LETTERS

Seeing double

A study of elderly Swedish twins—which attempted to unravel the contributions of genes, environment, and aging to cognitive ability—elicits critical comment, as well as suggestions for future research design.

Twin Studies, Heritability, and Intelligence

Unraveling the sources of phenotypic variability for human behavioral traits has been a notoriously elusive goal. Attempts to determine the influence of genes on traits such as general cognition are frustrated by the fact that we know little about how genes and environment combine to shape a developing individual. Although this influence may be quantified in a number of ways, we will use heritability in the narrow sense (*1*), which measures the additive contribution of genes to the variation in a trait observed among members of a specific population. Given a particular model, the heritability of a trait may be estimated from familial correlations, as in the recent study of 240 pairs of elderly twins by Gerald E. McClearn *et al.* (Reports, 6 June, p. 1560). However, there are many possible models, and we have little information about which is the most appropriate for a given trait.

Heritability studies based on twins are particularly suspect because twin correlations alone do not provide sufficient data to disentangle genetic from cultural influences. The most important factor missing from models of the type used by McClearn *et al.* (*2*) is a measure of the environmental correlations among monozygotic (c_{MZ}) and dizygotic (c_{DZ}) twins. The twin research design used by McClearn *et al.* cannot be used to estimate these correlations separately and assumes that they are equal. In an accompanying Perspective (6 June, p. 1522), Irving I. Gottesman summarizes McClearn *et al.*'s results by saying that the heritability of general cognitive ability is high, namely, 62%, but he does not point out that the 95% confidence interval is 29% to 73%. There is also no mention that the statistical model used does not account for environmental differences between monozygotic and dizygotic twins.

We have examined a number of different linear models in an attempt to determine the sensitivity of heritability estimates for IQ to underlying assumptions (Table 1). These estimates, which are commonly used in the literature, vary widely. The most important factor appears to be whether or not twins are used exclusively or whether the observed data include other familial correlations. Heritability estimated from all known familial correlations is substantially lower than heritability estimated from twin data alone. In particular, monozygotic twins resemble one another more closely than expected from the similarity among other relatives (as evidenced by the high estimates of c_{MZ} in Table 1). This may be because there is a substantial correlation in the environmental experiences of monozygotic twins or because of the existence of specific genetic interactions (epistasis) that make genetically identical individuals more alike, but which contribute little to the resemblance of other relatives.

Twin studies such as that of McClearn *et al.* use the most bare-boned models, which produce an inflated estimate of heritability. More detailed models [including our own (*3*)], which include data from a broader array of relatives (*4–6*), have led to much lower estimates for the heritability of intelligence.

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2. The twin correlations used in the report by McClearn *et al.* are based on the first principal component index of general cognitive ability, also discussed in the report. Results similar to those in Table 1 are obtained with the use of twin correlations from the Wechsler Adult Intelligence Scale given by McClearn *et al.* and from a paper by Bouchard and McGue (*6*).
3. S. P. Otto, F. B. Christiansen, M. W. Feldman, *Morrison Institute for Population and Resource Studies Paper No. 64* (Stanford University, Stanford, CA, 1995).
4. C. R. Cloninger, J. Rice, T. Reich, *Am. J. Hum. Genet.* **31**, 176 (1979).

Table 1. Heritability estimates for cognitive ability. The first three estimates are based on correlations among monozygotic and dizygotic twins ($r_{MZ} = 0.75$ and $r_{DZ} = 0.38$, respectively) from the report by McClearn *et al*. The last three estimates combine those twin data with 14 other familial correlations from the study by Bouchard and McGue (*6*). Estimates of heritability from twin data come from Falconer [h^2_F, (*1*), confidence limits based on ± 1.96 SE with the use of (A6.16) from (*7*)], Holzinger [h^2_H, cited in (*8*)], and McClearn *et al*. [h^2_M, maximum likelihood (ML) fit, as they report]. Estimates of heritability based on twins all include dominance and epistatic components to varying degrees and estimate narrow-sense heritability only if those components are small. Analyses of the full set of family correlations incorporate genetic and cultural transmission as well as parameters that estimate the common rearing environment experienced by siblings. These models assume that (i) cognitive ability of the parents has a direct influence on the cognitive development of the child and that the observed correlation among parents (*6*) is a result of assortative mating based on cognitive ability [h^2_{DP}, (*9*)], or that (ii) cognitive ability of the parents has an indirect influence on the child through correlations with other, unobserved traits and that parents mate assortatively on the basis of cognitive ability [h^2_{IP} (*4, 10*)]; or that (iii) cognitive ability is culturally inherited indirectly as in (ii), but assortative mating occurs within social groupings and does not depend directly on cognitive abilities [h^2_{IS}, (*5, 11*)].

Heritability measure	Data base	Estimate of heritability (95% confidence limits)	Environmental correlations	
			c_{MZ}	c_{DZ}
$h^2_F = 2(r_{MZ} - r_{DZ})$	Twin correlations	0.74 (0.26 to 1.22)	(Absent)	
$h^2_H = (r_{MZ} - r_{DZ}) / (1 - r_{DZ})$	Twin correlations	0.60	(Absent)	
h^2_M (ML fit)	Twin covariances	0.62 (0.29 to 0.73)	(Absent)	
h^2_{DP} (ML fit)	Family correlations	0.29 (0.25 to 0.35)	0.52	0.05
h^2_{IP} (ML fit)	Family correlations	0.33 (0.28 to 0.38)	0.65	0.00
h^2_{IS} (ML fit)	Family correlations	0.44 (0.35 to 0.48)	0.57	0.10

5. D. C. Rao, N. E. Morton, J. M. Lalouel, R. Lew, *Genet. Res. Camb.* **39**, 187 (1982).
6. T. J. Bouchard Jr. and M. McGue, *Science* **212**, 1055 (1981).
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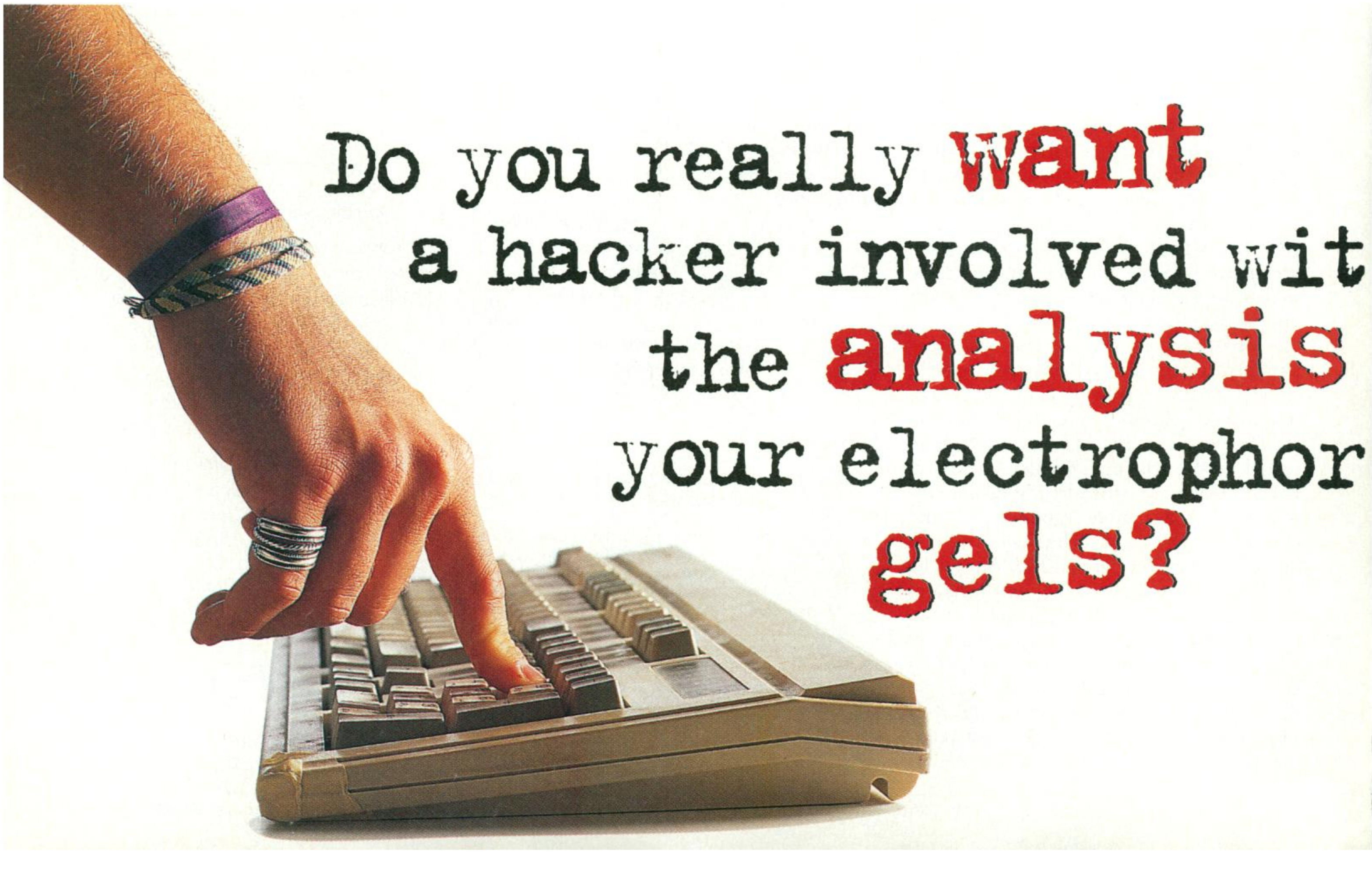
11. D. C. Rao, N. E. Morton, S. Yee, *ibid.* **28**, 228 (1976); N. E. Morton and D. C. Rao, *Yearb. Phys. Anthropol.* **21**, 12 (1978); D. C. Rao, N. E. Morton, C. R. Cloninger, *Genet. Res. Camb.* **33**, 175 (1979).

The assumption made by McClearn *et al.* that differences between identical and fraternal twins (in test scores, for example) are an expression of genetic differences may not be correct. Genetically identical twins often have similar physical characteristics (as expected). However, we have observed that such characteristics often elicit common reactions from caregivers and educators. For example, children with low muscle tone tend to be self-absorbed and elicit less enthusiastic interactions from caregivers because such children do not provide much feedback. Eager, outgoing infants and children often elicit robust, animated caregiver interaction (*1*).

That cognitive and behavioral patterns (such as test scores) may be stable over time might be a result of interaction patterns (that begin with caregiver responses to a



child's physical characteristics and continue throughout one's life. Behavioral genetic studies should therefore include the observation and coding of interaction patterns between children and caregivers.

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McClearn *et al.* compare cognitive test scores of aged Swedish identical and fraternal twins and conclude that the heritability of cognitive ability is 62%. But there is an extensive literature which documents that twins who look very much alike (most identical twins) experience much more similar treatment in their social environmental than do twins who do not (most fraternal twins). Because environmental treatment affects cognitive development, much (if not all) of the greater cognitive resemblance of identical twins may be a consequence of environmental, rather than genetic, similarity.

McClearn *et al.* suggest that heritability increases progressively from 20% in infancy to 60% in adulthood. Their 62% value for old age is said to contradict an "assumption in gerontology that environmental influence increases throughout the life-span." Earlier studies by McClearn and his colleagues (*1*), based on twin data from the same Swedish study (SATSA), indicated a heritability of 80%, which "does not differ across the age range of the SATSA sample, from 50 to 84" (*1*). In 1994 (*2*), a heritability of 80% was again reported by the group, but this finding was described as "consistent with the hypothesis that heritability increases throughout the life-span." And in 1995, Swedish twins aged 65 to 88 were said by the same group to show significantly lower heritability than younger twins (*3*). It is not easy to reconcile these discrepancies.

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The report by McClearn *et al.* that genetic influences on cognitive abilities persist into old age (in 240 senescent twin pairs) confirms our earlier finding in 134 pairs (ages 60 to 90). At first testing (in 1947), mean intrapair differences were larger for dizygotic than for monozygotic pairs in all six tests of the psychological battery and significantly larger ($P < 0.01$) on four of them (*1*). Although the number of surviving pairs retested in 1955 was small, dizygotic pairs still showed greater mean intrapair differences (not statistically significant) in five of six tests (*2*). We concluded "that biological similarities between one-egg twins persist into the senescent period, confirming the influence of basic hereditary factors upon intellectual function . . ." (*2*, p. 308). The same conclusion is reached by McClearn and his co-workers.

We also stated that (i) "no significant change in intrapair correlations was demonstrated during the followup period"; (ii) performance of 60-to-90-year-old twins equaled or exceeded the general population norms for 50-to-59-year-olds; (iii) "heterogeneous age changes . . . on the various tests . . . confirmed by factor analysis," . . . suggest the need for biologically oriented approaches to clarify cognitive changes; and (iv) because of the evidence for positive relationships between test score and survival, and for significant genetic influences on both, this new research road should be explored. To date, it still has not been adequately explored. The importance of understanding cognitive change in our increasingly aged population highlights the need for support of long-term studies.

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Response: Model-fitting meta-analyses of the huge literature of family, twin, and adoption data consistently yield significant estimates of genetic influence on cognitive ability, *g*, with effect sizes (heritabilities) typically estimated to be about 50%, meaning that about half of the observed variance in *g* can be attributed to genetic differences among individuals (*1*, *2*).

We gave results for *g* and specific cognitive abilities (verbal, spatial, speed of processing, and memory abilities) for 240 pairs of twins. Our results are similar to those in the literature, with our best estimate of heritability for *g*, for example, being 62%. There is an implicit, and not unreasonable, hypothesis in gerontology that environmental influence increases throughout the life-span, with a consequent decline in heritability. Our results for twins aged 80 and older indicate that, for cognitive functioning, there is still substantial genetic influence, even among the old-old and oldest-old. Since our report was published, yet another summary of the literature on genetics and IQ has appeared (*3*) that gives an estimate of broad-sense heritability of *g* of 48%, similar to that of previous meta-analyses.

Feldman and Kamin appear to have not changed their views since the early 1970s, despite new evidence confirming significant genetic influence on *g* and other behavioral traits. Feldman and Otto state that "there are many possible models." But fancy model-fitting simply refines the clear pattern of substantial genetic influence that emerges from examination of this body of data. If one considers the basic data for *g* (*4*), why are identical twins more similar than fraternal twins (0.86 rather than 0.60)? Why are identical twins reared apart so similar (0.78)? Why are first-degree relatives adopted apart so similar (0.24)? Feldman and Otto's estimate of heritability (their table 1) from the twin literature yield a number of 74% (first row). Their second estimate apparently uses a formula that has been shown to be inappropriate (*5*), although it provides an estimate of 60%. The third estimate, 62%, appears to be from our study, not from the entire twin literature. However, it is not clear where their other summary estimates come from. Family studies cannot disentangle genetic and environmental influence because family members share nurture as well as nature. Adoption studies directly test the effects of shared nature by studying genetically related individuals separated by adoption and directly test the effects of shared nurture by studying genetically unrelated individuals brought together by adoption. First-degree relatives adopted apart correlate 0.24 for *g*, whereas genetically unrelated individuals adopted together correlate near zero after adolescence. This straightforward comparison suggests a heritability estimate of about 50%, which corresponds to several model-fitting meta-analyses of the entire literature of twin and adoption data (*1*, *2*).

Feldman and Otto, as well as Kamin and Greenspan, raise the issue of the equal environments assumption of the method of studying twins. Much research supports the reasonableness of this assumption (*4*). Contrary to the hypothesis that twins who are more similar in appearance might be more similar in IQ, evidence indicates that identical twins who look more similar are not more similar in IQ or in personality (*6*). The adoption study design, which is a different design, yields results similar to those of the twin study design (*7*).

Although Kamin questions the evidence for the increase in heritability from infancy to adulthood, results from several dozen studies are clear on this unexpected conclusion (*2, 4*). Kamin says that our reported estimate of heritability differs from a previous report "from the same Swedish study." However, the SATSA study used a different and younger sample than our current study of twins 80 or more years of age (*8*). We are less concerned whether the heritability of *g* in old age is 40%, 60%, or 80% than with the conclusion that genetic factors still account for a substantial amount of variance of *g* even very late in life.

Greenspan's prescription is that "behavioral genetic studies should therefore include the observation and coding of interaction patterns between children and caregivers." We agree that these would be valuable data in a comprehensive design with young people. Interpretations of these interactions as "environmental" are contraindicated, however, by the demonstration that genetics plays a major role in family interactions, because parents respond to genetically influenced characteristics of their children (*9*).

Falek and Jarvik note that their study (*10*) yielded results consistent with ours, even though attrition resulted in relatively small sample sizes in advanced ages in this classic study. We agree strongly with the points in their letter and second their call for support of long-term studies that can tackle the important issue of cognitive status and change in our increasingly aged population.

We would like to take this opportunity to correct some errors in our report. On page 1561 (column 3) the percentages of different housing arrangements of twin pairs should have been 6 (service apartments) and 5 (institutional settings), respectively. On page 1562 (column 3, line 33), the confidence interval for general cognitive ability as indexed by the principal component should have ranged from 20 to 82%. In figure 2 (p. 1562), the dizygotic (DZ) intraclass correlation for spatial ability should have been 0.24, and the monozygotic (MZ) intraclass correlation for speed of processing should have been 0.65. In the legend for figure 3 (p. 1562), the *P* value for the Wechsler Adult Intelligence Scale should have been 0.82, and the chi-square and *P* values for speed of processing should have been 1.66 and 0.65, respectively. In note 32 (p. 1563), intraclass correlations for speed of processing for MZ and DZ twins should have been 0.65 and 0.23, respectively. The estimates of heritability, shared environment, and unshared environment were correct, as were the conclusions of the study.

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Letters to the Editor

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