BEHAVIORAL GENETICS[1]

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The history of the area known as behavioral genetics (psychological genetics, sometimes shortened to psychogenetics) is, as mirrored in the pages of earlier volumes of the *Annual Review of Psychology,* but a short one. Fuller's 1960 review (88), covering the period from 1955, showed that activity in the late 1950s was directed to practical human problems such as mental illness. A major technique in work with humans was the twin study; most animal work aimed simply to demonstrate genetic control, comparing strain, as the independent variable, with behavior, as the dependent. Crossbreeding of strains was done largely in the Mendelian tradition of trying to isolate single genes. Attempts to find gene-behavior pathways emphasized audiogenic seizures. Such work continues, but now a major part of psychogenetic research is biometric and quantitative.

McClearn & Meredith in 1966 (161) observed that strain differences were commonplace and to be regarded only as a starting point. Of major interest were advances in human cytogenetics which continue to provide insights. There were also some biochemical and single-gene studies. Most of the rat and mouse work reported were studies on strain differences, although selective and crossbreeding studies were more plentiful.

In 1970 McClearn wrote a distinguished review for a sister volume, the *Annual Review of Genetics* (157), with a useful historical introduction, but the purely psychological story was not resumed until 1971 with Lindzey, Loehlin, Manosevitz

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& Thiessen's long and comprehensive review (150), which chronicled the further decline of simple strain comparisons and the increase of quantitative genetic analyses. The growth of studies using physiological analyses and the increasing interest in evolution were stressed. Meanwhile Childs (38) reviewed behavioral genetics from a viewpoint appropriate to the *Annual Review of Medicine.*

The growing importance of the field is reflected in the increasing frequency of reviews which, according to the 1971 "master plan" of the *Annual Review of Psychology,* should appear at 3-year intervals. But increasing frequency means decreasing size, and the present review must be shorter than many of those mentioned. Hence, in addition to maintaining an enforced selectivity, we have relied much more on previous reviews for background references than did our predecessors, using the five cited above to the exclusion of other earlier sources unless absolutely necessary. But their total of 1213 citations, with surprisingly little overlap, makes them a valuable repository of information on which to base our current progress report, which reviews the literature from approximately late 1969 to early 1973.

Our organization is simple: the first section deals with work using infra-human animals as subjects, the second with work with humans. The animal section is organized broadly by methodology, progressing from relatively simple to more complex situations and designs, with a detour through a single phenotype, that relating to alcohol preference, as befits a topic putatively concerned more directly with real-life problems. This approach also governs the organization of the section on humans in terms of the traditional categorization of behavioral phenotypes, but it necessitates a terminal discussion of methodology.

ANIMAL RESEARCH

Reviews

Topics include developmental problems (94), learning (257), audiogenic seizures (90), alcoholism (159), aggression (160), genetic homeostasis (120), and evolutionary contexts (240, 269); a general review by Thiessen (239) provides a history of mouse strains.

Major Genes

Interest in major-gene analysis continues. Studies of neurological mutants in *Drosophila* by Benzer and his co-workers have reached an exciting stage (see 150, p. 73; 38, p. 380). By producing flies which are structural mosaics for genes controlling various abnormal behavior traits, including one named "drop dead," they have succeeded in mapping gene location with reference to known marker structures, on the two-dimensional surface provided by the egg, with such precision that the origin of the defect can be pin-pointed (118). Thus in the case mentioned it has been shown that it is the brain and not any other part of the fly that must be involved. Analysis by this powerful genetic mosaic technique of other more complex behavior is promised and will be eagerly awaited.

In mice a study of the effects of four alleles differing in respect to tyrosinase activity at the albino locus showed that behavioral differences were unrelated to

either eye pigmentation or tyrosinase activity (241). When pigmented and albino segregants were tested for descent from a visual cliff using different lighting and platform heights, albinos were slower under all conditions (187). To discuss these findings, simply in terms of acrophobia added to a postulated photophobia is to minimize developmental history. A major gene which affects sensory receptors from birth must express itself in behavioral differences at maturity. Debate continues (23, 268) over the efforts at conceptual clarification in this area (see 150, p. 72).

Using a developmental approach, van Abeelen & Kalkoven (1) compared normal and Nijmegen waltzer mice on reflex and behavioral measures at several ages. Unlike major reflex differences which disappeared by weaning, behavioral differences persisted into adulthood. The deficient performance of retinal degenerate segregants on jump-out and one-way avoidance learning was shown to be a function of poor visual capacity, (259) whereas albino mice, if tested in dim red light, were not deficient. Albino segregants were progressively inferior to pigmented mice in a water-escape task (78). Since initial scores were identical, light intensity is unlikely to have been important. Mutations producing obesity in mice were detailed in relation to food-intake regulation by Fuller (89).

Major-gene analysis has been approached through study of behavioral differences rather than mutation. Whitney (266) measured the tendency to vocalize among randomly bred mice, finding frequencies expected for a single locus with complete dominance. Collins (43) has now mapped the position of the audiogenic seizure-prone gene using this approach.

Selection

Progress in ongoing behavioral selection studies as well as new selections for behavioral characteristics, continues to be reported. The sophisticated selection experiment of DeFries and co-workers, in which mice were bidirectionally selected for open-field activity, exceeds ten generations (50). Realized heritability was reliably estimated at 13%. Because the selection lines (as well as controls) were replicated, it could be shown that the negative correlation between defecation and activity was not a fortuitously correlated response. Tests at S_{10} checked the situational generality of the selection response. Differentiation of strains by activity in boxlike and illuminated apparatus had elements in common with that in the open field. Activity in wheels did not differentiate the strains.

Mouse strains bidirectionally selected for rearing did not differ systematically in adrenal or thyroid activity (2). However, Blizard & Chai (16), selecting for release of radioactively labeled iodine from the thyroid, found their strains differed in open-field defecation and water-maze learning. The results complement work on the Maudsley strains of rats selected for open-field differences and found to differ in thyroid funtion (see 161, p. 536).

Lagerspetz & Lagerspetz (141) now report results to S_{19} of their bidirectional selection for aggression in mice, noting no change since S_7, though strain differences in aggressiveness may be reversed by social experiences. Mice were selected over seven generations for learning activity in a T-maze (119). The authors' analysis showed a small genetic component. In four generations of selection of mice for rapid water-escape learning, realized heritability was found to be low (78).

Roubicek et al (209) selected rats for high and low water intake for eight generations at an ambient temperature of 22°C and for nine at 35°C. Selections for high intake at high temperature and low intake at the low were successful, but selections for increased intake at the low temperature and for decreased at the high were much less so. However, transfer of animals selected for increased intake at low temperature to the hot environment produced an intake similar to that of the rats selected at that temperature, though the relationship between the two low intake lines under comparable conditions was less marked. This difference is reflected in the higher genetic correlation of intake in the two environments for the high lines compared with the low. No abnormalities in urine concentration, adrenals, pituitaries, or kidneys were found in these strains, which consequently should make good material for behavioral studies.

A preliminary communication (26), which nevertheless reported 19 generations of bidirectional selection for shuttle-box avoidance in the rat, indicated an asymmetry comparable to that found previously in a similar experiment (see 150, p. 55).

Imprinting is widely regarded as a biologically important behavior. Graves & Siegel (98) bidirectionally selected chicks over five generations and found asymmetry for response to selection favoring rapid approach, but not for slow approach. Three lines of domestic chickens, bidirectionally selected for mating behavior, were studied for the effects of androgen therapy on sexual behavior (162). Level of restored mating behavior depended on genotype.

Alcohol Preference

In an ongoing study, Eriksson (67, 69) bidirectionally selected for voluntary ethanol consumption in the rat. At S_{17} the low strain was more active in the open field. Examination of the metabolic differences between the strains (81) showed that the high alcohol strain had a higher brain serotonin content; the difference was more extreme after exposure to alcohol (6). This strain voluntarily drank to intoxication (68).

Whitney, McClearn & DeFries (267) reconsidered earlier reports and presented new data on alcohol preference in rats and mice. They calculated various heritabilities, all consistently low, and questioned the assumption that heritabilities must be population specific. A study using two mouse strains and their filial and backcross generations has reported slightly higher heritability indices and considerable directional dominance for nondrinking behavior (66). A similar analysis of morphine intake in the same strains encountered severe scalar problems (70). Good agreement with a two-gene model was found for preference and consumption in two strains and their filial and backcrosses, without demonstrated involvement of coat-color genes (91). Inter- and intra-strain differences in preferences in mice were also reported (71, 194).

Pursuing his search for underlying mechanisms, McClearn proposed a negative feedback model (158) and, with co-workers (223), reported that intraperitoneal injections of ethanol produced higher accumulations of blood acetaldehyde in a nonpreference strain. Odor has been implicated in ethanol aversion in BALB/c mice (178).

Three studies relate alcohol consumption to other behaviors in mice and rats. A factor analytic study related alcohol consumption to two factors, audiogenic reactivity and disorganized behavior (195). An analysis using filial and backcrosses found alcohol preference in mice to be negatively related to open-field defecation and urination (265). Higher voluntary intake, however, was found in strains of rats selected for high emotionality and high avoidance (212). At two symposia (80, 222) material on the value of animals in alcoholism research was presented.

Simple Strain Differences

Despite previous caveats, strain differences continue to be reported and some merit brief attention. However, the practice of using laboratory rodents "bought in," i.e. reared somewhere other than in the home laboratory, must raise doubts about the validity of the findings reported, depending, of course, on what is known about the phenotype investigated.

Interest has arisen in measurements of brain biochemistry. Comparisons among rat strains have included telencephalic serotonin metabolism (207), acetylcholinesterase (230), and tyrosinehydroxylase—a rate-limiting enzyme in the biosynthesis of brain amines—which was found to relate inversely to activity (221). Rick et al (204) found a similar association between activity and GABA. Studies using drugs affecting the cholinergic system supported the existence of a genetically controlled brain cholinergic mechanism which facilitates exploration in mice (3, 4).

Several studies have been based on rat strains selected for behavioral characteristics including derivations of the Maudsley and Roman strains (see 161, p. 535, 150, p. 55 respectively). The most recent of a series (102) has confirmed strain differences; emotionality, as measured by suppression of drinking by unsignaled shock, was unrelated to defecation (121), as was spontaneous activity and avoidance conditioning (211, 213).

In further reports from Schlesinger and co-workers (see 150, p. 41, and 157, p. 454), a heterogeneous and an inbred mouse strain were both found to be susceptible to audiogenic seizure priming, though optimum stimulus exposure varied between them (20). Substances known to affect levels of biogenic amines reduced susceptibility to seizure after priming without affecting priming itself.

Simple Genetic Analyses

By this rubric, we mean analyses uncomplicated by environmental variables, though genetic designs may be quite complex. When three mouse strains and their first filials were compared on water-escape learning, the filials performed as well as the fastest inbred strain (77); in another study two strains and their first filials were compared for activity by means of two types of automatic recorders (191). When saccharine preference was compared in four inbred mouse strains, broad heritabilities were found to be high (192). Comparisons involving filial and backcrosses showed extreme heterosis for nest building in mice, in a study which also presented a diallel analysis (143). The pattern of differences between the broad and narrow heritabilities supports the adaptive significance of this character, interesting for its a priori relationship to biological fitness.

Van Oortmerssen (183), from an ethological analysis based on extensive filial crosses, has suggested that some laboratory strains of mice arise from surface-nesting wild progenitors while others derive from hole-nesters. Developmental differences in passive avoidance in two strains of mice and their F_1s were shown to depend on shock level (233), and Hegmann (110) found strain differences in nerve conduction velocities which were faster in reciprocal F_1s obtained between two of the six strains. Wahlsten (258) "bought in" strains for an interesting attempt to study mechanisms underlying response to shock and avoidance learning by equating shock level eliciting jumping. Multivariate analyses of behavioral sequences in two mouse strains and their filial and backcrosses showed evidence of segregation of sequence elements (148).

Of two mouse studies based on parent-offspring covariation, one (245) demonstrated the absence of change in a measure of narrow heritability for runway learning, while the other (181) showed high narrow heritability for avoidance learning.

Studies with an Environmental Dimension

Of more interest than simple demonstrations of strain differences are recent attempts to show the limits of generality by utilizing more than one environment. For example, interstrain fighting in mice was shown to be dependent on infantile stimulation (198), and when male mice from two "bought in" strains were kept at five different temperatures and three different population densities to study the effects of these factors on aggression (100), the strain differences did not interact with environmental variables. An interesting limitation of generality arising from strain-measurement interaction was reported by Goodrick (95), who compared automatic and direct observations of open-field activity which varied differentially in two mouse strains. Differences in the effect of a companion on exploratory behavior were found in two strains of mice (227). Another social variable, litter size, was similarly studied (62). Factor analysis revealed dimensions interpreted as territorial marking, autonomic balance, acrophobia, and motor discharge. Although litter size directly affected the first two, the only interaction of litter size with genotype was in respect of motor discharge.

Vale et al (251) "bought in" male mice of five inbred strains that displayed differences in population density effects for number of aggressive attacks and for adrenal weight. Genotype-density interactions were also found, but the relationship between behavioral and physiological measures was unsystematic. Scott et al (219) linked the effects of amphetamine on fighting in mice to differences in its effect on body temperature in two strains. Their findings relate to explanations of the aggregation-toxicity effect of this drug which invoke body temperature.

Investigation of nest building at two ambient temperatures (152) showed for one strain a negative correlation with food consumption at the lower temperature. Vocalization in infant mice was shown to depend on strain and ambient temperature as well as age (14, 180).

Complex Processes, Including Genotype-Environment Interaction

With this section we begin the consideration of what we see as the central problem in behavioral genetics. Psychologists quite rightly regard their discipline as having

analytical difficulties; geneticists view their area similarly. The intersection of interests in behavioral genetics must therefore raise problems of considerable complexity, on either the genetic or the environmental side and often on both (25). Erlenmeyer-Kimling (72), deploring the lack of effort directed to the solution of these problems, blames the daunting nature of the task. We have necessarily concentrated on studies that emphasized these complexities, but we do not, of course, undervalue those that attempted to provide a framework on which to build further. Some of what has preceded may be regarded as falling into this latter category.

Vale and collaborators, who recognize the importance of such a positive approach (see 150, p. 76), have presented a series of intriguing sex X strain X hormone interaction studies of aggressive and sexual behavior in mice (248–250). Their results have led them to suggest that sexual behavior may in fact be less dimorphic than aggressive behavior.

Fulker (84) reanalyzed three studies—one on rats and two on mice—of prenatal and shock stress (244). Patterns of reciprocal F_1 differences yielded to a model in which the additive genetic effects were buffered by contribution from the maternal environment.

Henderson has continued his systematic attack on problems in this area (see 150). In a 6 X 6 diallel study of mice, additive genetic variation in a complex food-seeking task was released by environmental enrichment, although the underlying genetic pattern remained essentially the same under both environmental treatments (113). A similar finding emerged for spontaneous alternation (111). Brain weight showed greater heterosis in the environmentally enriched group (112). In another diallel study on enrichment, Henderson found that it led to only small changes in discrimination learning (114). These studies can be contrasted since aversive motivation was used in the second study.

In attempting the analysis of gene-action in relation to environmental determinants, the various approaches adopted by Broadhurst & Jinks (24) open several possibilities. Their implications have been reviewed by Fulker et al (87), who presented a preliminary multivariate analysis of an 8 X 8 diallel cross of rat strains, with infantile stimulation as an imposed environmental treatment. Genotype-environment interaction was considerable in the additive components of variation in open-field measures, but minimal in avoidance conditioning. As in Henderson's work with mice, directional dominance not interacting with treatment was found. More detailed analyses have revealed the subtlety of the interaction within avoidance acquisition (271) in that a progressive change in genetic control has indicated the possibility of two distinct behavioral processes underlying learning. Since they correspond to the two factors of Mowrer's theory, it might be argued that behavioral genetics is beginning to have something of direct interest to say to one of its parent disciplines.

Less systematic effects suggestive of similar mechanisms in mouse avoidance conditioning were found by Royce et al (210) in a 6 X 6 diallel, which also showed clear evidence of directional dominance for high avoidance.

Genetic analysis of factor scores confirmed the different genetic substrates for those two frequently combined measures of emotionality in the mouse, urination and defecation. The results of two studies (171, 182) using mouse diallels and

concerned with conditioning, maze learning, and activity were interpreted in part in terms of dominance relationships dependent on the particular crosses involved. But this epistasis (nonallelic interaction), evident in extensive biometric analyses, may be a function of the choice of extreme parental lines in relatively small diallel crosses.

Several other small diallels in mice have been reported (30, 44, 83, 206, 247). The use of the variance-covariance diagram of Jinks for the proper presentation of the results is a welcome sign of an increasing awareness of the power of this method.

As we have seen, *Drosophila* lend themselves to powerful analyses. The quantitative approach employs such behaviors as geotaxis (279) and locomotor activity, which Hay (107) used in a design employing all possible filial and backcrosses (14 in all) to establish directional dominance for high activity in *D. melanogaster*. This study also incorporated an environmental variable (culture) and provided the first unambiguous demonstration of duplicate gene interaction and dispersed gene distribution for a behavioral trait. Such genetic architecture is typical of fitness traits which determine uniformly high levels in free-mating populations. For a measure of preening, additive and dominance maternal effects were found among females, whereas among males maternal and progeny genotype interactions occurred. Diallels gave supporting evidence. In further work (109) genetic factors were implicated in social interactions in flies. Manning & Hirsch (153) investigated genetic mechanisms in a strain of *D. stimulans* selected for slow mating.

Population considerations often involve complex behavioral processes (see 150, p. 69); Ehrmann has now implicated olfactory cues in density dependent selection in *D. pseudoobscura* (63). In other *Drosophila* species similar effects are observed (64, 108), but frequency-dependent selection was not found in house flies (37). Using mice, Yanai & McClearn showed that females determine mating preference (282). In some strains they prefer to mate with males of different strains (281), and early exposure to adult males may play a part (283). An interstrain comparison of sexual performance in pairings forming a "diallel" pattern showed male rats' success to be inversely related to success in the female (164), whereas in a study of mouse aggression using filial and backcrosses, restriction to pairing within generations only made interpretation difficult (264). In a true diallel cross of mouse sexual behavior, Vale & Ray (246) reduced the problem by pairing uniformly with only one of the strains. Models for dyadic social interaction measurment in psychogenetics have been discussed (237).

As we have seen, many of these quantitative studies make inferences from their results that bear on natural selection and evolutionary processes. It may be necessary to use behavior putatively related to fitness to substantiate such inferences (238, 270).

Study of the relevance of laboratory populations to wild populations is likely to be enhanced by biometric (85) and other (49, 177) techniques for investigating the genetic architecture and gene flow of the latter. Meanwhile, comparisons of wild and domesticated rodent species have been made (93, 199, 226), with some results suggesting that laboratory strains compare more favorably than previous reports have indicated (21, 22, 154, 200, 232).

HUMAN RESEARCH

Reviews

A comprehensive account edited by Kaplan (134), one on twins (172), and others on abnormal psychology (136, 224, 228) have appeared.

Normal Cognitive Abilities

General intelligence continues to receive most attention. Because early work in this area set high standards, many publications attempt reinterpretation and synthesis of already existing data. Burt (28), Jensen (126), and Vandenberg (253) have drawn extensively on earlier studies to conclude that differences in IQ in the populations surveyed are largely under genetic control: broad heritability was found to account for 70 to 80% of the variation, family environmental variation for about half that remaining, and random environment for the rest. Jinks & Fulker (131) reanalyzed data from five major studies using biometric methods (155) to investigate the genetic architecture of IQ. Distortions due to sampling and to both gene-environment correlations and interactions were judged absent; they therefore concluded that at least 22–100 genes controlled IQ, that narrow heritability was 71% (assortative mating accounting for 14% of this), and that dominance variation was 15%. The average level of dominance was 0.74 or more, dominance being for high IQ, and increaser and decreaser genes were found on the average to be equally frequent. A simple account of this analysis may be found elsewhere (86). Substantial dominance variation detected by three different methods tends to confirm Burt & Howard's earlier finding (see 88, p. 56), while its direction conforms to the expectation that components of IQ have been of adaptive advantage during evolution. The influence of assortative mating was expected since the IQs of spouses correlate more than any other human behavioral trait (254). However, its influence has been most strikingly demonstrated by Eaves (56), who used the Reeds' extensive pedigree data from five generations (see 150, p. 51) and showed that only genetic models incorporating assortative mating fitted the data successfully.

Additional evidence (127) supports a fairly simple genetic model: genotype-environment interaction and genetic threshold effects were found to be absent, and all three fundamental components—phenotypic, genotypic, and environmental—were found to be normally distributed.

Maternal effects may complicate any simple model: such effects, probably of intrauterine origin, are clearly indicated by the association between birth weight and IQ, recently confirmed for multiple births (201, 214). Willerman and associates (274) suggested postnatal maternal effects on the measured IQ of children of interracial marriage, but the results rest on weakly supported assumptions. A further study employing a similar approach with white subjects presented more convincing evidence (273). Children resembled their high- and low-scoring mothers more than their high- and low-scoring fathers. However, the failure of maternal effects to influence parent-offspring correlations differentially indicates their probably minor importance as a systematic source of variation.

Fascinating evidence is emerging concerning the developmental stability of a genetic component in intelligence. Wilson & Harpring (275, 276) report impressive results from administering the Bayley scales to 261 pairs of MZ and DZ twins at frequent intervals during their first 2 years. The profiles of the developmental lags and spurts, responsible for the typically low correlations at different ages, were very similar, especially for MZ twins. The figures suggest that heritable factors account for about half the variation in profile similarity and demonstrate that even the fine detail of some aspects of mental development as measured by the Bayley scale is under relatively tight genetic control. In a study of development of IQ from 3 to 12 years using sibling-sibling and parent-offspring correlations, the average level of intelligence also appeared highly heritable, but not the pattern of development (156). However, these methods must be less powerful than the use of MZ twins. The design has also been criticized on other grounds (262). At the other extreme of age, a continuing study of senescent twins suggested that the genetic component in IQ remains high well into the eighth decade of life (124). Unfortunately, numbers were insufficient to warrant profile analysis.

A large genetic component in IQ variation extending over a wide age range may have important consequences for human population structure. One possible consequence is that natural selection may be acting on IQ through differential fertility. Previous studies (see 157, p. 458) and a later follow-up (261) suggest that selection differentials are positive, tending to increase IQ. Selection may not be acting uniformly throughout the full range of IQ (60, 76), however, and the possibility of dysgenic trends in some subpopulations should be noted (130). However, since there is good reason to believe, on both general evolutionary grounds and recent evidence (122), that the narrow heritability of fertility is low in some populations, there may be no long-term effects, whatever the size of the heritability of IQ and its correlation with family size.

Another consequence of a large genetic component in IQ is its possible influence on class structure and mobility (58, 242). Differences in IQ between father and son correlate +.37 with differences in social position (260). Partial and multiple correlations were used to unravel the interrelated factors of educational level, social class, and IQ. Eckland (59) sees these intergenerational consequences of IQ as most important.

Yet a third possible consequence is that genetic endowment might explain part of the 15 points average difference in measured IQ between white and black Americans. This claim by Jensen (125) provoked more debate than any other psychogenetic issue of our times; timely press releases by the journal's editorial staff ensured an instant controversy. Jensen's is a thorough account of the genetics of IQ and scholastic achievement which he related to the results of compensatory education. These, he claimed, were inconsistent with existing environmental explanations. The paper was soon reprinted (126) with invited criticism and a reply (104, see also 105). A fascinating blow-by-blow account of the original publication and its early aftermath can be found in *Genetics and Education* (129), which again reprints the article together with others and bibliographies relevant to the debate. Jensen's considered

reply (130) argues that no plausible combination of environmental factors known to influence IQ, and which differentiate blacks from whites, seems capable of producing a mean difference as large as 15 points. He finds most consistent with the evidence the hypothesis that from one half to three quarters of the difference is attributable to genetic factors. While he convincingly demolishes most current environmentalist explanations, we may be simply suffering from a dearth of good research into environmental variables. None so far for which there is evidence available is deemed adequate, but that is a far cry from proving that none exist. Moreover, we rely on a weak form of proof when we rely on the mere consistency of a genetic model with the data. Whereas the model for IQ based on white populations has been thoroughly tested, such is not the case here. We must avoid what Willerman (272) in a book review (73) calls "premature closure" (216). Diverse views on race and IQ may be found in (29, 188, 203), but for balanced views in small compass, we recommend Thoday & Gibson (243) and Cavalli-Sforza (33).

The need for more research is seen by many; the question is, of what kind? A major difficulty is that inferences about the relative contribution of heredity and environment to differences between groups are not possible simply from a knowledge of their relative contributions within them. DeFries (48) has formulated the problem showing the unknown parameters involved. What other (less indirect) evidence could be gathered?

Fostering studies have been suggested (19, 272). Certainly they produce most convincing direct evidence for the relative importance of genetic and environmental influences within subpopulations. The problem is that fostered children must carry their color across populations, unlike the class origins of such children within populations. But comparisons involving very young foster children would have merit. The study of interracial marriages is a related approach (274).

A second direct method, which employs blood groups as a covert means of identifying the extent of mixtures of genes which typically differentiate blacks and whites, and correlates these differences with IQ, has been elaborated by Heston et al (see 130, p. 224). Using simpler methods, it was found (184) that such blood groups correlate with a measure of mental ability in blacks, though not in whites; a Monte Carlo simulation (185) showed that problems of statistical inference involved in stepwise multiple regression were responsible for the high correlation and new data failed to show the relationship. Loehlin, Vandenberg & Osborne (151a) reanalyzed the original data together with new, calculating separate correlations for black and white subjects between composite mental test scores and the presence of blood group genes. Rank correlation between these correlations themselves and the rank order of decreasing frequency among blacks of the 16 genes studied indicated no association between the extent of white ancestry and mental ability. Before concluding that there was no evidence for a genetic component in the black-white difference, they tested the efficiency of their method and found that genes in one blood group system, known to have relatively greater frequency in Europe, failed to predict the presence of genes of greater European frequency in other systems. They observed that genes that themselves are no longer associated cannot be expected to be associated with other genes affecting IQ. Larger samples might help,

but the method does not look promising, especially in view of the very large overlap in the interracial frequencies.

A third direct line of research is to seek genotype-environment interactions to explain the difference. Scarr-Salapatek (215) carried out a twin study involving black and white children using aptitude and achievement tests. She compared heritabilities in the different races and social class combinations, and suggested that heritability was low in groups disadvantaged by either race or class, thus supporting the idea that disadvantaged children do not reach their full potential. While this may be true, her data cannot support the conclusion. Eaves & Jinks (57) showed that the study suffered from methodological weakness: the conclusions do not even suggest a genetic component within races, let alone any interaction with race or social class. Barker's rejoinder (12), insofar as can be judged, suggests that something may yet be salvaged! While Vandenberg (252) has reported a small twin study on black subjects' IQ, we clearly do not know enough to suggest genotype-environment interaction in black populations. Again the prospect is not promising, for Jensen (128) failed to find convincing interaction between race, class, and sex, and evidence from white populations suggests no genotype-environment interactions for IQ (127, 131).

Turning from general intelligence to other congitive abilities, available evidence on school achievement (104, 130) implies that genetic components account for only about 40% of variation, half that for IQ. This is partly because common family environment accounts for some 50% compared to about 10% for IQ. Such differences could explain why compensatory education affects attainment more than IQ.

Does sex-linkage underlie observed sex differences (99) in many special cognitive abilities (144)? Polygenic sex-linkage is indicated by parent-offspring and sibling correlations, the pattern depending on sex of parent and of offspring, as originally worked out by Mather & Jinks (see 155, p. 291). Stafford, who reported this pattern for spatial ability (see 161, p. 526), found a similar pattern for mathematical ability and suggested a major recessive gene (236). Further data relating to spatial abilities have been reported (103), and Bock & Kolakowski (17) demonstrated a possible single recessive sex-linked gene operating against a background of environmental and autosomal variation. Combining a variety of spatial test items to score 727 boys and girls on a normal ogive model of the test, they decomposed the distributions of scores for each sex separately into the two equal-variance normal distributions best fitting the data, equal variation being expected once allowance has been made for the effect of the major gene. They showed that their model strikingly fitted the data and applied equally well to parent-offspring correlations from their own and previous studies. Finally they noted that such a recessive gene might be more associated with fitness (rather than the more usual expectation of dominance), if natural selection acted primarily through the male. Evidence suggesting an autosomal linkage between a major gene for verbal ability and blood-group genes depends on the greater within-pair variance found for DZ twins discordant for blood groups (18).

Other investigated phenotypes of interest include musical ability, which showed (235) genotype X instruction interaction; number estimation (101); and laterality

(146), for which a model postulating one locus for cerebral dominance and one for relative hand control was shown to fit a variety of data (178a). Cultural and genetic factors could not be distinguished (140) for sensitivity to flickering light in part of a study of racial differences in Hawaii (40).

Mental Retardation

Given a large genetic component in cognitive abilities, it follows that much retardation, other than extreme forms attributable to major genes or traumata, will result from normal polygenic segregation. Thus, using a narrow heritability of 70% for IQ and allowing for assortative mating, it has been shown (86) that a polygenic model predicts within 1% the frequency of subnormality from subnormal matings in the Reeds' data. Combining the two approaches, Berman & Ford (15) predicted phenylketonurics' IQ from those of their parents and siblings. Discrepancies were related to phenylalanine levels in the blood. The complexity of genotype-environment interaction in this area is illustrated by a study which manipulated these levels by dietary regimens to find the optimal effect on IQ amelioration (92).

Among other factors of possible methodological importance in the genetics of subnormality are sex-linkage and inherited maternal effects, already mentioned as additional complications of the polygenic model of normal variation. Lehrke (144) proposed that sex-linkage for several cognitive abilities might explain the greater variability of males, resulting in their greater contribution to the retarded as well as to the gifted. His arguments have been criticized mainly on statistical grounds (see 145), but the possibility remains. Maternal effects, probably mediated via inherited intrauterine inadequacy, were detected by Ahern & Johnson (5) in the Reeds' data on IQ. Probably the best evidence for directional dominance for high IQ has come from Schull & Neel (see 150, p. 45), who, in their continued study of inbreeding in Japan (218), have shown effects of parental consanguinity on IQ and achievement (179), which, while nonsignificant, reproduced those previously found. Retardation should thus result from severe inbreeding. Seemanova (220) writing on incest among first-degree relatives, who have a very high inbreeding coefficient (¼), described an impressive series of 161 such relatives plus 95 half-sibling controls. Among the former, 40 showed moderate to severe retardation; among the latter, none.

Kessler & Moos, reviewing chromosomal abnormalities (137), concluded that, with the possible exception of space-form deficit in Turner's (XO) syndrome, such abnormalities are only associated with a general retardation, as in Down's syndrome (10). Concerning major-gene effects, for those who curled up with McKusick after the last annual review, the third edition is a must (163).

Normal Personality

It is well established that most aspects of personality are at least under some genetic control. Eysenck (74), Vandenberg (255), and Shields (224) have reviewed the area.

A methodologically elegant study is that reported by Hill & Hill (115, 116). It involved 97 married couples who had been tested with the Minnesota Multiphasic

Personality Inventory at school, thus ensuring no postmarriage biases. Twenty-eight of their children were then tested at the same age their parents had been, thus ensuring no age bias. Despite these small numbers, the results were judged free of common artifacts. Positive assortative mating was indicated for three psychoticism scales. A cross-correlation matrix yielded intriguing relationships such as the correlation (+0.3, on the average) of depression in wives with psychoticism in husbands, whereas the reciprocal correlations were essentially zero. Parent-offspring correlations for psychoticism scales, together with those for mania, showed greatest narrow heritability when corrected for assortative mating, as this design allows.

The multivariate methods developed by Vandenberg and collaborators (see 150, p. 50) in the cognitive domain have been adapted by Eaves (55) to analyze genetic and environmental covariance among MZ twins tested on Eysenck's PEN questionnaire. The structure of the within-pair matrix, assessed using principal components, indicated environmental covariance structure. Genetic covariance was also investigated using a kind of canonical analysis. He concluded that environmental influences determined variation in neuroticism and introversion-extraversion in a unitary manner, but several genetic variables determined psychoticism. A well-defined genotypic factor also emerged for neuroticism, but not for introversion-extraversion. Another study employing a dimensional approach has been reported (41).

The co-twin control method allows a control for genotype to permit a more accurate assessment of environmental influences. Developmental studies have used this method to relate personality differences to possible causal influences (8, 42). Babies better endowed became more secure and trusting infants and better coping, assertive toddlers. Wilson et al (277) found MZ twins were more alike than DZ twins in temper frequency, demands for attention, and crying; this difference between MZ and DZ twins was larger among babies than at later ages up to 6 years. Some of these findings were related to motor performance (276). Van den Daele (46) reported evidence of a genetically controlled regulation of infant responses to rocking and soothing sound; hypnotic susceptibility may be controlled similarly (174). In an extensive and massive series of twin studies in Sweden by Cederlöf and co-workers (see 35, 36), which previously attracted little attention, research is now focused (82) on twins discordant for mortality but concordant for smoking. Present evidence implicates factors other than smoking, such as accidents and suicide, in the greater morbidity found among smokers. Another co-twin control study (149) related dissatisfaction with life to incidence of heart disease.

In a small twin study of sexual behavior, Chilton (39) produced evidence that the age at which masturbation, orgasm, and other sexual activity began is heritable. Difficulties inherent in research into sexuality are apparent, but the study points to an important, neglected area.

Abnormal Personality

Some progress is being made in our understanding of schizophrenia, affective disorders, alcoholism, and the XYY syndrome; discussion here will be restricted to these topics. Two major books have appeared on schizophrenia: Kaplan's work (133) brings together some 30 contributors on a variety of topics related to the genetics of schizophrenia, and Gottesman & Shields' study (96) is based on the Maudsley

twin sample. Meehl's afterword (167) appraises this as a "definitive work, a magnum opus, a beautiful book indeed." It is also a gold mine, since it provides case histroies and diagnoses of all 24 MZ and 33 DZ twin pairs originally identified through an index case. These diagnoses were investigated in a cross-national study (225). Six diagnosticians, differing widely in their training and outlook, not surprisingly produced different concordance rates. But consensus diagnosis maximized the heritability, restoring some faith in the diagnosis of schizophrenia. Psychometric implications have been discussed (96, 132, 167). While concordance rates for MZ and DZ twins have been falling recently, perhaps due to better sampling procedures (see 150, p. 64), consensus diagnosis restores concordance values to the region of 46% for MZ and 14% for DZ (97), much closer to those in the older studies. Allen et al (7) also found more searching diagnostic methods increased concordance rates.

New data from Iceland (135), based on public records, gave typicaliy low concordance rates for many kinds of relatives (but not twins), and preliminary findings (13), have suggested that among children of two psychotic parents, the incidence of psychosis is about half that previously suggested. Inouye's data on MZ twins separated before the age of 5 years (123) yield the same incidence as that for twins reared together, suggesting that between family environmental influences are small in determining schizophrenia (see 150, p. 63), but Kety, Rosenthal & Wender and co-workers (138, 208) confirmed earlier findings (see 38, p. 386) that incidence of broad-spectrum schizophrenia among adopted children of schizophrenic parents was as high as that among home-reared children, though the incidence of severe cases was much less. Perhaps severity is modified by home environment while disposition is not. The same research program produced evidence (263) that it is the downward social mobility of schizophrenics rather than prevailing cultural influences, that produces the high incidence in low socioeconomic groups.

Fischer (79) used an ingenious design to investigate the importance of home environment to children of parents themselves MZ twins discordant for schizophrenia. The age-corrected risk for children of schizophrenic parents (9.5%) is not significantly different from the nonschizophrenic (12.3%), suggesting no obvious parental effects. However, the nonschizophrenic parents could hardly be regarded as a normal control group.

Attempts to fit genetic models to data on concordance rates continue (see 150, p. 65). But recent attention has been directed to threshold models of liability developed by Falconer (75) and others (61, 231) and used by Gottesman & Sheilds (see 157, p. 439), who later point out the difficulty of distinguishing a one-gene from a polygenic threshold model (97). Kidd & Cavalli-Sforza (139) encountered the same difficulty, not surprisingly, since the polygenic model includes the case of a single gene as an approximate special case. However, Reich et al's method (202) of multiple thresholds based on different degrees of severity should resolve the issue, provided that reliable population base rates for each threshold can be obtained, since this method has effectively distinguished between one-gene and polygenic models of animal phenotypes. Fulker (86) assessed gene action for liability, using a simple biometric extension of the threshold model, and detected additive variation but little or no variation due to dominance or between-family environment.

As others have pointed out (e.g. 150, p. 64), environmental factors in schizophrenia must be very important, since MZ discordance is about 50%. But the absence of a strong between-family environmental effect and the marked discordance point to physiological differences, possibly arising early in life. Mednick's prospective study (165, 166) of children at risk is one investigating this possibility. A distinctive premorbid condition is found in those adolescents who have suffered breakdowns, with birth anoxia implicated.

In a study of discordant twins (234), biochemical variables have recently received most attention. Interpretation was complicated by long-standing drug treatments, but the lactate-pyruvic ratio involved in brain oxygen metabolism differed within discordant pairs. Pollin (196) discussed evidence bearing on his suggestion that catecholamine levels may be important in a physiological predisposition to schizophrenia. From the same major study, another possible predisposing marker in discordant pairs was reported (280) in the form of reduced platelet monoamine oxidase activity. Findings from a new, prospective high-risk study by Anthony (9) delineate five premorbid syndromes.

Interest in population aspects of schizophrenia continues. Moran (173) discussed the role of heterozygote advantage and mutation in maintaining a high level of schizophrenia in the population. Lane (142) discussed the possibility that fertility may be even greater than previously supposed, especially among female schizophrenics.

Research on affective psychoses continues to be dominated by Perris's (see 38, p. 388) suggestion that unipolar (depression only) and bipolar (depression with mania) disorders are under two distinct forms of genetic control. Winokur (278) showed that when only bipolar cases were considered, evidence of a dominant sex-linked gene was striking, a father-son concordance of zero completing the expected familial pattern exactly. Mendlewicz et al (168) associated bipolar depression with color blindness in the pedigrees of seven families, again suggesting sex-linkage, but unipolar depression (11) more clearly followed a polygenic pattern of inheritance, since the disorder occurred with equal frequency in male and female lines. Perris, who originally favored a dominant sex-linked model for the unipolar disorder, now favors polygenic inheritance for both (193), as do Slater et al (229); thus Winokur's findings supporting sex-linkage for the bipolar disorder are atypical. Some bipolar fathers have bipolar sons which cannot happen with sex-linkage alone. Evidence of either heterogeneity of gentic control or a threshold mechanism for both bi- and unipolar depression has been presented (169, 197).

Questionnaire studies have established a genetic component in alcohol use (151, 190), but Schuckit et al (217) have presented the first clear evidence of a genetic factor in its abuse. Half-siblings were used together with objective criteria of alcoholism and other psychiatric disturbances were avoided. The frequency of alcoholism in half-sibs with an alcoholic parent who lived with the child was 46%, compared with 50% if they did not. When there was no alcoholic parentage, but the child was reared with an alcoholic step-father or mother, the figure was 14% compared with 8% where the step parent was not alcoholic. The appropriate comparisons clearly indicate that genetic factors are important and that the mere presence of an alcoholic

in the home unimportant. Good evidence of assortative mating for alcoholism and associated psychiatric disorders has been found (205).

The effects of sex chromosome aberrations on personality continue to be discussed (see 38, p. 394; 137), but most attention is focused on the XYY syndrome and its possible link with aggressive and criminal behavior. Two excellent reviews (117, 186) summarize and evaluate available data, the latter review covering a wealth of material on physical and developmental abnormalities. The controversy concerning the high frequency of XYY individuals in mental and penal institutions is far from resolved. Owen concluded that rates in the majority of either mental hospitals or prisons are no higher than reliable population rates. But Hook found that for institutions of a kind reserved for the criminally insane, rates are from 5 to 20 times higher than those in the general population. This discrepancy is partly due to the different population rates used and partly to Hook's rejection of lower rates in samples previously screened for abnormal height, but mainly to the different institutions selected for comparison. Some association between XYY and such special institutionalization remains a distinct possibility, although Hook suggests the risk is slight. Moreover, both reviews, in line with Kessler & Moos' earlier review of this area (see 150, p. 68), failed to reveal any consistent association with personality and behavior. Hook plans a study of 11,000 young males whose sex chromosome compositions are to be ascertained at birth.

Methodology

This final section will review briefly some methodological developments. Three texts on statistical genetics are noteworthy in this connection (34, 45, 155), and some work already discussed will be cited again.

The study of twins will continue to form an important part of most research designs. Their uses and limitations have been ably described by Vandenberg (255), who also has provided new evidence on intrapair attitudes. Bulmer's (27) excellent book is indispensable to understanding the biology of twinning.

A general approach to partitioning variation in individual differences in behavior has, it is argued (131), certain advantages over others. Assumptions are tested and components of the model used are estimated with simple but efficient methods. Components can then be validly used to explore genetic control, using conventional methods of biometric genetics. The approach is flexible and can be extended to the multivariate case (54). Eaves has also discussed the requirements of genetic design in terms of the relationships–twins, siblings, and other family constellations–involved (51, 52) and the sample sizes needed (53) if significant results are to be expected. Morton (175) described a number of powerful biometric methods applicable to psychogenetic problems and noted that forms of multiple regression are the obvious ways to identify behavioral patterns associated with single genes. With many genes and many sets of behavioral phenotypes, the natural extention is generalized canonical correlation, for which satisfactory methods now exist (31).

Haseman & Elston (106) have described a powerful method for detecting linkage between a major marker gene and either major-gene or polygenic variation by using siblings and regressing the square of the sib difference onto the estimated likelihood

of carrying the marker. Pakstis et al (189) used this method to detect linkage between activity measures in children and blood-group markers. Elston & Stewart (65) extended the model to cover more complex genetic situations in which extensive pedigree data are available (see also 176). The threshold models (231) which have been used in schizophrenia research may be extended to the biometric analysis of polygenic inheritance (86) or treated as special cases of complex segregation analysis (175). In the latter case the multiple threshold model of Reich et al (202) should help distinguish single-gene from polygenic models and thus resolve the protracted debate about the genetic determination of schizophrenia.

Morton (175) is skeptical about genetic analysis where genetic and environmental influences are correlated, whereas Cattell developed MAVA, multiple abstract variance analysis, for just such situations (see 88, p. 55). However, it seems likely that certain limited models may be possible (131, 170). Cattell's (32) major recent contribution has been a heroic extension of MAVA attempting to separate the genetic and environmental effects of both maturation and learning. To accomplish this would require employing MAVA to partition both independent and dependent variables within a number of subpopulations. Correlating appropriate pairs of components across such subsets would lead to a regression equation for estimating genetic and environmental scores for individual subjects, which could then be examined for the effects of maturation and learning.

The opportunity to test more complex and specific genetic hypotheses is now within reach. Methodology is now well worked out and theory is far ahead of experimental endeavor. We foresee a period of concentration on gathering empirical data which can then be consolidated into a comprehensive structure of knowledge.

We cannot conclude this review without mentioning Darlington's book (47), which, in the opinion of at least one of us, may prove to have been the most important citation of all.

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