BEHAVIORAL GENETICS[1]

GARDNER LINDZEY, JOHN LOEHLIN, MARTIN MANOSEVITZ AND DELBERT THIESSEN
University of Texas

INTRODUCTION

It is difficult to specify precisely the steps involved in the complex merging of psychology and genetics that has resulted in the field of behavioral genetics. Perhaps Calvin Hall's chapter on psychogenetics, which appeared in Stevens' *Handbook of Experimental Psychology* in 1951 (151), was the first explicit sign of the origin of the field. Certainly the publication in 1960 of *Behavior Genetics*, by Fuller & Thompson (125), indicated a fully developed self-awareness of an important new specialty. The appearance of *Annual Review* chapters in 1960 (115) and 1966 (261) made clear that behavioral genetics was now viewed, even by those not working actively in the area, as an interdisciplinary field of general interest and significance.

Recent years have witnessed a variety of developments that attest to the continued vigor of activities in this area. Efforts are currently well along in the establishment of a behavioral genetics society which will have a broad and interdisciplinary membership. A journal entitled *Behavior Genetics* has also been initiated and will provide a unified outlet for animal and human behavioral genetics research. While at the beginning of this decade there were no formal graduate programs in behavioral genetics, there are now several such programs (University of Colorado, University of Illinois, University of Minnesota, University of Texas) and others are being initiated. A number of summer training institutes and conferences for graduate students and postgraduates have been conducted in recent years, as well as a variety of symposia and research conferences, e.g. Center for Advanced Study in the Behavioral Sciences (175), Rockefeller University (134), and Dorado Beach (338). Of special interest is the invited address to the annual American Psychological Association Convention by the distinguished geneticist Theodosius Dobzhansky (84).

Examination of the literature during the past 5 years reveals a sharp and unmistakable increase in the number of relevant publications. It also ap-

[1] The preparation of this review was partly supported by the following grants: NIMH #11774; 14076; 16899; NSF-USDP #GU-1598, and a grant from the Russell-Sage Foundation. We would like to thank J. M. Horn for valuable criticism and suggestions and the statistics on McKusick's volume cited in the section on single-gene defects. The review of the literature for this chapter was concluded late in 1969.

pears that the research represented in these studies has been broadened significantly and now extends from the molecular to the societal. The most noticeable shifts in research orientation have involved a reduction in the number of simple comparisons of inbred strains of animals, and an increase in quantitative genetic analysis at both the animal and human levels, molecular and physiological studies, single gene analyses, and a growing interest in evolution.

Several major volumes and monographs appeared during the period covered by this review. Most of the research monographs will be referred to in the appropriate sections below. However, it may be useful to mention some publications of a more general nature at this point. While a few years ago teaching materials that could be used for undergraduate and graduate instruction in behavioral genetics were limited, there are now a number of appropriate volumes available: Parsons (307); Manosevitz, Lindzey & Thiessen (279); Hirsch (175); Spuhler (376); Glass (134); Vandenberg (410). The teacher faced with a classroom of students who have limited backgrounds in genetics should find Lerner's recent book (236) a useful means of giving students some familiarity with basic genetics. The instructor teaching introductory psychology who wishes to introduce his students to behavioral genetics may find the elementary discussion by Hirsch & Ksander (181) helpful.

Space limitations prohibit a comprehensive and detailed review of all the relevant literature during the past 5 years. We have attempted to comment upon those studies that are most significant, and at the same time we have tried to provide reasonable breadth of coverage. Several reviews of the literature in this area have been published (e.g. 35, 81, 91, 106, 137, 259, 260, 287, 412) and the reader may wish to consult them for additional information.

This chapter is arranged in five major sections: (*a*) pathways of gene expression; (*b*) sensory and cognitive abilities and learning; (*c*) personality, temperament, and social behavior; (*d*) evolution; and (*e*) research methodology.

PATHWAYS OF GENE EXPRESSION

Ultimately many or all gene effects on behavior will be linked to DNA structure, action, and processes of development. This section is devoted to reviewing three selected areas in behavioral genetics that promise early success in identifying such associations. The first part deals with audiogenic seizure susceptibility; the second with alcohol preference and aversion; and the third, with studies of the influence of external environment upon gene effects.

Seizure Susceptibility

Several species including domestic and *Peromyscus* mice, guinea pigs, rats, rabbits, and man are known to convulse occasionally when exposed to

a loud auditory stimulus (351). Without a doubt, the most systematic work bearing on the genetics and physiology of audiogenic seizure susceptibility has been done with *Mus musculus*. This section will review these findings. Other reviews with different emphases are available (126, 167).

Genetic evidence.—The transmission mechanisms for seizure proneness have never been clarified since the earlier suggestions that one, two, or several genes control audiogenic seizure susceptibility. The highly susceptible dilute color DBA and nonsusceptible and nondilute color C57 inbred lines have frequently been the object of investigation. The dilute locus (*dd*) in linkage group II has received special attention because of its apparent association with seizure susceptibility (188) and its relation to reduced phenylalanine hydroxylase activity (58), a predisposing condition for convulsions in man.

It is apparent that no single genetic model can incorporate all the data. Fuller & Sjursen (124) detected seizing in 11 mouse strains tested, including DBA/2J, LP/J, 129/J, RF/J, AKR/J, LG/J, SJL/J, CBA/J, SM/J, BALB/cJ, and C57BL/6J. The phenotypic patterns of susceptibility differ widely. For example, the first six strains mentioned are seizure susceptible beginning about 3 weeks of age, whereas the latter five first convulse around 4 weeks of age. It is unlikely that one particular genetic hypothesis would be valid for all strains. Moreover, Schlesinger, Elston & Boggan (350) point out that the genetic inference will vary for DBA/2J and C57BL/6J strains and their segregating groups depending on age and which aspect of the seizure is assessed (i.e. wild running, clonic or tonic seizures, or death). According to these investigators at least two separate genetic systems operate—one determining wild running and one affecting the tonic seizure. If a single locus is involved it cannot be the one associated with dilute coat color, as neither segregants between dilute and nondilute mice, nor inbred mice differing only at this locus, give the expected result (168, 350). Recently another allele, *asp*, located at a locus in linkage group VIII, has been implicated (60). This double recessive apparently accounts for much of the variance between DBA/2J and C57BL/6J mice. The genetic situation is complicated by the fact that activity differences among animals within a strain (DBA/1J) relate to death rate under auditory stress, and that nearly any inbred strain can be induced to seize with prior exposure to auditory stimuli during a critical age period. Strains are differentially affected by this treatment, again suggesting that the underlying genetics are complex.

Physiological evidence.—Research in the past 5 years has turned from the study of energy cycles, oxidative phosphorylating systems, and endocrine factors to the investigation of neurotransmitter substances and the peripheral processing of sensory data. Schlesinger and his associates have vigorously pursued the study of the neurotransmitter substances serotonin (5-HT), norepinephrine (NE), and gamma-aminobutyric acid (GABA)

(351). Using a variety of genetic and pharmacologic techniques, 5-HT, NE, and GABA levels in the brain have been elevated or decreased in an attempt to associate seizure susceptibility to these transmitters. In summary, these investigators find that decreases in 5-HT and NE, either singly or in combination, elevate seizure susceptibility in both genotypes, whereas an increase in these substances and GABA decreases seizure susceptibility. Moreover, the manipulation of these brain substances also effects metrazol-induced and electroconvulsive-induced seizures in a similar fashion, leading to the suggestion that seizure susceptibility is merely another index of brain excitation. Similar hypotheses were held in earlier years.

The second major strategy for investigating seizure proneness has been to subject mice to a "priming" auditory stimulus either before audiogenic seizures can normally be elicited or when the mice are anesthetized and cannot seize. Apparently any mouse strain subjected to auditory priming will seize at a later time when exposed to the same noise, including C57BL/6J, BALB/cJ, SJL/J, and various crosses between these strains (170). DBA/2J is apparently at such a high convulsive level that priming does not enhance susceptibility. In crosses between DBA/2J and C57BL/6J strains, primed F_1 offspring deviate in the direction of the C57BL/6J. Priming is ineffective before the ears become functional around 14 days of age and is maximally effective between the ages of 16 and 19 days (169). The priming effect is apparently long-term and can be restricted to one ear by temporarily blocking hearing in the other during the priming interval (123, 169). The ipsilateral effect and the fact that anesthetics that dampen the CNS have no inhibiting effect on priming strongly implicate peripheral mechanisms, possibly in the ear itself. The generality of the findings point to a fundamental mechanism of control. Peripheral mechanisms may not only regulate the onset of seizing but also its inhibition, inasmuch as Ralls (323) demonstrated that DBA/2J and BALB/cJ mice lose much of their auditory sensitivity with age.

Alcohol Preference and Aversion

There have been several recent studies of the genetic and physiological mechanisms underlying alcohol preference and aversion in mice, rats, and man (100, 238, 259, 331, 349). Work has focused on the nature of the genetic system and metabolic variations that influence alcohol ingestion.

Genetic evidence.—Both rats (99, 100) and mice (331, 349) respond to selection pressure with increases and decreases in alcohol preference. Selection is effective over a number of generations, and heritabilities are low to moderate. Studies of this kind continue to support the notion that alcohol ingestion is polygenetically controlled. Fuller (118) points out that mouse strain differences are partly attributable to the drinking schedule imposed and the number of choice solutions available during the test.

Sex differences are in some cases prominent in mouse and rat strains.

Eriksson & Pikkarainen (101), for example, point out that female C57BL, but not CBA female mice, drink more than their male counterparts. Strain differences in males account for about 69% of the variance, and in females about 94% of the total variance. Similar differences have been found for rats selected for high and low preference (100).

Other evidence indicates that Maudsley Reactive (MR) and Nonreactive (MNR) rat strains selected for emotional behavior in an open field also differ in their preference for a 5% ethanol solution (32). The MNR strain displayed a higher preference score. A 2×2 diallel cross gave evidence for an additive genetic system with high heritability (about 70%). Interestingly, Brewster reanalyzed earlier data collected by McClearn & Rodgers (262) on the C57BL/Crgl and A/Crgl mice, and data by Fuller (116) on C57BL/6J, C3HeB/FeJ, A/J, DBA/2J, and F_1 groups, and found high estimates of additive variance (82% and 86% respectively) and evidence for incomplete dominance. In another study (95) "domestic hooded rats" and "wild pack rats" (genus *Neotomo*) were found to differ in the amount of absolute alcohol consumed under forced and choice conditions.

Perhaps the most interesting recent genetic experiment is that by Fuller & Collins (122). Using C57BL/6J high-preferring mice and DBA/2J low-preferring mice, nonparametric analyses with F_1, F_2, and backcross generations revealed that for both total ethanol consumed and amounts relative to water, a two-unit model closely fits the data. This result contrasts with those discussed above but may indicate that a major portion of the phenotypic variance in some strains is due to major gene effects. In view of recent biochemical findings (see below) this observation may be highly significant.

Physiological evidence.—Early evidence suggested that mouse strain differences in alcohol preference corresponded to parallel differences in the liver enzyme alcohol dehydrogenase (ADH), with the C57 genotypes scoring higher on both variables than DBA and other strains (331). ADH activity is the first step in the metabolism of ethanol to acetaldehyde and therefore could be of critical importance. Although individual reports vary in their conclusion about metabolic differences between the C57 and other genotypes (206, 331, 349, 352), it is generally agreed that ADH activity and voluntary consumption of ethanol are at least partially related and that strains do not drink beyond their metabolic capacity (333). When, however, ethanol ingestion is high over long periods and coupled with lowered nutrition, striking pathologies appear in some strains (e.g. C3H/HeJ and C57BL/10J) that correspond to those often found among human alcoholics (333). Obvious intoxication can be produced in C57BL/6J animals by restricting their intake to a short period of the day (118). Mice tend to compensate for fluid deprivation by lowering their preference for this dipsogenic drug (387). C57BL/Crgl mice, but not RIII mice, return toward their original preference when fluid balance is re-established by an *ad libitum* choice schedule or by injection of isotonic saline.

Conversely, alchohol ingestion is increased severalfold when C57BL/Crgl females are nursing young (390). This elevation is related to increases in liver size, metabolic capacity to dispose of blood alcohol, and the number of mice being lactated (391). Population density or other stresses, on the other hand, tend to decrease, not increase, alcohol ingestion (331, 332, 389), which casts some doubt on the common assumption that alcoholism and anxiety are positively related. In mice, at least, metabolic considerations predominate.

While previous research has concentrated on ADH as the rate-limiting enzyme, investigation is now turning to the enzyme responsible for the oxidation of acetaldehyde, namely aldehyde dehydrogenase (ACDH). ADH and ACDH enzymes in combination may account for the major restrictive pathway of ethanol breakdown through the citric acid cycle (349, 352, 362). ACDH may assume the primary importance, as drinking and nondrinking strains of mice differ by 300% in this enzyme (362).

In any case, it is interesting to speculate about the relation between the two crucial enzymes discussed and the two genetic units suggested by biometric analysis of the behavioral variance (122).

Gene Regulation

Recent developments in microbiology and developmental genetics offer behavior genetics new challenges. Not only must behavior geneticists concern themselves with transmission of genetic information but also with regulators of gene expression. Perhaps not more than 10% to 20% of the assembly of genes are active at any one time, depending upon the tissue and environment considered (21). Attention, therefore, is turning to models of gene regulation that may account for some of the dynamic qualities of behavior (33, 286).

The regulatory mechanism best known involves the molting patterns of *Diptera* and midge (*Chironomus*) from larva through pupa and adulthood. The sequence depends upon the molting hormone, ecdysone, acting on loci of chromosomes I and IV (21, 57). Hormones in mammals which appear to stimulate messenger RNA production and protein synthesis probably underlie many important behavior processes (382).

Not many clear examples of gene induction and behavior are yet known. Environmental stimuli such as avoidance training can alter the kinds and amounts of RNA and protein formed in the brain (127, 424). Hydén (192), in particular, details how environmental influences can alter primary genetic action. In one experiment "right-handed" rats were trained to use their nonpreferred paw to obtain food. There were significant increases in RNA in the motor cortex associated with the change in preference, and the bases used to form RNA (adenine, guanine, cytosine, and uracil) showed unique changes. The (G+C)/(A+U) ratio decreased from 1.72 to 1.51. Moreover, the nuclear RNA formed was said to have a DNA-like base ratio composition.

Apparently genes in neural centers appropriate to the behavior involved are differentially activated and expressed as messenger RNA.

Studies using other techniques can be interpreted in a similar fashion. Drugs that selectively interfere with DNA transcription, RNA translation, or protein synthesis are known to disrupt learning, memory, and reproductive functions (18, 110, 298, 392). Similarly, amino acid substitutions in the adrenocorticotropic hormone (ACTH) can limit extinction of an avoidance response in rats (28). It is just such gene regulated changes that would be expected to influence behavior and evolutionary processes.

Biochemical brain changes related to enriched or deprived environmental and social settings (especially those involving enzyme effects) strongly suggest a regulatory function at the level of DNA (11, 69, 340). In fact, the immense increase in the amount of DNA material in higher species without substantial change in the number of biochemicals present may be interpreted as evidence that such gene-level regulatory processes are of considerable importance in the process of evolution (33).

SENSORY AND COGNITIVE ABILITIES AND LEARNING

In this section we will review studies which focus primarily on the genetic basis of behavioral capacities, ranging from single-gene studies of sensory functions in inbred mouse strains to general intelligence in man.

Single-Gene Effects on Sensory and Motor Functions

Locomotor coordination.—Behavioral observations on neurological mutant mouse strains have provided a useful tool for investigating locomotor behavior. Fox (111), using a battery of reflexological and behavioral tests, has been able to classify mutants as static, in which vestibulocerebellar lesions can be detected early in the neonatal period (*twirler* and *waltzer*), and progressive, in which the defect begins later and the condition of the mice deteriorates (*ducky, dystonia musculorom, reeler, jumpy,* and *jittery*). Thiessen (384) has studied several developmental features of the *wabbler-lethal* mouse, concluding that behavioral observations can sometimes be used to identify the mutant before brain lesions (myelin degeneration) are obvious. Sidman et al (368) have developed a sensitive conditioned suppression method for studying auditory thresholds and have applied their technique to the study of *pirouette, quaking,* and *reeler* mutants. *Pirouette* mice have an elevated auditory threshold which accompanies a loss of outer hair cells in the cochlea. Lastly, in a careful series of studies, Erway, Hurley & Fraser (105) have demonstrated that a swimming disability of *pallid* can be rectified by supplements of manganese during prenatal development and is related to the re-establishment of otoliths in the vestibular portion of the inner ear. A phenocopy can be produced by a dietary deficiency of manganese. This is one of the rare cases in which the phenocopy-inducing agent has a reciprocal effect on the mutant itself.

Retinal degeneration.—In most C3H inbred stocks, as well as several other commonly used strains, photoreceptor cells degenerate by the second postnatal week (217, 366), resulting in a 10^{-5} reduction in visual sensitivity, although the spectral sensitivity curve is unaltered (365). Unfortunately, the C3H line has been used in visual experiments by investigators unaware of the magnitude of the defect; it appears likely that the major learning disabilities found can be attributed to this mutant gene (30, 31). It is probable that the peripheral lesion is followed by degeneration of higher visual centers (150). Several lessons can be gained from a study of selected neurological mutants, not the least of which is that subtle aspects of behavior may be understood better once major or gross relationships are identified.

Albinism.—Albinism results from an inherited loss of melanin formation in skin, hair, and eyes. Oculocutaneous albinism in mice, and probably other species including man, is apparently due to the lack of tyrosinase synthesis and hence a failure of tyrosine to be converted to dopa, dopaquinone, and melanin (433). At the *C* locus in mice, the allelic series, *agouti, chinchilla, himalayan, extreme dilution,* and *albino,* regulate the number and size of malanosomes and the amount of melanin deposited on each. Tyrosinase activity is highest in *agouti* and totally absent in *albino* (433). Several reviews of the genetic and physiological control of coat color in mammals are available (79, 322, 388).

Albinism in man has been known for over a century to result in photophobia (109), and since the early studies of Keeler & King (219) it has been suggested that albinism and behavior are related. Mice, in particular, have become favorite subjects for investigation, because much is known about coat color variations in this species and mice offer possibilities for precise genetic control (433).

Several experiments with mice and rats suggest that some strain differences in behavior may be related to albinism (70, 83, 239, 240, 363). Much of this research was stimulated by the finding of Winston & Lindzey (430) that albino inbred strains and albino mice derived from F_2 and backcross generations were deficient in water escape performance. The increased latency to escape water has been amply verified (119, 388, 420), and it is generally agreed that albinism is associated with reduced activity under bright, white-light conditions (75, 119, 171, 388). Importantly, such genetic differences in activity often disappear under red or dim light (75, 119, 388), implicating photophobia as the primary mechanism of control. In strong confirmation, DeFries (73) selected for high and low activity from an F_3 population containing albino segregants and found that he was concomitantly selecting for high and low frequency of albinism.

Photophobia cannot account for all differences between albino and pigmented mice, as many behavioral variations can be found in situations where vision is relatively unimportant. For example, albino F_2 mice are inefficient in active avoidance learning but highly efficient in passive avoid-

ance learning (431); they also appear to be less sensitive in their geotaxic response to gravity (388), and respond less readily to an audiogenic seizure stimulus (145). In other segregating populations albino mice show a fear of heights (3) and a slight advantage in sexual competition (242). Albinism even leads to a reduction in alcohol preference in a two-bottle water-alcohol preference situation (171). Clearly the albino effect is extensive and cannot always be related to the absence of visual pigment. It would be interesting to examine all the alleles at this locus in an attempt to find a relation between tyrosinase activity and some of the behaviors implicated in the above studies.

Screening on multiple measures.—A number of investigators have examined inbred mouse strains on batteries of different behavioral measures. While for convenience these studies are considered here, many of the individual measures could properly be discussed with studies of motivation and temperament later in this review. Abeelen (1) has studied a number of different behaviors in several groups of mice that differ at single loci. It is Abeelen's hope to construct an "ethogram" of natural behavior by observing natural units of behavior. Approximately 25 species specific behaviors were recorded for the following alleles: *maltese dilution*, *pink-eyed dilution*, *looptail*, *jerky*, and *waltzer*. The latter three are neurological mutants. Only the pink-eyed gene among the non-neurological mice had obvious effects on behavior, among which was a reduction in reconnoitering, a decrease in lifting of one forepaw and tail rattling, and an increase in gnawing at a wall. Unfortunately the number of animals tested was often small and the background genotypes were not standardized, so it is difficult to interpret these findings.

Thiessen, Owen & Whitsett (388) have screened 14 genes segregating on the C57 background for open-field activity, negative geotaxis, water-escape latency, and wheel revolutions. As with many other studies, these loci are related to changes in coat color. The genes studied were *black and tan*, *tanoid*, *intermediate agouti*, *yellow*, *viable yellow*, *brown*, *buff*, *himalayan*, *caracul-J*, *misty*, *white*, *steel-Dickie*, *tortoise shell*, and *dominant spotting*. Both dominant and recessive genes were included in this survey, and several chromosome units were represented. Some gene specificity was evident, which gave the investigators hope that "behavioral genes" could be mapped on chromosomes.

Werboff, Anderson & Ross (420) studied brown, black, and albino littermates obtained from a four-way cross between inbred mouse strains. The animals were tested for open-field activity, visual and spatial learning in a water maze, submersion time in water, and amphetamine effects on submersion time and water escape. Albino mice with weights attached to their tail submerged less readily than black mice, and were poorer learners on visual and spatial problems. The results could indicate a pleiotropic effect on several homeostatic mechanisms.

Normal Human Abilities

The broad spectrum of human abilities which have received attention from the behavior geneticist during the period under review ranges from general intelligence to such simple sensory capacities as color vision and taste sensitivity. There have been a number of excellent reviews and discussions of this research (180, 190, 198-200, 378, 395, 406, 408, 409).

General intelligence.—Perhaps the most important paper on this topic during the period under review is that of Burt (48), who presents data on the resemblance in IQ of 53 pairs of identical twins reared apart, as well as summarizing his own work and that of others on the correlation in IQ of persons of differing degrees of relationship. Not only is his group of separated pairs the largest so far reported, but all twins were separated within the first 6 months of life, and there was no selection for environmental similarity—in fact, there was a slight negative correlation between the socioeconomic status of homes in which separated twins were reared. This appears to have resulted from the scientifically fortunate circumstance that in many of his cases one twin was given up for adoption by impoverished parents unable to care for both, the one twin thus being reared in a relatively well-to-do environment, while the other remained in a lower-class home. The Stanford-Binet IQ's of the separated twins correlated .86. This is not very much lower than the figure of .92 obtained by Burt for 95 identical twin pairs who were reared together. In reply to critics (244, 381), Burt (49) reports that for the subgroup of 27 pairs separated within the first month, the IQ correlation of .84 is essentially as high as for the entire group. [Along the same lines, Vandenberg & Johnson (414) reanalyzed data from separated identical twins in studies prior to Burt's, to show that twins who are separated relatively late do not turn out to be more alike in IQ than those separated relatively early.] Prenatal environmental effects are not ruled out as contributing something to the resemblance of the separated pairs in Burt's study, but if one places very much weight on prenatal environment as a cause of similarity, it creates difficulties elsewhere—for example, why should parent and child be almost as similar in IQ as siblings, and why are not fraternal twins more alike than they are? As for the cultural environment, Burt does not exclude its effects altogether; in fact, he presents evidence of a modest correlation of .26 between rated differences in cultural conditions and IQ differences for the separated monozygotic pairs. (Note that such a correlation says nothing about the relative importance of heredity and environment: it merely indicates that the MZ IQ differences that *do* exist—which are necessarily environmental—show some association with ratings of home conditions.)

A number of studies during the period present data further supporting the well-established generalization that identical twin pairs tend to be more similar on measures of general ability than same-sex fraternal twin pairs.

Huntley (190), with a composite vocabulary test given to 85 identical and 135 fraternal pairs, obtained correlations of .83 and .66, respectively; Nichols (300), with the National Merit Scholarship Qualifying Text (NMSQT), obtained correlations of .87 and .62 for 315 and 209 male pairs, and correlations of .88 and .65 for 372 and 273 female pairs. Schoenfeldt (354), for a general intellectual factor from the Project TALENT battery, obtained correlations of .78 and .39 for 150 and 53 male pairs, and .81 and .52 for 187 and 103 female pairs. Obviously there is some study-to-study fluctuation in these correlations, which could result from differences in the measuring instruments, differences in the populations, differences in the accuracy of zygosity determination, and sampling error. As discussed in a subsequent section of this review, estimating heritability from twin data is a somewhat tricky business, but on typical assumptions these twin correlations suggest heritabilities a little lower than the .86 "broad sense" heritability suggested directly by Burt's data—perhaps more on the order of .75 (the authors give various estimates ranging from .60 to .88). Since any heritability estimate is specific to a population and a measure, some variation is to be expected.

Heritability coefficients do not provide much information about the nature of the genetic determinants of IQ. One potential source of such information lies in studies of the effects of inbreeding. If a substantial proportion of the genes involved show dominance, then the mating of related persons will increase the probability of matching up recessive alleles. If the direction of dominance is predominantly toward high intelligence, the trait will tend to show "inbreeding depression" among the offspring of related marriages. Schull & Neel (355), who were carrying out large scale studies of the genetic effects of the atomic bombings in Japan, took advantage of the relatively high frequency of cousin marriages in that country to give a Japanese version of the Wechsler Intelligence Scale for Children (WISC) to 2111 Hiroshima school children, half of them the product of marriages of related individuals (in the range of first to second cousins) and half whose parents were unrelated. After controlling statistically for socioeconomic differences between the groups, they found an average depression of 3-5% in the offspring of first cousins, slightly greater for verbal than for performance IQ. Thus there is some evidence for directional dominance of genes influencing intelligence within the normal range. It is also interesting to note that IQ proved to be one of the most sensitive indicators of inbreeding depression in their studies, which also included a variety of other psychometric and anthropometric measures.

Special abilities.—Another way of trying to understand the genetic and environmental influences on general intelligence is to break it down into more specialized component abilities. A good deal of effort has been expended along these lines in the period under review. Nichols (300) reports an analysis of the subtests of the NMSQT after partialling out statistically the large general factor. While the results were rather erratic, there was some indica-

tion that the specific abilities were of about the same level of heritability as the general factor. There was no consistency across the sexes as to the relative heritability of the specific abilities. For the Project TALENT twin sample, Schoenfeldt (354) reports data on three differential aptitude factors: visual reasoning, perceptual speed, and memory. He found somewhat lower heritabilities for these than for his general intelligence factor, with the median coefficient in the .50s; again there was no consistency across the sexes. Vandenberg and various associates have been especially active in this field, examining the performance of high school age twins from the Louisville area on the Wechsler Adult Intelligence Scale (WAIS) (26), the Differential Aptitude Tests (DAT) (27, 409), the Primary Mental Abilities (PMA) (255, 408), a battery of spatial visualization tests (411), Guilford's divergent thinking tests (416), and a variety of other measures. Some of the same tests have also been given to a small Atlanta twin sample by Osborne & Greger (305, 306). The results of all these studies may be summarized by saying that identical pairs are typically more similar than fraternal pairs on cognitive ability traits, and that there are no very clear indications that the similarity is consistently greater for some abilities than for others, when measured with equal reliability. The lack of agreement across the sexes in the Nichols and Schoenfeldt studies is perhaps best treated as a particular case of this general phenomenon, which surely constitutes a challenge for the theorist. We will encounter it again when we consider personality.

Several studies with older (197, 311) and younger twins (113, 416) suggest that rather similar conditions prevail across the life span.

In addition to analyzing the variance of single traits into genetic and environmental components, one can analyze the covariance between traits in much the same way; it is often instructive to ask whether what is shared by two correlated traits is primarily common genes or a common set of environmental influences. Furthermore, one can readily extend this logic to a factor analysis or other multivariate analysis of a whole matrix of covariances (or correlations). Analyses along these lines have been reported for the PMA tests (255, 405), the WISC (408), the DAT (27), and a Finnish test battery (311). In some analyses, a large general factor (Spearman's "g"?) emerged in the genetic covariance; however, this was not the case in others. Differences in method of analysis may have been partially responsible—but in any case, for dependable results these approaches probably require somewhat larger sample sizes than have been used so far.

Sensory capacities.—Active work continues with color vision and the ability to taste low concentrations of phenylthiocarbamide (PTC). No attempt will be made here to provide a serious review of this extensive literature; an illustrative example or two must suffice.

A good deal of recent effort has been directed toward mapping the frequency of color and taste insensitivities in various human populations, among which considerable variation exists. Interest in this area has been

stimulated by the suggestion of Post (320) that the relatively high incidence of red:green color blindness in long-civilized peoples may represent a relaxation of selection against such defects in these populations. Evidence indicating a low incidence of color-vision defect in primitive peoples continues to be reported (e.g. 90, 326), but there are some discrepancies, and some strongly skeptical views persist (4).

Research on sensitivity to the taste of PTC and other thiourea derivatives is being carried out on various fronts. A particularly extensive research program is that of Kaplan and his associates (summarized in 208), using 6-*n*-propylthiouracil (PROP), a substance which has technical advantages over PTC but is otherwise similar. Taste thresholds for another unrelated bitter substance (quinine) and a sour substance (hydrochloric acid) were measured in the same subjects. A twin-sibling study (210) showed the threshold for tasting PROP, but not for the other substances, to be under a high degree of genetic control. Many variables affecting the taste threshold for PROP seem not to be closely related to the single taster gene. For example, the thresholds for the two bitter substances PROP and quinine were correlated across individuals and tended to behave similarly with respect to age and smoking, the menstrual cycle, and food preferences. An interesting exception was provided by duodenal ulcer patients, who were relatively sensitive tasters of PROP, but not of quinine or hydrochloric acid (in comparison to gastric ulcer patients or normal controls).

Mental Deficiency

Two categories of mental retardation are often distinguished: severe retardation, with IQ less than 50, and mild retardation, with IQ from 50 to 70 (or whatever is specified as the lower limit of normal intelligence). Major chromosomal anomalies or single gene defects tend to produce severe retardation; most mild retardation is taken as merely the low end of the normal distribution of intelligence, whose genetic basis is presumably polygenic (for discussions, see 200, 438). After glancing at some studies of familial incidence of mental retardation, the present review will focus on the more severe forms, concerning which there is more genetic information. But even here the treatment will necessarily be brief. Fuller reviews and discussions are available (e.g. 20, 439) and other references are cited below.

The massive study of Reed & Reed (325) began with the grandparents of 289 Minnesota mental defectives initially studied between 1911 and 1918. More than 82,000 of the descendants of these grandparents were traced, some over as many as seven generations. The bulk of the Reeds' report consists of detailed pedigree charts of these kindreds, including a large number of persons for whom IQ scores were available through schools and other agencies. Summary data on the frequency of retardation among the offspring of consanguineous unions and various types of marriages of normals and retardates are presented and discussed. There is also information on assortative mating, sibling and twin differences, and the relation between IQ

and family size (slightly positive, when childless individuals are properly taken into account).

Another study of fertility and mental deficiency is reported by Åkesson (7), based on 7533 people living in a random sample of rural parishes in southern Sweden. He found 88 mild and 44 severe mental defectives in this population, or 1.8%. The mildly retarded produced decidedly more offspring than a comparison group of normals. However, if the severely retarded group (who were "practically infertile") are added, the retarded appear to be only slightly overproducing. In another population study, this time in west Sweden, Åkesson (8) found 105 cases of severe mental deficiency among 17,303 inhabitants, or .6%, agreeing well with the previous study. About 20% could be identified as to probable cause (with trisomy-21 the most common); the remaining 80% were of unknown etiology.

Chromosomal anomalies.—After the dramatic discoveries of the preceding 5 years, the last 5 have been relatively quiet. Still, a large literature is developing in this area, which we cannot hope to survey in detail. Fortunately, there are available a number of excellent books, reviews, and bibliographies (51, 63, 209, 312, 380, 401, 436). The major trisomies of autosomal chromosomes 13, 18, and 21 appear to be firmly established, as well as partial deletions of the short arm of chromosome 5 (*"cri du chat"* syndrome), and of the short and long arms of chromosome 18. All produce severe mental defect, among other symptoms. Those that survive presumably represent relatively benign conditions. Some 20% of spontaneously aborted fetuses show chromosomal anomalies (51, 404), and the more gross aberrations may not even get to that stage. Many new syndromes are being reported, some with quite complicated chromosomal aberrations postulated, involving several breaks and insertions (e.g. 76). Some of these anomalies are balanced (i.e. merely involve a rearrangement of normal genetic material) and seem to have no obvious phenotypic effects, although they can lead to unbalanced conditions in offspring. Others are unbalanced (i.e. involve a duplication or deletion of genetic material) and do more or less severe damage, almost invariably including mental defect. Lejeune (234) has suggested than an excess and a deficiency of particular chromosomal material may tend to have opposite somatic effects (e.g. a partial monosomy of chromosome 21 led to hypertony, a prominent nose, hypocanthus, a narrow pelvic angle, and other "antimongol" symptoms); mental retardation, however, shows up in either case.

Anomalies of the sex chromosomes have less severe effects on intelligence: an extra X or Y is compatible with normal intelligence or mild retardation. However, a multiple duplication like XXXY or XXXX (or more) usually leads to serious mental deficiency (234). For one ability, the capacity for visualizing spatial relationships, there has been discussion of a simple mode of sex-linked inheritance. Garron (129) provides an up-to-date review of the evidence on this topic.

Mosaicisms (i.e. mixtures of two or more cell lines with different chromosome complements) are increasingly often reported, particularly among the sex chromosomes, but for autosomes too. Such mosaicisms may vastly complicate interpretations, since the cell proportions may be quite different in different organs, and may change over time. But by the same token, transient mosaicisms should sometimes be capable of providing tests of hypotheses about critical developmental periods.

Some chromosomal anomalies are inherited (via translocations), but the majority are sporadic, of unknown origin. An external environmental agent (radiation? viruses?) is often suspected (71, 330, 402). Some such theories would predict an association between different chromosomal anomalies. While associations of this kind have been reported (401), there is also negative evidence (160).

Single-gene defects.—Among the 1545 syndromes known or suspected to be due to a single gene which are described in the second edition of McKusick's *Mendelian Inheritance in Man* (268), 134 have mental retardation listed as one of their symptoms. Seven of these syndromes involve autosomal dominant genes, 112 involve autosomal recessives, and 16 are X-linked. In total, McKusick's book lists 793 autosomal dominant, 629 autosomal recessive, and 123 X-linked syndromes. Thus those single-gene syndromes which include mental retardation constitute only about 1% of the dominant category, while they account for 18% and 13% of the other two. No doubt the shortage of dominant syndromes is in part due to the difficulty of recognizing such syndromes in disorders which effectively prevent the reproduction of affected individuals, such as severe congenital mental defect. But it is conceivable that the mode of gene action is also involved: according to McKusick, recessive genes typically act on enzyme systems, dominant genes on structural protein—and this also might have some relevance to theories of intellectual development.

Morton and his colleagues (82, 297) have attempted to estimate the role of major genes in the etiology of severe mental defect. After excluding cases due to known chromosomal anomalies, trauma, infection, and the like, they examine "high-risk" families, with more than one severe defective in a sibship. From the relative frequency of such sibships, and the degree of parental consanguinity, they arrive at estimates of the number of gene loci involved. Based on a Wisconsin population which involved 49 high-risk families, and Penrose's earlier Colchester data, they conclude: that on the order of 114 recessive loci contribute to severe mental defect; that these are maintained by mutation, not heterozygote advantage; and that they account for only about 12% of severe mental defect. The remaining 88% of sporadic cases must thus be due to other causes, perhaps including unrecognized exogenous factors, small chromosomal aberrations, and the heterozygous expression of usually recessive genes.

Space does not permit a systematic review of the literature on individual

single-gene defects producing mental retardation. Some examples may be found described and discussed in (14, 187, 293). And readers wishing to curl up with the literature on gangliosidosis, the Prader-Willi syndrome, or the oasthouse urine disease can get a start in McKusick (268).

Animal Learning

Maze learning.—Behavioral genetic studies of maze learning have used rats, mice, and dogs. Most of the studies have focused on strain differences and little work going beyond this has been reported (96, 155, 263, 314). Studies with mice have shown strain differences for T-maze reversal learning in four inbred strains, but no differences in performance as a function of intertrial interval (53). Heritabilities based on the four inbred strains ranged from .59-.70 for the various intertrial intervals. A study by Werboff & Anderson (419) on water maze learning evaluated the preference and use of visual or spatial cues in two inbred strains, A/J, C57BL/6J. Both strains of mice showed spatial preference but this was stronger in the pigmented C57BL/6J strain.

Avoidance conditioning.—Of all learning studies, avoidance conditioning studies have been the most popular. Several studies to determine optimal parameters for avoidance conditioning in mice have been reported. Royce (342) found that 3-sec CS-US intervals, 120-sec intertrial interval, and 400 V levels were optimal for avoidance conditioning, but that considerable variation exists around these parameters. There was also some limited evidence that CS-US interval may interact with genotype.

Several studies have demonstrated strain differences in active and passive avoidance conditioning (31, 52, 356, 425, 431). Bovet and his colleagues have reported a series of studies on genetic and environmental determinants of learning and memory. On the basis of studies using massed and distributed practice, extended training sessions with variable rest periods, one-trial avoidance learning, ECS, and various drugs, they suggest that a two-stage concept of memory storage, with the C3H/He as a "short term memory" strain and the DBA/2J as a "long term memory" strain, appeared to be a plausible interpretation. As discussed in a preceding section, the interpretation of such experiments is complicated by sensory defects in some of the strains involved. Research by this group using light or tone as the CS in avoidance conditioning studies indicated that strains may respond differently to various CS (303). Thus studies using various CS and genotypes would be useful. Data (from the DBA/2J inbred strain only) on active avoidance learning (jumping response) and passive avoidance, as reported by Wimer et al (425), using massed and distributed practice and immediate post-trial etherization, are contradictory to those reported by Bovet et al (31). The nature of the conditioned avoidance response (CAR) and other procedural differences may be responsible for the inconsistency. One might

expect that considerable work in the next few years will be devoted to reducing these differences. In a study (353) using seven inbred mouse strains and four F_1 hybrids (males only), in which the CAR was jumping onto a shelf, strain differences and heterosis were demonstrated. In some of these studies the same inbred strains of mice were tested (31, 353). Although there are differences in procedures and the CAR being measured, there is some agreement as to which strains are fast and which are slow learners. Roberts' work (327) on the effects of electric shock on skin resistance (presumably a measure of arousal) in inbred mice showed significant strain differences in skin resistance. On the basis of his results, it appears that there are genetic differences in aversiveness of grid shock and that strain differences in avoidance conditioning could be partly a result of differences in shock aversiveness. In a study (431) in which subjects were given the option of avoiding actively or passively, it was shown that albino mice do not differ from non-albinos in avoidance conditioning. However, albinos differ in the response mode of avoiding aversive stimulation; they usually avoid passively whereas non-albinos may avoid actively or passively.

Further evidence on the importance of genetic factors in CAR has come from selection experiments. Bovet et al (31) reported progress after only three generations of selection for higher avoidance scores, starting with a heterogeneous SWR mouse base population. They do not report the method of selecting breeding pairs or the criterion by which high CAR subjects were identified. A selection study reported by Bignami (23) used a random bred foundation stock of Wistar rats. Selection progress was made in both high-avoidance (RHA) and low-avoidance (RLA) lines by the fifth generation. An experiment by Broadhurst & Bignami (37) tested the two lines for CAR and in the open field test of emotionality, to determine if avoidance conditioning was associated with open-field reactivity. In the Maudsley lines the reactive strain had been shown to be low in CAR, and the nonreactive strain to be high in CAR. These results could have been due to fortuitous joint selection or to pleitrophy. The association between avoidance learning and open-field emotionality was not found with the Bignami strains. Thus, the two selection experiments appear to have selected for different and non-correlated characteristics. A subsequent factor analytic study (182) using the RHA and RLA lines from the eighth generation showed that at least one difference between the two lines is in activity.

PERSONALITY, TEMPERAMENT, AND SOCIAL BEHAVIOR

In this section we will review studies which focus primarily on genetic factors that influence temperamental and motivational aspects of behavior and social interaction. The range is from insects to man, and from normal behavior to pathological reaction. The reader is also referred to the earlier section "Pathways of Gene Expression" for relevant material on audiogenic seizures and alcohol preference.

Animal Studies of Temperament, Motivation, and Social Behavior

Emotionality.—Behavioral genetic analyses of emotional behavior have typically been concerned with open-field activity and defecation in mice and rats. Although other dependent measures of emotionality have been used by comparative psychologists, such measures have not been used extensively in behavioral genetic analyses. Strain differences in open-field behavior of mice and rats have been reported in previous *Annual Reviews* (115, 261) and additional reports of strain differences have appeared in the last few years (e.g. 5, 78, 374, 423).

A genetic analysis of repeated open-field tests using F_2 and F_3 mice (161) showed that h^2 decreased on Day 2 for both defecation and activity, but the decrease in h^2 of open-field activity was due to a decrease in V_A and an increase in V_E+V_{NA}, while the decrease in the h^2 estimate for defecation was largely due to a decrease in V_A. Genetic correlations of Day 1 and Day 2 activity and defecation measures showed that activity and defecation on Day 2 are influenced by some of the same genes, but defecation on Day 1 appeared to be associated with different genes.

Results from five generations of artificial selection for high and low open-field activity using mice were reported by DeFries & Hegmann (74). Differences in defecation were associated with the significant differences obtained for activity. The results of their selection study were similar to those obtained in their preliminary genetic analysis based upon P_1, P_2, F_1, F_2, B_1, B_2, and F_3 generations. This study makes a valuable contribution by comparing various methods of estimating genetic parameters.

Bruell's (43) diallel study of the mode of inheritance of defecation in a maze revealed that in both inbred and hybrid subjects males defecated more than females, and that inbred females defecated more than hybrid females, but that hybrid males defecated more than inbred males. Thus, the mode of inheritance was heterotic but in different directions for males and females. Exploratory activity in the maze was positively correlated with defecation in males but negatively for females. It was suggested that these results are not as puzzling as they may appear at first. The observed sex differences are consistent with an interpretation of defecation as a territory marking response.

Broadhurst (36) summarized his previously reported diallel study of open-field defecation and activity using covariance-variance diagrams to present his results. Estimates of genetic parameters obtained through various techniques were compared, and genotype-environment interactions in developmental stability and change were investigated. He reported that for defecation heritability in the narrow sense decreased across the four daily trials. A finding similar to that of Hegmann & DeFries (161) was reported and in both studies it was suggested that this is a result of decreased additive variance.

Henderson (164) also performed a diallel analysis of open-field defecation

and activity using four inbred mouse strains. In addition he used three levels of pretesting stimulation. He found that pretesting experiences influenced the overall mode of inheritance of open-field defecation (but not activity), and that the pretesting treatments interacted with genotype. He suggested that inferences about mode of inheritance should be made with caution until a variety of pretesting experiences have been studied.

A comprehensive review of the literature on perinatal factors that influence emotionality showed that many pre- and postnatal factors affect emotionality and interact with genotype (203). This review should be consulted for a discussion of the methodological problems and typical findings in the area.

Hoarding.—A number of experiments, using various inbred and crossbred mice, were conducted by Manosevitz and his associates on the determinants of hoarding (271, 272, 274, 275, 277, 278). A major consideration in these studies was the interaction between genetic and experiential factors. Strain differences were demonstrated in several studies (246, 271, 274, 275), and genetic analyses were performed to estimate various genetic parameters (278). The only significant heritable component was additivity although there was some suggestion that dominance might be present. Coefficients of genetic determination were estimated to be 37 percent in the F_2 generation and 49 percent in the parental lines (271). Heritability was estimated to vary between 29 and 54 percent (277). These results suggest that a partial answer to Bindra's (24) question "What makes rats hoard?" may be found in the genetic endowments of the subjects studied.

Several studies on hoarding have been concerned with various events, such as water immersion, neonatal irradiation, environmental enrichment, and aperiodic feeding experiences, that could affect hoarding performance, and these have been summarized by Manosevitz & Lindzey (278). These studies have provided some indication of the magnitude and general nature of genetic and environmental factors influencing hoarding. The frequent presence of genotype-treatment interactions (273) indicates that environmental experiences do not equally affect all genotypes.

Single-gene effects on motivation and temperament.—Studies relating single alleles of mice to temperament are those of Hawkins (158, 159) relating *yellow lethal* to reduced activity, Bartke & Wolff (19) implicating this same allele in estrus synchrony, and Iversen et al (194) correlating *hairless* to reduced spontaneous activity. Keeler and his colleagues are attempting to relate color changes in foxes, presumably under simple genetic control, to tameness and drug response (218, 220).

Recent efforts to identify single-gene influences on insect behavior are reviewed by Manning (270), Thiessen, Owen & Whitsett (388), and Wilcock (422). With rare exceptions the work has not been extensive, which is unfortunate in view of the fact that at least 675,000 species are available for

study—more than all other animal species combined. Rothenbuhler (341) summarizes his work on hygienic nest behavior of hymenoptera, demonstrating the influence of interaction of two genes—one influencing the opening of diseased larvae cells and the other influencing the removal of the diseased larvae. This work is a model of elegance and clearly demonstrates how a complex behavior can be resolved into individual parts and related to separate genetic units.

Animal social behavior.—A wide variety of behaviors are often considered to be "social," including dominance, affiliation, and imprinting. However, these domains have not been extensively studied in a behavioral genetics context. This remains an area where important contributions could be made by behavioral geneticists. A number of significant investigations are identified in the volume edited by Glass (134), and another valuable source is the report by Scott & Fuller (356) of their extensive study of social behavior of dogs.

Strain differences in sociability have been reported (251), and these differences in affiliation do not merely reflect differences in activity, exploration, fearfulness (defecation), or freezing behavior. Social bonds formed through imprinting were studied using purebred and crossbred chicks (142), and population heterosis was shown for all three components of the imprinting response. Several studies on social dominance in chickens were reported (65, 304). A bidirectional selection study for high and low social dominance showed that large differences were produced in five generations of selection (66). Inbreeding reduced social dominance and aggressiveness (65). A further study of high and low socially dominant chickens showed that these differences may be due to differences in physiological responsiveness to social stimuli (304).

Social dominance in three inbred mouse strains (A, C3H/Bi, DBA/8) was measured by Lindzey, Manosevitz & Winston (247) using three behavioral indices of dominance: tube dominance, food competition, and a measure of spontaneous fighting (aggression). Large and reliable strain differences in all three measures were observed. It appears that tube dominance performance is inversely related to food competition and aggression. A recent study using a group of random bred mice showed that food competition performance was associated with general activity and emotionality (276).

A partial replication and extension of an earlier study (250) on social dominance was reported using different sublines of the original strains (249). Mice from three inbred strains (A/J, C3H/HeJ, DBA/1J) were tested in the tube test of social dominance. The A/J strain was the most socially dominant, defecated more in the open field, was less active in the open field, and was lower in total body weight. This study using different sublines did not replicate the social dominance differences observed earlier between the DBA and the C3H strains.

A series of studies by Fuller on the effects of isolation and the post-isola-

tion syndrome using beagles and terriers have shown large breed differences in the responses to social isolation (117, 121).

With an increasing emphasis on the importance of social behavior for evolution, we may expect to see a continued increase in the breadth and intensity of behavioral genetic research in this area.

Normal Human Personality, Interests, and Social Behavior

During the past 5 years the total volume of available data on the inheritance of normal human personality, interests, and social behavior has probably at least tripled. It would be pleasant to say that this influx of new data has settled conclusively most of the outstanding questions in the field. It would also be untrue. Still, some progress has been made, even if in many cases the best we can say is that things are a lot harder than we thought. There are several good reviews and general discussions of work in this area (254, 395-397, 406, 407).

The largest body of new data is derived from questionnaire studies of adolescent US twins, including major studies by Vandenberg, Gottesman, Nichols, and Schoenfeldt. Other ages have not been neglected, however: Bruun and his associates have worked with adults in Finland, Koch and Scarr with US preschool and elementary school children, and Vandenberg's group and Freedman with US infant twins.

Studies of adolescent twins.—Gottesman reports data from a sample of 79 MZ and 68 DZ twin pairs from the Boston area on the Minnesota Multiphasic Psychological Inventory (MMPI) and the California Psychological Inventory (CPI) (135, 136). Vandenberg and his associates have given various twin samples in the Louisville area several personality and interest questionnaires, including the Myers-Briggs Type Indicator, the Stern Activities Index, the Comrey personality and attitude scales, and the Minnesota Vocational Interest Inventory (415, 416); their sample sizes range from 40 and 27 pairs to 111 and 90 pairs.

Nichols (301) mailed a battery of personality and interest inventories to a group of 1200 questionnaire-diagnosed MZ and DZ twins from among the large number of US high school juniors taking the nationwide National Merit Scholarship Qualifying Test in 1962, and received reasonably complete returns from both twins of about 70% of the group: 516 MZ and 334 DZ pairs. Included in the battery were the CPI, the Holland Vocational Preference Inventory, and a variety of other questionnaires, rating scales, and check lists. Schoenfeldt (354) obtained questionnaire diagnoses from 337 MZ and 156 DZ pairs from among the 400,000 high school students studied in Project TALENT, and reports results for a number of personality and interest factor measures derived from the original test battery used in that study.

Several fairly clear conclusions emerge from these investigations: 1. There is ample confirmation of the fact that pairs of identical twins tend on the average to be more similar in their scores on personality and interest

measures than do pairs of same-sex fraternal twins. Table 1 shows the median intraclass correlations obtained in various studies, for male and female identical and fraternal twin pairs. There is some variation, but overall a correlation of about .46 for identical and about .28 for fraternal pairs may be taken as representative. 2. Twin pairs of the two sexes show no striking difference in general similarity of personality traits. There is some indication of higher female correlations among the fraternals in Nichols' data, but the other samples do not support this. 3. The correlations for either identical or fraternal pairs on personality and interest measures are substantially lower than those typically found for ability measures. This is no doubt partly due to the lower reliabilities characteristic of personality measurement. But even corrected for attenuation, assuming typical reliabilities of around .75 for scales of this kind, the resulting correlations of .61 and .37 are lower than comparable figures from the ability domain. 4. Environment appears to carry somewhat greater weight than the genes in accounting for individual variation in personality traits. Holzinger's heritability coefficient based on the above corrected correlations is .38; Jensen's is .48. If one allows for a possible greater similarity of identical twin environments, the heritability estimates would be lower than this. 5. On the whole, there is little solid evidence that some personality and interest traits are consistently more influenced by the genes than others are. For example, there is little agreement in trait heritabilities across the two sexes. Thompson & Wilde (397) report the rank-order agreement between the heritability estimates for males and for females in eight studies in the literature, including most of those covered in the present review. They obtained Spearman rho's from .04 to .26, with a median of .15. Since a certain amount of purely artifactual consistency is to be expected, stemming from differences in the reliability of the scales, this level of agreement is not very impressive. Schoenfeldt (354) reports a similar lack of agreement between the sexes for the heritabilities of personality and interest factors in the Project TALENT data (not included in Thompson & Wilde's survey). These results might be interpreted as representing consistent sex differences in heritability but, alas, there is little empirical support for even this much consistency. Nichols (301) reports the correlations between the CPI heritabilities obtained in his sample and Gottesman's for males and females separately to be −.22 for males and −.24 for females. (As Nichols remarks: "Perhaps the best that can be said about these two correlations is that neither is significantly different from zero.") Thus, for the present, the most economical interpretation of the data would seem to be that while the genotype may have an appreciable effect on personality, the network of causal pathways between genotype and phenotype is so complex in this realm that the effect of genotype is spread almost evenly across the broad phenotypic measures that personality and interest questionnaires provide.

Younger and older twins.—Are things simpler, earlier? Several studies

from the period under review used younger twins. Scarr (345, 346, 348) used a small sample of girl pairs of elementary school age, 24 MZ and 28 DZ, and obtained experimenter's ratings and observations in standardized interview and experimental situations, as well as mother's ratings on the Adjective Check List (ACL). The median of the MZ and DZ correlations which were reported are .39 and .23 for measures and ratings based on the experiments and interviews, and .40 and .11 for those derived from the mothers' ACL. On the whole, allowing for sampling differences, these appear comparable to the figures cited earlier for high school age twins. Koch's detailed study of 5- and 6-year-old pairs (227) was chiefly focused on comparisons between different twin and non-twin groups, but she does report evidence that her identical pairs saw more resemblances between themselves and their twins than did fraternal pairs, and also that they shared more common experience and were psychologically closer in various respects.

Two studies report on even younger twins. Brown, Stafford & Vandenberg (39) provide some preliminary data on a group of 140 twin pairs being followed longitudinally. Only the 74 oldest pairs, aged 3 to 6 years, had been blood-typed at the time of the report. Eight variables were assessed, based on interviews with the mothers. The identical twins were more alike on seven of the eight, most strikingly so on feeding and sleeping problems. The more personality-like variables such as readiness to smile, temper, and dominance ("takes toys from twin") tended to show lower heritabilities. Freedman (113) worked with a small sample of 20 pairs during their first year. Diagnosis of zygosity at the end of the study revealed 9 to be identical and 11 fraternal. Films of the infants in standard situations were rated by judges, each judge seeing only one member of a given pair. On two items of prototypic social behavior, smiling and fear of a stranger, identical pairs were significantly more similar than fraternals.

These data on younger twins, taken together, tend to support the common observation that the greater resemblance of identical twins has early roots, but the data are insufficient to cast much light on the differential influence of heredity and environment on different traits, and thus to assist much in the interpretation of findings at later ages. A really large study of twins followed through the early years of life would be most welcome.

Partanen and his associates (311) report correlations on several personality traits in adult Finnish males, based on a questionnaire filled out by 157 MZ and 189 DZ pairs. The identical twin correlations on each of four personality measures were higher than the fraternal twin correlations; the median of their correlations (corrected for unreliability) were .54 and .36, or reasonably close to those found with US adolescent twins.

Twins reared apart.—In sharp contrast to the statistical studies described previously is the detailed case-history information presented by Juel-Nielsen (205) on 12 Danish pairs of identical twins reared apart from infancy (or early childhood, for some pairs). His personality measures were mainly pro-

jective tests, which are reported and analyzed on a case-by-case basis. In addition to these, the author reports his general impressions, based on interviews, of the areas in which the separated twins showed most and least resemblance in personality. According to him, the twins showed little resemblance in their modes of interpersonal interaction, their views on religious and social issues, their fields of interest, and the sort of persons they married. The twins also tended to be different in ambition, aggressiveness, and emotional control, and in matters of taste and dress. By contrast, the twins of a pair displayed marked resemblance in expressive movements:

TABLE 1. MEDIAN TWIN INTRACLASS CORRELATIONS FOR PERSONALITY AND INTEREST INVENTORY SCALES IN VARIOUS STUDIES

Inventory	Identical pairs		Fraternal pairs		Source
	Male	Female	Male	Female	
CPI	.53	.50	.25	.35	Nichols (301)
CPI	.45	.53	.30	.32	Gottesman[a]
VPI	.42	.42	.19	.28	Nichols (301)
TALENT	.44	.43	.33	.33	Schoenfeldt (354)
Comrey	.42	.42	.22	.19	Vandenberg et al (413)
Mean	.45	.46	.26	.29	

[a] Data reported in (136) in another form. Intraclass correlations supplied in a personal communication.

Juel-Nielsen reports being impressed repeatedly by similarities of gait, of carriage, of small gestures, of facial expression, of smiles and laughter, of tone of voice. A number of striking concordances in complaints of a psychosomatic type were also noted. In other respects their personalities tended to show an intermediate degree of resemblance, with a mixture of similarities and differences, often related to life events. A large amount of detailed information about these events is provided in almost 300 pages of case-history material on the twins. For any researcher in search of hypotheses, there is a mine of gold here.

PERSONALITY DISORDERS

Psychiatric genetics during the past 5 years could easily qualify for a whole review of its own. Consequently, the present discussion is highly selective. The psychoses, especially schizophrenia, have received the lion's share of research attention and will receive most space here.

Before becoming enmeshed in the details of particular disorders, we will mention a few items of broader interest. One is the volume on psychiatric illnesses of the handbook *Humangenetik* (20). Chapters by Zerbin-Rudin on the psychoses and Strömgren on the neuroses are of particular relevance here. Another item is a collection of Japanese studies in psychiatric genetics,

edited by Mitsuda (289). A third item is a recent book by Rosenthal (337) on genetic theory and abnormal behavior.

Finally, while the present review is organized under fairly conventional diagnostic labels, it should be made clear that how best to classify personality disorders is a very controversial matter among genetically oriented investigators in this area. Some use as their unit of analysis the presence of any psychiatric disorder. Others stick to classic Kraepelinian categories. Others subdivide these. Still others think in terms of a large number of highly specific genetic entities. To quote the population geneticist Morton on this point: "We may predict that in the future major genes (very likely as many different ones as have been identified in mental defect) will be found to account for many cases of mental disorders, leaving a component due to polygenes and nongenetic mechanisms that will defy analysis" (296, p. 183). Perhaps so. Only time (and the data) will tell.

Schizophrenia.—The landmark publication in this area during the past 5 years is clearly the Rosenthal & Kety volume on *The Transmission of Schizophrenia* (338), the proceedings of a 1967 conference at Dorado Beach, Puerto Rico, in which major US and European investigators from both genetic and environmentalist camps summarized their own research or reviewed relevant bodies of evidence. Included among the papers are reports of initial results from several important new studies, noted below. For some other major symposia and reviews in the past 5 years see References 61, 138, 334, 437. The puzzle of genes and schizophrenia is far from fitted together, but during the period under review some critical pieces took fairly clear shape.

The presence of a genetic predisposition to schizophrenia-like disorders may now be regarded as firmly established. Heston (172) matched 58 offspring of hospitalized schizophrenic mothers, given up for adoption at birth between 1915 and 1945, with 58 control adoptees of nonpsychotic parentage. Extensive follow-up beginning in 1964 located 47 of the experimental group and 50 of the control group, then at ages ranging from 20 to 50. Five members of the experimental group had become schizophrenic; none of the control group had. The frequency of schizophrenia in the experimental group was about as great as that expected among children of a schizophrenic mother reared in their own homes. There was a good deal of other assorted psychopathology in the experimental group as well: 24 other members of this group showed evidence of sociopathic personality, mental deficiency, or neurotic personality disorder, as opposed to a total of 9 such cases in the control group.

Support for Heston's main finding comes from studies of adopted children by Rosenthal, Kety, and their colleagues, preliminary results of which were reported at the Dorado Beach conference. The principal studies are being carried out in Denmark, because of the existence of superb medical and other social records there. One study (339) is essentially similar to Heston's, ex-

cept that children of either a psychotic mother or father are included, and in most cases the birth and adoption of the child occurred before the parent became overtly ill. At the time of report, 39 experimental subjects and 47 control adoptees had been examined by psychiatrists and psychologists completely blind as to their status. The results confirmed Heston's. Seven subjects diagnosed schizophrenic or borderline schizophrenic came from the experimental group, and only one (a borderline) from the control group. The Danish results, however, did not support Heston's finding of a good deal of criminal behavior, mental defect, and the like in the experimental group. Two other adoption studies by the same group (225, 418), one carried out in Denmark and one in the US, found considerable amounts of schizophrenia and related disorders in the biological families of adopted individuals who become schizophrenic, and little in their adoptive families. Another investigator, Karlsson (213), in a genealogical study in Iceland, found similar results using the biological and foster siblings of adopted schizophrenics. He also reports adoption data consistent with Heston's and Rosenthal's.

Taken together, these studies give clear confirmation of some degree of inherited predisposition to schizophrenia-like disorders. However, they suggest also that this predisposition does not always become manifest in clinical schizophrenia. More direct evidence on this point comes from the studies of monozygotic twins to be considered next.

Early studies found quite a high incidence of schizophrenia in the co-twins of MZ schizophrenic index cases (Kallmann's figure of 69% is representative). A trickle of lower concordance figures beginning to appear 5 years ago is now a flood. The difference at least partly reflects a difference in method. Most of the early studies started with severe hospitalized schizophrenics; the newer studies have searched whole populations or started with milder psychiatric cases. Tienari (399) in Finland found only one clearly psychotic co-twin of 16 schizophrenics who were members of identical twin pairs, or 6% concordance. Adding four cases with some borderline schizophrenic symptoms raises the figure to 31%. Kringlen (229-231) in Norway, with 55 MZ pairs found concordance for 25%-38%, depending on definition of cases. Fischer et al (108) in Denmark found 25 pairs with 36%-56% concordant. Gottesman & Shields (139) in England, with a psychiatric outpatient series found 42% of 24 MZ pairs concordant. Finally, reports based on the large US veterans twin sample (319) indicate that in this group of twins (from which any early psychotics would be excluded), out of 80 MZ pairs with at least one member diagnosed schizophrenic, in only 11, or 14%, was the second twin also schizophrenic. The twins, age 38 to 48 when studied, were nearing the end of the risk period for schizophrenia.

Taking all these results together, it is clear that the genetic predisposition to schizophrenia is of far less than perfect penetrance, since the same genotype which leads to schizophrenia in the one MZ twin fails to do so in the other at least as often as not. Thus while a predisposition seems to be inherited, environment (in some form) obviously plays a large role in determining whether it will be expressed as clinical schizophrenia.

It should not be concluded that the nonschizophrenic MZ co-twins of schizophrenics are perfectly normal. Some degree of schizoid personality, neuroticism, or general eccentricity is commonly found in such individuals, although a few apparently normal co-twins have been reported (229, 318, 399). It is tempting to consider these personality deviations as milder manifestations of the schizophrenic genotype (173, 315), but some could conceivably reflect the effects of intimate association with the psychotic or prepsychotic twin. A small amount of critical data on this point is supplied by Mitsuda (290), who located in Japan eight cases of that very rare species identical twin pairs reared apart from infancy in which at least one member has become schizophrenic. Five of the eight co-twins were schizophrenic, two were said to be schizoid, and one was normal. Again, it appears that certain genotypes predispose to schizophrenia, but may be expressed in milder personality deviation, or even be compatible with normality.

Discordant MZ pairs have been examined in several studies in the hope of identifying the environmental factors that have led to the difference in outcome (229, 318, 399). No conclusive results seem yet to have emerged. A rather characteristic finding is that the schizophrenic-to-be is the more submissive and introverted of the pair, but whether this is a contributory factor or merely an early symptom of the schizophrenia is not yet clear.

Another promising approach to investigating the expression of the schizophrenic genotype has been taken by Mednick & Schulsinger (284, 285). These investigators are following 207 offspring of Danish schizophrenic mothers. All subjects were given extensive initial tests and interviews. Perhaps half of these children, in their teens at the start of the study, are expected eventually to show some degree of socially deviant behavior, with about 30 becoming schizophrenic. After 5 years of follow-up, approximately 20 subjects have shown some more or less severely deviant behavior, although none is yet unambiguously schizophrenic. Further developments in this study will be awaited with great interest.

There is no evidence that anyone has given up his pet genetic hypothesis about schizophrenia during the past 5 years. Eloquent voices have been raised on behalf of a single partially dominant gene (97, 371), a dominant and a recessive gene (213), two recessive genes (47), a polygenic system (140), and some sort of generalized genetic disharmony (25). The notion of schizophrenia as a heterogeneous group of genetic diseases has also been popular (102, 290). Along this last line, Hanhart (153) has urged the study of schizophrenia in isolated human groups, where the chances of finding just one genetic variety would presumably be enhanced. He reports data from a Swiss isolate with considerable inbreeding and a relatively high frequency of schizophrenia, and suspects a recessive gene with reduced penetrance.

Regrettably, the earlier cited evidence from discordant MZ twins concerning the varied and uncertain manifestation of the schizophrenic genotype(s) suggests that, barring some biochemical breakthrough, none of the above genetic hypotheses is in imminent danger of extinction.

Since schizophrenics tend to have fewer offspring than nonschizophrenics,

the gene(s) involved should be selected against. Yet they are apparently much more frequent than can be explained plausibly by new mutations. It has been suggested that the schizophrenic gene(s) in other contexts may confer a reproductive advantage (191). This idea has been widely discussed in the period under review (25, 103, 135, 207, 213, 232) but no solid evidence has yet emerged. Some data *have* been reported suggesting that the reproductive disadvantage of schizophrenics is rapidly diminishing (104), due largely to modern treatment methods which allow the schizophrenic to spend more time in the community. The social implications of this trend deserve attention.

Affective psychoses.—While genetic research with the affective psychoses has been somewhat less active than that with schizophrenia, there have been several substantial investigations and some good reviews and symposia (62, 437). Perhaps the most striking results from a genetic standpoint are those of Perris (313). He investigated the hypothesis of two different genetic entities in the affective psychoses, one marked by both manic and depressive phases ("bipolar"), and the other by recurrent depressive (or manic) phases only ("unipolar"). Among the relatives of 138 bipolar index cases he found 58 bipolar and 3 unipolar cases, and among the relatives of 139 unipolar depressive cases he found 44 unipolar and only 2 bipolar cases—a remarkably clear separation. (He only found 17 unipolar manic index cases, and they did not yield very clearcut results.) The two varieties of affective psychosis differed in several respects: most notably in age of onset, with unipolar cases occurring later. In addition, the risk of psychosis among relatives was lower for the unipolar cases. In general the study appears to have been carefully done, but Perris did all his own diagnoses, and it is difficult to rule out the possibility of some contamination, particularly in the diagnosis of the relatives.

In a similar study in Switzerland, Angst (15) found most bipolar illness occurred among the relatives of bipolar probands, in agreement with Perris; however, unipolar illness occurred fairly often among the relatives of both kinds of probands. Angst also confirms Perris's finding of a lower psychiatric risk among relatives of unipolar probands, as does Asano (16), in a Japanese sample restricted to cases with an early age of onset (under 40). In Great Britain, Hopkinson & Ley (183) found a lower risk of affective disorders in relatives of probands with relatively late illness, and some tendency toward a bimodal distribution of age of onset. However, in a substantial US study of affective psychosis by Winokur and his associates (427) there was no difference in average age of onset between familial and nonfamilial cases of affective disorder, which is somewhat out of line with the other studies. Winokur's group did find that most of their cases displaying manic symptoms fell in a group with highly positive family histories (426), which is consistent with Perris's results.

Thus on the whole, despite some inconsistencies, there appears to be a

fair amount of support for the hypothesis of two fairly distinct entities among the affective psychoses, one marked by the presence of both mania and depression, a higher risk in relatives, and earlier onset, and the other by recurrent depression, less risk in relatives, and later onset. Winokur proposes that a dominant form of genetic transmission may be involved in the first type (426). However, Slater & Tsuang (372) failed to find support for dominant transmission in manic-depressive families, using a method based on the frequency with which cases are found only on one side of the family.

Psychoneuroses and character disorders.—Shields & Slater (364) report higher concordance among identical than among fraternal twin pairs for anxiety states and for personality disorders, but not for other neuroses. Mitsuda and his co-workers (291) found that none of 56 cases of neurosis diagnosed as hysteria or depressive reaction had cases of schizophrenia among their close relatives, as opposed to 23 such families among 100 cases of neurosis diagnosed as anxiety reaction, obsessional state, or oversensitivity. The possibility must be considered that some of the latter group represent milder expressions of genotypes which might under other conditions have led to schizophrenia.

Guze (149, 432) reports an elevated incidence of hysteria in female relatives of hysterics, and in male relatives an excess of other psychiatric disorders, mostly sociopathy and alcoholism. The incidence of the latter disorders was even higher in the *husbands* of his index cases, suggesting the possibility that it is hysterical females, not hysterical genes, that drive men to drink. Substantially higher identical than fraternal twin concordance for criminality was reported from Denmark (157), while studies of obsessional neurosis yielded somewhat mixed results (133, 193, 335, 344). Some limited new twin data on homosexuality suggests the involvement of both genetic and environmental factors (174). The US veterans twin sample gave a much lower MZ/DZ concordance ratio for psychoneurosis than for schizophrenia (319). On the whole, a mixed bag, with some evidence of the importance of the genes, but every reason to suppose that the environment plays a major role as well in these disorders.

Single-gene and chromosomal defects.—Much less is known about the influence of genetic and chromosomal anomalies on personality and social behavior than on intelligence. Two exceptions to this generalization will be discussed briefly. One concerns genetic and chromosomal effects on sex role identification. The other concerns the XYY chromosome constitution and criminal behavior.

Representative of work on genetic and chromosomal sex is the research of Money and his associates (294). A particularly illuminating instance is provided by the testicular feminizing syndrome (292). This condition, believed to be due to a single gene, appears to involve an insensitivity of the body cells to male hormones. Affected individuals, genetic males, have (internal)

male gonads, but the androgens secreted by the testicular tissue do not have their normal masculinizing effects, and the individuals develop as phenotypic females, except that they do not have female internal reproductive organs, do not menstruate, and are of course infertile. The interesting fact about such patients from our present standpoint is that their sex role behavior and attitudes appear to be entirely feminine: as little girls they show no signs of tomboyishness, and when they grow up they fall in love with masculine males, get married, and settle down to cheerful domesticity. They also tend to show the typical feminine pattern of cognitive abilities, with verbal abilities superior to perceptual-spatial ones (282). Thus their genetic maleness appears to have no direct psychological effects; presumably it normally acts only via the male sex hormones. However, there may be a direct effect of Y chromosome genes in growth, since these girls tend to be somewhat taller than average.

Much research and legal and popular discussion have stemmed from the reports by Casey (54) and Jacobs (196) of a number of men with an extra Y chromosome found in special security institutions for mentally subnormal men with aggressive and criminal tendencies. It was noted that many of these individuals were exceptionally tall, and soon investigators on several continents were busily examining the chromosomes of tall prisoners and mental patients and finding some cases of XYY. Court Brown (64) and Kessler & Moos (224) provide good reviews of these studies, and Borgaonkar (29) provides an extensive bibliography. On the whole, it appears that if one surveys 100 penitentiary inmates over 6 feet tall he can expect to find two or three men with an XYY karyotype. It is clear that this is well above general population levels: Court Brown found one XYY male in a survey of 1185 male births, and none in an industrial population of 371 adult males selected for a height of 6 feet or more (64). Nine XYY cases were found among 8215 males karyotyped in 15 French laboratories for a variety of reasons (324). Three cases in approximately 2000 male births were found in a US study (256). A somewhat higher figure of 4 in 1066 male births has been reported in one study (361), but even this is far below typical findings in criminal populations.

Obviously, only a small fraction of serious criminals have an extra Y chromosome. Furthermore, neither tallness nor mental subnormality nor criminal tendencies invariably accompany an XYY karyotype. Indeed it has recently been proposed (50) that there are at least two quite different XYY syndromes, one marked by various genital anomalies, but not by tallness or psychopathic behavior, and the other by tallness and psychopathic tendencies, along with normal genital development. Judging from estimated population frequencies, most XYY males fit neither syndrome, but lead ordinary lives in society (162). One such individual, discovered by chance (421), was 5′11½″ tall, had an IQ of 97, was physically unremarkable, and had exhibited a cheerful disposition and a mild and law-abiding temperament since childhood.

EVOLUTION

Gene Diversity and Adaptation

Many forms of behavior that are critical for individual survival and population adaptation are under genetic control. Mating speed and duration of mating in *Drosophila* species show extensive genetic variation (215, 309, 310), and respond to directional selection (223, 258). Similar genetic determination is evident for mating ability in chickens (369), aggressive behavior in mice (212, 233, 239), nest building in rabbits (435), and sex behavior in mice (264-267, 400).

Dobzhansky and his collaborators have been the most effective in identifying genetic variation in *Drosophila* and relating this variation to population survival. Using mass screening devices developed by Hirsch, these investigators have identified positive and negative geotaxis and phototaxis in many strains of *D. pseudoobscura* and *D. persimilis* from various geographic localities (375). Some strains are positive, others neutral, and still others negative. *D. pseudoobscura* has been selected for high and low characteristics on both of these responses (84, 85, 88). Realized heritability of both traits was extremely low—8 to 10% for the phototaxic and 2 to 3% for the geotaxic behavior. In spite of this, clear separation of selected lines was obtained after 16 generations, and when selection was relaxed the populations again converged, almost as rapidly as they were separated (88). Apparently geotaxic and phototaxic neutrality are the optimal responses among these populations—any radical deviation in either direction is counterbalanced by natural selection. When only a few "migrants" are permitted between populations separated for their responses to gravity and light, the migrants will alter the recipient populations in the direction of the donor populations, even though phenotypically the migrants are the most deviant individuals of the donor groups (87, 89). In other words, the phenotype of the migrants is not a good index of their genetic architecture, in accord with the finding of low heritabilities for these traits. Dobzhansky views these experiments as models of social mobility that may have application to human populations.

One explanation for the disproportionate influence of rare genotypes on population parameters is found in recent studies by Ehrman and associates (93, 94). Rare *Drosophila* males mate more frequently than males that are common. The phenomenon has now been observed in strains of *D. melanogaster*, *D. pseudoobscura*, *D. persimilis*, *D. tropicalis*, *D. willistoni*, and *D. equinoxialis*, and in strains carrying different chromosome arrangements (AR and CH), strains selected for different behavior, and in those containing mutant markers. For whatever reason, rare male genotypes are reproductively more fit until the numbers of each in the population are equal, at which time mating becomes random. The effect is less pronounced for females. Obviously this mechanism could lead to balanced polymorphism without heterozygous advantage. The exact sensory mechanisms underlying

the phenomenon are unknown, although olfactory and tactual cues have been implicated (93).

Ethological Isolation

Genetic groups, especially sympatric populations, may become reproductively isolated because of behavioral differences. This is in fact one basis for speciation and is of increasing interest to behavior geneticists. Isolation may occur because of differential selection pressure in a diversified environment (131, 141, 243), because of divergent evolution from founding stocks that contain few individuals with unique genotypes (184, 185, 308), because of inadvertent selection during domestication (321), because of differences in reproductive behavior (222) and physiologies (40, 281), because of different capacities for food utilization under population stress (237), and because of assortative mating for various traits (13, 86). Obviously the various reasons for isolation are not independent and must be studied in their interactions. Like other traits, isolating mechanisms can come under the control of selection in the laboratory (77, 221). As yet speciation has not occurred under laboratory conditions, but it might be a reasonable goal.

While isolating mechanisms are better studied in organisms with a quick turnover of generations, some work with higher organisms shows promise of revealing critical ethological factors. Bruell (44) has proposed designs for evaluating the effects of inbreeding on wild populations. Emphases are on comparisons between sympatric mouse populations in the wild and in the laboratory. Selander & Yang (359) have studied several rural populations of domestic mice using blood esterases and hemoglobin as genetic markers to evaluate geographic variations in polymorphisms. Significantly, isolated breeding populations with characteristic gene frequencies appear even within individual barns with no physical barriers. These "tribes," "demes," and other breeding units are apparently isolated by male territorial and aggressive behavior. Surprisingly, up to 25% inbreeding is tolerated within subgroups. Some investigators believe that research into ethological isolating devices and modes of adaptation is of paramount importance for the continuing vigor of behavioral genetics (280, 385).

Heterosis

The terms heterosis or hybrid vigor are used to refer to the observation that crossbred F_1 offspring from two inbred parental lines either deviate significantly from the expected midparent value or exceed the extreme parental value. Both definitions have been used by behavioral geneticists. There are several competing theories of heterosis, and behavioral geneticists have done little to resolve these issues. Hybrid vigor is negatively associated with inbreeding or reduced genetic variation; indeed the opposite of hybrid vigor is called inbreeding depression. Falconer (107) suggests that when inbred lines are crossed the F_1 offspring will show heterosis for those char-

acters that showed inbreeding depression in the inbred lines. It appears that inbreeding allows more deleterious recessive genes to be expressed, and thus crossing, which increases genetic heterozygosity, should mask more of the recessive deleterious genes (245), and this may be what one observes in heterosis.

Hybrid vigor has intrigued geneticists for many years, and in agriculture and animal breeding extensive and valuable economic use has been made of this phenomenon. However, at this time the amount of research devoted directly to investigating *behavioral* heterosis has not been extensive. Reviews of this literature may be found in a chapter by Bruell (42) and Lindzey's American Psychological Association presidential address (245).

Heterosis has been observed for many phenotypes including morphological and reproductive characters such as: litter size, longevity, body size, egg size and production, and disease resistance, using a variety of organisms such as: chickens, mice, rats, silkworms, honeybees, and *Drosophila* (245). Most studies that have shown evidence for behavioral heterosis have been concerned with simple nonsocial responses such as appetitive and aversive learning (59, 429), activity (41, 42, 295), emotionality (43), and exploration (41). A study by Barnett & Scott (17) was concerned with nest building, food and wood gnawing, and activity, responses that appear to be closely related to adaptation. The investigators found evidence for "behavioural vigour" or heterosis in activity, gnawing, and nest building. In studies of heterosis, both behavioral and nonbehavioral, those characteristics that show heterosis seem to be associated with adaptation to the environment, natural selection, and thus evolution. Regrettably, social behavior, which from an evolutionary perspective is most important, seems rarely to have been the object of behavioral heterosis studies.

Evidence for behavioral inbreeding depression in man has been discussed by Spuhler (376), but evidence for behavioral heterosis in man is difficult to obtain. An interesting study by Hulse (189) provides some limited evidence for morphological heterosis in humans. He found that offspring from marriages between members of different Swiss villages were taller and more robust than offspring of within-village marriages. Lindzey (245) has discussed the evidence for inbreeding depression as a result of incestuous matings and consanguineous marriages. On the basis of the results presented in that review one could reasonably infer that heterosis for human traits, behavioral and nonbehavioral, should be observable under appropriate circumstances.

A major publication on behavioral heterosis during the period under review was Bruell's chapter (42). In it he reviewed several diallel studies of heterosis, and discussed genetic mechanisms that may underlie heterosis, and the relationship between evolution and heterosis. He also provided several suggestions for the design of future experiments on behavioral heterosis. In another important paper, Parsons (307) reviewed several studies that used mice and *Drosophila*, and examined the relationship between behavioral heterosis and homeostasis, i.e. reduced variability in hybrids. It

appears that heterosis is often associated with homeostasis and that this association is stronger when the hybrid crosses are between unrelated strains. Only a few studies (163, 164, 428, 429) have directly investigated the ability of hybrid organisms to withstand various environmental changes or stresses, which has been called buffering (235). Additional studies of this problem would be a valuable contribution to the study of behavioral homeostasis and heterosis. Since heterosis and inbreeding depression are so closely related to evolution it can be expected that behavioral geneticists in the future will devote considerable experimental effort to these phenomena.

RESEARCH METHODOLOGY

In this section various techniques used in behavioral genetic analyses are briefly discussed and a few comments are made regarding trends in the use of these techniques. Examples of substantive findings yielded by these methods have been presented throughout this review.

Single-Gene Techniques

Single-gene methodology is currently drawing increased attention, and the mouse has established itself as the chief species for study. Three major reviews have appeared (261, 388, 422) and several discussions point to the usefulness of single gene analyses (1, 269, 288, 386). By substituting single genes within a population, an investigator can preset biochemical substrate, relate behavioral effects to chromosome positions, and correlate allelic variations in physiology and morphology to phenotypic components of behavior.

In a critique, Wilcock (422) described the major techniques of unifactorial study and stipulated their limitations. The primary methods of assessment are (*a*) to trace backward in development a gene mutation until its effects vanish, thus indicating the point of genetic influence (e.g. 384); (*b*) to compare single-gene variations at a particular point in time and assess the differences in the phenotype (e.g. 1); (*c*) to screen alleles on a battery of behavioral tests and look for patterns and specificity of influence (e.g. 388); and (*d*) to resolve biometrical traits into their separate genetic components (e.g. 176, 329, 341, 393). Each approach has its special advantages as well as deficiencies.

Genes of interest are maintained in stocks of animals by preserving spontaneous mutations in inbred lines, by introducing mutations in inbred lines with repeated backcrossing from a mutant source, and by crossing mutant and nonmutant stocks and recovering segregants in F_2 or other heterotypic generations (144). The first method reduces the possibility of linked genes affecting the phenotype, while the second and especially the third method allow linked genes to influence the results. Several catalogs of single genes are available (e.g. 257, 379), and an annotated bibliography of mutant mice has been compiled (367). In the mouse, well over 300 mutant genes occupying more than 250 loci are known (143). Of these at least 55 are nonlethal coat color genes (433), in many respects ideal for behavioral study.

Moreover, over 100 neurological mutants are recognized in the mouse (367) affecting almost every conceivable biological function relevant to behavior. In *Drosophila* at least 479 loci have been identified (12), and in man a minimum of 123 loci have been identified on the sex chromosome and more than 1400 loci are noted for non-sex chromosomes (268). Here, then, is the genetic raw material for investigation, especially abundant in mouse, *Drosophila*, and man.

Relatively little of the quantity and diversity of single genes has been explored experimentally. Investigations of neurological mutants in mice have been most productive, and we will focus upon these here. Other significant investigations are cited at the end of this section.

Neurological mutants.—Neurological mutant mice can be classified roughly according to (*a*) the area of the central nervous system affected (367); (*b*) the level of defect (e.g. 365); (*c*) the age of onset (e.g. 111); or (*d*) the type of behavior involved (e.g. 111, 248, 384). It seems that most known mutant disorders arise during early development (e.g. before 30 days of age) and are associated with smaller brain and body size and are related to locomotor disabilities (especially swimming). Often the defects are pleiotropic to changes in coat color and spotting.

The variety of neurological mutants affecting balance and locomotion is very great. A partial listing can be found in McClearn & Meredith (261) and a more or less complete cataloging of such mutants is available (367). New discoveries and studies include *dancer*, *ducky*, *dystonia musculorom*, *jimpy*, *jittery*, *Nijmegen waltzer*, *pallid*, *pirouette*, *quaking*, *reeler*, *retinal degeneration*, *rotating*, *shambling*, *Snell's waltzer*, *twirler*, *wabbler-lethal*, *waltzer*, and *whirler* (2, 80, 111, 248, 343, 366).

Screening techniques.—Somewhat paradoxically, methods devised to understand polygenetically controlled behavior have introduced new means of studying and classifying single-gene differences. Hirsch (176) has devised elegant techniques of mass screening and genetic analysis that function in this way. He and his colleagues (176, 179, 181, 186, 316) have used genetic markers, chromosomal inversions, and balanced lethal systems to study geotaxis in *Drosophila*. As a result, negative and positive geotaxic behaviors have been related to three of the four fly chromosomes. This refinement, as Hirsch and others see it, is a prelude to resolving chromosomal influence into smaller and smaller units, much as Thoday (393) and others have done for bristle number in *Drosophila*. Eventually, individual genes may be isolated following this strategy.

Seymour Benzer (22), a Nobel laureate in genetics, has amplified Hirsch's mass screening techniques to study single-gene mutations in *D. melanogaster*. He induces the mutations with ethyl methane sulfonate (ENS) and screens the mutants and normals on successive light stimulus tests. The technique, which Benzer calls "countercurrent distribution," may offer a useful method

for fractionating populations according to genetic diversity on a number of sensory-motor dimensions. Benzer's technique has been used by others (211) to study induced neurological mutants in *D. melanogaster*. Of the four dominant mutants examined (mapped on the X chromosome) which cause shaking following etherization, marked activity, increased wing scissoring and increased fly specking (defecation) following disturbance, none reacted in any abnormal way to a light gradient.

An elegant yet simple single-gene method of great potential has been developed by Adler (6) and his associates to study chemotaxis in *E. coli* bacteria. A capillary tube containing a solution of a chemical attractant is pushed into a suspension of bacteria on a slide and the number of bacteria attracted is counted. Using this straightforward technique combined with specific monounit mutants, Adler was able to conclude that the periphery of *E. coli* contains at least five chemoreceptors which direct movements of flagella toward different chemical substances. These receptors respond to galactose, glucose, ribose, aspartate, and serine—chemicals of interest to bacteria. The receptor mechanisms are independent of metabolic processes needed for survival and membrane transport systems. The methodology could be applied to the study of other sensory systems. In general the research steps of Hirsch, Benzer, Adler, and others significantly advance the concepts of taxes and innate releasing mechanisms established by Loeb and European ethologists and promise new and exciting developments in the understanding of single-gene processes.

Designs and Models

Along with the growing use of single-gene analyses and mutants as techniques in behavioral genetic analyses there is continued use of a variety of crossing techniques. Genetic analyses using two inbred strains (P_1, P_2), F_1, F_2, and backcrosses (B_1, B_2) to estimate components of variation, heritability, and other genetic parameters have been used by a number of investigators (e.g. 148, 278, 299, 394). A computer program which can be used to estimate the various genetic parameters and test the assumptions of the classical model is available (214). In studies reported by Newell (299) and DeFries & Hegmann (74) various biometrical analyses were used and compared. Such comparative analyses of biometrical methods should be useful in future behavioral genetic research. In a study of audiogenic seizures (60) three additional segregating generations, B_1F_1, B_2F_1, B_1B_2, were added to the usual generations. A goodness of fit test was used to assess the congruence between expected and observed proportions of seizures in each generation.

The "Tryon effect."—The absence of greater phenotypic variance in the segregating F_2 generation compared to the F_1 generation was discussed by Hirsch (178), and he suggested that this was due to inadequate sampling of the spectrum of possible F_2 genotypes. Tellegen (383) has suggested that as

long as sampling of the F_2 subjects is random, an unbiased estimate of the population variance should be obtained. He has also shown that as n, the number of relevant genes, increases, the probability of observing the Tryon effect increases, but as the degree of genetic variation relative to environmental variation increases, the probability that the Tryon effect will be observed decreases. The absence of the Tryon effect was reported by DeFries & Hegmann (74) and their discussion of this effect is similar to that provided by Tellegen.

The diallel cross.—In the past 5 years there has been increased interest in the diallel cross as a technique in behavioral genetics. A variety of organisms have been studied with the diallel method including rats (34, 36), mice (43, 59, 164), and fruit flies (114, 307). Broadhurst (34) provides an introduction to the diallel cross and discusses the advantages, limitations, procedures, and inferences that can be made using the technique. He illustrates the design from his own work on open-field behavior in rats. It is likely that this technique may be used more frequently in the future; indeed, Henderson (164) has suggested that generally the use of inbred or random-bred mice should be "abandoned" and replaced by diallel designs.

However, other promising new alternatives to the diallel cross are being proposed. The quantitatively oriented behavioral geneticist should find several papers by the Birmingham biometricians of considerable interest (e.g. 45, 202, 204, 216, 283).

Although selection is probably the technique most generally identified with behavior geneticists, it has not been used extensively in recent years. Broadhurst (36) and Roberts (328) have discussed some of the reasons for this. Broadhurst (36), who in his own work has used artificial selection extensively, writes " . . . despite this widespread interest [in selection experiments] the harvest yielded by so much work has been relatively barren" (p. 807). Selection experiments have been carried out using diverse behavioral phenotypes and organisms, e.g. maze learning, avoidance learning, activity, alcohol consumption, and emotionality in rats; aggressiveness in mice; geotaxis, phototaxis, and mating speed in *Drosophila.* Roberts (328) provides a discussion of genetic concepts that underlie selection studies.

It should be noted that some interest is now being shown in using wild *Mus musculus* (44) and other noninbred and nondomesticated mice. Bruell provides cogent reasons for increased concern with organisms that are not peculiar to the laboratory. He has argued that domesticated inbred mice, commonly used, represent a highly selective sample of the total genetic variation of mice. As behavioral geneticists become more interested in evolution and behavioral population genetics, continued use of domesticated inbred mouse strains will become more restricting.

Genotype x environment interaction.—The frequent observation of genotype x environment (or treatment) interactions, in a wide variety of be-

havioral traits, raises formidable problems in the design and strategy of research and interpretation of research findings. One may view the ubiquitous G x E interactions as either trivial or else as a basic challenge to psychology as a science with general laws of behavior. Examination of these problems and some solutions and research strategies can be found in the reviews by Henderson (165), Manosevitz (273), and Vale & Vale (403).

Psychologists have generally been concerned with the effects of specific treatments (main effects) upon behavior, while behavioral geneticists have typically been concerned with the effects of genes upon behavior. While some investigators have been directly concerned with genotype x environment or treatment interactions, there have been few systematic programs of research on such interactions. Interested readers should find the following an adequate starting point for further inquiry into this area (72, 120, 132, 152, 164, 165, 177, 273, 307, 336, 378, 395, 396). Suggestions have been made to show how uniformity within an array of complex interactions may be found (165, 403), and a scheme for classifying the various kinds of interactions has been reviewed by Manosevitz (273). Suggestions found in the paper by Vale & Vale (403) for searching for the "underlying variables," "mechanisms," or "processes" of the interaction should stimulate not only a new look at G x E interactions but also systematic studies in which these interactions are investigated and efforts are made to identify their bases.

Genotype x age interaction.—Behavioral genetic research on genetic variation and development has been concerned with (*a*) genetic differences in developmental rate; (*b*) interactions between genetic factors and age of subjects at time of testing; and (*c*) interaction between genetic variation and early experience treatments. Genetic differences in developmental rate and behavioral stability have been studied by comparing strains or mutant stocks with respect to a variety of behaviors (38, 83, 112, 358, 417). Generally it has been shown that genetic factors are important determinants of developmental rate and behavioral stability.

The importance of genotype x age interactions has been emphasized by Henderson (166), and interested readers should find his review of these studies helpful. He has suggested that investigators become alert to detecting genotype x age x treatment interactions in their research. Studies on the effects of early experiences and their interaction with genetic factors as determinants of adult behavior have been studied by several investigators (e.g. 164, 275). Generally these studies show that responses to early experience and the course of subsequent development of the organism are partly determined by genetic factors and the age of the organism when he is exposed to the treatment.

Twin and family studies.—A number of methodological developments in human behavior genetics are noted elsewhere in this review in connection with substantive studies. The present section will discuss some general

methodological trends, and some particular issues of method not covered elsewhere. The reader's attention is also invited to a general discussion of twin methodology by Allen (9), and to a symposium on the use of computers in human genetics (370).

An authoritative discussion of twin concordance rates is presented by Allen, Harvald & Shields (10). They distinguish between "pairwise" and "probandwise" concordance rates. The former (which is the definition used elsewhere in the present review) refers to the proportion of affected pairs that are concordant; the latter, to the probability of having an affected co-twin. They note that the pairwise rate in a sample is a biased estimator of the corresponding population parameter, and present a method of correction. A related discussion of twin models is presented by Gedda & Brenci (130).

A striking (and welcome) trend in twin studies is toward the use of large samples which are essential for quantitative estimates of heritabilities, or for multivariate analyses. A 1965 survey by the World Health Organization (434) counted 23 current or planned twin studies in the areas of "psychiatry, psychology, or normal development" in the US, Europe, and Japan. Seventeen of these involve over 500 twin pairs, and six over 2000. Such large-scale studies usually require questionnaire diagnosis of twin zygosity, based on physical characteristics, mistaken identity, and the like. Several recent validations of questionnaires against blood typing methods have been undertaken, with reassuring results. Nichols & Bilbro (302), with the US National Merit Scholarship sample, found agreement between a mail questionnaire and blood diagnosis in 78 of 84 blood-tested pairs, or 93%. Hauge and his co-workers (157) with the Danish twin sample found agreement on 313 of 335 pairs, again about 93%. A study with a sample of twins from among US veterans (195) achieved approximately the same level of agreement for a mail questionnaire against a combined serological and morphological criterion, for 232 pairs. Since blood tests themselves are typically 97-98% accurate, and since the errors are probably reasonably independent, this suggests that the questionnaires are actually achieving around a 95% level of diagnostic accuracy, which should be quite satisfactory for most large-scale studies.

A number of writers have discussed the estimation of heritability coefficients from twin and family data (67, 140, 199, 200, 254, 300, 354). As several have pointed out, this is only a special case of the more general problem of assigning the phenotypic variance of a trait in some population to various genetic and environmental sources. The more recent approaches take into account (to varying degrees) such factors as assortative mating, nonadditive genetic variance, heredity-environment correlation and interaction, and (for twins) the possible greater similarity of environments between members of identical pairs than between members of fraternal pairs. Since it is clear that one cannot estimate all of these parameters from twins alone, more complex designs involving other family members as well are

being explored (98). Cattell's pioneering effort in this direction, Multiple Abstract Variance Analysis, has been criticized (253), and elaborated (55). In a major paper, Jinks & Fulker (201) examine thoroughly the whole area of quantitative estimates of variance components in human behavior, from a biometrical genetics point of view. While it is possible to find fault with the assumptions underlying any particular one of these techniques, it is clear that there are many options open in designing quantitative studies of the genetic and environmental influences on human traits, options which represent special cases of a very general approach.

Two factors mentioned above which have come in for special attention recently are assortative mating and the equivalence of identical and fraternal twin environments. Anyone wanting to incorporate assortative mating into a quantitative model will wish to consult a paper on its genetic implications by Crow & Felsenstein (68), which provides a simple Wrightian approach to Fisher's classic work. Other papers review assortative mating with respect to education (226), IQ (128), and physical characteristics (377) and discuss theories of mate selection (92). Work on assortative mating with respect to normal and neurotic personality traits has also been appearing (46, 56, 154, 228).

The assumption that fraternal twins have just as similar environments as do identical twins is basic to many methods of estimating heritability from twin data. It has been a major focus of attack for critics of such methods. Additional ammunition for the critics is provided in several recent studies showing that compared to same-sex fraternal pairs, identical twin pairs have more close friends in common (373), play together more as children (227), and are responded to more similarly by teachers and peers (360). Some evidence for the defense is reported by Scarr (347), who suggests that, at any rate, parents' identification of twins as identical or fraternal may not be crucial. She looked at cases where parents were mistaken concerning the zygosity of their twins, and found that this seemed to make little difference in their resemblance. There were only 11 mistaken pairs in her sample, however, so this approach deserves repetition on a larger scale.

In general, the causal sequence in development is hard to unravel. Do identical twin pairs play together more because they are more alike, or are they more alike because they play together more? Studies of separated twins, or longitudinal studies, should be able to get some leverage on this distinction. A statistical approach is also possible, if one assumes a constant increase in environmental similarity relevant to several traits of differing heritability (254). Stability of relationships across cultures may also provide a clue (146, 147).

Potentially powerful tools for tearing apart complex causal influences are presented by multivariate statistical methods. For a sampling of such techniques applied to human behavior-genetic problems, in the realm of abilities, the reader is referred to the concluding section of Vandenberg's

recent volume (410); such techniques have also been applied in the personality realm (252, 311, 398).

IN CONCLUSION

It seems evident that the field of behavioral genetics has displayed an accelerating rate of growth that is matched by few psychological specialties. It provides a major point of intersection between psychology and the biological sciences and in recent years has involved extensive exchanges with other behavioral sciences. The early mission of behavioral geneticists centered about the demonstration that genetic variation was an important determinant of most (all?) domains of behavior and, in large part, this goal seems to have been accomplished. Contemporary investigation is much more likely to be concerned with the mechanisms or pathways involved in genetic determination and the attempt to arrive at quantitative estimates of the magnitude of genetic influence under stated conditions. The literature we have reviewed provides clear evidence of the growing influence of both molecular biology and biometrical developments upon research in this area.

LITERATURE CITED

1. Abeelen, J. H. F. van. 1965. An Ethological Investigation of Single-Gene Difference in Mice. PhD thesis. Univ. Nijmegen, Netherlands
2. Abeelen, J. H. F. van. 1966. Behavioural profiles of neurological mutant mice. *Genetica* 37:149–58
3. Abeelen, J. H. F. van, Kroes, H. W. 1967. Albinism and mouse behaviour. *Genetica* 38:419–29
4. Adam, A. 1969. A further query on color blindness and natural selection. *Soc. Biol.* 16:197–202
5. Ader, R. 1969. Adrenocortical function and measurement of "emotionality." *Ann. N.Y. Acad. Sci.* 159:791–805
6. Adler, J. 1969. Chemoreceptors in bacteria. *Science* 166:1588–97
7. Åkesson, H. O. 1967.A study of fertility and mental deficiency. *Acta Genet. (Basel)* 17:234–42
8. Ibid. Severe mental deficiency in a population in Western Sweden, 243–47
9. Allen, G. 1965. Twin research: problems and prospects. *Progr. Med. Genet.* 4:242–69
10. Allen, G., Harvald, B., Shields, J. 1967. Measures of twin concordance. *Acta Genet. (Basel)* 17:475–81
11. Altman, J., Das, G. D., Chang, J. 1966. Behavioral manipulations and protein metabolism of the brain: effects of visual training on the utilization of leucine-H^3. *Physiol. Behav.* 1:111–15
12. Altman, P. L., Dittmer, D. S., Eds. 1964. *Biology Data Book.* Washington: Fed. Am. Soc. Exp. Biol. 633 pp.
13. Anderson, W. W., Ehrman, L. 1969. Mating choice in crosses between geographic populations of *Drosophila pseudoobscura*. *Am. Midl. Natur.* 81:47–53
14. Anderson, V. E., Siegel, F. 1968. Studies of behavior in genetically defined syndromes in man. In *Progress in Human Behavior Genetics*, ed. S. G. Vandenberg, 7–17. Baltimore: Johns Hopkins Press. 356 pp.
15. Angst, J. 1966. Zur Ätiologie und Nosologie endogener depressiver Psychosen. *Monogr. Gesamtgeb. Neurol. Psychiat.* Heft 112, 118 pp.
16. Asano, N. 1967. Clinico-genetic study of manic-depressive psychosis. In *Clinical Genetics in Psychiatry*, ed. H. Mitsuda, 262–75. Tokyo: Igaku Shoin. 408 pp.
17. Barnett, S. A., Scott, S. G. 1964. Behavioural "vigour" in inbred and hybrid mice. *Anim. Behav.* 12:325–37
18. Barondes, S. H., Cohen, H. D. 1969. Comparative effects of acetoxycycloheximide and puromycin on learning and memory in mice. In *Biochemical Factors Concerned in the Functional Activity of the Nervous System*, ed. D. Richter, 17. New York: Pergamon. 234 pp.
19. Bartke, A., Wolff, G. L. 1966. Influence of the lethal yellow (A^y) gene on estrous synchrony in mice. *Science* 153:79–80
20. Becker, P. E., Ed. 1967. *Humangenetik* (Band V/2.) *Psychiatrische Krankheiten.* Stuttgart: Georg Thieme Verlag. 613 pp.
21. Beerman, W. 1965. Cytological aspects of information transfer in cellular differentiation. In *Molecular and Cellular Aspects of Development*, ed. E. Bell, 204–12. New York: Harper & Row. 353 pp.
22. Benzer, S. 1967. Behavioral mutants of *Drosophila* isolated by countercurrent distribution. *Proc. Nat. Acad. Sci. USA* 58:1112–19
23. Bignami, G. 1965. Selection for high rates and low rates of avoidance conditioning in the rat. *Anim. Behav.* 13:221–27
24. Bindra, D. 1948. What makes rats hoard? *J. Comp. Physiol. Psychol.* 41:397–402
25. Bleuler, M. 1968. A 23-year longitudinal study of 208 schizophrenics and impressions in regard to the nature of schizophrenia. In *The Transmission of Schizophrenia*, ed. D. Rosenthal, S. S. Kety, 3–12. Oxford: Pergamon. 433 pp.
26. Block, J. B. 1968. Hereditary components in the performance of twins on the WAIS. See Ref. 14, 221–28
27. Bock, R. D., Vandenberg, S. G. 1968. Components of heritable variation in mental test scores. See Ref. 14, 233–60
28. Bohus, B., de Wied, D. 1967. Failure of -MSH to delay extinction of conditioned avoidance behavior in rats with lesions in the parafascicular nuclei of the thalamus. *Physiol. Behav.* 2:221–23

29. Borgaonkar, D. S. 1969. 47, XYY bibliography. *Ann. Génét.* 12:67–70
30. Bovet, D., Bovet-Nitti, F., Oliverio, A. 1968. Memory and consolidation mechanisms in avoidance learning of inbred mice. *Brain Res.* 10: 168–82
31. Bovet, D., Bovet-Nitti, F., Oliverio, A. 1969. Genetic aspects of learning and memory in mice. *Science* 163: 139–49
32. Brewster, D. J. 1968. Genetic analysis of ethanol preference in rats selected for emotional reactivity. *J. Hered.* 59:283–86
33. Britten, R. J., Davidson, E. H. 1969. Gene regulation for higher cells: a theory. *Science* 165:349–57
34. Broadhurst, P. L. 1967. An introduction to the diallel cross. In *Behavior-Genetic Analysis*, ed. J. Hirsch, 287–304. New York: McGraw-Hill. 522 pp.
35. Broadhurst, P. L. 1968. Genetics: genetics and behavior. *Int. Encycl. Soc. Sci.* 6:96–102
36. Broadhurst, P. L. 1969. Psychogenetics of emotionality in the rat. *Ann. N.Y. Acad. Sci.* 159:806–30
37. Broadhurst, P. L., Bignami, G. 1965. Correlative effects of psychogenetic selection: a study of the Roman high and low avoidance strains of rats. *Behav. Res. Ther.* 2:273–80
38. Broadhurst, P. L., Jinks, J. L. 1966. Stability and change in the inheritance of behaviour in rats: a further analysis of statistics from a diallel cross. *Proc. Roy. Soc. B* 165: 450–72
39. Brown, A. M., Stafford, R. E., Vandenberg, S. G. 1967. Twins: behavioral differences. *Child Develop.* 38: 1055–64
40. Bruce, H. M. 1968. Absence of pregnancy-block in mice when stud and test males belong to an inbred strain. *J. Reprod. Fert.* 17:407–8
41. Bruell, J. H. 1964. Heterotic inheritance of wheelrunning in mice. *J. Comp. Physiol. Psychol.* 58:159–63
42. Bruell, J. H. 1967. *Mice and the study of evolution.* Presented at Am. Psychol. Assoc. meeting, Washington, D. C.
43. Bruell, J. H. 1969. Genetics and adaptive significance of emotional defecation in mice. *Ann. N.Y. Acad. Sci.* 159:825–30
44. Bruell, J. H. 1970. Behavioral population genetics and wild *Mus musculus*. In *Contributions to Behavior-Genetic Analysis: The Mouse as a Prototype*, ed. G. Lindzey, D. D. Thiessen, 261–91. New York: Appleton-Century-Crofts. 334 pp.
45. Bucio Alanis, L., Perkins, J. M., Jinks, J. L. 1969. Environmental and genotype-environmental components of variability: V. Segregating generations. *Heredity* 24:115–27
46. Buck, C. W., Ladd, K. L. 1965. Psychoneurosis in marital partners. *Brit. J. Psychiat.* 111:587–90
47. Burch, P. R. J. 1965. Genetics of schizophrenia. (Letter to editor) *Brit. J. Psychiat.* 111:1119
48. Burt, C. 1966. The genetic determination of differences in intelligence: a study of monozygotic twins reared together and apart. *Brit. J. Psychol.* 57:137–53
49. Ibid. 1967. The genetic determination of intelligence: a reply. 58:153–62
50. Carakushansky, G., Neu, R. L., Gardner, L. I. 1969. Testicular agenesis in a boy with XY/XYY mosaicism. *Ann. Génét.* 12:111–14
51. Carr, D. H. 1969. Chromosomal abnormalities in clinical medicine. *Progr. Med. Genet.* 6:1–61
52. Carran, A. B. 1967. Passive avoidance and strain differences associated with differences in emotionality: a test of Mowrer's theory. *Psychon. Sci.* 7:263–64
53. Ibid. 1969. Biometrics of reversal learning in mice: I. Effects of ITI and strain. 16:248–49
54. Casey, M. D., Segall, L. J., Street, D. R. K, Blank, C. E. 1966. Sex chromosome abnormalities in two state hospitals for patients requiring special security. *Nature* 209: 641–42
55. Cattell, R. B. 1965. Methodological and conceptual advances in evaluating hereditary and environmental influences and their interaction. In *Methods and Goals in Human Behavior Genetics*, ed. S. G. Vandenberg, 95–139. New York: Academic. 351 pp.
56. Cattell, R. B., Nesselroade, J. R. 1967. Likeness and completeness theories examined by sixteen personality factor measures on stably and unstably married couples. *J. Pers. Soc. Psychol.* 7:351–61
57. Clever, U. 1966. Gene activity patterns and cellular differentiation. *Am. Zool.* 6:33–41

58. Coleman, D. L. 1960. Phenylalanine hydroxylase activity in dilute and non-dilute strains of mice. *Arch. Biochem.* 91:300–6
59. Collins, R. L. 1964. Inheritance of avoidance conditioning in mice: a diallel study. *Science* 143:1188–90
60. Collins, R. L., Fuller, J. L. 1968. Audiogenic seizure prone (asp): a gene affecting behavior in linkage group VIII of the mouse. *Science* 162:1137–39
61. Coppen, A., Walk, A., Eds. 1967. Recent Developments in Schizophrenia. *Brit. J. Psychiat.* Special Publ. No. 1. 158 pp.
62. Coppen, A., Walk, A., Eds. 1968. Recent Developments in Affective Disorders. *Brit. J. Psychiat.* Special Publ. No. 2. 150 pp.
63. Court Brown, W. M. 1967. *Human Population Cytogenetics.* Amsterdam: North-Holland. 107 pp.
64. Court Brown, W. M. 1968. Males with an XYY sex chromosome complement. *J. Med. Genet.* 5:341–59
65. Craig, J. V., Baruth, R. A. 1965. Inbreeding and social dominance ability in chickens. *Anim. Behav.* 13:109–13
66. Craig, J. V., Ortman, L. L., Guhl, A. M. 1965. Genetic selection for social dominance ability in chickens. *Anim. Behav.* 13:114–31
67. Crow, J. F. 1969. Genetic theories and influences. *Harvard Educ. Rev.* 39:301–9
68. Crow, J. F., Felsenstein, J. 1968. The effect of assortative mating on the genetic composition of a population. *Eugen. Quart.* 15:85–97
69. Das, G. D., Altman, J. 1966. Behavioral manipulations and protein metabolism of the brain: effects of restricted and enriched environments on the utilization of leucine-H^3. *Physiol. Behav.* 1:109–10
70. Davidson, P. W., Walk, R. D. 1969. Differential visual depth discrimination of hooded as compared to albino rats. *Psychon. Sci.* 14:207–8
71. Day, R. W. 1966. The epidemiology of chromosome aberrations. *Am. J. Hum. Genet.* 18:70–80
72. DeFries, J. C. 1967. Quantitative genetics and behavior: overview and perspective. See Ref. 34, 322–39
73. DeFries, J. C. 1969. Pleiotropic effects of albinism on open-field behaviour in mice. *Nature* 221:65–66
74. DeFries, J. C., Hegmann, J. P. 1970. Genetic analysis of open-field behavior. See Ref. 44, 23–56
75. DeFries, J. C., Hegmann, J. P., Weir, M. W. 1966. Open-field behavior in mice: evidence for a major gene effect mediated by the visual system. *Science* 154:1577–79
76. DeGrouchy, J., Aussannaire, M., Brissaud, H. E., Lamy, M. 1966. *Aneusomie de recombinaison*: three further examples. *Am. J. Hum. Genet.* 18:467–84
77. Del Solar, E. 1968. Selection for and against gregariousness in the choice of oviposition sites by *Drosophila pseudoobscura*. *Genetics* 58:275–82
78. Denenberg, V. H. 1965. Behavioral differences in two closely related lines of mice. *J. Genet. Psychol.* 106:201–5
79. Deol, M. S. 1963. Inheritance of coat colour in laboratory rodents. In *Animals for Research*, ed. W. Lane-Petter, 177–97. New York: Academic. 531 pp.
80. Deol, M. S., Lane, P. W. 1966. A new gene affecting the morphogenesis of the vestibular part of the inner ear in the mouse. *J. Embryol. Exp. Morphol.* 16:543–58
81. Deutsch, M., Katz, I., Jensen, A. R., Eds. 1968. *Social Class, Race and Psychological Development.* New York: Holt, Rinehart & Winston. 531 pp.
82. Dewey, W. J., Barrai, I., Morton, N. E., Mi, M. P. 1965. Recessive genes in severe mental defect. *Am. J. Hum. Genet.* 17:237–56
83. Dixon, L. K., DeFries, J. C. 1968. Development of open-field behavior in mice: effects of age and experience. *Develop. Psychobiol.* 1:100–7
84. Dobzhansky, T. 1967. Of flies and men. *Am. Psychol.* 22:41–48
85. Dobzhansky, T. 1967. Étude génétique des réactions de Drosophiles à la lumière et à la pesanteur. *Ann. Biol.* 6:483–97
86. Dobzhansky, T., Ehrman, L., Kastritsis, P. A. 1968. Ethological isolation between sympatric and allopatric species of the *obscura* group of *Drosophila*. *Anim. Behav.* 16:79–87
87. Dobzhansky, T., Spassky, B. 1967. Effects of selection and migration on geotactic and phototactic be-

haviour of *Drosophila*. I. *Proc. Roy. Soc. B.* 168:27–47

88. Dobzhansky, T., Spassky, B. 1969. Artificial and natural selection for two behavioral traits in *Drosophila pseudoobscura*. *Proc. Nat. Acad. Sci.* 62:75–80
89. Dobzhansky, T., Spassky, B., Sved, J. 1969. Effects of selection and migration on geotactic and phototactic behaviour of *Drosophila*. II. *Proc. Roy. Soc. B* 173:191–207
90. Dutta, P. C. 1966. A review of the inherited defective colour-vision variability and selection relaxation among the Indians. *Acta Genet. (Basel)* 16:327–39
91. Eckland, B. K. 1967. Genetics and sociology: a reconsideration. *Am. Sociol. Rev.* 32:173–94
92. Eckland, B. K. 1968. Theories of mate selection. *Eugen. Quart.* 15: 71–84
93. Ehrman, L. 1970. Stimulation of the mating advantage in mating of rare *Drosophila* males. *Science* 167:905–6
94. Ehrman, L., Spassky, B., Pavlovsky, O., Dobzhansky, T. 1965. Sexual selection, geotaxis, and chromosomal polymorphism in experimental populations of *Drosophila pseudoobscura*. *Evolution* 19:337–46
95. Eimer, E. O., Senter, R. J. 1968. Alcohol consumption in domestic and wild rats. *Psychon. Sci.* 10: 319–20
96. Elliot, O., Scott, J. P. 1965. The analysis of breed differences in maze performance in dogs. *Anim. Behav.* 13:5–18
97. Elston, R. C., Campbell, M. A. 1970. Schizophrenia: evidence for the major gene hypothesis. *Behav. Genet.* 1:3–10
98. Elston, R. C. Gottesman, I. I. 1968. The analysis of quantitative inheritance simultaneously from twin and family data. *Am. J. Hum. Genet.* 20:512–21
99. Eriksson, K. 1968. Genetic selection for voluntary alcohol consumption in the albino rat. *Science* 159:739–41
100. Eriksson, K. 1969. Factors affecting voluntary alcohol consumption in the albino rat. *Ann. Zool. Fennici* 6:227–65
101. Eriksson, K., Pikkarainen, P. H. 1968. Differences between the sexes in voluntary alcohol consumption and liver ADH-activity in inbred strains of mice. *Metabolism* 17: 1037–42
102. Erlenmeyer-Kimling, L. 1968. Studies on the offspring of two schizophrenic parents. See Ref. 338, 65–83
103. Erlenmeyer-Kimling, L. 1968. Mortality rates in the offspring of schizophrenic parents and a physiological advantage hypothesis. *Nature* 220:798–800
104. Erlenmeyer-Kimling, L., Nicol, S., Ranier, J. D., Deming, W. E. 1969. Changes in fertility rates of schizophrenic patients in New York state. *Am. J. Psychiat.* 125:916–27
105. Erway, L., Hurley, L. S., Fraser, A. 1966. Neurological defect: manganese in phenocopy and prevention of genetic abnormality of inner ear. *Science* 152:1766–68
106. Ewing, A. W., Manning, A. 1967. The evolution and genetics of insect behaviour. *Ann. Rev. Entomol.* 12:471–94
107. Falconer, D. S. 1960. *Introduction to Quantitative Genetics*. New York: Ronald. 365 pp.
108. Fischer, M., Harvald, B., Hauge, M. 1969. A Danish twin study of schizophrenia. *Brit. J. Psychiat.* 115:981–90
109. Fitzpatrick, T. B., Quevedo, W. C., Jr. 1965. Albinism. In *The Metabolic Basis of Inherited Diseases*, ed. J. B. Stanbury, J. B. Wyngaarden, D. S. Frederickson, 324–40. New York: McGraw-Hill. 2nd ed. 1477 pp.
110. Flexner, L. B. 1966. Loss of memory in mice as related to regional inhibition of cerebral protein synthesis. *Tex. Rep. Biol. Med.* 24:3–19
111. Fox, M. W. 1965. Neuro-ontogeny of neuromuscular mutant mice. *J. Hered.* 56:55–60
112. Fox, M. W. 1965. Reflex-ontogeny and behavioural development of the mouse. *Anim. Behav.* 13:234–41
113. Freedman, D. 1965. An ethological approach to the genetical study of human behavior. See Ref. 55, 141–61
114. Fulker, D. W. 1966. Mating speed in male *Drosophila melanogaster*: a psychogenetic analysis. *Science* 153: 203–5
115. Fuller, J. L. 1960. Behavior genetics. *Ann. Rev. Psychol.* 11:41–70
116. Fuller, J. L. 1964. Measurement of alcohol preference in genetic ex-

periments. *J. Comp. Physiol. Psychol.* 57:85–88
117. Fuller, J. L. 1967. Experiential deprivation and later behavior. *Science* 158:1645–52
118. Fuller, J. L. 1967. Effect of drinking schedule upon alcohol preference in mice. *Quart. J. Stud. Alc.* 28:22–26
119. Fuller, J. L. 1967. Effects of the albino gene upon behaviour of mice. *Anim. Behav.* 15:467–70
120. Fuller, J. L. 1968. Genotype and social behavior. See Ref 134, 111–17
121. Fuller, J. L. Clark, L. D. 1968. Genotype and behavioral vulnerability to isolation in dogs. *J. Comp. Physiol. Psychol.* 66:151–56
122. Fuller, J. L. Collins, R. L. 1967. Ethanol preference in hybrids between a low and high preference strain of mice. *Genetics* 56:560–61
123. Fuller, J. L., Collins, R. L. 1968. Mice unilaterally sensitized for audiogenic seizures. *Science* 162:1295.
124. Fuller, J. L., Sjursen, F. H., Jr. 1967. Audiogenic seizures in eleven mouse strains. *J. Hered.* 58:135–40
125. Fuller, J. L., Thompson, W. R. 1960. *Behavior Genetics.* New York: Wiley. 396 pp.
126. Fuller, J. L., Wimer, R. E. 1966. Neural, sensory, and motor functions. See Ref. 143, 609–28
127. Gaito, J., Ed. 1966. *Macromolecules and Behavior.* New York: Appleton-Century-Crofts. 197 pp.
128. Garrison, R. J., Anderson, V. E., Reed, S. C. 1968. Assortative marriage. *Eugen. Quart.* 15:113–27
129. Garron, D. C. 1970. Sex-linked, recessive inheritance of spatial and numerical abilities, and Turner's syndrome. *Psychol. Rev.* 77:147–52
130. Gedda, L., Brenci, G. 1966. Theoretical models in twin research. *Acta Genet. Med. Gemellol.* 15:219–23
131. Gee, J. H. 1969. Effect of daily synchronization of sexual activity on mating success in laboratory populations of two species of *Dacus* (Diptera: Tephritidae). *Aust. J. Zool.* 17:619–24
132. Ginsburg, B. E. 1968. Breeding structure and social behavior of mammals: a servomechanism for the avoidance of panmixia. See Ref. 134, 117–28
133. Gittleson, N. L. 1966. Depressive psychosis in the obsessional neurotic. *Brit. J. Psychiat.* 112:883–87
134. Glass, D. C., Ed. 1968. *Biology and Behavior: Genetics.* New York: Rockefeller Univ. Press and Russell Sage Found. 260 pp.
135. Gottesman, I. I. 1965. Personality and natural selection. See Ref. 55, 63–80
136. Gottesman, I. I. 1966. Genetic variance in adaptive personality traits. *J. Child Psychol. Psychiat.* 7:199–208
137. Gottesman, I. I. 1968. A sampler of human behavioral genetics. *Evol. Biol.* 2:276–320
138. Gottesman, I. I., Shields, J. 1966. Contributions of twin studies to perspectives on schizophrenia. In *Progr. Exp. Pers. Res.* 3:1–84
139. Gottesman, I. I., Shields, J. 1966. Schizophrenia in twins: 16 years' consecutive admissions to a psychiatric clinic. *Brit. J. Psychiat.* 112:809–18
140. Gottesman, I. I., Shields, J. 1967. A polygenic theory of schizophrenia. *Proc. Nat. Acad. Sci.* 58:199–205
141. Grant, B., Mettler, L. E. 1969. Disruptive and stabilizing selection on the "escape" behavior of *Drosophila melanogaster*. *Genetics* 62:625–37
142. Graves, H. B., Siegel, P. B. 1968. Chick's response to an imprinting stimulus: heterosis and evolution. *Science* 160:329–30
143. Green, E. L., Ed. 1966. *Biology of the Laboratory Mouse.* New York: McGraw-Hill. 2nd ed. 706 pp.
144. Green, E. L., Doolittle, D. P. 1963. Systems of mating used in mammalian genetics. In *Methodology in Mammalian Genetics*, ed. W. J. Burdette, 3–55. San Francisco: Holden-Day. 646 pp.
145. Gutherz, K., Thiessen, D. D. 1966. Albinism and audiogenic seizures in the mouse. *Psychon. Sci.* 6:97–98
146. Guttman, R. 1965. A design for the study of the inheritance of normal mental traits. See Ref. 55, 199–222
147. Guttman, R., Guttman, L., Rosenzweig, K. A. 1967. Cross-ethnic variation in dental, sensory and perceptual traits: a nonmetric multibivariate derivation of distances for ethnic groups and traits. *Am. J. Phys. Anthropol.* 27:259–76
148. Guttman, R., Lieblich, I., Naftali, G. 1969. Variation in activity scores and sequences in two inbred mouse strains, their hybrids, and backcrosses. *Anim. Behav.* 17:374–85

149. Guze, S. B. 1967. The diagnosis of hysteria: what are we trying to do. *Am. J. Psychiat.* 124:491–98
150. Gyllensten, L., Lindberg, J. 1964. Development of the visual cortex in mice with inherited retinal dystrophy. *J. Comp. Neurol.* 122:79–89
151. Hall, C. S. 1951. The genetics of behavior. In *Handbook of Experimental Psychology*, ed. S. S. Stevens, 304–29. New York: Wiley. 1436 pp.
152. Hamburg, D. A., Lunde, D. T. 1967. Relation of behavioral, genetic, and neuroendocrine factors to thyroid function. In *Genetic Diversity and Human Behavior*, ed. J. N. Spuhler, 135–70. Chicago: Aldine. 291 pp.
153. Hanhart, E. 1965. Die genetischen probleme der schizophrenien. *Acta Genet. Med. Gemellol.* 14:13–40
154. Hare, E. H., Shaw, G. K. 1965. A study in family health: (2) a comparison of the health of fathers, mothers and children. *Brit. J. Psychiat.* 111:467–71
155. Harrington, G. M. 1968. Genetic-environmental interaction in "intelligence": I. biometric genetic analysis of maze performance of *Rattus norvegicus*. *Develop. Psychobiol.* 1:211–18
156. Haseman, J. K., Elston, R. C. 1970. The estimation of genetic variance from twin data. *Behav. Genet.* 1: 11–19
157. Hauge, M., Harvald, B., Fischer, M., Gotlieb-Jensen, K., Juel-Nielsen, N., et al. 1968. The Danish twin register. *Acta Genet. Med. Gemellol.* 17:315–32
158. Hawkins, J. D. 1965. Effects of the A^y substitution on mouse activity level: spurious pleiotropy. *Psychon. Sci.* 3:269–70
159. Ibid. 1966. Developmental effects of the A^y substitution on mouse activity level. 4:105–6
160. Hecht, F., Nievaard, J. E., Duncanson, N., Miller, J. R., Higgins, J. V., et al. 1969. Double aneuploidy: the frequency of XXY in males with Down's syndrome. *Am. J. Hum. Genet.* 21:352–59
161. Hegmann, J. P., DeFries, J. C. 1968. Open-field behavior in mice: genetic analysis of repeated measures. *Psychon. Sci.* 13:27–28
162. Heller, J. H. 1969. Human chromosome abnormalities as related to physical and mental dysfunction. *J. Hered.* 60:239–48
163. Henderson, N. D. 1966. Inheritance of reactivity to experimental manipulation in mice. *Science* 153:640–52
164. Henderson, N. D. 1967. Prior treatment effects on open field behaviour of mice—a genetic analysis. *Anim. Behav.* 15:364–76
165. Henderson, N. D. 1968. The confounding effects of genetic variables in early experience research: can we ignore them? *Develop. Psychobiol.* 1:146–52
166. Henderson, N. D. 1969. Prior-treatment effects on open-field emotionality: the need for representative design. *N.Y. Acad. Sci.* 159:860–68
167. Henry, K. R. 1966. *Audiogenic Seizures.* PhD thesis. Univ. North Carolina, Chapel Hill
168. Henry, K. R., Bowman, R. E. 1968. The dilute locus, pyridoxine deficiency, and audiogenic seizures in mice. *Proc. Soc. Exp. Biol. Med.* 128:635–38
169. Henry, K. R., Bowman, R. E. 1969. *Early exposure to intense acoustic stimuli and susceptibility to audiogenic seizures.* Presented at AAAS, Boston
170. Henry, K. R., Bowman, R. E. 1970. Behavior-genetic analysis of the ontogeny of acoustically primed audiogenic seizures in mice. *J. Comp. Physiol. Psychol.* 70:235–41
171. Henry, K. R., Schlesinger, K. 1967. Effects of the albino and dilute loci on mouse behavior. *J. Comp. Physiol. Psychol.* 63:320–23
172. Heston, L. L. 1966. Psychiatric disorders in foster home reared children of schizophrenic mothers. *Brit. J. Psychiat.* 112:819–25
173. Heston, L. L. 1970. The genetics of schizophrenic and schizoid disease. *Science* 167:249–56
174. Heston, L. L., Shields, J. 1968. Homosexuality in twins. *Arch. Gen. Psychiat.* 18:149–60
175. Hirsch, J., Ed. 1967. *Behavior-Genetic Analysis.* New York: McGraw-Hill. 522 pp.
176. Ibid. Behavior-genetic analysis at the chromosome level of organization, 258–69
177. Ibid. Behavior-genetic analysis, 416–35
178. Hirsch, J. 1967. Behavior-genetic, or "experimental," analysis: the chal-

lenge of science versus the lure of technology. *Am. Psychol.* 22:118–30

179. Hirsch, J. 1967. Tropisms are forced movements. *Am. Zool.* 7:422
180. Hirsch, J. 1967. Intellectual functioning and the dimensions of human variation. See Ref. 152, 19–31
181. Hirsch, J., Ksander, G. 1969. Studies in experimental behavior genetics: V. Negative geotaxis and further chromosome analyses in *Drosophila melanogaster*. *J. Comp. Physiol. Psychol.* 67:118–22
182. Holland, H. C., Gupta, B. D. 1966. Some correlated measures of activity and reactivity in two strains of rats selectively bred for differences in the acquisition of a conditioned avoidance response. *Anim. Behav.* 14:574–80
183. Hopkinson, G., Ley, P. 1969. A genetic study of affective disorder. *Brit. J. Psychiat.* 115:917–22
184. Hosgood, S. M. W., MacBean, I. T., Parsons, P. A. 1968. Genetic heterogeneity and accelerated responses to directional selection in *Drosophila*. *Molec. Gen. Genet.* 101: 217–26
185. Hosgood, S. M. W., Parsons, P. A. 1967. Genetic heterogeneity among the founders of laboratory populations of *Drosophila melanogaster*. *Aust. J. Biol. Sci.* 20:1193–1203
186. Hostetter, R. C., Hirsch, J. 1967. Genetic analysis of geotaxis in *Drosophila melanogaster*: complementation between forward and reverse selection lines. *J. Comp. Physiol. Psychol.* 63:66–70
187. Hsia, D. Y.-Y. 1967. The hereditary metabolic diseases. See Ref. 175, 176–93
188. Huff, S. D., Fuller, J. L. 1964. Audiogenic seizures, the dilute locus, and phenylalanine hydroxylase in DBA/1 mice. *Science* 144: 304–5
189. Hulse, F. S. 1958. Exogamie et hétérosis. *Arch. Suisses Anthropol. Génét.* 22:103–25
190. Huntley, R. M. C. 1966. Heritability of intelligence. In *Genetic and Environmental Factors in Human Ability*, ed. J. E. Meade, A. S. Parkes, 201–18. Edinburgh: Oliver & Boyd. 242 pp.
191. Huxley, J., Mayr, E., Osmond, H., Hoffer, A. 1964. Schizophrenia as a genetic morphism. *Nature* 204: 220–21
192. Hydén, H. 1969. Biochemical aspects of learning and memory. In *On the Biology of Learning*, ed. K. H. Pribram, 95–125. New York: Harcourt, Brace & World. 225 pp.
193. Inouye, E. 1965. Similar and dissimilar manifestations of obsessive-compulsive neurosis in monozygotic twins. *Am. J. Psychiat.* 121:1171–75
194. Iverson, O. H., Fagereng, E., Pjerknes, R., Veres, G. 1967. Diurnal variations in spontaneous bodily activity in hairless mice measured with activograph. *Acta Pathol. Microbiol. Scand.* 70:526–30
195. Jablon, S., Neel, J. V., Gershowitz, H., Atkinson, G. F. 1967. The NAS-NRC twin panel: methods of construction of the panel, zygosity diagnosis, and proposed use. *Am. J. Hum. Genet.* 19:133–61
196. Jacobs, P. A., Brunton, M., Melville, M. M., Brittain, R. P., McClemont, W. F. 1965. Aggressive behavior, mental sub-normality and the XYY male. *Nature* 208:1351–52
197. Jarvik, L. F. 1967. Survival and psychological aspects of ageing in man. *Symp. Soc. Exp. Biol.* 21: 463–82
198. Jarvik, L. F., Erlenmeyer-Kimling, L. 1967. Survey of familial correlations in measured intellectual functions. In *Psychopathology of Mental Development*, ed. J. Zubin, G. A. Jervis, 447–59. New York: Grune & Stratton. 658 pp.
199. Jensen, A. R. 1967. Estimation of the limits of heritability of traits by comparison of monozygotic and dizygotic twins. *Proc. Nat. Acad. Sci.* 58:149–56
200. Jensen, A. R. 1969. How much can we boost IQ and scholastic achievement? *Harvard Educ. Rev.* 39:1–123
201. Jinks, J. L., Fulker, D. W. 1970. Comparison of the biometrical, genetical, MAVA, and classical approaches to the analysis of human behavior. *Psychol. Bull.* 73: 311–49
202. Jinks, J. L., Perkins, J. M., Breese, E. L. 1969. A general method of detecting additive, dominance and epistatic variation for metrical traits: II. Application to inbred lines. *Heredity* 24:45–57
203. Joffe, J. M. 1969. Perinatal determinants of emotionality. *Ann. N.Y. Acid. Sci.* 159:668–80
204. Jones, R. 1965. Analysis of variance

of the half diallel table. *Heredity* 20:117–21

205. Juel-Nielsen, N. 1965. *Individual and Environment.* Copenhagen: Munksgaard. 158 pp. +292 pp. appendix
206. Kakihana, R., Brown, D. R., McClearn, G. E., Tabershaw, I. R. 1966. Brain sensitivity to alcohol in inbred mouse strains. *Science* 154:1574–75
207. Kaplan, A. R. 1967. Genetics and schizophrenia. *Eugen. Quart.* 14: 296–98
208. Kaplan, A. R. 1968. Physiological and pathological correlates of differences in taste acuity. See Ref. 410, 31–66
209. Kaplan, A. R. 1969. The use of cytogenetical data in heredity counseling. *Am. J. Ment. Defic.* 73:636–53
210. Kaplan, A. R., Fischer, R., Karras, A., Griffin, F., Powell, W. et al. 1967. Taste thresholds in twins and siblings. *Acta Genet. Med. Gemellol.* 16:229–43
211. Kaplan, W. D., Trout, W. E., III. 1969. The behavior of four neurological mutants of *Drosophila. Genetics* 61:339–409
212. Karczmar, A. G., Scudder, C. L. 1969. Aggression and neurochemical changes in different strains and genera of mice. In *Aggressive Behaviour*, ed. S. Garattini, E. B. Sigg, 209–27. New York: Wiley. 385 pp.
213. Karlsson, J. L. 1966. *The Biologic Basis of Schizophrenia.* Springfield, Ill.: Thomas. 87 pp.
214. Katzenmeyer, C., Whitney, G. D., Newell, T. G. 1969. Computers in behavior science: estimation of genetic parameters by computer. *Behav. Sci.* 14:160–63
215. Kaul, D., Parsons, P. A. 1966. Competition between males in the determination of mating speed in *Drosophila pseudoobscura. Aust. J. Biol. Sci.* 19:945–47
216. Kearsey, M. J. 1965. Biometrical analysis of a random mating population: a comparison of five experimental designs. *Heredity* 20: 205–35
217. Keeler, C. 1966. Retinal degeneration in the mouse is rodless retina. *J. Hered.* 57:47–50
218. Keeler, C., Fromm, E., Wade, L. 1968. Melanin, adrenalin and the legacy of fear. *CSH Bull. Current Res.* 7:15–21
219. Keeler, C., King, H. D. 1942. Multiple effects of coat color genes in the Norway rat, with special reference to temperament and domestication. *J. Comp. Psychol.* 34:241–50
220. Keeler, C., Ridgeway, S., Lipscomb, L., Fromm, E. 1968. The genetics of adrenal size and tameness in colorphase foxes. *J. Hered.* 59: 82–84
221. Kessler, S. 1966. Selection for and against ethological isolation between *Drosophila pseudoobscura* and *Drosophila persimilis. Evolution* 20:634–45
222. Kessler, S. 1968. Speed of mating and sexual isolation in *Drosophila. Nature* 220:1044–45
223. Kessler, S. 1969. The genetics of *Drosophila* mating behavior. II. the genetic architecture of mating speed in *Drosophila pseudoobscura. Genetics* 62:421–33
224. Kessler, S., Moos, R. H. 1970. The XXY karyotype and criminality: a review. *J. Psychiat. Res.* 7:153–70
225. Kety, S. S., Rosenthal, D., Wender, P. H., Schulsinger, F. 1968. The types and prevalence of mental illness in the biological and adoptive families of adopted schizophrenics. See Ref. 338, 345–62
226. Kiser, C. V. 1968. Assortative mating by educational attainment in relation to fertility. *Eugen. Quart.* 15:98–112
227. Koch, H. L. 1966. *Twins and Twin Relations.* Chicago: Univ. Chicago Press. 302 pp.
228. Kreitman, N. 1968. Married couples admitted to mental hospital. *Brit. J. Psychiat.* 114:699–718
229. Kringlen, E. 1967. *Heredity and Environment in the Functional Psychoses.* London: Heinemann. 201 pp.
230. Kringlen, E. 1968. An epidemiological-clinical twin study on schizophrenia. See Ref. 338, 49–63
231. Kringlen, E. 1968. Clinical variability in schizophrenic twin partners. See Ref. 410, 127–35
232. Kuttner, R. E., Lorincz, A. B. 1966. Schizophrenia and evolution. *Eugen. Quart.* 13:355–56
233. Lagerspetz, K. M. J. 1969. Aggression and aggressiveness in laboratory mice. See Ref. 212, 77–85
234. Lejeune, J. 1967. Chromosomal studies in psychiatry. *Recent Advances Biol. Psychiat.* 9:13–20

235. Lerner, I. M. 1954. *Genetic Homeostasis*. New York: Wiley. 134 pp.
236. Lerner, I. M. 1968. *Heredity, Evolution and Society*. San Francisco: Freeman. 307 pp.
237. Les, E. P. 1968. Cage population density and efficiency of feed utilization in inbred mice. *Lab. Anim. Care* 18:305–13
238. Lester, D. 1966. Self-selection of alcohol by animals, human variation, and the etiology of alcoholism. *Quart. J. Stud. Alc.* 27:395–438
239. Levine, L., Barsel, G. E., Diakow, C. A. 1965. Interaction of aggressive and sexual behavior in male mice. *Behaviour* 25:272–80
240. Levine, L., Barsel, G. E., Diakow, C. A. 1966. Mating behaviour of two inbred strains of mice. *Anim. Behav.* 14:1–6
241. Levine, L., Diakow, C. A., Barsel, G. E. 1965. Interstrain fighting in male mice. *Anim. Behav.* 13:52–58
242. Levine, L., Krupa, P. L. 1966. Studies on sexual selection in mice III. effects of the gene for albinism. *Am. Natur.* 100:227–34
243. Levins, R. 1968. *Evolution in Changing Environments*. Princeton: Princeton Univ. Press. 120 pp.
244. Lewis, D. G. 1966. Commentary on 'The genetic determination of differences in intelligence: a study of monozygotic twins reared together and apart' by Cyril Burt. *Brit. J. Psychol.* 57:431–33
245. Lindzey, G. 1967. Some remarks concerning incest, the incest taboo, and psychoanalytic theory. *Am. Psychol.* 22:1051–59
246. Lindzey, G., Manosevitz, M. 1964. Hoarding in the mouse. *Psychon. Sci.* 1:35–36
247. Lindzey, G., Manosevitz, M., Winston, H. D. 1966. Social dominance in the mouse. *Psychon. Sci.* 5:451–45
248. Lindzey, G., Thiessen, D. D. 1970. Genetic aspects of negative geotaxis in mice. *Behav. Genet.* 1:21–34
249. Lindzey, G., Thiessen, D. D., Blum, S., Tucker, A. 1969. Further observations on social dominance in mice. *Psychon. Sci.* 14:245–46
250. Lindzey, G., Winston, H., Manosevitz, M. 1961. Social dominance in inbred mouse strains. *Nature* 191: 474–76
251. Lindzey, G., Winston, H. D., Roberts, L. E. 1965. Sociability, fearfulness, and genetic variation in the mouse. *J. Pers. Soc. Psychol.* 1:642–45
252. Loehlin, J. C. 1965. A heredity-environment analysis of personality inventory data. See Ref. 55, 163–70
253. Loehlin, J. C. 1965. Some methodological problems in Cattell's Multiple Abstract Variance Analysis. *Psychol. Rev.* 72:156–61
254. Loehlin, J. C. 1970. Psychological genetics, from the study of human behavior. In *Handbook of Modern Personality Theory*, ed. R. B. Cattell. Chicago: Aldine. In press
255. Loehlin, J. C., Vandenberg, S. G. 1968. Genetic and environmental components in the covariation of cognitive abilities: an additive model. See Ref. 410, 261–85
256. Lubs, H., Ruddle, F. 1969. Major chromosomal abnormalities in 4000 consecutive newborns. (Abstract) *Clin. Res.* 17:316
257. Lyon, M. F. (Collator) 1969. *Mouse News Letter* 41. Harwell, Didcot, Berkshire, England, M.R.C. Radiobiological Research Unit
258. MacBean, I. T., Parsons, P. A. 1967. Directional selection for duration of copulation in *Drosophila melanogaster*. *Genetics* 56:233–39
259. McClearn, G. E. 1968. Genetics and motivation of the mouse. *Neb. Symp. Mot.* 16:47–84
260. McClearn, G. E. 1970. Behavioral genetics. *Ann. Rev. Genet.* 4:437–68
261. McClearn, G. E., Meredith, W. 1966. Behavioral genetics. *Ann. Rev. Psychol.* 17:515–50
262. McClearn, G. E., Rodgers, D. A. 1959. Differences in alcohol preference among inbred strains of mice. *Quart. J. Stud. Alc.* 20:691–95
263. McGaugh, J. L., Cole, J. M. 1965. Age and strain differences in the effect of distribution of practice on maze learning. *Psychon. Sci.* 2: 253–54
264. McGill, T. E. 1965. Studies of the sexual behavior of male laboratory mice: effects of genotype, recovery of sex drive, and theory. In *Sex and Behavior*, ed. F. A. Beach, 76–88. New York: Wiley. 592 pp.
265. McGill, T. E. 1969. An enlarged study of genotype and recovery of sex drive in male mice. *Psychon. Sci.* 15:250–51
266. McGill, T. E. 1970. Genetic analysis of male sexual behavior. See Ref. 44, 57–88
267. McGill, T. E., Ransom, T. W. 1968. Genotypic change affecting con-

clusions regarding the mode of inheritance of elements of behaviour. *Anim. Behav.* 16:88–91

268. McKusick, V. A. 1968. *Mendelian Inheritance in Man.* Baltimore: Johns Hopkins Press. 2nd ed. 521 pp.
269. Manning, A. 1965. *Drosophila*, and the evolution of behaviour. *Viewpoints Biol.* 4:123–67
270. Manning, A. 1967. Genes and the evolution of insect behavior. See Ref. 175, 44–60
271. Manosevitz, M. 1965. Genotype, fear, and hoarding. *J. Comp. Physiol. Psychol.* 60:412–16
272. Ibid. 1967. Hoarding and inbred strains of mice. 63:148–50
273. Manosevitz, M. 1969. A note on genotype-environment interaction. *Tex. Rep. Biol. Med.* 27:1089–1103
274. Manosevitz, M. 1970. Prolonged aperiodic feeding and adult hoarding in mice. *J. Comp. Physiol. Psychol.* 70:228–34
275. Manosevitz, M., Campenot, R. B., Swencionis, C. F. 1968. Effects of an enriched environment upon hoarding. *J. Comp. Physiol. Psychol.* 66:319–24
276. Manosevitz, M., Fitzsimmons, C. I., McCanne, T. R. 1970. Correlates of food competition behavior. *Psychon. Sci.* 19:141–42
277. Manosevitz, M., Lindzey, G. 1967. Genetics of hoarding: a biometrical analysis. *J. Comp. Physiol. Psychol.* 63:142–44
278. Manosevitz, M., Lindzey, G. 1970. Genetic variation and hoarding. See Ref. 44, 91–113
279. Manosevitz, M., Lindzey, G., Thiessen, D. D., Eds. 1969. *Behavioral Genetics: Methods and Research.* New York: Appleton-Century-Crofts. 771 pp.
280. Marler, P. 1966. Mechanisms of animal behavior: recent work and theories. *Science* 152:933–34
281. Marsden, H. M., Bronson, F. H. 1965. Strange male block to pregnancy: its absence in inbred mouse strains. *Nature* 207:878
282. Masica, D. N., Money, J., Ehrhardt, A. A., Lewis, V. G. 1969. IQ, fetal sex hormones and cognitive patterns: studies in the testicular feminizing syndrome of androgen insensitivity. *Johns Hopkins Med. J.* 124:34–43
283. Mather, K. 1967. Complementary and duplicate gene interactions in biometrical genetics. *Heredity* 22:97–103
284. Mednick, S. A., Schulsinger, F. 1965. A longitudinal study of children with a high risk for schizophrenia: a preliminary report. See Ref. 55, 255–95
285. Mednick, S. A., Schulsinger, F. 1968. Some premorbid characteristics related to breakdown in children with schizophrenic mothers. See Ref. 338, 267–91
286. Meissner, W. W. 1965. Functional and adaptative aspects of cellular regulatory mechanisms. *Psychol. Bull.* 64:206–16
287. Menkes, J. H., Migeon, B. R. 1966. Biochemical and genetic aspects of mental retardation. *Ann. Rev. Med.* 17:407–30
288. Merrell, D. J. 1965. Methodology in behavior genetics. *J. Hered.* 56: 262–66
289. Mitsuda, H., Ed. 1967. *Clinical Genetics in Psychiatry.* Tokyo: Igaku Shoin. 408 pp.
290. Ibid, 3–21
291. Mitsuda, H., Sakai, T., Kobayashi, J. 1967. A clinico-genetic study on the relationship between neurosis and psychosis. See Ref. 289, 27–35
292. Money, J., Ehrhardt, A. A., Masica, D. N. 1968. Fetal feminization induced by androgen insensitivity in the testicular feminizing syndrome: effect on marriage and maternalism. *Johns Hopkins Med. J.* 123:105–14
293. Money, J., Lewis, V., Ehrhardt, A. A., Drash, P. W. 1967. IQ impairment and elevation in endocrine and related cytogenetic disorders. See Ref. 198, 22–27
294. Money, J., Mittenthal, S. 1970. Lack of personality pathology in Turner's syndrome: relation to cytogenetics, hormones and physique. *Behav. Genet.* 1:43–56
295. Mordkoff, A. M., Fuller, J. L. 1959. Heritability in activity within inbred and crossbred mice. *J. Hered.* 50:6–8
296. Morton, N. E. 1967. Population genetics of mental illness. *Eugen. Quart.* 14:181–84
297. Morton, N. E. 1967. The detection of major genes under additive continuous variation. *Am. J. Hum. Genet.* 19:23–34
298. Nakajima, S. 1969. Interference with relearning in the rat after hippocampal injection of actinomycin

D. *J. Comp. Physiol. Psychol.* 67: 457–61

299. Newell, T. G. 1970. Three biometrical genetic analyses of activity in the mouse. *J. Comp. Physiol. Psychol.* 70:37–47
300. Nichols, R. C. 1965. The national merit twin study. In *Methods and Goals of Human Behavior Genetics*, ed. S. G. Vandenberg, 231–43. New York: Academic. 351 pp.
301. Nichols, R. C. 1969. The resemblance of twins in personality and interests. See Ref. 55, 580–96
302. Nichols, R. C., Bilbro, W. C., Jr. 1966. The diagnosis of twin zygosity. *Acta Genet. (Basel)* 16: 265–75
303. Oliverio, A. 1968. Transfer of avoidance learning in different strains of inbred mice. *Life Sci.* 7:1157–62
304. Ortman, L. L., Craig, J. V. 1968. Social dominance in chickens modified by genetic selection—physiological mechanisms. *Anim. Behav.* 16: 33–37
305. Osborne, R. T., Gregor, A. J. 1966. The heritability of visualization, perceptual speed and spatial orientation. *Percept. Mot. Skills* 23:379–90
306. Osborne, R. T., Gregor, A. J., Miele, F. 1967. Heritability of numerical facility. *Percept. Mot. Skills* 25: 659–66
307. Parsons, P. A. 1967. *The Genetic Analysis of Behaviour*. London: Methuen. 174 pp.
308. Parsons, P. A., Hosgood, S. M. W., Lee, B. T. O. 1967. Polygenes and polymorphism. *Mol. Gen. Genet.* 99:165–70
309. Parsons, P. A., Kaul, D. 1966. Mating speed and duration of copulation in *Drosophila pseudoobscura*. *Heredity* 21:219–25
310. Parsons, P. A., Kaul, D. 1967. Variability within and between strains for mating behaviour parameters in *Drosophila pseudoobscura*. *Experientia* 23:1–4
311. Partanen, J., Brunn, K., Markkanen, T. 1966. *Inheritance of drinking Behavior*. Helsinki: Finnish Found. Alc. Stud. 159 pp.
312. Penrose, L. S., Smith, G. F. 1966. *Down's Anomaly*. London: Churchill. 218 pp.
313. Perris, C. 1966. A study of bipolar (manic-depressive) and unipolar recurrent depressive psychoses. *Acta Psychiat. Scand.* Suppl. 194
314. Petrinovich, L., Bradford, D., McGaugh, J. L. 1965. Drug facilitation of memory in rats. *Psychon. Sci.* 2:191–92
315. Planansky, K. 1966. Schizoidness in twins. *Acta Genet. Med. Gemellol.* 15:151–66
316. Platt, S. A., Hirsch, J. 1968. The analysis of (typologically conceived) tropisms. *Am. Zool.* 8:59
317. Pollin, W., Allen, M. G., Hoffer, A., Stabenau, J. R., Hrubec, Z. 1969. Psychopathology in 15,909 pairs of veteran twins: evidence for a genetic factor in the pathogenesis of schizophrenia and its relative absence in psychoneurosis. *Am. J. Psychiat.* 126:597–609
318. Pollin, W., Stabenau, J. R. 1968. Biological, psychological and historical differences in a series of monozygotic twins discordant for schizophrenia. See Ref. 338, 317–32
319. Pollin, W., Stabenau, J. R., Hoffer, A., Mosher L. R., Spillman, B. 1968. The NIMH study of a series of monozygotic twins discordant for schizophrenia. See Ref. 410, 137–49
320. Post, R. H. 1962. Population differences in red and green color vision deficiency: a review, and a query on selection relaxation. *Eugen. Quart.* 9:131–46
321. Price, E. 1967. The effect of reproductive performance on the domestication of the prairie deermouse, *Peromyscus maniculatus bairdii*. *Evolution* 21:762–70
322. Quevedo, W. C. 1969. The control of color in mammals. *Am. Zool.* 9: 531–40.
323. Ralls, K. 1967. Auditory sensitivity in mice: *Peromyscus and Mus musculus*. *Anim. Behav.* 15:123–28
324. Recherche Coopérative sur Pro gramme No. 85 (C.N.R.S.). 1968. Fréquence de la constitution XYY dans la population générale. *Ann. Génét.* 11:245–46
325. Reed, E. W., Reed, S. C. 1965. *Mental Retardation: A Family Study*. Philadelphia: W. B. Saunders. 719 pp.
326. Roberts, D. F. 1967. Red/green color blindness in the Niger delta. *Eugen. Quart.* 14:7–13
327. Roberts, L. E. 1967. Central, peripheral, and artifactual determinants of skin resistance in the mouse. *J. Comp. Physiol. Psychol.* 64:318–28
328. Roberts, R. C. 1967. Some concepts

and methods in quantitative genetics. See Ref. 175, 214–57

329. Robertson, A. 1967. The nature of quantitative genetic variation. In *Heritage From Mendel*, ed. R. A. Brink, 265–80. Madison: Univ. Wisconsin Press, 455 pp.
330. Robinson, A., Goad, W. B., Puck, T. T., Harris, J. S. 1969. Studies on chromosomal nondisjunction in man. III. *Am. J. Hum. Genet.* 21: 466–85
331. Rodgers, D. A. 1966. Factors underlying differences in alcohol preference among inbred strains of mice. *Psychosom. Med.* 28:498–513
332. Rodgers, D. A., Thiessen, D. D. 1964. Effects of population density on adrenal size, behavioral arousal, and alcohol preference of inbred mice. *Quart. J. Stud. Alc.* 25:240–47
333. Rodgers, D. A., Ward, P. A. Thiessen, D. D., Whitworth, N. S. 1967. Pathological effects of prolonged voluntary consumption of alcohol by mice. *Quart. J. Stud. Alc.* 28: 618–30
334. Romano, J., Ed. 1967. *The Origins of Schizophrenia.* Amsterdam: Excerpta Med. Found. 288 pp.
335. Rosenberg, C. M. 1967. Familial aspects of obsessional neurosis. *Brit. J. Psychiat.* 113:405–13
336. Rosenthal, D. 1968. The genetics of intelligence and personality. See Ref. 134, 69–78
337. Rosenthal, D. 1970. *Genetic Theory and Abnormal Behavior.* New York: McGraw-Hill
338. Rosenthal, D., Kety, S. S., Eds. 1968. *The Transmission of Schizophrenia.* Oxford: Pergamon. 433 pp.
339. Rosenthal, D., Wender, P. H., Kety, S. S., Schulsinger, F., Welner, J., et al. 1968. Schizophrenics' offspring reared in adoptive homes. See Ref. 338
340. Rosenzweig, M. R., Bennett, E. L. 1969. Effects of differential environments on brain weights and enzyme activities in gerbils, rats, and mice. *Develop. Psychobiol.* 2: 87–95
341. Rothenbuhler, W. C. 1967. Genetic and evolutionary considerations of social behavior of honeybees and some related insects. See Ref. 175, 61–106
342. Royce, J. R. 1966. Optimal stimulus parameters in avoidance conditioning of inbred strains of mice. *Multivar. Behav. Res.* 1:209–17
343. Sackler, A. M., Weltman, A. S. 1967. Metabolic and endocrine differences between the mutation whirler and normal female mice. *J. Exp. Zool.* 164:133–40
344. Sakai, T. 1967. Clinico-genetic study on obsessive-compulsive neurosis. See Ref. 289, 323–31
345. Scarr, S. 1966. Genetic factors in activity motivation. *Child Develop.* 37:663–73
346. Scarr, S. 1966. The origins of individual differences in adjective check list scores. *J. Consult. Psychol.* 30:354–57
347. Scarr, S. 1968. Environmental bias in twin studies. *Eugen. Quart.* 15: 34–40
348. Scarr, S. 1969. Social introversion-extraversion as a heritable response. *Child Develop.* 40:823–32
349. Schlesinger, K. 1966. Genetic and biochemical correlates of alcohol preference in mice. *Am. J. Psychiat.* 122:767–73
350. Schlesinger, K., Elston, R. C., Boggan, W. 1966. The genetics of sound induced seizure in inbred mice. *Genetics* 54:95–103
351. Schlesinger, K., Griek, B. J. 1970. The genetics and biochemistry of audiogenic seizures. See Ref. 44, 219–57
352. Schlesinger, K., Kakihana, R., Bennett, E. L. 1966. Effects of tetraethylthiuramdisulfide (Antabuse) on the metabolism and consumption of ethanol in mice. *Psychosom. Med.* 28:514–20
353. Schlesinger, K., Wimer, R. 1967. Genotype and conditioned avoidance learning in the mouse. *J. Comp. Physiol. Psychol.* 63:139–41
354. Schoenfeldt, L. F. 1968. The hereditary components of the project TALENT two-day test battery. *Meas. Eval. Guid.* 1:130–40
355. Schull, W. J., Neel, J. V. 1965. *The Effects of Inbreeding on Japanese Children.* New York: Harper & Row. 419 pp.
356. Scott, J. P., Fuller, J. L. 1965. *Genetics and the Social Behavior of the Dog.* Chicago: Univ. Chicago Press. 468 pp.
357. Scudder, C. L., Avery, D., Karczmar, A. G. 1969. Study of avoidance conditioning in five genera of mice. *Anim. Behav.* 17:77–86
358. Scudder, C. L., Karczmar, A. G., Lockett, L. 1966. Behavioural developmental studies on four genera

and several strains of mice. *Anim. Behav.* 15:353–63

359. Selander, R. L., Yang, S. Y. 1970. Biochemical genetics and behavior in wild house mouse populations. See Ref. 44, 293–334
360. Sells, S. B., Roff, M. 1967. Peer acceptance-rejection and personality development. *Final rep., Project OE* 5-0417, *Off. Educ., US Dep. HEW*. 466 pp.
361. Sergovich, F., Valentine, G. H., Chen, A. T. L., Kinch, R. A. H., Smout, M. S. 1969. Chromosome aberrations in 2159 consecutive newborn babies. *New Engl. J. Med.* 280:851–55
362. Sheppard, J. R., Albersheim, P., McClearn, G. E. 1968. Enzyme activities and ethanol preference in mice. *Biochem. Genet.* 2:205–12
363. Sheridan, C. L. 1965. Interocular transfer of brightness and pattern discriminations in normal and corpus callosum-sectioned rats. *J. Comp. Physiol. Psychol.* 59:292–94
364. Shields, J., Slater, E. 1966. La similarité du diagnostic chez les jumeaux et le problème de la spécificité biologique dans les névroses et les troubles de la personnalité. *Evolut. Psychiat. (Paris)* 31:441–51
365. Sidman, R. L., Appel, S. H., Fuller, J. F. 1965. Neurological mutants of the mouse. *Science* 150:513–16
366. Sidman, R. L., Green, M. C. 1965. Retinal degeneration in the mouse. *J. Hered.* 56:23–29
367. Sidman, R. L., Green, M. C., Appel, S. H. 1965. *Catalog of the Neurological Mutants of the Mouse.* Cambridge: Harvard Univ. Press. 82 pp.
368. Sidman, M., Ray, B. A., Sidman, R. L., Klinger, J. M. 1966. Hearing and vision in neurological mutant mice: a method of their evaluation. *Exp. Neurol.* 16:377–402
369. Siegel, P. B. 1965. Genetics of behavior: selection for mating ability in chickens. *Genetics* 51:1269–77
370. Sing, C. F., Griffith, R. W., Schull, W. J. 1967. The use of computers in human genetics. *Am. J. Hum. Genet.* 19:183–268
371. Slater, E. 1968. A review of earlier evidence on genetic factors in schizophrenia. See Ref. 338, 15–26
372. Slater, E., Tsuang, N. T. 1968. Abnormality on paternal and maternal sides: observations in schizophrenia and manic-depression. *J. Med. Genet.* 5:197–99
373. Smith, R. T. 1965. A comparison of socioeconomic factors in monozygotic and dizygotic twins, testing an assumption. See Ref. 55, 45–61
374. Southwick, C. H., Clark, L. H. 1968. Interstrain differences in aggressive behavior and exploratory activity of inbred mice. *Commun. Behav. Biol.* 1:49–59
375. Spassky, B., Dobzhansky, T. 1967. Responses of various strains of *Drosophila pseudoobscura* and *Drosophila persimilis* to light and to gravity. *Am. Natural.* 101:59–63
376. Spuhler, J. N., Ed. 1967. *Genetic Diversity and Human Behavior.* Chicago: Aldine. 291 pp.
377. Spuhler, J. N. 1968. Assortative mating with respect to physical characteristics. *Eugen. Quart.* 15: 128–40
378. Spuhler, J. N., Lindzey, G. 1967. Racial differences in behavior. See Ref. 175, 366–414
379. Staats, J. 1966. The laboratory mouse. See Ref. 143, 1–9
380. Stiles, K. A., Isoun, M. F. 1966. Bibliography of mongolism (Down's syndrome). *Acta Genet. Med. Gemellol.* 15:247–72
381. Stott, D. H. 1966. Commentary on 'The genetic determination of differences in intelligence: a study of monozygotic twins reared together and apart' by Cyril Burt. *Brit. J. Psychol.* 57:423–29
382. Tata, J. R. 1966. Hormones and the synthesis and utilization of ribonucleic acids. *Progr. Nucl. Acid Res.* 5:191–250
383. Tellegen, A. 1968. Note on the Tryon effect. *Am. Psychol.* 23:585–87
384. Thiessen, D. D. 1965. The wabbler-lethal mouse: a study in development. *Anim. Behav.* 13:87–100
385. Thiessen, D. D. 1970. Philosophy and method in behavior genetics: its relation to biology. In *Scientific Psychology: Some Perspectives*, ed. A. R. Gilgen. New York: Academic. In press
386. Thiessen, D. D. 1970. A reply to Wilcock on gene action and behavior. *Psychol. Bull.* In press
387. Thiessen, D. D., McClearn, G. E. 1965. Thirst and alcohol preference of inbred strains of mice. *J. Comp. Physiol. Psychol.* 59:436–38
388. Thiessen, D. D., Owen, K., Whitsett, M. 1970. Chromosome mapping of

behavioral activities. See Ref. 44, 161–204

389. Thiessen, D. D., Rodgers, D. A. 1965. Alcohol injection, grouping, and voluntary alcohol consumption of inbred strains of mice. *Quart. J. Stud. Alc.* 26:378–83.
390. Thiessen, D. D., Whitworth, N. S., Rodgers, D. A. 1966. Reproductive variables and alcohol consumption of the C57BL/Crgl female mouse. *Quart. J. Stud. Alc.* 27:591–95
391. Thiessen, D. D., Whitworth, N., Rodgers, D. A. 1967. Reproductive functions and metabolic capacity as determinants of alcohol preference in C57BL female mice. *J. Comp. Physiol. Psychol.* 63:151–54
392. Thiessen, D. D., Yahr, P. 1970. Central control of territorial marking in the Mongolian gerbil. *Physiol. Behav.* 5:275–78
393. Thoday, J. M. 1967. New insights into continuous variation. In *Proc. 3rd Int. Congr. Hum. Genet. 1966*, 339–50. Baltimore: Johns Hopkins Press. 578 pp.
394. Thomas, K. 1969. Selection and avoidance of alcohol solutions by two strains of inbred mice and derived generations. *Quart. J. Stud. Alc.* 30:849–61
395. Thompson, W. R. 1967. Some problems in the genetic study of personality and intelligence. See Ref. 175, 344–65
396. Thompson, W. R. 1968. Genetics and social behavior. See Ref. 134, 79–101
397. Thompson, W. R., Wilde, G. J. S. Behavior genetics. In *Handbook of Psychology*, ed. B. B. Wolman. New York: Wiley
398. Tienari, P. 1966. On intrapair differences in male twins with special reference to dominance-submissiveness. *Acta Psychiat. Scand.* Suppl. 188, 42. 166 pp.
399. Tienari, P. 1968. Schizophrenia in monozygotic male twins. See Ref. 338, 27–36
400. Tucker, G. R., McGill, T. E. 1966. Hormonal reactivation of castrated male mice of different strains. *Psychol. Rep.* 19:810
401. Turpin, R., Lejeune, J. 1969. *Human Afflictions and Chromosomal Aberrations.* Oxford: Pergamon. 392 pp. (Transl. of *Les Chromosomes Humains*, Gauthier-Villars, 1965)
402. Uchida, I. A., Holunga, R., Lawler, C. 1968. Maternal radiation and chromosomal aberrations. *Lancet* ii:1045–49
403. Vale, J. R., Vale, C. A. 1969. Individual differences and general laws in psychology: a reconciliation. *Am. Psychol.* 24:1093–1108
404. Valenti, C. 1969. Discussion of Dr. Jarvik's paper. In *Neurobiological Aspects of Psychopathology*, ed. J. Zubin, C. Shagass, 275–80. New York: Grune & Stratton. 429 pp.
405. Vandenberg, S. G. 1965. Innate abilities, one or many? *Acta Genet. Med. Gemellol.* 14:41–47
406. Vandenberg, S. G. 1966. Contributions of twin research to psychology. *Psychol. Bull.* 66:327–52
407. Vandenberg, S. G. 1967. Hereditary factors in normal personality traits (as measured by inventories). *Recent Advan. Biol. Psychiat.* IX; 65–104
408. Vandenberg, S. G. 1967. Hereditary factors in psychological variables in man, with a special emphasis on cognition. See Ref. 376, 99–133
409. Vandenberg, S. G. 1968. The nature and nurture of intelligence. See Ref. 134, 3–58
410. Vandenberg, S. G., Ed. 1968. *Progress in Human Behavior Genetics.* Baltimore: Johns Hopkins Press. 356 pp.
411. Vandenberg, S. G. 1969. A twin study of spatial ability. *Multivar. Behav. Res.* 4:273–94
412. Vandenberg, S. G. 1969. Human behavior genetics: present status and suggestions for future research. *Merrill-Palmer Quart.* 15:121–54
413. Vandenberg, S. G., Comrey, A. L., Stafford, R. E. 1967. Hereditary factors in personality and attitude scales, a twin study. *Res. Rep. No. 16, Louisville Twin Study, Univ. Louisville Sch. Med.*
414. Vandenberg, S. G., Johnson, R. C. 1968. Further evidence on the relation between age of separation and similarity in IQ among pairs of separated identical twins. See Ref. 410, 215–19
415. Vandenberg, S. G., Stafford, R. E. 1967. Hereditary influences on vocational preferences as shown by scores of twins on the Minnesota Vocational Interest Inventory. *J. Appl. Psychol.* 51:17–19
416. Vandenberg, S. G., Stafford, R. E., Brown, A. M. 1968. The Louisville Twin Study. See Ref. 410, 153–204
417. Vestal, B. M., King, J. A. 1968. Relationship of age at eye opening

to first optokinetic response in deermice (*Peromyscus*). *Develop. Psychobiol.* 1:30–34

418. Wender, P. H., Rosenthal, D., Kety, S. S. 1968. A psychiatric assessment of the adoptive parents of schizophrenics. See Ref. 338, 235–50
419. Werboff, J., Anderson, A. 1967. Spatial and visual performance and learning in mice. *Percept. Mot. Skills* 24:67–72
420. Werboff, J., Anderson, A., Ross, S. 1967. Mice of a four-way cross: coat color associated with behavior and response to *d*-amphetamine. *J. Psychol.* 66:99–117
421. Wiener, S., Sutherland, G. 1968. A normal XYY man. *Lancet* ii:1352
422. Wilcock, J. 1969. Gene action and behavior: an evaluation of major gene pleiotropism. *Psychol. Bull.* 72:1–29
423. Wilcock, J., Broadhurst, P. L. 1967. Strain differences in emotionality: open-field and conditioned avoidance behavior in the rat. *J. Comp. Physiol. Psychol.* 63:335–37
424. Wilson, J. E., Zemp, J. W., Glassman, E. 1969. Increased RNA synthesis in the brains of mice undergoing conditioned avoidance training. In *Biochemical Factors Concerned in the Functional Activity of the Nervous System*, ed. D. Richter, 218. New York: Pergamon. 234 pp.
425. Wimer, R. E., Symington, L., Farmer, H., Schwartzkroin, P. 1968. Differences in memory processes between inbred mouse strains C57BL/6J and DBA/2J. *J. Comp. Physiol. Psychol.* 65:126–31
426. Winokur, G., Clayton, P. 1967. Family history studies I. two types of affective disorders separated according to genetic and clinical factors. *Recent Advan. Biol. Psychiat.* IX:35–50
427. Winokur, G., Ruangtrakool, S. 1966. Genetic versus nongenetic affective disorder. *Am. J. Psychiat.* 123:703–5
428. Winston, H. D. 1963. Influence of genotype and infantile trauma on adult learning in the mouse. *J. Comp. Physiol. Psychol.* 56:630–35
429. Ibid. 1964. Heterosis and learning in the mouse. 57:279–83
430. Winston, H. D., Lindzey, G. 1964. Albinism and water escape performance in the mouse. *Science* 144:189–91
431. Winston, H., Lindzey, G., Connor, J. 1967. Albinism and avoidance learning in mice. *J. Comp. Physiol. Psychol.* 63:77–81
432. Woerner, P. I., Guze, S. B. 1968. A family and marital study of hysteria. *Brit. J. Psychiat.* 114:161–68
433. Wolfe, H. G. Coleman. D. L. 1966. Pigmentation. See Ref. 143, 405–25
434. World Health Organization. 1966. The use of twins in epidemiological studies. *Acta Genet. Med. Gemellol.* 15:109–28
435. Zarrow, M. X., Denenberg, V. H., Kalberer, W. D. 1965. Strain differences in the endocrine basis of maternal nest-building in the rabbit. *J. Reprod. Fert.* 10:397–401
436. Zellweger, H. 1969. Chromosomal aneuploidy, mosaicism and eugenics. *Acta Genet. Med. Gemellol.* 18:69–76
437. Zerbin-Rubin, E. 1967. Endogene psychosen. In *Humangenetik* (Band V/2) *Psychiatrische Krankheiten*, ed. P. E. Becker, 446–577. Stuttgart: Georg Thieme Verlag. 613 pp.
438. Zigler, E. 1967. Familial mental retardation: a continuing dilemma. *Science* 155:292–98
439. Zubin, J., Jervis, G. A., Eds. 1967. *Psychopathology of Mental Development.* New York: Grune & Stratton. 658 pp.