Intelligence

Ian J. Deary

Centre for Cognitive Ageing and Cognitive Epidemiology, Department of Psychology,
University of Edinburgh, Edinburgh EH8 9JZ, United Kingdom; email: i.deary@ed.ac.uk

Annu. Rev. Psychol. 2012. 63:453–82

The *Annual Review of Psychology* is online at psych.annualreviews.org

This article's doi:
10.1146/annurev-psych-120710-100353

0066-4308/12/0110-0453$20.00

Keywords

IQ, cognitive ability, psychometrics, behavior genetics, cognitive epidemiology, twins, education, health

Abstract

Individual differences in human intelligence are of interest to a wide range of psychologists and to many people outside the discipline. This overview of contributions to intelligence research covers the first decade of the twenty-first century. There is a survey of some of the major books that appeared since 2000, at different levels of expertise and from different points of view. Contributions to the phenotype of intelligence differences are discussed, as well as some contributions to causes and consequences of intelligence differences. The major causal issues covered concern the environment and genetics, and how intelligence differences are being mapped to brain differences. The major outcomes discussed are health, education, and socioeconomic status. Aging and intelligence are discussed, as are sex differences in intelligence and whether twins and singletons differ in intelligence. More generally, the degree to which intelligence has become a part of broader research in neuroscience, health, and social science is discussed.

Contents

INTRODUCTION

Some people are cleverer than others. The ways in which this occurs, and the causes and consequences of these individual differences, are the topics of this review.

It is some time since the *Annual Review of Psychology* contained an article that dealt substantially with human intelligence differences. Therefore, the period that is covered is broadly the past ten years: the first decade of the twenty-first century. Historically, this is neat, because the foundations of the scientific study of human intelligence differences were laid in the first decade of the twentieth century. Then, there were the statistical developments, empirical discoveries, and conceptual innovations of Spearman (1904) and the intelligence testing technology begun by Binet (1905). Some key questions that Spearman and Binet addressed are still lively topics of research: Along which dimensions of mental abilities do people differ? Do these differences matter? And what are the causes of these differences? These and other questions—such as the effect of aging on intelligence—are included in the present review.

One should be explicit about the difficulties that accompany the topic of human intelligence differences (which will normally, here, be shortened to just intelligence). The study of individual differences has never been in the mainstream of psychology. With respect to psychology's two cultures—experimental and differential (Cronbach 1957)—differential is the small minority. Not only that, but the statistical procedures used by differential psychologists are relatively abstruse to those outside the field. These factors—and the fact that intelligence-type tests have been used so widely in practical settings and have been the subject of controversies—contribute to the range of attitudes that intelligence research attracts from lay and professional outsiders. These attitudes include interest (research reports on intelligence often attract much media interest), indifference (much of mainstream psychology and wider social science ignores individual differences in intelligence), and hostility (the emotional heat generated by some aspects of intelligence research is matched by few other topics in psychology). However, this review also describes how researchers from a number of other disciplines—e.g., neuroscience and epidemiology—are newly and keenly including intelligence as a topic in their research. Intelligence is rarely discussed for long before the word "controversial" appears; this is another difficulty. Because there is controversy attached to some research topics in intelligence, it is important that there are clear and even-handed accounts of what is known and what is unknown about it. The present piece attempts to be both, with examples of influential studies and pointers to areas of disagreement.

RECENT BOOKS ON INTELLIGENCE

The Remarkable 1990s

Books in the past 10 years followed a remarkable decade, because the 1990s witnessed several important publishing events in intelligence. Carroll's (1993) *Human Cognitive Abilities* was his decades-in-preparation analysis of over 400 intelligence data sets that synthesized human cognitive differences in a three-level psychometric hierarchy. Jensen's (1998) *The g Factor* was a massive review of the construct of general intelligence (*g*). Brand's (1996) book of the same name—*The g Factor*—was withdrawn by Wiley after some of the author's remarks in the press about group differences. The book was given a postmortem review in *Nature* (Mackintosh 1996), which commented that, "This seems a singularly cack-handed attempt at censorship... How is it that they found out about the repellant nature of Brand's views only *after* they had printed and distributed copies of the book" (p. 33). Of these books, Carroll's is singled out here for its usefulness in cementing a psychometric structure for intelligence that brought "harmony where there had been discord," although similar models had been available for decades. However, the intelligence publishing event of the 1990s was Herrnstein & Murray's (1994) *The Bell Curve*, which spawned an industry of mainly hostile reaction that continues into the twenty-first century. The book is an unusual mixture of overview, empirical analyses, statistical tutorials, policy reflections, and appendixes and notes (approximately 300 pages of the latter). For example, Part II of the book was almost 150 pages of analyses of the white people in the National Longitudinal Survey of Youth 1979. The authors ran regression models that enquired about the relative importance of measured intelligence and parental socioeconomic status for important social outcomes such as poverty, education, employment, injury, marriage, divorce, childbearing, welfare dependency, parenting, crime, and citizenship.

Intelligence Books from the Past Decade

Because of space limitations, it is possible only to select some books about intelligence from the previous decade. Inevitably, someone's favorite book on intelligence will be missing. However, the following have been chosen because they provide interesting and still-useful accounts of different aspects of intelligence.

With regard to general books on intelligence, there are two books for the lay person or beginning student: Deary's (2000) *Intelligence: A Very Short Introduction* and Cianciolo & Sternberg's (2004) *Intelligence: A Brief History* are both elementary introductions to the science of the area, and they also cover some of the controversies in intelligence research. Bartholomew's (2004) *Measuring Intelligence* is a useful view of intelligence research from the perspective of a statistician, making it more objective than most accounts. Probably the best book on human intelligence differences to appear for many years, Hunt's (2011) *Human Intelligence* is superb. It is written by someone almost equally at home in experimental and differential psychology, with an engaging wit, comprehensive knowledge, and clear writing style. Most of all, it is written with great even-handedness; even for the controversial areas of intelligence it is strongly recommended as an excellent account of the science of intelligence research to date. It also has the merit of not avoiding the technicalities involved in intelligence research, and it explains them well.

The previous decade saw some interesting books on the history of intelligence. Carson's (2007) *The Measure of Merit* is a detailed look at the origins of the idea of merit and mental testing, especially in France and the United States, and begins, historically, well prior to current intelligence testing technology. It is light on the psychometric aspects and arguments of intelligence, but very careful and painstaking in, for example, describing how the military were won over to mental testing in World War I. Wright Gillham's (2001) *A Life of Sir Francis Galton: From African Exploration to the Birth of*

Eugenics is probably the best biography of the Victorian genius to date and includes his seminal work on intelligence. Remarkably, this pioneer of intelligence research and statistics was a significant contributor to many other scientific areas; so much so that, despite the importance of Galton's contribution to intelligence and cognate topics, these take up a small section of the book. White's (2006) *Intelligence, Destiny and Education* is an intriguing examination of the originators of intelligence testing. His thesis is that these researchers were largely characterized by a nonconformist religious background, which in turn led them to emphasize gifted and "feeble-minded" individuals (and less so the middle of the continuum), heredity, and the usefulness of examinations. Although intriguing, the thesis is not convincing.

In addition to history books there have also been several of what might be characterized as broadly anti-IQ-type books. These include Murdoch's (2007) *IQ: A Smart History of a Failed Idea*, Nisbett's (2009) *Intelligence and How to Get It*, Shenk's (2010) *The Genius In All of Us: Why Everything You've Been Told About Genetics, Talent and Intelligence is Wrong*, and Stanovich's (2009) *What Intelligence Tests Miss: The Psychology of Rational Thought*. All are readable, often highly so. Murdoch reviews the history and major areas of controversy in intelligence research, with clear antagonism toward IQ testing and a preference for theories that emphasize multiple mental abilities. Stanovich, in particular, emphasizes aspects of reasoning that are poorly correlated with intelligence and often lacking in people who are intelligent. Shenk emphasizes gene-environment interactions in human life, downplays main effects of intelligence, and argues that the attainment of excellence may be found more in effort. Of these three books, Stanovich's and Shenk's stand out as having the better empirical bases. However, the empirical surveys take them where they will. Stanovich could easily have emphasized the value of rational thought without the negative reference to intelligence tests in the title. None of the four authors is a researcher on intelligence, and it is interesting to reflect on how many other areas of psychological research attract such attention from journalists and psychologists from outside their fields.

There have been several books on the causes of intelligence differences. Deary's (2000) *Looking Down On Human Intelligence: From Psychometrics to the Brain* was a relatively pessimistic survey of what had been contributed by studying cognitive components of intelligence and also the relatively limited findings that had come from biological approaches to intelligence during the twentieth century. Geary's (2005) *The Origin of Mind: Evolution of Brain, Cognition, and General Intelligence* took a more evolutionary approach and tried to fit intelligence differences within broader ideas in the development of brain and mind. Garlick's (2010) *Intelligence and the Brain: Solving the Mystery of Why People Differ in IQ and How a Child Can Be a Genius* had a particular thesis concerning white matter and processing speed and how they produce intelligence, but it was probably at too general a level and with too little empirical information to validate the interesting ideas fully. Duncan's (2010) *How Intelligence Happens* is a welcome account of intelligence from an experimental psychologist who acknowledges the existence and omnipresence of Spearman's *g*, with biological leanings. Flynn's (2007) *What Is Intelligence?* was an interesting culmination of his work since he had demonstrated the Flynn effect of rising intelligence. I have largely avoided edited books in this survey. However, Wilhelm & Engle's (2005) *Handbook of Understanding and Measuring Intelligence* and Sternberg & Grigorenko's (2002) *The General Factor of Intelligence: How General Is It?* both remain useful edited compilations of different viewpoints on the causes of intelligence differences.

Books on intelligence that concern individual, long-term cohort studies appeared in the past decade. The latest monograph from Schaie (2005)—*Developmental Influences on Adult Intelligence: The Seattle Longitudinal Study*—was an update on the Seattle longitudinal aging study. This cross-sequential study started in the 1950s, and this is an indispensable book about cognitive aging. Deary et al.'s (2009c) *A Lifetime of Intelligence: Follow-up Studies of the Scottish Mental Surveys of 1932 and 1947* was a summary of 10 years of work on follow-up studies of the population-wide intelligence tests that took place in Scotland. Because these two cover aspects of aging and intelligence, it should be noted that Salthouse's (2010) *Major Issues in Cognitive Aging* appeared recently and is a highly recommended source of information and toolkit for those concerned with aging aspects of intelligence.

Lynn & Vanhanen's (2002) *IQ and the Wealth of Nations* attempted to analyze prosperity at the national level and IQ. This type of global, country-level epidemiology has many critics. Lynn followed this up with three other books on international aspects of intelligence and productivity. At the personal level of success, Saunders's (2010) *Social Mobility Myths* summarized his and others' research, with a conclusion that social mobility is far greater and more meritocratic in U.K. society than most people—especially politicians and sociologists—think, and that mobility substantially depends on intelligence and effort.

THE PSYCHOMETRIC STRUCTURE OF INTELLIGENCE

Following Carroll's (1993) synthetic account of the psychometric structure of intelligence, there has been a broad consensus that meaningful variance among people exists at three levels: third-level general cognitive ability (*g*), second-level broad domains of cognitive functioning (group factors), and first-level test-specific variation. To explain these levels simply, consider the answer to the following question: Why are some people good at explaining the meanings of words in their first language? The answers are that people who are good at one mental task tend to be good at other types of mental task (third level; *g*); people who are good within one domain (e.g., verbal ability) tend to be good at other tasks in that domain; and people have strengths in specific, narrow mental skills. Thus, when a diverse battery of mental tests is applied to a sample of the population, some of the between-subject variation is shared by all tests, some is shared by tests that have family resemblances within a cognitive domain, and some is specific to the individual test. *g* often accounts for nearly half the variance when a broad battery of cognitive tests is applied to a representative sample of the adult population. Relatively little of the variance lies at the domain level. Researchers do not always agree on the nature of the domains—they can vary in number, name and content between samples depending on the battery applied—and there have long been worries about whether the nature of *g* might vary between cognitive batteries.

The latter worry was addressed directly using over 400 subjects from the Minnesota Study of Twins Reared Apart (Johnson et al. 2004). The subjects had taken three large cognitive test batteries, originating from different theoretical orientations: the Hakstian and Cattell Comprehensive Ability Battery (14 tests); the Hawaii Battery, with Raven's Matrices added (17 tests); and the Wechsler Adult Intelligence Scale (11 tests). Each battery had a strong *g* factor, and the correlations among the three *g* factors—from a hierarchical confirmatory factor analysis—were 0.99, 0.99, and 1.00. That is, the individual differences in *g* were identical from the three different batteries, leading the authors to title the paper "Just one *g*." The result was replicated in a sample of over 500 Dutch seamen who had been tested on five different cognitive batteries (Johnson et al. 2008c). These two papers are important contributions to the psychometric structure of intelligence. As the authors stated, "our results provide the most substantive evidence of which we are aware that most psychological assessments of mental ability of any breadth are consistently identifying a common underlying component of general intelligence. These results provide evidence both for the existence of a general intelligence factor and for the consistency and accuracy of its measurement" (Johnson et al. 2008c, p. 91).

The subjects' mental test data from the Minnesota Study of Twins Reared Apart were also used to tweak Carroll's (1993) proposed three-level hierarchy of intelligence differences (Johnson & Bouchard 2005a). The authors inquired whether there might usefully be proposed some more general—but not as general as *g*—latent traits between the several second-level factors (group factors, or cognitive domains) and *g*, thus giving a four-level hierarchy. Carroll's results had suggested no. The second-level factors were called crystallized (12 tests), fluency (4), fluid (12), memory (6), perceptual speed (11), and visualization (14). Johnson and Bouchard's proposed candidates for new, third-level general factors were the Cattell-Horn fluid and crystallized factors and Vernon's verbal-educational (v:ed) and perceptual (k:m; spatial:mechanical) factors. Vernon's factors arose because, contra Spearman, he thought that, "It may be concluded that *no* test measures nothing but *g* and a specific factor, since the type of test material employed always introduces some additional common element" (Vernon 1956, p. 144). Therefore, according to Vernon, the v:ed domain influenced all tests involving verbal material and those that required the manipulation of words. The k:m domain influenced tests that required the mental manipulation of shapes, spatial imagination, and mechanical knowledge. However, better fitting than any of these three models was one that included three factors at the third level: verbal, perceptual, and image rotation. All three loaded very highly on *g*. The authors called this the VPR model of intelligence. The verbal and perceptual, and perceptual and image rotation, factors correlated very highly, and verbal and image rotation less so. The same four-level model was tested versus the other three using Thurstone's data on 60 mental tests from 1941 and again was found to fit best (Johnson & Bouchard 2005b).

Whereas the hierarchical structure of intelligence differences does allow researchers and interested others to focus on the variance accounted for in cognitive abilities at different levels of generality, there is still some impetus from those looking for an alternative formulation of intelligence or for additions to individual general intelligence. There was an attempt to operationalize Gardner's (1983, 1993) multiple intelligences as tests and to examine their intercorrelations and correlations with a standard psychometric intelligence test (Wonderlic Personnel Test) (Visser et al. 2006). There were clear results. As operationalized by these authors, most of the Gardner mental skills were correlated substantially with psychometric intelligence; formed a substantial *g* factor; and musical and body-kinesthetic intelligence were more separate and intrapersonal intelligence harder to measure. One group of investigators claimed to have found a "group intelligence" that can explain how well a group performs on tasks but is not just the mean or maximum of the individual general intelligence of the members of the group (Woolley et al. 2010). Group intelligence was higher in groups where turn-taking in speaking was relatively evenly distributed among members and in groups whose members had higher mean social sensitivity. Their practical suggestions were that it might be easier to boost the intelligence of a group than of an individual and that it might be useful to introduce group intelligence testing for teams of workers. Replication of this finding is necessary. The study has possible limitations in that the individual intelligence test was a single test, versus a range of tests for group intelligence, which, therefore, would have more general variation and be more likely to be correlated with a criterion measure. Furthermore, there was more overlap in content between the group intelligence test and the criterion tasks than looked likely with the individual intelligence measure.

It is often stated that the major historical challenge to Spearman's conception of intelligence differences as being largely based on *g* was from Thurstone and his Primary Mental Abilities (PMAs). This is only partly correct. It was clear from the late 1930s and certainly by the 1940s that the PMAs were not independent and that Thurstone's own data contained a statistical *g* factor. Probably the strongest psychometric challenge to Spearman's account of intelligence differences was from Godfrey Thomson (Bartholomew et al. 2009). Thomson never denied Spearman's positive manifold of correlations among mental tests, but he suggested a radically different reason for its occurring. Instead of *g*—perhaps, according to Spearman, the result of people having generally more or less of mental energy or power—Thomson found that the universally positive correlations among tests could also arise from each test's sampling a subset of numerous, independent mental bonds; thus his "bonds" or "sampling" theory of intelligence. The Spearman-Thomson debates lasted from the First World War until almost the end of World War II. A fresh look at Thomson's ideas concluded that his model of intelligence was not inferior to Spearman's, either on statistical or biological grounds, though that was partly because both were vague biologically (Bartholomew et al. 2009). A related development is the mutual interaction model of intelligence, which also posits the emergence of a general factor without a general cause (van der Maas et al. 2006). The basic idea is that a statistical *g* emerges through the mutual interaction, over the course of their development, of several cognitive processes.

COGNITIVE CORRELATES OF INTELLIGENCE

Those taking a reductionist view of intelligence have not always gone straight down to biology. Three levels of reduction have been visited that stop short of, say, genetics or brain imaging: other psychometric measures; measures from experimental psychology, especially reaction times; and measures from psychophysics, including inspection time. With the increased accessibility of brain imaging, studies that use these sorts of tasks and their related constructs along with intelligence are probably declining from a peak between the 1970s and 2000 (Deary 2000). Much interest has focused, in the broadly psychometric-experimental levels, on processing speed and working memory as potential explanatory variables for intelligence. However, there are new findings to report from each of these areas, some of which are being incorporated within brain imaging and genetic studies of intelligence. That is, a study may be potentially more informative if it includes intelligence phenotypes, a biological marker, and an additional, potentially explanatory psychological construct.

Odd as it is to report, and in spite of there being a hundred years of research in these areas, an indication of the true correlation between intelligence and sensory discrimination and reaction time appeared only in the past decade. Spearman (1904) proposed what he termed a functional correspondence between general intelligence and sensory discrimination. Ninety-seven years after this, it was reported, in a large sample (N = 899) of healthy adults in the United States, that general intelligence (from a battery of 13 tests) correlated 0.21 with pitch discrimination and 0.31 with color discrimination (Acton & Schroeder 2001). These bivariate correlations between general intelligence and sensory discrimination do not actually test what Spearman (1904) hypothesized; namely, that whatever was common to discrimination measures was almost perfectly correlated with whatever is common to cognitive test measures. Acton & Schroeder's sample was reanalyzed using structural equation modeling, and the correlation between general intelligence and general sensory ability latent traits was 0.68; in a separate Scottish sample of children, it was 0.92 (Deary et al. 2004a). Spearman was substantially correct, although we still do not know what causes the correlation between these two latent traits.

With regard to reaction time, the first large (N = 900) population-representative study of its correlation with intelligence found, in a very narrow age cohort about 56 years old, as follows: four-choice reaction time mean = −0.49; four-choice reaction time intraindividual variability = −0.26; simple reaction time mean = −0.31; and simple reaction time intraindividual variability = −0.26 (Deary et al. 2001). A series of empirical reviews—but not formally conducted or presented meta-analyses—of processing speed and intelligence correlations gathered findings from 172 studies containing over 50,000 subjects in total (Sheppard

2008). The correlation was slightly lower than that of Deary et al. (2001) but included convenience samples; that is, samples of participants where no attempt is made to match to population characteristics and that often involve students or other relatively cognitively homogeneous groups. Much of Sheppard's account concerns cognitive components—such as the reaction time for processing in short-term memory or speed of retrieval from long-term memory—but I consider these to be a distraction when the correlation with straightforward choice reaction time is so high. The cognitive components claimed to be isolable from reaction time models tend to be unreliable and not, in any case, to improve the correlation with intelligence (Deary 2000, chapter 6). This was emphasized by Lohman (1994) in his overview of such attempts: "attempts to isolate component scores that decompose individual differences on homogeneous tasks into process measures cannot succeed, and so our efforts should be directed elsewhere" (p. 9). The mean correlation between general intelligence and visual inspection time—a psychophysical task that does not involve reaction speed but, instead, records correct discriminations based on a simple stimulus that is presented for different durations—was −0.36 (Sheppard 2008); people with higher intelligence test scores were more efficient in accumulating accurate information from briefly presented stimuli. The equivalent correlation with auditory inspection time was −0.31.

Therefore, the current situation is that apparently lower-level mental tasks—such as sensory discrimination, visual processing and reaction time—have fairly well-established significant and far-from-trivial correlations with intelligence. Less is known about why these correlations occur, and that is largely because of lack of understanding of the causes of individual differences in these so-called elementary cognitive tasks. Although reaction time tasks are quite widely applied in mental testing, current research is in a state whereby there is less interest in these sorts of tasks to explore the origins of intelligence differences. On the one hand, this might reflect a correct judgment that explanations have not been and will not be forthcoming from that research route. On the other hand, it might be that researchers in intelligence have prematurely left this field to explore other routes that promise more by way of a reductionistic account of intelligence differences; for example, in brain imaging and molecular genetics.

THE BIOLOGY OF INTELLIGENCE

Around 2000, there were two overviews of the biological correlates of human intelligence differences (Deary 2000, Jensen 1998). They dealt with the same assortment of biological tools: genetics (behavioral and molecular studies), brain imaging (structural and functional), the brain's electrical responses (analyses of the electroencephalograph [EEG] and evoked responses), nerve conduction velocity, and an assortment of less-studied approaches. Little was certain at that time. Two things seemed relatively firm: People with higher measured intelligence tended to have larger brains, and intelligence differences had a substantial heritability. An emerging finding was that older people with the e4 allele of the gene for apolipoprotein E (*APOE*) tended to have lower cognitive ability. Apart from these findings, most approaches suffered from lack of replicability. There were many, mostly modestly sized, studies reporting correlations between a biological variable and intelligence, but typically these were either not replicated or attempted replications tended to be too different to be characterized as such (Deary 2000, Jensen 1998). EEG and brain-evoked response studies suffered particularly in that regard. In looking forward to the research after 2000, my opinion was that, "it is tempting to say that the hope lies mainly in the less-put-to-the-test approaches of functional brain scanning and molecular genetics" (Deary 2000, p. 312). This prognostication, made a decade ago, is both correct and wrong. Probably it is correct to have identified brain imaging and genetics as the two techniques that would be most applied to human intelligence research and would deliver the most solid findings. However, it backed the wrong horse in both cases. Behavioral genetics and structural brain imaging have added more than their respective molecular and functional counterparts in our search for the causes of human intelligence. A recent review of the biological foundations of intelligence—from the point of view of neuroscience—covers behavioral and molecular genetic studies and structural and functional brain imaging, and it provides more detail than space allows in the present account (Deary et al. 2010a).

Genetics

This overview of advances in the genetic contributions to human intelligence differences divides the research into behavioral and molecular approaches. Behavioral studies use twin-, adoption-, and family-based designs to obtain estimates of the proportion of the population variance in intelligence caused by genetic (mostly additive) differences and by the shared and nonshared aspects of the environment. Molecular genetic studies use candidate gene or genomewide association techniques. Candidate gene studies are hypothesis driven. On the basis of prior findings or on the known function of a gene's protein, a gene that is polymorphic (has multiple alleles and might thereby express different phenotypes) is selected and a test is made to discover whether people with different alleles tend to differ on intelligence generally or on specific cognitive domains. Genomewide association studies (widely referred to using the acronym GWAS) are hypothesis free: One tests the association between a phenotype (e.g., an intelligence test score or *g* factor) and a large number of genetic variants spread across the human genome. Then one tries to find out which of the many nominally significant associations are replicable and then what they might mean mechanistically (if they replicate). There have been at least five reviews of the genetic studies of human intelligence in the past few years (Deary et al. 2006, 2009b; Lee et al. 2010; Payton 2009; Plomin & Spinath 2004). The broad conclusions are that the heritability of intelligence is now well established, with some important moderation by age and other factors; that multivariate behavior genetic studies have been informative about the causes of correlations between intelligence and some other variables with which intelligence correlates; and that molecular genetic studies—with the exception of variation in *APOE*—have yet to identify variations in specific genes that are firmly associated with intelligence differences.

Behavior genetics. Individual studies from the past decade may be used to exemplify important advances or consolidations in the understanding of the environmental and genetic contributions to intelligence differences.

Much of the additive genetic effect on intelligence is attributable to the general cognitive ability (*g*) factor, as is much of the genetic influence on specific cognitive tests. A Dutch study administered the 11 subtests of the Wechsler Adult Intelligence Scale and Raven's Matrices (a test of nonverbal reasoning) to 194 twin pairs in their later teens (Rijsdijk et al. 2002). The variation in full-scale IQ (almost equivalent to *g*) attributable to additive genetic factors was 82%. The additive genetic contributions to individual tests were examined according to the psychometric three-level hierarchy, as described by Carroll (1993). Additive genetic contributions to the *g* factor, which capture the variance shared by all 12 tests, accounted for a mean of 31% (range = 8 to 53) of the variation in individual test scores. Additive genetic contributions to cognitive domains—verbal comprehension, freedom from distraction, perceptual organization—accounted for a mean of 12% (range = 1 to 30). Additive genetic contributions to test-specific variation accounted for a mean of 14% (range = 0 to 38). Therefore, these diverse mental tests correlate phenotypically largely due to genetic causes of *g*. In the VPR model of intelligence, based on data from the Minnesota Study of Twins Reared Apart, the additive genetic contributions were calculated for general intelligence (*g*) and for cognitive domains at different levels of generality-specificity (Johnson et al. 2007). For *g*, at the fourth stratum in their hierarchical model, which contained the variance shared by all tests, additive genetic causes accounted for 77% of the variance. The third stratum, one step below *g*, had factors that accounted for variance shared by tests drawing on broad verbal, perceptual, and image rotation abilities, and the additive genetic contribution to these was 78%, 77%, and 76%, respectively. The second stratum had still-more-specific factors accounting for variance shared by tests drawing on the following abilities, with the percentages of variation accounted for by additive genetic causes given in parentheses: narrow verbal (79%), scholastic (69%), fluency (79%), number (72%), content memory (33%), perceptual speed (67%), spatial (76%), and rotation (75%). There are two remarkable points here. The first is the consistently high genetic contribution to variance at the second to fourth strata. Of course, it must be emphasized that much of the genetic influence on the second and third strata derives from the genetic influence on *g*, because factors at these levels load highly on it. The second is the relatively high environmental contribution to content memory.

The heritability of intelligence is not the same at different ages. A Dutch study with 209 pairs of twins examined intelligence at ages 5, 7, and 10 years (RAKIT battery) and again at 12 years (Wechsler Intelligence Scale for Children-Revised) (Bartels et al. 2002). Across these four ages, from 5 to 12, the percentages of variation in intelligence accounted for were as follows: additive genetic effects = 26, 39, 54, 64; shared environment effects = 50, 30, 25, 21 (the latter three values were nonsignificant); and unique environment effects (includes error) = 24, 31, 21, 15. Similarly, the Twins Early Development Study in the United Kingdom found that, for general intelligence, the heritability was 23% in early childhood (with shared environment accounting for 74% of the variance) and that this increased to 62% by middle childhood (with shared environment only 33% by this stage) (Davis et al. 2009). When extended to adult samples, Dutch twin family studies demonstrate that the percentages of variance accounted for by additive genetic effects rise to over 80% for verbal IQ and almost 70% for performance IQ (Posthuma et al. 2001). Lower estimates, however, were indicated from the results of the Vietnam Era Twin Registry, which estimated that genetic factors cause 49% of variation in the Armed Forces Qualification Test in young adulthood and 57% in late middle age, with the genetic effects also explaining almost all of the 0.74 correlation between the two administrations of the test in this longitudinal study (Lyons et al. 2009). Results from repeated cognitive testing of participants in the Swedish Adoption Twin Study of Aging indicate that genetic influences remain substantial into old age, with a lowering of the contribution (though it is still the majority) at about 80 years (Reynolds et al. 2005). A review of twins studies with older people estimated the heritability of *g* to be about 80% at 65 years and about 60% at age 82 (Lee et al. 2010).

In old age, the genetic contribution is almost entirely to the level (intercept) of intelligence rather than its change (slope) (Lee et al. 2010). Latent growth curve models of the Swedish Adoption Twin Study of Aging showed that genetic effects were largely to the intercepts and not the slopes. Any small genetic effect on the slope tended to be on the smaller quadratic rather than the much larger linear effect (Reynolds et al. 2005). Among the cognitive domains, the genetic effects on memory increased with age, but those on processing speed and fluid ability decreased somewhat.

The heritability of intelligence might be moderated by environmental factors. This idea was tested in 229 pairs of seven-year-old twins from the National Collaborative Perinatal Project, in which over half the sample was black and there was a high proportion of poor families (Turkheimer et al. 2003). To give just one interesting result, when socioeconomic status was split into high and low, the percentage of variance in intelligence accounted for by genetic factors was 71% and 10%, respectively. The shared environment effects were 15% and

58%, respectively. Life events have also been found to moderate genetic and environmental contributions to general intelligence variation in adults (Vinkhuyzen et al. 2011).

Multivariate genetic-environmental studies have been useful in exploring the causes of the associations between intelligence and some of its correlates. To explain, behavior genetic methods may be used to describe the proportions of environmental and genetic influences on single phenotypes, and they can also be used to describe the proportions of environmental and genetic influences on the correlations between two or more phenotypes. For example, an adolescent twin sample of over 500 twin pairs examined the correlations between psychometric intelligence and processing speed measures including choice reaction time and inspection time (Luciano et al. 2004a). A general genetic factor influenced intelligence and processing speed tests. More specific genetic factors accounted for some test covariance and test-specific variance. Environmental effects were mostly nonshared and test specific. A bivariate environmental-genetic study using Australian and Dutch participants in a twin family design explored causal hypotheses concerning the correlation between inspection time and intelligence; in the literature there were competing suggestions about which caused the other (Luciano et al. 2005). The best model was one of pleiotropy; that is, a common set of genes influences both intelligence and inspection time, but neither of these variables mediated the genetic influence on the other. For processing speed and other cognitive models of intelligence this is instructive: It suggests that these so-called elementary cognitive tasks are aspects of *g* rather than causes of it. Strong genetic correlations exist between IQ scores from the Wechsler battery and the heavily genetically influenced general executive function and the updating specific aspect of executive function (Friedman et al. 2008). There is mixed evidence about whether the small but consistent correlation between intelligence and birth weight (in the normal range; Shenkin et al. 2004) is caused by shared genetic factors (Luciano et al. 2004b). Brain volume is highly heritable (Thompson et al. 2001), and it correlates modestly with intelligence (McDaniel 2005). A bivariate genetic-environmental study of this correlation showed that the correlations between intelligence and the total volumes of gray matter and white matter were caused by genetic factors (Posthuma et al. 2002).

Molecular genetics. With almost-equal justification, this section of the overview could be very long or very short: very long, because dozens of candidate genes have been reported as being associated with intelligence; very short, because almost none of them has been replicated. As a compromise, this section attempts to steer a course between the two extremes of giving a list of type 1 statistical errors and bleakly stating that we, as yet, know nothing about the genes that influence intelligence differences.

Candidate gene studies of intelligence differences have been disappointing. A review surveyed a period of about 14 years in which there were more than 200 studies on approximately 50 genes with polymorphisms that might be related to intelligence (Payton 2009). It was concluded that, as yet, no individual genetic variants are conclusively related to intelligence or its change with age in healthy individuals. An exception is the gene for apolipoprotein E (*APOE*), which is involved in cholesterol transport and neuronal repair. Possession of the e4 allele for this gene was the subject of a meta-analysis of 77 studies with nearly 41,000 healthy individuals (Wisdom et al. 2011). People with the e4 allele have, on average, lower general cognitive function, with an effect size (*d*) of about −0.05. The effect is found in late-middle and old age, and the effect appears to strengthen with age. There may also be a very small effect of a polymorphism in the gene for catechol-O-methyl transferase (Barnett et al. 2008). A meta-analysis of polymorphisms in the gene for dystrobrevin-binding protein 1, which is associated with risk of schizophrenia, showed effect sizes for two single-nucleotide polymorphisms around the 0.1 value (Zhang et al. 2010). It is also worth mentioning the Val66Met polymorphism of the gene for brain-derived neurotrophic factor, which might have a small effect on intelligence, although there are uncertainties about the direction of association (Payton 2009).

A fascinating molecular genetic contribution was an interaction between variants in the gene for fatty acid desaturase 2 and the effect of breastfeeding on children's intelligence (Caspi et al. 2007). Though this was shown in two cohorts, another large cohort has not replicated the direction of association (Steer et al. 2010). And, though maternal intelligence did not appear to explain the effects in the original study, a meta-analysis of breastfeeding effects on children's intelligence did show that maternal intelligence accounted for much of the apparent protective effect of breastfeeding, leaving it nonsignificant overall (Der et al. 2006).

With regard to GWAS, the past decade has seen a remarkable maturing of genetic studies of intelligence. The first genomewide scans for intelligence used a few to many hundreds of microsatellite markers and hundreds of sibling pairs or families in linkage analyses (e.g., Luciano et al. 2006, Posthuma et al. 2005). Linkage refers to the fact that genetic loci that are relatively close to each other on the same chromosome tend to be inherited together. Fundamentally, then, linkage analyses explore whether, among related individuals, certain genetic marker loci tend to co-occur with phenotypic characteristics. These types of analyses, for example, indicated the possible importance of variants in the chromosome 6p region. Linkage designs have, for the present, been pushed to the periphery of genetic research on intelligence with the coming of so many GWAS that test unrelated subjects. However, linkage designs might well reappear in the search for rare variants that influence intelligence. The first genome-wide studies of intelligence to use hundreds of thousands of single-nucleotide polymorphisms have appeared. Even with this density of genotyping, a few hundreds or thousands of subjects, and attempts at replication, it is becoming clear that the effects of individual genetic variants will be very small, and for replicable discoveries to be made, the scale of this research will have to be far larger (Cirulli et al. 2010, Davis et al. 2010).

Through the use of a novel statistical technique applied to genomewide data, the first purely biological evidence for the substantial heritability of fluid and crystallized general intelligence has appeared (Davies et al. 2011). The study analyzed more than 500,000 single-nucleotide polymorphisms in over 3,500 older people from Scotland and England. The authors used a new technique that employs information from all of the half-million-plus genetic variants to calculate "relatedness" in these unrelated individuals. The method afforded an estimate of the narrow-sense heritability of intelligence based purely on these biological data. The heritability estimate was 40% for crystallized intelligence and 51% for fluid general intelligence. This means that unknown causal genetic variants in linkage disequilibrium with the assessed single-nucleotide polymorphisms account for much of the genetic variation in intelligence. The results point to the possibility of many genes of small effect contributing to human intelligence variation. Moreover, the authors were also able to use the purely biological information from the Scottish and English samples significantly to predict about 1% of the variance in intelligence scores in an independent Norwegian sample.

The best genetically oriented research on intelligence now appreciates that individual gene effects are likely to be small or very small; examines hundreds of thousands of single-nucleotide polymorphisms on every participant (and, indeed, uses the fact that single-nucleotide polymorphisms that occur quite close together show linkage disequilibrium—that is, there is a greater than zero chance of certain variants being inherited together—to impute these effectively to provide a few million); gathers in consortia that can bring together several or even many thousands of participants; often involves researchers with a primary interest in medical or other social variables but who happened to have collected intelligence data on their participants; and has rigorous approaches to significance and replication. This has arisen after finding that no studies to date at the smaller scale—either candidate gene or genomewide—have found large or medium effects. The same maturing of studies has occurred with other quantitative traits, such as height. A consortium of studies on height, which reported results on over 180,000 individuals, found significant effects at more than 180 genetic loci, which together explained 10% of the phenotypic variation (Lango Allen et al. 2010). At present, at least three international consortia of studies are conducting genomewide association analyses of intelligence-related phenotypes. All have participant numbers well into five figures; none has reported findings to date.

If it is the case that genomewide searches for intelligence do not throw up any large or medium effects—or even a collection of smaller effects that seem to account for most of the additive genetic effect that is apparent from behavioral genetic studies—then the genetic contributions lie elsewhere. One option is in a very large number of very small effects, as suggested by Davies et al. (2011). A second is in rare variants (Penke et al. 2007), where the idea is that the accumulation of these across generations causes differences in intelligence. Another is in what are called copy number variations: deletions and duplications of stretches of DNA. Although based on very few subjects and requiring replication, one study examined this possibility and found that people with more rare deletions had lower Wechsler Adult Intelligence Scale scores (Yeo et al. 2011).

Brain Imaging

In the early years of brain imaging and intelligence—the 1980s and 1990s—there were studies using positron and single-photon emission tomography. Both techniques were expensive and involved the administration of radioactive substances to subjects. These techniques have been eclipsed by magnetic resonance imaging (MRI) in its various structural and functional forms. Although it is still not inexpensive to scan each subject's brain, its safety and relatively low cost have meant that almost all the brain imaging studies of intelligence have used MRI. Below, some studies exemplifying key empirical advances in the association between brain structure and functions are described. First, though, attention is directed toward an adventurous and helpful synthesis of brain imaging–intelligence work. The parieto-frontal integration theory of intelligence (P-FIT) brought together 37 studies that had employed neuroimaging techniques to investigate differences in intelligence and reasoning (Jung & Haier 2007). They included structural and functional MRI, diffusion tensor MRI, magnetic resonance spectroscopy, and positron emission tomography studies, all of which they thought could be used to sketch a coherent account of what it means to have a brighter brain. Congruent brain imaging–intelligence associations overlapped on how the association cortices were linked by key white matter pathways (particularly the arcuate fasciculus and superior longitudinal fasciculus). In the P-FIT account, abstraction and elaboration of incoming sensory information are conducted in the parietal cortex; the parietal interacts with the frontal cortex, which tests hypotheses concerning a problem; following a best solution, the anterior cingulate constrains the selection of responses and inhibits competing ones; and the whole process depends for its efficiency on intact white matter connections between the regions. In addition to the P-FIT paper—and its associated discussion—readers are directed to a recent special issue of the journal *Intelligence* that was devoted to brain imaging, as introduced by Haier (2009).

Structural brain imaging. Only in the past decade has there been enough evidence to conclude with confidence something that had been mooted, debated, and, at times, ridiculed for over a century: People with larger brains do tend to have higher intelligence test scores. In a meta-analysis of 37 samples examining whole-brain volume in healthy subjects (total N = 1,530), the raw correlation was 0.29 (0.33 after correction for range restriction; McDaniel

2005). A more recent review reckoned the correlation between whole-brain size and general intelligence is about 0.4 and that the correlation between external head size and intelligence (based on 59 samples with a total N = 63,405) is 0.20 (Rushton & Ankney 2009).

That being established, there has been interest in whether certain brain regions and the pathways between them are associated with intelligence differences—see the P-FIT theory above (Jung & Haier 2007)—and more generally the extent to which intelligence is localized or distributed through the brain. One issue has been whether intelligence is more strongly associated—and where in the brain—with gray or white matter. Certainly, gray matter volume seems to be correlated significantly with intelligence. In 216 children and adolescents, there were positive correlations between general intelligence (from a short Wechsler battery) and brain cortical thickness distributed through frontal, parietal, temporal, and occipital brain regions (Karama et al. 2009). These were in agreement with, if partly more extensive than, the regions identified in the P-FIT theory. Data from 65 men and women showed that cortical thickness (gray matter) was associated with intelligence, particularly in the prefrontal and posterior temporal areas (Narr et al. 2007). The study had also found an intelligence–overall brain volume correlation of 0.36 (0.37 for overall gray matter; 0.26 for intracranial white matter volume). This correlation between overall brain volume and intelligence is almost identical to McDaniel's (2005) estimate and that from a study of over 200 young adults, in which the correlation was 0.35 (Choi et al. 2008). Intelligence scores and voxel-based morphometry analyses of brain MRI data from twins provided evidence for an intelligence differences–associated network of frontal-occipital-parahippocampal gray matter and connecting white matter of the superior occipito-frontal fasciculus and corpus callosum (Hulshoff Pol et al. 2006). Investigators using voxel-based morphometry analysis of MRIs of 48 adult human brains to separate gray and white matter identified positive correlations between *g* from the Wechsler Adult Intelligence Scale and gray matter volumes in the frontal, temporal, occipital, and some sublobar (lentiform nucleus, thalamus, etc.) brain regions (Colom et al. 2006). More sophisticated than these cross-sectional studies, a study of 307 children aged 7 to 19—who had been imaged between one and three times with a two-year interval—investigated the association between general intelligence and the developmental trajectory of brain cortical thickness (Shaw et al. 2006). An examination of their findings—where, for illustration, intelligence was divided into superior, high, and average groups—revealed that the superior intelligence group had a distinct trajectory whereby their cortical thickness was the lowest of the three groups at age 7, highest at about age 12, and average again at age 19.

The above studies were performed on healthy individuals. Validation of the principal P-FIT ideas was found from an innovative study of 241 patients with brain lesions who underwent brain imaging (with voxel-based lesion-symptom mapping) and were tested on the Wechsler Adult Intelligence Scale battery (Gläscher et al. 2010). Conceptually, this is what they did: They divided the brain into voxels and asked what the association was between having a lesion in a given voxel and the score obtained on the *g* factor from the Wechsler battery. The significant associations with *g* were with damage in a frontal-parietal network and the white matter tracts that connected them, with the superior longitudinal/arcuate fasciculus again being prominent.

The P-FIT ideas include both gray and white matter in the brain as contributing intelligence differences. The past decade has seen increasing evidence of correlations between white matter–related brain-imaging variables and intelligence. People with more lesions in the white matter tend to have lower intelligence, although this has been found—with small effect sizes—mainly in older people, in whom these lesions tend to accumulate and can be rated using brain MRI images (Frisoni et al. 2007). Eleven studies show, overall, some evidence of an association between n-acetylaspartate—a brain metabolite measured by magnetic resonance spectroscopy—and cognitive abilities, but the associations do not always go in the same direction (Jung et al. 2009). The development of diffusion tensor MRI has meant that indices of brain white matter integrity can be produced in vivo. Several studies show that people with higher intelligence tend to have greater white matter integrity—typically assessed using a parameter called fractional anisotropy—which accords with a distributed and connectionist view of what it means to be higher in intelligence (Deary et al. 2010a provide an overview). Brain white matter integrity is highly heritable, and the modest phenotypic correlation with intelligence appears to be caused by shared genetic factors (Chiang et al. 2009).

Functional brain imaging. Early in the past decade there were two high-profile articles that made many intelligence researchers sit up and take notice because of their striking titles: "A neural basis for general intelligence" (Duncan et al. 2000) and "Neural mechanisms of general fluid intelligence" (Gray et al. 2003). In the first study, a PET experiment on only 13 subjects devised high- and low-*g*-demanding versions of three tasks and looked for the brain regions that were consistently associated with greater activation in the high-*g* task versions. The answer was the lateral frontal cortex, and the authors argued against a diffuse neural recruitment mechanism for *g* differences, but instead, "*g* reflects the function of a specific neural system, including as one major part a specific region of the lateral frontal cortex" (Duncan et al. 2000, p. 459). It is clear from the P-FIT theory (Jung & Haier 2007) and lesion-based studies (Gläscher et al. 2010) that Duncan did identify an important brain region for intelligence and that it is just one region in a network. In the second study (Gray et al. 2003), the straightforward result was that neural activity (inferred from the blood oxygen–level dependent signal in functional MRI) in the lateral prefrontal cortex almost completely attenuated the association between intelligence (Raven's Advanced Progressive Matrices) and the ability, correctly, to ignore "lure" stimuli in a 3-back working memory task. Gray and colleagues (2003) concluded this was "the first direct support for a major hypothesis about the neurobiological basis of *gf*" (p. 319). An attempt to replicate this result beyond lure detection in an n-back task was not successful (Waiter et al. 2009). Both of these studies had interesting findings based on well-aimed hypotheses. However, their ambitious titles must be read with appropriate skepticism: They appear to offer too broad a conclusion from small studies that are the beginnings rather than the end of a long research effort to explain intelligence differences.

A very valuable integrative review, which included functional brain imaging and EEG techniques, converges on an account of the intelligent brain that is distributed (Neubauer & Fink 2009). It also provides much diverse support for the view that the intelligent brain is more efficient.

Fluctuating Asymmetry

On the basis of the idea that intelligence is an indicator of fitness, there has been considerable activity in examining whether it associated with other aspects of bodily fitness. One aspect is health, and that is covered here under the section on cognitive epidemiology; another is fluctuating asymmetry (Van Valen 1962): the degree to which the same bodily parts on the two sides of the body show an absolute deviation from being identical in size. This is covered here because it is an aspect of research into the biology of intelligence that emerged only in the previous decade or so. Except for the original report of two samples (Furlow et al. 1997)—each with just over 100 participants—in which intelligence correlated just over 0.2 with symmetry, all studies have appeared in the twenty-first century. A meta-analysis of 14 samples (published and unpublished) with a total N of 1,871 estimated that the correlation between intelligence and fluctuating asymmetry was −0.12 to −0.20 (Banks et al. 2010). That is, people with higher intelligence test scores tend to be more symmetrical. To explain these findings, Banks et al. appealed to an account which suggests that both intelligence and symmetry are markers of a general fitness latent trait that is associated with survival and reproductive success (Miller 2000, Keller & Miller 2007). However, a first attempt to examine the genetic correlation between intelligence and symmetry found neither a genetic nor a phenotypic correlation (Johnson et al. 2008b).

PREDICTIVE VALIDITY OF INTELLIGENCE

Intelligence predicts important things in life. The predictive validity of intelligence for education, occupational success, and social mobility was well documented prior to the past decade. That said, there was some new research in these areas. Some progress was also made in assessing the association between intelligence and related personal-social constructs. A useful meta-analysis showed that the correlation between intelligence and ego-development stage is between 0.20 and 0.34, making them related but not identical concepts (Cohn & Westenberg 2004). Long-term follow-up studies of the British cohorts born in 1958 and 1970 showed strong associations between higher childhood intelligence and more socially liberal attitudes at age about 30 years (Deary et al. 2008, Schoon et al. 2010). The most novel contribution of intelligence as a predictor was the emergence and growth of the new research field of cognitive epidemiology; the first years of the twenty-first century established intelligence as a predictor of health, illness, and death.

Education, Occupation, and Social Mobility

Although they have been studied for many years, it is useful to see a meta-analytic approach to the associations of intelligence with some of its well-known correlates in the field of socioeconomic success. Intelligence had average correlations (95% confidence intervals, total number of studies, total number of subjects) as follows: education = 0.46 (0.36 to 0.75, 59, 84,828); occupation = 0.37 (0.28 to 0.57, 45, 72,290); and income = 0.21 (−0.01 to 0.40, 31, 58,758) (Strenze 2007). With only the objectively defined better studies included, with sample-size weighting, and with correction for unreliability and dichotomization, the effect sizes were estimated as, respectively, 0.56, 0.45, and 0.23. In this meta-analysis, education was assessed using educational level. If objective results are used from national examinations, the correlation between intelligence and education is considerably higher. For example, the prospective correlation between the general intelligence latent trait from the Cognitive Abilities Test at age 11 years and a general educational latent trait (based on English national General Certificate of Secondary Education scores at age 16 years; N = 13,248) was 0.81 (Deary et al. 2007a). By way of balance—albeit in a study with two orders of magnitude fewer subjects—it is noted that self-discipline (as rated by the person or by others) can have stronger associations with educational outcomes than intelligence has (Duckworth & Seligman 2005). There is also evidence for some people's being more motivated than others in certain intelligence testing situations, which could inflate intelligence–life outcomes correlations (Duckworth et al. 2011). In another investigation of real-life outcomes of intelligence, a large study of trainee truckers showed that intelligence was associated with "preferences and choices in ways that favor economic success" (Burks et al. 2009). Those with higher intelligence were more patient, were better at taking calculated risks, were better at predicting how other people would act and how they should act as a result, and persevered longer in a job when there was a financial penalty for leaving.

Much remains to be discovered about social mobility. The United Kingdom, because it has various prospective cohorts that have been studied from youth and are now at various stages of adulthood and old age, has been especially informative, but of course these results do not necessarily generalize to other countries or cultures. However, even using the same cohort's data, different researchers have come to different conclusions. For example, consider some analyses from the past decade on the U.K.'s National Child Development Study (the 1958 British Birth Cohort), which gathered prospective data on all children born in Great Britain in one week in March 1958. Some researchers emphasized that, with respect to social class destinations at age 33, there were still substantial effects of parental social class after adjusting for intelligence and academic effort (Breen & Goldthorpe 2002). With the same data, others emphasized that whereas parental social class accounted for about 25% of people's own social class at age 33, intelligence, motivation, and qualifications accounted for over 60% (Saunders 2002). Others have shown that social mobility from all the social classes is driven about equally by intelligence from childhood (Nettle 2003). General findings—using structural equation modeling—from analyses of various British cohorts (born in 1921, 1936, the 1950s, and 1970) are that education tends to mediate the influence of childhood intelligence on adult socioeconomic status; the effect of intelligence on education is stronger (insofar as they can be compared numerically) than that of parental social class; and that childhood behavioral disturbance is correlated significantly with intelligence and contributes, at most, only small amounts of additional (beyond intelligence and parental social class) variance to education or adult social class (Deary et al. 2005a; Johnson et al. 2010; von Stumm et al. 2009, 2010). Most of these latter analyses were completed in men because of the difficulty in assessing socioeconomic status among women at historical times when women either tended not to be in employment to the extent that they are now or tended to be less likely to attain employment in accordance with their abilities.

Health, Illness, and Death

Prior to the past decade, health was not an outcome that counted intelligence as one of its determinants to any extent. That has changed. Two of the prominent social correlates of intelligence—education and social class (parental and own in adulthood)—were already known to be associated with health inequalities; therefore, it is not surprising to see intelligence added to the list. The first journal report of an intelligence-death association was with Australian male Vietnam veterans; those whose mental test scores were lower on entry to the armed services were more likely to have died (principally from external causes) by midlife (O'Toole & Stankov 1992). Subsequent findings took place in the next decade. A follow-up of over 2,000 subjects of the Scottish Mental Survey of 1932 (of people born in 1921) found that a standard deviation disadvantage in childhood (age 11 years) intelligence was associated with 21% (95% confidence interval 25% to 16%) lower survival up to age 76 (Whalley & Deary 2001). This was a new and healthy development for intelligence research: its being included in large-scale epidemiological health research with population-representative samples. A few years later the term "cognitive epidemiology" was coined (Deary & Der 2005) to describe this field of research. A glossary of the field is available (Deary & Batty 2007), and a special issue of the journal *Intelligence* (2009) appeared, with 13 new empirical studies on the topic.

The association between lower intelligence test scores in childhood or youth and dying earlier has been replicated in many studies, typically involving the follow-up of thousands of subjects for up to several decades. The largest single study to date included about one million men (Batty et al. 2009). This was conducted in Sweden and was possible because almost all Swedish men are conscripted into military or civil service in young adulthood. For decades, young Swedish males have taken the same set of mental tests. Thereafter, the unique code number that is assigned to Swedish citizens was used prospectively to link these cognitive test data with data held in education, health, and other public databases. A systematic review and meta-analysis has identified 16 independent studies of the intelligence-mortality association and found that one standard deviation advantage in intelligence was associated with 24% lower risk of death (95% CI = 23% to 25%) over a follow-up range of 17 to 69 years (Calvin et al. 2011). This paper and an extensive narrative review with discussion (Deary et al. 2010b) debate possible causes of the association and the range of causes of death with which intelligence is significantly associated. The latter include deaths from cardiovascular disease, suicide, homicide, and accidents, but not cancer.

Prospectively, physical and mental health outcomes are both associated with childhood or early adult intelligence, especially cardiovascular disease among the physical illnesses (Deary et al. 2010b). This applies to most categories of psychiatric disorders, and a standard deviation disadvantage in intelligence at about age 20 has been associated with, for example, about 50% greater risk of hospitalization for schizophrenia, mood disorder, and alcohol-related disorders (Gale et al. 2010) and for personality disorders (Moran et al. 2009). Lower childhood intelligence is also associated in early to middle adulthood with more self-reported psychological distress (Gale et al. 2009) and with a greater risk of vascular dementia (McGurn et al. 2008).

Although demonstrating, replicating, and partially refining the association between intelligence and illness and mortality has been a substantial achievement in the past ten years, the major interest now lies in understanding the association. Finding its mechanisms will be important for applying these discoveries in public health. The attempt to understand has seen differential psychologists work more closely with epidemiologists than before and has also seen the two professions challenge each other. One challenge came from what is arguably cognitive epidemiology's most comprehensive theoretical statement, in which it was suggested that the associations between health and education and socioeconomic status might be caused by intelligence (Gottfredson 2004). Direct tests of this idea with large epidemiological samples from the United Kingdom suggest that the hypothesis has some merit but does not fully account for the effects (Batty et al. 2006, Singh-Manoux et al. 2005). A promising route toward understanding has been the finding that childhood intelligence is associated with many health behaviors during the life course: smoking, alcohol intake, physical activity, and dietary choices (Batty et al. 2007, Deary et al. 2010b, Weiser et al. 2009); risk factors for cardiovascular disease, including the metabolic syndrome as well as its elements such as poor glucose regulation, higher blood pressure, high waist-hip ratio, and disadvantageous lipid profile (Batty et al. 2008, Power et al. 2010); and a disadvantageous diurnal cortisol profile in middle age (Power et al. 2008).

There are other suggestions to explain the intelligence-mortality association. For example, it has been suggested that education and adult social class mediate and explain the association, perhaps in association with stress (Sapolsky 2005). Substantial mediation has been found statistically, but it is moot whether these are explanatory factors or partial surrogates for intelligence (Calvin et al. 2011). The idea that intelligence—even in childhood—relates to later health because it is an index of general bodily "system integrity" achieved some validation when it was found that reaction time variance largely accounted for the association (Deary & Der 2005). The idea needs further development: theoretically, to elaborate more fully the notion of system integrity; and empirically, to identify more marker variables of the construct and the testing of their association with each other, intelligence, and mortality, illness, and health.

INTELLIGENCE AND AGING

This is a topic that is growing in importance in intelligence research, and it also has significant public policy relevance (Beddington et al. 2008). People are living longer, the proportion of older people in the population is growing, and losing cognitive ability is an especially feared aspect of growing older. Research includes the following questions: How do aspects of intelligence change with age? Do all domains of cognitive ability decline together? How much stability and variation is there in intelligence across the life course? Is age kinder to the initially more able? Are there discoverable (and ideally modifiable) determinants of individual differences in the aging of intelligence? And can the decline in age-sensitive aspects intelligence be ameliorated? My opinion is that cognitive aging should be an integrated part of broader intelligence research, not a topic on its own: Intelligence as a whole should be seen as a life-course topic, with developmental psychology, individual differences, and aging aspects contributing to a rounded account of how and why intelligence differences develop, are maintained, and decline (e.g., Foresight Mental Capital Wellbeing Project 2008). Here, some advances over the past decade are highlighted.

Aging research on intelligence has been modeled using the hierarchical account of intelligence differences (Carroll 1993). This is the hierarchy that was described at the start of this article, whereby cognitive ability variance may be portioned into variance shared by all tests (*g*), variance shared by tests assessing the same cognitive domain, and variance specific to each test. A comprehensive example is a combined analysis of 33 cross-sectional studies, involving 16 cognitive tasks, with a total N of about 7,000 (Salthouse 2004). The covariance of the 16 tasks formed a hierarchy with *g* at the pinnacle and group factors of reasoning, spatial ability, memory, processing speed, and vocabulary. Apart from vocabulary tests—which tended to peak at about age 60—the other types of test showed mean declines from young or middle adulthood. It is notable, too, that Salthouse modeled processing speed as one of the domains of intelligence that ages, alongside others at the second level, instead of its being the cause of other domains' aging effects, as he has done previously. The largest effect of age was on general intelligence, with additional, specific smaller effects on memory and processing speed. In a longitudinal study, Wilson et al. (2002) also found that when one aspect of intelligence declines, the other aspects tend to go also. A common factor representing people's aging slopes on seven different cognitive domains accounted for 62% of the variance. A study that is congruent with these, and extended their findings, was by Tucker-Drob (2011). He reported analyses on over 1,200 people aged from 18 to 95 years and who had been tested over a period of up to seven years on a dozen mental tests. Again, the analyses employed the three-level hierarchical model of cognitive differences. The domains of function tested were abstract reasoning, spatial visualization, episodic memory, and processing speed. A general factor—common to all the domains—accounted for an average of 39% of the differences in individual variables, 33% was accounted for at the domain level, and a mean of 28% was test specific. It is notable that the general effect on 'aging' was found even in younger and middle-age groups as well as in older people.

The past decade has seen the longest follow-up studies of intelligence differences, with follow-up studies of the Scottish Mental Surveys of 1932 and 1947. When the same intelligence test is administered at age 11 years and again to individuals when they are in their late seventies, the correlations are between 0.6 and 0.7 (Deary et al. 2000, 2004b) and are still above 0.5 when the individuals are in their late eighties (Gow et al. 2011). Obviously, these correlations imply that at least one-quarter to one-half of the variance in intelligence is stable across most of the human life course. The obverse is that there is also considerable change in the rank order of intelligence across the life course, and there is a lively and varied set of research directions seeking the determinants—psychosocial and biomedical—of aging-associated cognitive change. Overviews of this research are provided by Deary et al. (2009c), Foresight Mental Capital and Wellbeing Project (2008), and Plassman et al. (2010). Plassman and colleagues systematically reviewed observational studies and randomized controlled trials and covered genetics, environmental toxins, medical factors, social and behavioral factors, and nutrition. Support for most factors was limited, although it was better for the risk factors of smoking, the

APOE e4 allele, and some medical conditions. Among the determinants of age-related cognitive change, there is still controversy about whether people with relatively high childhood intelligence have more gentle cognitive decline in middle and old age, with some studies suggesting they do (Richards et al. 2004) and some suggesting that there is no such association (Gow et al. 2011).

With regard to ameliorating the aging of intelligence among people without cognitive pathology, there is still discussion about how to separate normal and pathological cognitive aging, though with a suggestion that the former might be a specific target for therapeutics (Shineman et al. 2010). There is still uncertainty, too, about whether retaining engagement in physical, social, and intellectual activity helps to preserve what intelligence we have (Bielak, 2010), although some are positive about this (Hertzog et al. 2009).

CONTROVERSY OR CONSENSUS?

Two long-running controversies in intelligence research attracted much empirical attention in the past decade: whether—and if so, why—there are twin-singleton or male-female differences in intelligence. Both are discussed below. There was also a major re-examination of the issue of race differences in intelligence, and readers are referred to the target article and its subsequent discussion (Rushton & Jensen 2005).

Two analyses of whole-population or population-representative sets of twins found that, for children aged 11 years in Scotland in the 1930s, 1940s, and 1960s, twins had mean IQ scores of about one-third of a standard deviation lower than singletons (Deary et al. 2005b, Ronalds et al. 2005). However, a population-representative study of 11-year-olds tested on the Cognitive Abilities Test in the United Kingdom in 2004 showed no significant difference: Twins were only about 1% of a standard deviation lower than singletons on general intelligence (Calvin et al. 2009). Studies of more recently born Dutch twins also suggest a much lower—or no—cognitive deficit among twins and that any small deficit probably disappears by adolescence (Webbink et al. 2008). These large, new analyses of recently born children probably override the recent opinion that although there was heterogeneity in studies of singleton–twin intelligence differences, this might not be explained by a date-of-study effect (Voracek & Haubner 2008). This disappearance of the "cognitive cost" of being a twin is fascinating. Some have put this down to better perinatal care, but this has not been demonstrated clearly.

Sex differences in intelligence remains a hot topic, and the past decade saw much debate as well as substantial new analyses. Four issues appear to be important in assessing this question. First is the quality of the samples: Some have much better population representativeness than others. Second is the age of the samples: Some suggest that mean differences appear only after puberty. Third is the type of mental ability: The issue addressed here will be general intelligence rather than abilities such as spatial or verbal that, for whatever causes, tend to be accepted as having more replicable sex differences. Fourth is the research issue at hand: There are questions about whether the mean and/or the variance of intelligence is different between males and females. These will all be kept in mind and referred to as some recent research results on sex differences in intelligence are discussed.

In whole populations of Scottish 11-year-olds from the 1930s and 1940s there was more variance among males in intelligence by comparison with females, but little difference in mean intelligence; proportionately, more males were at both ends of the intelligence distribution and fewer were in the middle (Deary et al. 2003, Johnson et al. 2008b). This pattern was replicated in a population-representative sample of almost one-third of a million boys and girls tested on the Cognitive Abilities Test in the United Kingdom in the early 2000s: There were trivial differences in mean general intelligence and proportionately more boys than girls at the extremes (Strand et al. 2006). This pattern was replicated in the same test in a population-representative sample of 300,000+ students in grades 3 to 11 in the United States, with the authors concluding that, "The results showed an astonishing consistency in sex differences across countries, grades, cohorts, and test forms" (Lohman & Lakin 2009).

Whereas these epidemiological-quality data in children make the state of sex differences clear, the picture is less clear among adults. On the basis of meta-analyses of studies using Raven's Progressive Matrices—a widely used nonverbal intelligence test with items composed using abstract line drawings and demanding inductive reasoning—it was argued that men have a higher mean level of general intelligence than do women (Irwing & Lynn 2005, Lynn & Irwing 2004). It was argued against this that the better-quality studies tended to show no differences and that many of the samples might be biased toward less selectivity among women, which would reduce their mean scores as a group (Blinkhorn 2005). To test this idea, a novel design was applied to data from the U.S. National Longitudinal Survey of Youth 1979 (Deary et al. 2007b). When brother-sister pairs ($N = 1{,}292$ pairs) were compared on a general cognitive ability (*g*) factor derived from the Armed Services Vocational Aptitude Battery and the shorter Armed Forces Qualification Test, the male mean advantage was trivial (less than 7% of a standard deviation), but males once again had substantially greater variance.

A suggestion was made that an apparent male advantage in tests of general intelligence could in part be due to the combination of the following factors: Males have greater variance in intelligence, and recruitment into studies and into subsequent follow-ups of existing studies is restricted by intelligence and sex, with more women and people of higher intelligence tending to take part (Dykiert et al. 2009). The authors argued that the combination of these factors could produce an apparently higher male intelligence mean even when none existed in the population. This was tested in the British Cohort Study 1970 and found to be true: Sex differences in intelligence at age 10 became more biased toward higher male means when only those subjects who took part in subsequent waves were tested. This was extended further—to include a mathematical model of the recruitment process and to analyses of both the 1958 and 1970 British birth cohorts—and it was again found that sample restriction by these means can bring about apparently higher mean intelligence in males even when none is present in the original samples (Madhyastha et al. 2009).

CONCLUDING REMARKS

A desultory reading of this necessarily selective overview of intelligence research in the first decade of the twentieth century—about one hundred years after the first scientific research on intelligence—will lead some readers to echo Jean-Baptiste Alphonse Karr: "*Plus ça change, plus c'est la même chose.*" They would be wrong, for two reasons.

First, yes, some issues do seem to have continued for a very long time: the psychometric structure of intelligence, sex differences in intelligence, environment and genetic contributions to intelligence, the aging of intelligence, and whether intelligence drives educational attainment and social mobility. In all of these, the basics of what we know now were available empirically decades ago. However, there was often so much opposition to the findings—creating uncertainty—that more, newer, and better research has now far more firmly founded the conclusions that can be offered in these areas. In addition, research in most of these areas has added important details to the basic findings that were available early on. In part, these have come from better samples, better research designs, and more sophisticated and appropriate statistical modeling. On that last point, it remains the case that fully to engage in the discussions about intelligence research it is necessary to be numerate, and to quite an advanced level in specialist areas such as aging and environment-genetics.

Second, there are some genuinely new developments. The importance of intelligence for health, illness, and mortality is new. It adds a great deal to the predictive validity of intelligence. The brain associations of intelligence are new: They might not be large in effect size, but they offer foundational findings for a developing biological account of what it means to have higher levels of intelligence. The molecular genetics knowledge about intelligence—albeit largely knowing what the molecular genetics of intelligence is *not* like—is new. Because longitudinal cohorts—first studied in childhood—are still being studied at older ages, we now have a far better knowledge about how intelligence plays out into social as well as health outcomes in middle and older ages. In health, genetics, biology, and social science we are seeing and will see the incorporation of measured intelligence as a phenotype in better samples, of more epidemiological quality: larger, more population-representative, followed up for longer, and better characterized in terms of other phenotypes and genetic information. The past decade has seen the first studies of the stability and change in intelligence that have lasted more than 70 years, health-related studies of over one million men, brain imaging studies with numbers in three figures (instead of two or one), and the first genetic studies using hundreds of thousands of genetic markers. The forthcoming consortia on the genetics of intelligence will have sample sizes in at least five figures, and large studies that will include intelligence and brain imaging and genetics will increasingly appear. In social matters we shall know more too: As cohorts such as the United Kingdom's 1946, 1958, and 1970 cohorts grow older, we shall know more about the lifelong social mobility of these individuals (and their children) and the part played by intelligence in concert with other factors.

Real progress in the cognitive correlates of intelligence has been made over the past decade: It is clear that there is a sensory discrimination–intelligence correlation; the inspection time–intelligence correlation has been firmed up in larger samples and in adults and children; a much larger reaction time–intelligence correlation has emerged than had been appreciated heretofore; and working memory has large correlations and fresh evidence of apparent causal associations with intelligence. Nevertheless, this area is not as active as it was. A mountaineering analogy might help. For those seeking a causal account of intelligence differences, genetic and brain imaging approaches are far more reachable handholds and footholds than they were a decade ago. Perhaps researchers are wondering whether they can simply bypass the intermediate cognitive construct footholds, some of which might be illusory (based on unvalidated constructs) and some of which might take the researcher laterally rather than vertically (because they end up redescribing intelligence in terms at the same explanatory level).

If reorienting is the correct word—or perhaps it is synthesis or consolidation—then a reorienting of intelligence research is possible because of the research in the past decade or two, in terms of a life-course model. The fairly newly appreciated very long-term stability of intelligence, the influences of birth weight and perinatal growth, and the associations among intelligence, sensation, and health all point toward the need for intelligence researchers to be integrated with a wide range of cognate scientists interested in general health and well-being across the human life course. Intelligence is part of health. Whether it is called intelligence, or cognitive capital, or cognitive reserve, and whether we explain these links with ideas of general bodily system integrity or common cause (or a set of common causes), the development, adult operation, and eventual trajectory of decline in people's intelligence will be a combination of shared influences with the rest of the body and—who knows how much?—influences that are specific to the brain. An attempt at displaying the integrated science that lifelong intelligence research should be is shown in a U.K. government report (Foresight Mental Capital and Wellbeing Project 2008, appendix B, p. 53).

And before one hears the old saws that there is more to life than being clever—sure there is, like being happy, healthy, and free—and that there is more to achieving one's desired position than being clever—that's trivially true, as studies looking at personality traits and effort and motivation, for example, show—we should remember that research on the nature, causes, and consequences of intelligence is about something that people value and that has a big influence on people's lives. This brings with it the responsibility to be broad-minded and intelligent in researching intelligence.

SUMMARY POINTS

1. Intelligence differences continue to be a focus for lively research in psychology and also of considerable interest to nonspecialist psychologists, academics in other fields, and the public.
2. The past decade produced many books on intelligence, from introductory accounts to specialist discussions of specific issues. There are also historical accounts and books challenging the measurement and study of intelligence differences.
3. There is new research on the psychometric structure of intelligence. The *g* factor from different test batteries ranks people in the same way. There is still debate about the number of levels at which the variations in intelligence is best described. There is still little empirical support for an account of intelligence differences that does not include *g*.
4. There has been progress in establishing that sensory discrimination, inspection time, and reaction time are all associated with intelligence and achieving estimates of the population effect sizes. However, they now attract less attention as possible ways to understand intelligence differences, although sensory discrimination does attract attention as part of the common cause account of aging and intelligence.
5. The biology of intelligence is the subject of much research. Behavior genetics research continues to refine what we know about environmental and genetic contributions to intelligence, such as moderating effects of age and social circumstances, and the shared genetic influences of intelligence with, for example, brain size, processing speed, and birth weight. Molecular genetic research on intelligence has had a dry time with candidate gene studies and is now poised to take on sufficiently powered genomewide association studies. Brain imaging studies of intelligence are providing more replicated findings that are cohering around an account of a defined but distributed network in the brain that works more efficiently in people with higher intelligence scores.
6. New work on education and social mobility and social position as the outcomes of intelligence differences has plotted people's life courses from impressive longitudinal studies. Health outcomes are a new and burgeoning outcome for intelligence differences, and it is only in the past decade that the new field of cognitive epidemiology has emerged.
7. Aging is another expanding focus for intelligence research, with new findings. Also, this field increasingly takes a life-course view and is becoming more integrated with the study of intelligence differences in younger adulthood and in child development.
8. Controversial issues continue to be studied in intelligence. One such issue is the changing twin–singleton intelligence difference. Also, sex differences in intelligence continue to attract new research, with studies of both mean and variance differences.

FUTURE ISSUES

1. Molecular genetics studies of intelligence are required that are sufficiently powered to discover the likely very small effects of single-nucleotide polymorphisms in genomewide studies. Thereafter, there will almost certainly be a continued search for other loci of intelligence differences that will require gene sequencing studies, the analysis of copy number variations, gene methylation studies, and transcriptomics, proteomics, and other related studies.
2. Structural and functional brain imaging studies of intelligence are increasing in power and will continue to do so, and will also become more genetically informed.
3. The incorporation of intelligence tests into more cohort studies will enhance the study of social mobility, health, and other life outcomes across the human life course.
4. Intelligence research is increasingly becoming, and should continue to become, an integrated part of understanding the causes and consequences of human development and human lifelong health and well-being.

DISCLOSURE STATEMENT

The author is not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGMENTS

The work was undertaken within the University of Edinburgh Center for Cognitive Ageing and Cognitive Epidemiology, part of the cross-council Lifelong Health and Wellbeing Initiative (G0700704/84698). Funding from the Biotechnology and Biological Sciences Research Council (BBSRC), Engineering and Physical Sciences Research Council (EPSRC), Economic and Social Research Council (ESRC), and Medical Research Council (MRC) is gratefully acknowledged.

LITERATURE CITED

Acton GS, Schroeder DH. 2001. Sensory discrimination as related to general intelligence. *Intelligence* 29:263–71

Banks GC, Batchelor JH, McDaniel MA. 2010. Smarter people are (a bit) more symmetrical: a meta-analysis of the relationship between intelligence and fluctuating asymmetry. *Intelligence* 38:393–401

Barnett JH, Scoriels L, Munafo MR. 2008. Meta-analysis of the cognitive effects of the catechol-O-transferase gene Val158Met polymorphism. *Biol. Psychiatry* 64:137–44

Bartels M, Rietveld MJH, Van Baal GCM, Boomsma DI. 2002. Genetic and environmental influences on the development of intelligence. *Behav. Genet.* 32:237–49

Bartholomew DJ. 2004. *Measuring Intelligence: Facts and Fallacies*. Cambridge, UK: Cambridge Univ. Press

Bartholomew DJ, Deary IJ, Lawn M. 2009. A new lease of life for Thomson's bonds model of intelligence. *Psychol. Rev.* 116:567–79

Batty DJ, Deary IJ, Schoon I, Gale CR. 2007. Mental ability across childhood in relation to risk factors for premature mortality in adult life: the 1970 British Cohort Study. *J. Epidemiol. Comm. Health* 61:997–1003

Batty GD, Der G, Macintyre S, Deary IJ. 2006. Does IQ explain socioeconomic inequalities in health? Evidence from a population based cohort study in the west of Scotland. *Br. Med. J.* 332:580–84

Batty GD, Gale CR, Mortensen LH, Langenberg C, Shipley MJ, et al. 2008. Pre-morbid intelligence, the metabolic syndrome and mortality: the Vietnam Experience Study. *Diabetologia* 51:436–43

Batty GD, Wennerstad KM, Davey Smith G, Gunnell D, Deary IJ, et al. 2009. IQ in late adolescence/early adulthood and mortality by middle age: cohort study of one million Swedish men. *Epidemiology* 20:100–9

Beddington J, Cooper CL, Field J, Goswami U, Huppert FA, et al. 2008. The mental wealth of nations. *Nature* 455:1057–60

Bielak AAM. 2010. How can we not "lose it" if we still don't understand how to "use it"? *Gerontology* 56:507–19

Binet A. 1905. New methods for the diagnosis of the intellectual level of subnormals. *L'Annee Psychol*. 12:191–244. Transl. ES Kite, 1916, in *The Development of Intelligence in Children*. Vineland, NJ: Publ. Training School Vineland

Blinkhorn S. 2005. Intelligence: a gender bender. *Nature* 438:31–32

Brand CR. 1996. *The g Factor*. Chichester, UK: Wiley. (This book was withdrawn by the publisher shortly after publication.)

Breen R, Goldthorpe JH. 2002. Merit, mobility and method: another reply to Saunders. *Br. J. Sociol*. 53:575–82

Burks SV, Carpenter JP, Goette L, Rustichini A. 2009. Cognitive skills affect economic preferences, strategic behavior, and job attachment. *Proc. Natl. Acad. Sci. USA* 106:7745–50

Calvin C, Deary IJ, Fenton C, Roberts BA, Der G, et al. 2011. Intelligence in youth and all-cause mortality: systematic review with meta-analysis. *Int. J. Epidemiol*. In press

Calvin C, Fernandes C, Smith P, Visscher PM, Deary IJ. 2009. Is there still a cognitive cost of being a twin in the UK? *Intelligence* 37:243–48

Carroll JB. 1993. *Human Cognitive Abilities: A Survey of Factor Analytic Studies*. Cambridge, UK: Cambridge Univ. Press

Carson J. 2007. *The Measure of Merit: Talents, Intelligence, and Inequality in the French and American Republics, 1750–1940*. Princeton, NJ: Princeton Univ. Press

Caspi A, Williams B, Kim-Cohen J, Craig IW, Milne BJ, et al. 2007. Moderation of breastfeeding effects on the IQ by genetic variation in fatty acid metabolism. *Proc. Natl. Acad. Sci. USA* 104:18860–65

Chiang M-C, Barysheva M, Shattuck DW, Lee AD, Madsen SK. 2009. Genetics of brain fiber architecture and intellectual performance. *J. Neurosci*. 29:2212–24

Choi YY, Shamosh NA, DeYoung CG, Lee MJ, Lee J-M, et al. 2008. Multiple bases of human intelligence revealed by cortical thickness and neural activation. *J. Neurosci*. 28:10323–29

Cianciolo AT, Sternberg RJ. 2004. *Intelligence: A Brief History*. Malden, MA: Blackwell

Cirulli ET, Kasperaviciute D, Attix DK, Need AC, Ge D, et al. 2010. Common genetic variation and performance on standardized cognitive tests. *Eur. J. Hum. Genet*. 18:815–20

Cohn LD, Westenberg PM. 2004. Intelligence and maturity: meta-analytic evidence for the incremental and discriminant validity of Loevinger's measure of ego development. *J. Personal. Soc. Psychol*. 86:760–72

Colom R, Jung RE, Haier RJ. 2006. Distributed brain sites for the *g*-factor of intelligence. *NeuroImage* 31:1359–65

Cronbach LJ. 1957. The two disciplines of scientific psychology. *Am. Psychol*. 12:671–84

Davies G, Tenesa A, Payton A, Yang J, Harris SE, et al. 2011. Genome-wide association studies establish that human intelligence is highly heritable and polygenic. *Mol. Psychiatry*. doi:10.1038/mp.2011.85. In press

Davis OS, Butcher LM, Docherty SJ, Meaburn EL, Curtis CJ, et al. 2010. A three-stage genome-wide association study of general cognitive ability: hunting the small effects. *Behav. Genet*. 40:759–67

Davis OS, Haworth CM, Plomin R. 2009. Dramatic increase in heritability of cognitive development from early to middle childhood: an 8-year longitudinal study of 8700 pairs of twins. *Psychol. Sci*. 20:1301–8

Deary IJ. 2000. *Looking Down on Human Intelligence: From Psychometrics to the Brain*. Oxford, UK: Oxford Univ. Press

Deary IJ. 2001. *Intelligence: A Very Short Introduction*. Oxford, UK: Oxford Univ. Press

Deary IJ, Batty GD. 2007. Cognitive epidemiology: a glossary. *J. Epidemiol. Comm. Health* 61:378–84

Deary IJ, Batty GD, Gale CR. 2008. Bright children become enlightened adults. *Psychol. Sci*. 19:1–6

Deary IJ, Bell PJ, Bell AJ, Campbell ML, Fazal ND. 2004a. Sensory discrimination and intelligence: testing Spearman's other hypothesis. *Am. J. Psychol*. 117:1–19

Deary IJ, Corley J, Gow AJ, Harris SE, Houlihan LM, et al. 2009a. Age-associated cognitive decline. *Br. Med. Bull*. 92:135–52

Deary IJ, Der G. 2005. Reaction time explains IQ's association with death. *Psychol. Sci*. 16:64–69

Deary IJ, Der G, Ford G. 2001. Reaction times and intelligence differences: a population-based cohort study. *Intelligence* 29:389–99

Deary IJ, Irwing P, Der G, Bates TC. 2007b. Brother-sister differences in the *g* factor in intelligence: analysis of full, opposite-sex siblings from the NLSY1979. *Intelligence* 35:451–56

Deary IJ, Johnson W, Houlihan LM. 2009b. Genetic foundations of human intelligence. *Hum. Genet*. 126:215–32

Deary IJ, Pattie A, Wilson V, Whalley LJ. 2005b. The cognitive cost of being a twin: two whole-population surveys. *Twin Res. Hum. Genet*. 8:376–83

Deary IJ, Penke L, Johnson W. 2010a. The neuroscience of human intelligence differences. *Nat. Rev. Neurosci*. 11:201–11

Deary IJ, Spinath FM, Bates TC. 2006. Genetics of intelligence. *Eur. J. Hum. Genet*. 14:690–700

Deary IJ, Strand S, Smith P, Fernandes C. 2007a. Intelligence and educational achievement. *Intelligence* 35:13–21

Deary IJ, Taylor MD, Hart CL, Wilson V, Davey Smith G, et al. 2005a. Intergenerational social mobility and mid-life status attainment: influences of childhood intelligence, childhood social factors, and education. *Intelligence* 33:455–72

Deary IJ, Thorpe G, Wilson V, Starr JM, Whalley LJ. 2003. Population sex differences in IQ at age 11: the Scottish Mental Survey 1932. *Intelligence* 31:533–42

Deary IJ, Weiss A, Batty GD. 2010b. Intelligence and personality as predictors of illness and death: how researchers in differential psychology and chronic disease epidemiology are collaborating to understand and address health inequalities. *Psychol. Sci. Publ. Interest* 11:53–79

Deary IJ, Whalley LJ, Lemmon H, Crawford JR, Starr JM. 2000. The stability of individual differences in mental ability from childhood to old age: follow-up of the 1932 Scottish Mental Survey. *Intelligence* 28:49–55

Deary IJ, Whalley LJ, Starr JM. 2009c. *A Lifetime of Intelligence: Follow-up Studies of the Scottish Mental Surveys of 1932 and 1947*. Washington, DC: Am. Psychol. Assoc.

Deary IJ, Whiteman MC, Starr JM, Whalley LJ, Fox HC. 2004b. The impact of childhood intelligence in later life: following up the Scottish Mental Surveys of 1932 and 1947. *J. Personal. Soc. Psychol*. 86:130–47

Der G, Batty GD, Deary IJ. 2006. Effect of breast feeding on intelligence in children: prospective study, sibling pairs analysis, and meta-analysis. *Br. Med. J*. 333:945–48

Duckworth AL, Quinn PD, Lynam DR, Loeber R, Stouthamer-Loeber M. 2011. Role of test motivation in intelligence testing. *Proc. Natl. Acad. Sci. USA* 108:7716–20

Duckworth AL, Seligman MEP. 2005. Self-discipline outdoes IQ in predicting academic performance of adolescents. *Psychol. Sci*. 16:939–44

Duncan J. 2010. *How Intelligence Happens*. New Haven, CT: Yale Univ. Press

Duncan J, Seitz RJ, Kolodny J, Bor D, Herzog H, et al. 2000. A neural basis for general intelligence. *Science* 289:457–60

Dykiert D, Gale CR, Deary IJ. 2009. Are apparent sex differences in mean IQ scores created in part by sample restriction and increased male variance? *Intelligence* 37:42–47

Flynn J. 2007. *What Is Intelligence?* Cambridge, UK: Cambridge Univ. Press

Foresight Mental Capital and Wellbeing Project. 2008. *Final Project Report—Executive Summary*. London: Gov. Off. Sci.

Friedman NP, Miyake A, Young SE, DeFries JC, Corley RP, et al. 2008. Individual differences in executive functions are almost entirely genetic in origin. *J. Exp. Psychol.: Gen*. 137:201–25

Frisoni GB, Galluzzi S, Pantoni L, Filippi M. 2007. The effect of white matter lesions on cognition in the elderly: small but detectable. *Nat. Clin. Pract. Neurol*. 3:620–27

Furlow B, Armijo-Prewitt T, Gangestad SW, Thornhill R. 1997. Fluctuating asymmetry and psychometric intelligence. *Proc. R. Soc. Lond. B* 264:823–29

Gale CR, Batty GD, Tynelius P, Deary IJ, Rasmussen F. 2010. Intelligence in early adulthood and subsequent hospitalization for mental disorders. *Epidemiology* 21:70–77

Gale CR, Hatch SL, Batty GD, Deary IJ. 2009. Intelligence in childhood and risk of psychological distress in adulthood: the National Child Development Survey and the 1970 British Cohort Study. *Intelligence* 37:592–99

Gardner H. 1983. *Frames of Mind*. New York: Basic Books

Gardner H. 1993. *Multiple Intelligences*. New York: Basic Books

Garlick D. 2010. *Intelligence and the Brain: Solving the Mystery of Why People Differ in IQ and How a Child Can Be a Genius*. Burbank, CA: Aesop

Geary DC. 2005. *The Origin of Mind: Evolution of Brain, Cognition, and General Intelligence*. Washington, DC: Am. Psychol. Assoc.

Gläscher J, Rudrauf D, Colom R, Paul LK, Tranel D, et al. 2010. Distributed neural system for general intelligence revealed by lesion mapping. *Proc. Natl. Acad. Sci. USA* 107:4705–9

Gottfredson LS. 2004. Intelligence: is it the epidemiologists' elusive "fundamental cause" of social class inequalities in health? *J. Personal. Soc. Psychol*. 86:174–99

Gow AJ, Johnson W, Pattie A, Brett CE, Roberts B, et al. 2011. Stability and change in intelligence from age 11 to ages 70, 79, and 87: the Lothian Birth Cohorts of 1921 and 1936. *Psychol. Aging* 26:232–40

Gray JR, Chabris CF, Braver TS. 2003. Neural mechanisms of general fluid intelligence. *Nat. Neurosci*. 6:316–22

Haier RJ. 2009. Neuro-intelligence, neuro-metrics and the next phase of brain imaging studies. *Intelligence* 37:121–23

Herrnstein RJ, Murray C. 1994. *The Bell Curve: Intelligence and Class Structure in American Life*. New York: Free Press

Hertzog C, Kramer AF, Wilson RS, Lindenberger U. 2009. Enrichment effects on adult cognitive development. *Psychol Sci. Publ. Int*. 9:1–65

Hulshoff Pol H, Schnack HG, Posthuma D, Mandl RCW, Barré WF, et al. 2006. Genetic contributions to human brain morphology and intelligence. *J. Neurosci*. 26:10235–42

Hunt E. 2011. *Human Intelligence*. Cambridge, UK: Cambridge Univ. Press

Intelligence. 2009. Special issue. Intelligence, health and death: the emerging field of cognitive epidemiology. *Intelligence* 37:6(whole issue)

Irwing P, Lynn R. 2005. Sex differences in means and variability on the progressive matrices in university students: a meta-analysis. *Br. J. Psychol*. 96:505–24

Jensen AR. 1998. *The g Factor*. New York: Praeger

Johnson W, Bouchard TJ. 2005a. The structure of human intelligence: It is verbal, perceptual, and image rotation (VPR), not fluid and crystallized. *Intelligence* 33:393–416

Johnson W, Bouchard TJ. 2005b. Constructive replication of the visual [*sic*]-perceptual-image rotation model in Thurstone's (1941) battery of 60 tests of mental ability. *Intelligence* 33:417–30

Johnson W, Bouchard TJ, Krueger RF, McGue M, Gottesman II. 2004. Just one *g*: consistent results from three test batteries. *Intelligence* 32:95–107

Johnson W, Bouchard TJ, McGue M, Segal NL, Tellegen A, et al. 2007. Genetic and environmental influences on the Verbal-Perceptual-Image Rotation (VPR) model of the structure of mental abilities in the Minnesota Study of Twins Reared Apart. *Intelligence* 35:542–62

Johnson W, Carothers A, Deary IJ. 2008a. Sex differences in variability in general intelligence: a new look at the old question. *Perspect. Psychol. Sci*. 3:518–31

Johnson W, Gow AJ, Corley J, Starr JM, Deary IJ. 2010. Location in cognitive and residential space at age 70 reflects a lifelong trait over parental and environmental circumstances: the Lothian Birth Cohort 1936. *Intelligence* 38:402–11

Johnson W, Segal NL, Bouchard TJ. 2008b. Fluctuating asymmetry and general intelligence: no genetic or phenotypic association. *Intelligence* 36:279–88

Johnson W, te Nijenhuis J, Bouchard TJ. 2008c. Still just 1 *g*: consistent results from five test batteries. *Intelligence* 36:81–95

Jung RE, Gasparovic C, Chavez RS, Caprihian A, Barrow R, et al. 2009. Imaging intelligence with magnetic resonance spectroscopy. *Intelligence* 37:192–98

Jung RE, Haier RJ. 2007. The Parieto-Frontal Integration Theory (P-FIT) of intelligence: converging neuroimaging evidence. *Behav. Brain Sci*. 30:135–54, discussion 154–87

Karama S, Ad-Dab'bagh Y, Haier RJ, Deary IJ, Lyttelton OC, et al. 2009. Positive association between cognitive ability and cortical thickness in a representative US sample of healthy 6 to 18 year-olds. *Intelligence* 37:145–55

Keller MC, Miller G. 2007. Resolving the paradox of common, harmful, heritable mental disorders: Which evolutionary genetic models work best? *Behav. Brain Sci*. 29:385–404

Lango Allen H, Estrada K, Lettre G, Berndt SI, Weedon MN, et al. 2010. Hundreds of variants clustered in genomic loci and biological pathways affect human height. *Nature* 467:832–38

Lee T, Henry JD, Trollor JN, Sachdev PS. 2010. Genetic influences on cognitive functions in the elderly: a selective review of twin studies. *Brain Res. Rev*. 64:1–13

Lohman DF. 1994. Component scores as residual variation (or why the intercept correlates best). *Intelligence* 19:1–11

Lohman DF, Lakin JM. 2009. Consistencies in sex differences on the Cognitive Abilities Test across countries, grades, test forms, and cohorts. *Br. J. Educ. Psychol*. 79:389–407

Luciano M, Posthuma D, Wright M, de Geus EJC, Smith GA, et al. 2005. Perceptual speed does not cause intelligence, and intelligence does not cause perceptual speed. *Biol. Psychol*. 70:1–8

Luciano M, Wright MJ, Duffy DL, Wainwright MA, Zhu G, et al. 2006. Genome-wide scan of IQ finds significant linkage to a quantitative trait locus on 2q. *Behav. Genet*. 36:45–55

Luciano M, Wright MJ, Geffen GM, Geffen LB, Smith GA, et al. 2004a. A multivariate genetic analysis of cognitive abilities in an adolescent twin sample. *Aust. J. Psychol*. 56:79–88

Luciano M, Wright M, Martin N. 2004b. Exploring the etiology of the association between birthweight and IQ in an adolescent twin sample. *Twin Res*. 7:62–71

Lynn R, Irwing P. 2004. Sex differences on the progressive matrices: a meta-analysis. *Intelligence* 32:481–98

Lynn R, Vanhanen T. 2002. *IQ and the Wealth of Nations*. Westport, CT: Praeger

Lyons MJ, York TP, Franz CE, Grant MD, Eaves LJ, et al. 2009. Genes determine stability and the environment determines change in cognitive ability during 35 years of adulthood. *Psychol. Sci*. 20:1146–52

Mackintosh NJ. 1996. Science struck dumb. *Nature* 381:33

Madhyastha TM, Hunt E, Deary IJ, Gale CR, Dykiert D. 2009. Recruitment modeling applied to longutidinal studies of group differences in intelligence. *Intelligence* 37:422–27

McDaniel MA. 2005. Big-brained people are smarter: a meta-analysis of the relationship between in vivo brain volume and intelligence. *Intelligence* 33:337–46

McGurn B, Deary IJ, Starr JM. 2008. Childhood cognitive ability and risk of late-onset Alzheimer and vascular dementia. *Neurology* 71:1051–56

Miller GF. 2000. *The Mating Mind*. New York: Doubleday

Moran P, Klinteberg BA, Batty GD, Vagero D. 2009. Childhood intelligence predicts hospitalization with personality disorder in adulthood: evidence from a population-based study in Sweden. *J. Personal. Disord*. 23:535–40

Murdoch S. 2007. *IQ: The Smart History of a Failed Idea*. Hoboken, NJ: Wiley

Narr KL, Woods RP, Thompson PM, Szeszko P, Robinson D, et al. 2007. Relationships between IQ and regional cortical gray matter thickness in healthy adults. *Cereb. Cortex* 17:2163–71

Nettle D. 2003. Intelligence and class mobility in the British population. *Br. J. Psychol*. 94:551–61

Neubauer AC, Fink A. 2009. Intelligence and neural efficiency. *Neurosci. Biobehav. Rev*. 33:1004–23

Nisbett R. 2009. *Intelligence and How to Get It*. New York: Norton

O'Toole BI, Stankov L. 1992. Ultimate validity of psychological tests. *Personal. Individ. Differ*. 13:699–716

Payton A. 2009. The impact of genetic research on our understanding of normal cognitive aging: 1995 to 2009. *Neuropsychol. Rev*. 19:451–77

Penke L, Denissen JJA, Miller GF. 2007. The evolutionary genetics of personality. *Eur. J. Personal*. 21:549–87

Plassman BL, Williams JW, Burke JR, Holsinger T, Benjamin S. 2010. Systematic review: factors associated with risk for and possible prevention of cognitive decline in later life. *Ann. Int. Med*. 153:182–93

Plomin R, Spinath F. 2004. Intelligence: genetics, genes and genomics. *J. Personal. Soc. Psychol*. 86:112–29

Posthuma D, de Geus EJC, Barré WF, Hulshoff Pol HE, Kahn RS, et al. 2002. The association between brain volume and intelligence is of genetic origin. *Nat. Neurosci*. 5:83–84

Posthuma D, de Geus EJC, Boomsma DI. 2001. Perceptual speed and IQ are associated through common genetic factors. *Behav. Genet*. 31:593–602

Posthuma D, Luciano M, de Geus EJC, Wright MJ, Slagboom PE, et al. 2005. A genomewide scan for intelligence identifies quantitative trait loci on 2q and 6p. *Am. J. Hum. Genet*. 77:318–26

Power C, Jefferis BJ, Manor O. 2010. Childhood cognition and risk factors for cardiovascular disease in midadulthood: the 1958 British Birth Cohort Study. *Am. J. Public Health* 100:129–36

Power C, Li L, Hertzman C. 2008. Cognitive development and cortisol patterns in mid-life: findings from a British birth cohort. *Psychoneuroendocrinology* 33:530–39

Reynolds CA, Finkel D, McArdle JJ, Gatz M, Berg S, et al. 2005. Quantitative genetic analysis of latent growth curve models of cognitive abilities in adulthood. *Dev. Psychol*. 41:3–16

Richards M, Shipley B, Fuhrer R, Wadsworth MEJ. 2004. Cognitive ability in childhood and cognitive decline in midlife: longitudinal birth cohort study. *Br. Med. J.* 328:552–56

Rijsdijk FV, Vernon PA, Boomsma DI. 2002. Application of hierarchical genetic models to Raven and WAIS subtests: a Dutch twin study. *Behav. Genet*. 32:199–210

Ronalds GA, De Stavola BL, Leon DA. 2005. The cognitive cost of being a twin: evidence from comparisons within families in the Aberdeen children of the 1950s cohort. *Br. Med. J.* 331:1306

Rushton JP, Ankney CD. 2009. Whole brain size and general mental ability: a review. *Int. J. Neurosci*. 119:691–731

Rushton JP, Jensen AR. 2005. Thirty years of research on race differences in cognitive ability. *Psychol. Publ. Policy Law* 11:235–94

Salthouse TA. 2004. Localizing age-related individual differences in a hierarchical structure. *Intelligence* 32:541–61

Salthouse TA. 2010. *Major Issues in Cognitive Aging*. Oxford, UK: Oxford Univ. Press

Sapolsky RM. 2005. The influence of social hierarchy on primate health. *Science* 308:648–52

Saunders P. 2002. Reflections on the meritocracy debate in Britain: a response to Richard Breen and John Goldthorpe. *Br. J. Sociol*. 53:559–74

Saunders P. 2010. *Social Mobility Myths*. London: Civitas

Schaie KW. 2005. *Developmental Influences on Adult Intelligence: The Seattle Longitudinal Study*. Oxford, UK: Oxford Univ. Press

Schoon I, Cheng H, Gale CR, Batty GD, Deary IJ. 2010. Social status, cognitive ability, and educational attainment as predictors of liberal social attitudes and political trust. *Intelligence* 38:144–50

Shaw P, Greenstein D, Lerch J, Clasen L, Lenroot R, et al. 2006. Intellectual ability and cortical development in children and adolescents. *Nature* 440:676–79

Shenk D. 2010. *The Genius in All of Us: Why Everything You've Been Told About Genetics, Talent and Intelligence is Wrong*. London: Icon

Shenkin SD, Starr JM, Deary IJ. 2004. Birth weight and cognitive ability in childhood: a systematic review. *Psychol. Bull*. 130:989–1013

Sheppard LD. 2008. Intelligence and information processing: a review of 50 years of research. *Personal. Individ. Differ*. 44:533–49

Shineman DW, Salthouse TA, Launer LJ, Hof PR, Bartzokis G, et al. 2010. Therapeutics for cognitive aging. *Ann. N. Y. Acad. Sci*. 1191:E1–10

Singh-Manoux A, Ferrie JE, Lynch JW, Marmot M. 2005. The role of cognitive ability (intelligence) in explaining the association between socioeconomic position and health: evidence from the Whitehall II prospective cohort study. *Am. J. Epidemiol*. 161:831–39

Spearman C. 1904. "General intelligence" objectively determined and measured. *Am. J. Psychol*. 15:201–93

Stanovich KE. 2009. *What Intelligence Tests Miss: The Psychology of Rational Thought*. New Haven, CT: Yale Univ. Press

Steer CD, Davey Smith G, Emmett PM, Hibbeln JR, Golding J. 2010. FADS2 polymorphisms modify the effect of breastfeeding on child IQ. *PLoS One* 5:e11570

Sternberg RJ, Grigorenko EL. 2002. *The General Factor of Intelligence: How General Is It?* Mahwah, NJ: Erlbaum

Strand S, Deary IJ, Smith P. 2006. Sex differences in cognitive abilities test scores: a UK national picture. *Br. J. Educ. Psychol*. 76:463–80

Strenze T. 2007. Intelligence and socioeconomic success: a meta-analytic review of longitudinal research. *Intelligence* 35:401–26

Thompson PM, Cannon TD, Narr KL, van Erp T, Poutanen VP, et al. 2001. Genetic influences on brain structure. *Nat. Neurosci*. 4:1253–58

Tucker-Drob EM. 2011. Global and domain-specific changes in cognition throughout adulthood. *Dev. Psychol.* 47:331–43

Turkheimer E, Haley A, Waldron M, D'Onfrio BM, Gottesman II. 2003. Socioeconomic status modified heritability of IQ in young children. *Psychol. Sci.* 14:623–28

van der Maas HLJ, Dolan CV, Grasman RP, Wicherts JM, Huizenga HM, Raijmakers ME. 2006. A dynamical model of general intelligence: the positive manifold of intelligence by mutualism. *Psychol. Rev.* 13:842–60

Van Valen L. 1962. A study of fluctuating asymmetry. *Evolution* 16:125–42

Vernon PE. 1956. *The Measurement of Abilities*. London: Univ. London Press

Vinkhuyzen AAE, van der Sluis S, Posthuma D. 2011. Life events moderate variation in cognitive ability (*g*) in adults. *Mol. Psychiatry* 16:4–6

Visser BA, Ashton MC, Vernon PA. 2006. Beyond *g*: putting multiple intelligences to the test. *Intelligence* 34: 487–502

von Stumm S, Gale CR, Batty GD, Deary IJ. 2009. Childhood intelligence, behaviour, and locus of control as determinants of intergenerational social mobility: the British Cohort Study 1970. *Intelligence* 37:329–40

von Stumm S, Macintyre S, Batty GD, Clark H, Deary IJ. 2010. Intelligence, social class of origin, childhood behavior disturbance and education as predictors of status attainment in midlife in men: the Aberdeen Children of the 1950s study. *Intelligence* 38:202–11

Voracek M, Haubner T. 2008. Twin-singleton differences in intelligence: a meta-analysis. *Psychol. Rep.* 102:951–62

Waiter GD, Deary IJ, Staff RT, Murray AD, Fox HC, et al. 2009. Exploring possible neural mechanisms of intelligence differences using processing speed and working memory tasks: an fMRI study. *Intelligence* 37:199–206

Webbink D, Posthuma D, Boomsma DI, de Geus EJC, Visscher PM. 2008. Do twins have lower cognitive ability than singletons? *Intelligence* 36:539–47

Weiser M, Zarka S, Wrbeloff N, Kravitz E, Lubin G. 2009. Cognitive test scores in male adolescent cigarette smokers compared to non-smokers: a population-based study. *Addiction* 105:358–63

Whalley LJ, Deary IJ. 2001. Longitudinal cohort study of childhood IQ and survival up to age 76. *Br. Med. J.* 322:819–22

White J. 2006. *Intelligence, Destiny and Education: The Ideological Roots of Intelligence Testing*. London: Routledge

Wilhelm O, Engle RW, eds. 2005. *Handbook of Measuring and Understanding Intelligence*. London: Sage

Wilson RS, Beckett LA, Barnes LL, Schneider JA, Bach J, et al. 2002. Individual differences in rates of change in cognitive abilities of older persons. *Psychol. Aging* 17:179–93

Wisdom NM, Callahan JL, Hawkins KA. 2011. The effects of apolipoprotein E on non-impaired cognitive function: a meta-analysis. *Neurobiol. Aging* 32:63–74

Woolley AW, Chabris CF, Pentland A, Hashmi N, Malone TW. 2010. Evidence for a collective intelligence factor in the performance of human groups. *Science* 330:686–88

Wright Gillham N. 2001. *A Life of Sir Francis Galton: From African Exploration to the Birth of Eugenics*. Oxford, UK: Oxford Univ. Press

Yeo RA, Gangestad SW, Liu J, Calhoun VD, Hutchison KE. 2011. Rare copy number deletions predict individual variation in intelligence. *PLoS One* 6:e16339

Zhang J-P, Burdick KE, Lencz T, Malhotra AK. 2010. Meta-analysis of genetic variation in DTNBP1 and general cognitive ability. *Biol. Psychiatry* 68:1126–33

Annual Review of
Psychology

Volume 63, 2012

Contents

Prefatory

Developmental Psychobiology

Memory

Judgment and Decision Making

Brain Imaging/Cognitive Neuroscience

Neuroscience of Learning

Comparative Psychology

Emotional, Social, and Personality Development

Adulthood and Aging

Development in Societal Context

Social Development, Social Personality, Social Motivation, Social Emotion

Social Neuroscience

Personality Processes

Work Attitudes

Job/Work Analysis

Education of Special Populations

Human Abilities

Research Methodology

Neuroscience Methods

Indexes

Errata

An online log of corrections to *Annual Review of Psychology* articles may be found at http://psych.AnnualReviews.org/errata.shtml