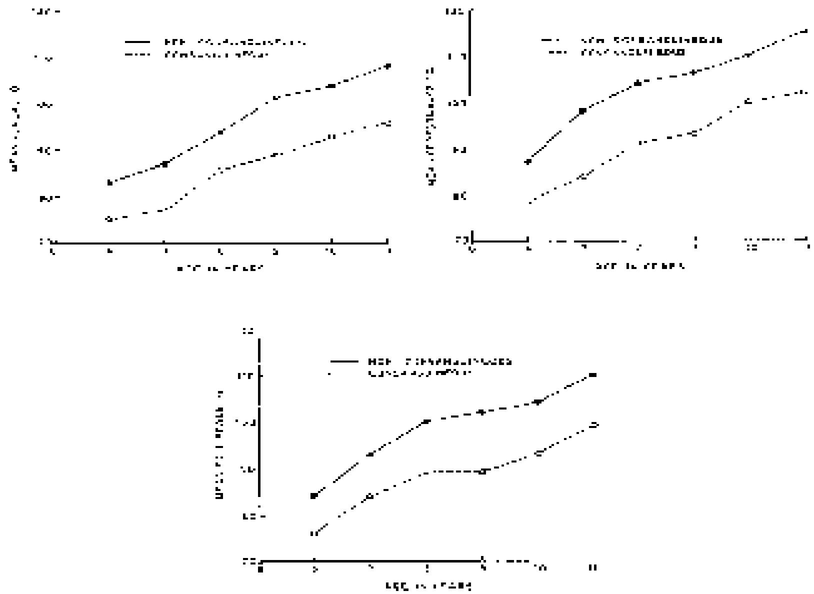


Fig. 1. Trend in the variation of mean IQs (verbal, performance, full-scale) among children of nonconsanguineous and consanguineous groups with their age.

Table I. Correlation (r) and Regression Coefficient (b) of Inbreeding and IQ

Types of IQ	$r \pm$ SE	$b \pm$ SE	a
Verbal	−.40 ± .08	−149.17 ± 34.55*	96.78
Performance	.48 ± .08	−207.4 ± 38.30*	103.98
Full-scale	.45 ± .08	−187.57 ± 34.85*	99.76

* Significant at $p < .001$.

first cousins (inbreeding coefficient, $F = 0.0625$) were selected for further study because marriages of the other consanguineous types were too infrequent. A total of 100 (50 noninbred and 50 inbred) individuals was screened for the study. All subjects were between 6 and 11 years of age. The means and standard deviations of age (in years) of the noninbred and inbred groups are 8.08 (SD = 1.25) and 7.7 (SD = 1.28) respectively—a nonsignificant difference ($t = 1.49$, df = 98, $p > .05$).

Mental Test. The WISC (R)-74, which includes five verbal subtests (information, similarities, arithmetic, vocabulary, comprehension) and five performance subtests (picture completion, picture arrangement, block design, object assembly, mazes), was administered to the subjects. The instructions in the test manual were translated into Urdu and Hindi separately. The subjects were tested in groups of three by the first author.

RESULTS

Figure 1 shows the probable linear relationship of IQ with age. A significant ($p < .001$) and negative correlation of IQ with inbreeding is found (Table I). The coefficients of linear regression (b) of the three IQs (verbal, performance and full scale IQ) on inbreeding, as computed here, are also significant in each case, at $p < .001$. The distribution of IQ in the two samples, however, follows a more or less normal pattern (Table II), with weighted means of 99.6 ± 2.0 and 88.1 ± 1.37 among noninbred and inbred children, respectively, the difference being significant ($p < .001$). A similar trend is also found for both verbal and performance IQs

Effects of Inbreeding on Raven Matrices

Nirupama Agrawal, S. N. Sinha, and Arthur R. Jensen

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Indian Muslim school boys, ages 13 to 15 years, whose parents are first cousins, were compared with classmates whose parents are genetically unrelated on the Raven Standard Progressive Matrices, a nonverbal test of intelligence. The inbred group (N = 86) scored significantly lower and had significantly greater variance than the noninbred group (N = 100), both on raw scores and on scores statistically adjusted to control for age and socioeconomic status. Genetic theory predicts both of these effects for a polygenic trait with positive directional dominance.