Book Reviews

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IQ and Human Intelligence. By Nicholas J. Mackintosh. Oxford: Oxford University Press, 1998. Pp. 419. $98.00 (cloth); $35.00 (paper).

To many geneticists, general cognitive ability (*g*), usually called "intelligence" or "IQ," might seem to be a most unlikely trait to take seriously. More than any other trait, it is associated with controversy, both scientific and political, which was seen most recently after the publication of *The Bell Curve* (Herrnstein and Murray 1994). How can something as nebulous as *g* be measured? How can it possibly be useful in genetic analyses?

Books such as this by Nicholas Mackintosh will aid the scientific rehabilitation of *g* (also see Brody 1992; Seligman 1992; Sternberg and Grigorenko 1997; Jensen 1998). Mackintosh is Chair of the Department of Experimental Psychology at Cambridge University and is a distinguished animal-learning theorist. That he comes to examine the field of intelligence as an outsider with no ax to grind will increase the book's impact for a wide audience, with his endorsement of the importance of *g* and the genetics that underlies it. Mackintosh's book is a good introduction to this field, in its description of the evidence for *g*'s validity, reliability, stability, and heritability. The book also includes a good summary of what we know (and, mostly, do not know) about environmental effects on *g;* it tackles the fraught issue of group differences and is especially helpful in its attempt to bring together *g* and cognitive psychology, two fields of mental functioning that have kept their distance. Because the book provides a broad overview of these areas, it does not discuss all issues related to genetics (see below), nor does it address neuroscience research on learning and memory, as seen in synaptic plasticity such as long-term potentiation, an area in which rapid progress is being made in genetic analysis (Migaud et al. 1998).

Measures of cognitive abilities as diverse as spatial ability (Mervis et al. 1999 [in this issue]), verbal ability, and memory intercorrelate moderately, with correlation coefficients of ~.50. However, it is not known what lies at the core of this general cognitive ability, whether it is a general process, such as executive function or a speedy brain, or whether it derives from overlapping component processes. Mackintosh favors the latter view. Regarding genetic research, Mackintosh agrees with other reviews in concluding that *g* is substantially heritable. More quantitative genetic research is available for *g* than for any other dimension or disorder—not just for behavior but for any domain of the life sciences. Dozens of studies, including >8,000 parent-offspring pairs, >25,000 pairs of siblings, >10,000 twin pairs, and 100s of adoptive families, all converge on the conclusion that the heritability of *g* is ~50% (Bouchard and McGue 1981). Sorting the results by age suggests that heritability increases from ~20% in infancy to ~40% in childhood, to ⩾60% later in life (McGue et al. 1993), even for individuals ⩾80 years of age (McClearn et al. 1997). Nearly all of the genetic variance is additive—that is, genetic effects add up rather than interact across loci and, thus, "breed true" from parent to offspring.

An interesting finding from genetic research, which Mackintosh mentions, only in passing, as posing a problem in the estimation of the heritability of *g*, is that there is greater assortative mating for *g* than for any other behavioral trait; that is, spouse correlations are only ~.1 for personality and only ~.2 for height or weight, but the correlation for assortative mating for *g* is ~.4. In addition to indicating that people are able to make judgments about *g* in real life, this finding suggests that assortative mating may contribute to the substantial additive genetic variance for *g,* because positive assortative mating for a character can increase its additive genetic variance.

One of the most surprising genetic findings about *g* during the past decade is not mentioned by Mackintosh, even though this finding has major implications for his attempt to understand the cognitive processes that underlie *g*. Work on genetic influences on intelligence has, to date, focused on *g;* we know much less about the genetic and environmental origins of individual differences in such specific cognitive abilities as spatial ability, verbal ability, memory, and processing speed. Specific cognitive abilities show substantial genetic influence, although it is less than that for *g* (Plomin and DeFries 1998). To what extent do different sets of genes affect these phenotypically different abilities? A technique called "multivariate genetic analysis" examines covariance among specific cognitive abilities and yields a statistic called "the genetic correlation," which is the extent to which genetic effects on one trait correlate with genetic effects on another trait, independent of the heritability of the two traits. In other words, although cognitive abilities are moderately heritable, the genetic correlations between them could be anywhere from .0, indicating complete independence, to 1.0, indicating that the same genes influence a variety of cognitive abilities. Multivariate analyses have shown that there is substantial genetic influence on each of these individual abilities, although it is less than that for *g* (Plomin and DeFries 1998). More surprisingly, such analyses have consistently found that genetic correlations among specific cognitive abilities are very high—close to 1.0 (Petrill et al. 1997).

These genetic results have major implications for current theories of cognitive neuroscience. According to one theory,

the brain works in a modular fashion—that is, cognitive processes are specific and independent. Implicit in this perspective is a bottom-up reductionist view of genetics, in which individual modules are the targets of gene action. The findings from multivariate genetic analyses suggest a top-down view, in which genetic effects operate primarily on *g*, rather than suggest a bottom-up view, in which genetic effects are specific to modules. Given that the brain has evolved to learn from a variety of experiences and to solve a variety of problems, perhaps it makes sense that it would function holistically. However, finding genetic correlations near 1.0 does not prove that genetic effects are limited to a single general cognitive process that works in a top-down way. Another alternative is that specific cognitive abilities, as they are currently assessed, might tap many of the same modular processes that are each affected by different sets of genes. This alternative hypothesis could be tested by means of multivariate genetic research on measures of modular processes, such as neuroimaging measures of brain function (Watkins et al. 1999 [in this issue]; Kosslyn and Plomin, in press).

Another direction for genetic research, one that is too new to be mentioned in Mackintosh's book, is the attempt to identify specific genes responsible for the heritability of *g*. DNA associations with *g* have begun to be reported (Chorney et al. 1998), including initial results from a systematic genome scan for association, by means of DNA pooling (Fisher et al. 1999). Neuroscience research with knockout animal models of learning and memory is likely to accelerate research on the molecular genetics of *g*, especially as neuroscientists come to appreciate the broad relevance of *g*. Finding specific genes associated with *g* will facilitate more-precise answers to questions such as modularity. For example, to what extent are genes that are associated with modular processes, such as long-term potentiation, also associated with *g*? Finding genes for *g* will have implications for society as well as for science (Plomin, in press). If, as I predict, *g* will soon take center stage in genetic research on the neuroscience of learning and memory, Mackintosh's excellent overview of research on *g* will be of great help to geneticists and others with an interest in the workings of learning, memory, and intelligence.

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References

Bouchard TJ Jr, McGue M (1981) Familial studies of intelligence: a review. Science 212:1055–1059

Brody N (1992) Intelligence, 2d ed. Academic Press, New York

Chorney MJ, Chorney K, Seese N, Owen MJ, Daniels J, McGuffin P, Thompson LA, et al (1998) A quantitative trait locus (QTL) associated with cognitive ability in children. Psychol Sci 9:159–166

Fisher PJ, Turic D, Williams NM, McGuffin P, Asherson P, Ball D, Craig I, et al (1999) DNA pooling identifies QTLs on chromosome 4 for general cognitive ability in children. Hum Mol Genet 8:915–922

Herrnstein RJ, Murray C (1994) The bell curve: intelligence and class structure in American life. Free Press, New York

Jensen AR (1998) The *g* factor: the science of mental ability. Westport, CT, Praeger

Kosslyn S, Plomin R. Towards a neuro-cognitive genetics: goals and issues. In: Dougherty D, Rauch SL, Rosenbaum JF (eds) Psychiatric neuroimaging strategies: research and clinical applications. American Psychiatric Press,Washington, DC (in press)

McClearn GE, Johansson B, Berg S, Pedersen NL, Ahern F, Petrill SA, Plomin R (1997) Substantial genetic influence on cognitive abilities in twins 80+ years old. Science 276:1560–1563

McGue M, Bouchard TJ, Iacono WG, Lykken DT (1993) Behavioral genetics of cognitive ability: a life-span perspective. In: Plomin R, McClearn GE (eds) Nature, nurture, and psychology. American Psychological Association, Washington, DC, pp 59–76

Mervis CB, Robinson BF, Pani JR (1999) Visuospatial construction. Am J Hum Genet 65:000–000 (in this issue)

Migaud M, Charlesworth P, Dempster M, Webster LC, Watabe AM, Makhinson M, He Y, et al (1998) Enhanced long-term potentiation and impaired learning in mice with mutant postsynaptic density-95 protein. Nature 396:433–439

Petrill SA, Saudino KJ, Cherny SS, Emde RN, Hewitt JK, Fulker DW, Plomin R (1997) Exploring the genetic etiology of low general cognitive ability from 14 to 36 months. Dev Psychol 33:544–548

Plomin R. Genetics and general cognitive ability. Nature (in press)

Plomin R, DeFries JC (1998) Genetics of cognitive abilities and disabilities. Sci Am (May), pp 62–69

Seligman D (1992) A question of intelligence: the IQ debate in America. Carol Publishing, New York

Sternberg RJ, Grigorenko EL (1997) Intelligence: heredity and environment. Cambridge University Press, Cambridge

Watkins KE, Gadian DG, Vargha-Khadem F (1999) Functional and structural brain abnormalities associated with a genetic disorder of speech and language. Am J Hum Genet 65:000–000 (in this issue)

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A Means to an End: The Biological Basis of Aging and Death. By William C. Clark. New York: Oxford University Press, 1999. Pp. 234. $27.50 (cloth).

The questions of how and why we age have great intrinsic intellectual appeal and major societal implications. William Clark, an Emeritus Professor of Immunology at UCLA, has written a popular book in an attempt to introduce the subject to nonspecialists. That he himself is a nonspecialist is probably a good thing, since there is the potential to bring a fresh new perspective. He has succeeded in producing a very readable review that does indeed outline the major ideas. Unfortunately, although he quite properly emphasizes the evolutionary theory of *why* we age, his language in many sections of the book indicates a belief that a genetic program has evolved to *produce* senescence.

All serious students of the evolutionary biology of aging would agree that the senescent phenotypes that emerge in age-structured populations are the result of a decline in the force of natural selection with respect to the age of gene effects (Rose 1991). Two classes of gene action are envisaged. The first class, originally outlined by Haldane and Medawar (1952), includes