356). Quantitative-genetic analyses strongly support the hypothesis that a mutation in one single, pleiotropic gene is responsible for this substrain divergence. We have investigated the protein profile of the cortex and hippocampus of adult and newborn animals by means of 2-D gel electrophoresis. Our results show that a hippocampus-specific protein is expressed later in the mutated substrain possessing the smaller IIPMF projections and having the poorer radial-maze performance.

Arthur R. Jensen.[188] Wanted: A Unified Theory of Individual and Group Differences. A trite truism in quantitative genetics states that the degree to which the mean difference *between* population groups in a trait is genetic cannot be inferred solely from a knowledge of the heritability of the trait *within* each group. In fact, this is only the special, limiting case of a theory that views both *between*-groups heritability (BGH) and *within*-groups heritability (WGH) within one and the same analytic framework. The truism is indeed absolutely true only when we have no prior knowledge about the groups other than the WGH of each group on a single measurement of the trait in question. Recent developments in quantitative genetic analysis allow statistically testable inferences of BGH, given certain kinds of prior knowledge and allowing certain assumptions dictated by Occam's razor. The necessary conditions for decomposing the difference *between* group means into its genetic and environmental components and estimating BGH are illustrated with mental test data on large samples of MZ and DZ twins of two racial groups (black and white). The results, with socioeconomic status controlled, are interpreted as indicating a large BGH for these data. A wholly environmental interpretation of the between-groups difference in psychometric *g* can be maintained only by violating Occam's razor.

Victor Jockin[189] and Matt McGue.[189] A Multivariate Genetic Analysis of Child-Rearing and Personality.[190] Measures of family environment and child-rearing practices are typically subject to modest genetic influence (Plomin and Bergeman, 1991, *Behav. and Brain Sci.*, 14, 373–427). Using data from adult twins Chipuer *et al.*, (1993, *Dev. Psychol.*, 29, 110–118) found that covariance between dimensions of family environment and personality was largely genetic in origin, though most of the genetic variance affecting family environment dimensions was unique to those factors. Adult twins from the Minnesota Twin Registry reported their child-rearing opinions and practices, completed the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982, unpublished manuscript), and supplied a host of demographic and socioeconomic information. Multivariate genetic analyses are used to further explore the relationship among parental attitudes, child-rearing opinions, and practices, personality, and socioeconomic variables.

B. Johansson,[191–193] K. E. Whitfield,[192, 193] G. E. McClearn,[191–193] F. Ahern,[192] S. Berg,[191–193] J. Nesselroade,[194, 197] N. Pedersen,[195] and R. Plomin.[192, 193, 196] Genetic and Environmental Influences on Memory in the Oldest Old: Results from the OCTO Twin Study. Variability in memory performance as a function of the type of memory test employed is a well-accepted aspect of research on memory. An unresolved issue, however, is whether estimates of heritability for memory measures in the oldest old differ from those in adulthood and late adulthood. The purpose of this investigation was to examine the origins of individual differences in very late life in different aspects of memory performance using quantitative genetic methods. Monozygotic (149) and dizygotic (204) twins, aged 80 and above, in the OCTO Twin Study in Sweden were examined with eight measures: Digit Span forward and backward, Thurstones picture memory, MIR (including recognition, relocate, and recall), Prose Recall, and Digit Symbol. The results indicate high phenotypic intercorrelations. Prose Recall and Digit Span forward were the only two measures that had a significant shared environmental component but no significant genetic component. The heritability estimates for the other tests varied from .19 to .52. While phenotypically memory in late life seems to be a relatively unitary construct, sources of individual variability differ according to the measure employed.

K. S. Kendler,[198, 199] R. E. Straub,[198] C. J. MacLean,[198, 199] and D. Walsh.[200] A Possible Susceptibility Locus for Schizophrenia in the 6p22-25 Region in Irish High-Density Families.[201] Although family, twin, and adoption studies provide substantial evidence that genetic factors play a major role in the etiology of schizophrenia, several lines of evidence suggest that the mode of transmission is likely to be complex. Linkage studies of schizophrenia have been impeded by many factors including incomplete penetrance, phenocopies, uncertain phenotypic boundaries and probable genetic heterogeneity and/or oligogenic transmission. The Irish Study of High Density Schizophrenia Families ascertained pedigrees containing two or more individuals with schizophrenia (S) or poor-outcome schizoaffective disorder (SAD) from 39 psychiatric institutions throughout Ireland and Northern Ireland. Following up on unpublished results (Diehl and Kendler), we report here evidence, in 264 multiplex small to moderate-sized Irish pedigrees, suggestive of a possible susceptibility locus in the 6p22-25 region. Using an additive genetic model, and a broad schizophrenia spectrum diagnostic model, the strongest evidence for linkage was to marker D6S296, which produced a LOD score under

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