Advances in Neurology, Vol. 42: Progress in Aphasiology, edited by F. C. Rose.
Raven Press, New York © 1984.

Genetics of Handedness in Relation to Language Disorder

I. C. McManus

Department of Psychology, Bedford College, University of London; and Department of Psychiatry, St. Mary's Hospital Medical School, London, England

. . . nature's simple equations in the mind's precincts . . .
R. S. Thomas

In the second half of the 19th century, two major biologic discoveries were made. Broca described the dichotomous division of the population according to cerebral language dominance, and Mendel described the laws of the discrete, particulate inheritance of biologic characteristics and the "simple equations" governing them. Since that time, a number of attempts have been made at integrating these two discoveries. In this chapter, I describe a simple model for interpreting the available data.

A major intervening variable in the relationship between genes and language dominance is handedness. This is due in part to its moderate correlation with language dominance and its clear familial tendencies but also is a reflection of the inherent difficulties of studying language dominance *per se* within families. A genetic analysis of a characteristic is not possible unless chromosomal reassortment has occurred during meiosis, but such analysis necessarily entails a knowledge of the phenotypes of related individuals (parent/offspring or siblings); this is difficult to obtain. For instance, it is not easy to find cases of aphasia attributable to unilateral lesions for whom there is a relative with a similar lesion. As a result, phenotypic handedness, which is relatively easily and reliably assessible for whole families, acts as a surrogate or model of the far more interesting phenotype of language dominance. It is, therefore, a lateral step from most genetic models of handedness to a model of language dominance; usually only indirect (i.e., strictly nongenetic) evidence can be found for the genetic model of language dominance.

A consequence of the above is that much of the current chapter concentrates on handedness and will only eventually be extrapolated to language dominance and aphasia. Unless we have an adequate genetic model of the voluminous data available on handedness, genetic models of the sparse data on language dominance and aphasia probably are premature.

THE PHENOTYPE OF HANDEDNESS

The keystone of genetic analysis is an appropriate description of the phenotypes, as may be immediately realized by considering the problems that would have arisen in analyzing the genetic basis of the mental subnormality now known to be due to phenylketonuria, had the blonde hair of these children been taken as the primary phenotype for analysis.

In the case of handedness, two phenotypic approaches may be found in the literature. Early studies tended to take the stated handedness of individuals at face value, using simply the layman's criterion of the hand used for writing. The use of this description resulted in two awkward results for any simple genetic model: the incidence of left-handedness differed between studies (implying different allele frequencies in different populations), and the incidence differed within studies between parents and offspring (implying strong selective pressures, usually in favor of left-handedness). While neither implication is impossible, neither seemed to be reasonable.

A second approach originated in the doubts raised as to the utility of a simple dichotomization into right- and left-handers. Annett (1) explicitly suggested that a third category of mixed handers could be identified. It should be noted that these mixed handers were not the relatively rare ambidexters but rather a group of individuals who were neither pure right-handers nor pure left-handers; they represented some 32% of the population. Annett (2) developed this position, suggesting that handedness represented a continuum between pure right- and pure left-handedness, and that this continuum was seen in the distribution of skill asymmetries and in the distribution of grip strength asymmetry [a result based on Woo and Pearson's (31) analysis of Galton's anthropologic laboratory data of 1884].

According to Annett's (2) theory, it becomes impossible to classify individuals as being either right- or left-handed; rather, they are merely more or less right-handed. The incidence of apparent left-handedness in a population, therefore, depends on the particular threshold or criterion chosen, and the incidence thus can vary between studies, and can even vary within the same individuals in the same study if a different criterion is applied. Annett's approach solves the problem found in earlier studies of different incidences of sinistrality, but it subsequently fails itself, in that degree of handedness becomes an unmeasurable variable, manifesting only in the form of degree of skill asymmetry. Furthermore, it is necessary, for a number of reasons, to characterize the distribution as a mixture of several normal distributions (see, e.g., ref. 4). However, a statistical analysis of skill asymmetry data (21) finds that Annett's models of the skill distribution are inadequate as a description of actual data, and that the skill distributions are more compatible with a dichotomous description of the population (i.e., left- or right-hand writers), with a degree of variability introduced, due to either measurement errors or real individual differences, which give the superficial impression of a single continuous unimodal distribution.

My own interpretation of the phenotypic description of handedness follows the commonsense view in arguing that the clearest difference in skill between the hands

is in writing ability, the vast majority of individuals being unable to write at all well with their nondominant hand. That a few individuals are not as clearly lateralized as the rest should not mean that we dispose of the conceptual advantages of a simple dichotomy; to do so would be to reproduce the "black-is-white" fallacy of Flew (10). Laterality researchers have continually searched for the elusive grail of the ideal measure of handedness [in part, as Bishop (6) has suggested, in the hope that clinically useful correlates would then emerge]. Nevertheless, it remains true that, of all the tasks that have been studied using either questionnaires or actual performance of skilled tasks, asymmetry of writing ability shows the greatest relative difference between hands: the least incidence of "either" responses on questionnaires (in approximately 2,000 laterality inventories, no subject has reported that he could write equally well with either hand) and the best test-retest reliability of all inventory or skill measures. The distribution of writing ability, therefore, is grossly bimodal, with minimal overlap between the distributions. Nevertheless, despite these strong features in its favor as a measure of handedness, writing hand suffers from several difficulties, which may be circumvented by careful statistical analysis:

1. Some individuals have been forced by teachers or parents to change from writing with the left hand to writing with the right hand [a practice that has now almost totally ceased in the West, although reports of it still occur from Russia (30) and Albania (9)]. This problem may be partially assessed in surveys by asking individuals whether or not they have ever been forced to change writing hand through either social pressure or injury, and reclassifying the positive responders.
2. For various reasons, individuals may conceal their true handedness in questionnaire surveys; in the earlier years of this century, left-handedness represented a social stigma; hence its incidence was lower than might be expected. In recent years, left handedness has come to be associated with creativity and hence is socially desirable.
3. Left- or right-handers may respond at a differential rate in questionnaire surveys either for similar reasons to those given above or because, in general, left-handers as a group seem to be interested in why they are different from the rest of the population.

As a result of these problems, we must distinguish an individual's manifest writing hand (i.e., as apparent in a survey) and his true writing hand. In a population, we must distinguish the manifest incidence and the true incidence of left-handedness. By assuming, in the first instance at least, that all populations have the same true incidence of left-handedness, and that parents and offspring also have the same true incidence of left-handedness, we may fit genetic models of handedness. Whether or not the fit of models is improved by allowing a secular increase in the incidence of left handedness then becomes an empirically answerable question.

It should be apparent that many of the difficulties associated with the classification of writing hand could be avoided if writing ability were assessed by performance rather than by inventory. However, since genetic studies are of necessity large, and

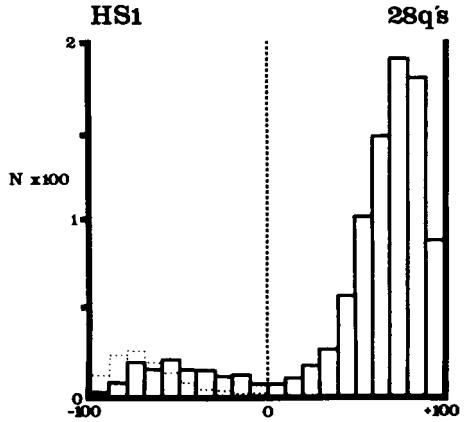

FIG. 1. Distribution of laterality scores of 948 undergraduates on a 28-item handedness inventory (15), each item being scored on a five-point scale. A score of −100 indicates that the subject responded "always left hand" on all items; a score of +100 indicates a response of "always right hand" on all items.

since genetically related individuals must be studied, this desirable feature is rarely attained.

This categorical approach to classification does not necessarily lack the desirable features of Annett's continuum of handedness. Figure 1 shows the responses of 948 subjects to a 28-item handedness inventory. Although the distribution is bimodal, within each mode there is a great amount of variability; indeed, it could be argued that there is a broad continuum across the whole distribution. It is important to note that the hand used for writing discriminates between the two modes almost perfectly (15), with almost no errors of classification. We may distinguish, therefore, between differences in direction of lateralization (right or left) and differences in degree of lateralization within each of the modes (Fig. 2); statistical methods for their differentiation in the case of overlapping distributions are described elsewhere (22). Since there is no correlation between the degree of handedness of parents and offspring (McManus, genetic model), and since degree of handedness within a direction group shows no relation to familial sinistrality (15), we may conclude that there is no inheritance of degree of handedness. In contrast, large numbers of studies have found strong familial trends in direction of handedness. It can be argued, therefore, that while Annett was right to stress that handedness is a continuum, that continuum is not itself homogenous. As a result, we may distinguish direction and degree of handedness. The former is dichotomous, has a familial component, and hence is of genetic interest, whereas degree of handedness is continuous, may represent either true individual differences or perhaps errors of measurement, and has no genetic component.

Finally, the distribution of Fig. 1 provides little support for the concept that the population contains a separate group who may be called ambidexters; if they do exist, they are sufficiently rare to mean they are unlikely to interfere with the genetic studies described.

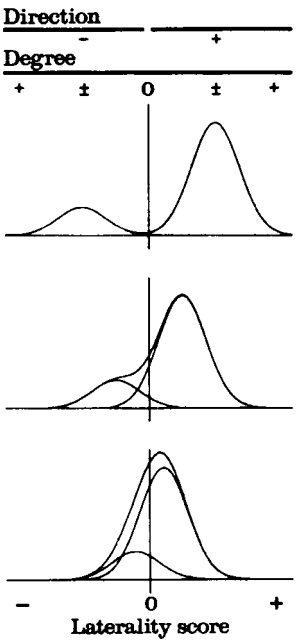

FIG. 2. The author's conception of the phenotypic description of handedness. Strongly lateralized scales (such as handedness inventories) show a bimodal distribution, in which the two modes are sufficiently far apart to mean that they hardly overlap at all (*b*). As a result, two independent measures, the direction of handedness (a categorical variable) and the degree of handedness (which is a continuous variable), may be derived (*a*). As the modes of the two distributions come closer together, it becomes more difficult to distinguish the two separate components in the overall distribution (*c* and *d*), such that *d* gives the superfical impression of being a single normal distribution.

A GENETIC MODEL OF HANDEDNESS

A genetic model of handedness has several biologic constraints, a more detailed account of which may be found elsewhere (19). In particular, the model must be compatible with the concept of fluctuating asymmetry, which says that the baseline for any lateralized biologic characteristic is a racemic mixture, in which 50% of a population are dominant on each side. Furthermore, any genetic model must be compatible with what I have called "Morgan's principle," which suggests that it is extremely unlikely that paired alleles can directly produce enantiomorphic phenotypes (25). Such a constraint rejects many of the earlier genetic models of handedness, such as those of Chamberlain (8), Rife (27), Ramaley (26), Trankell (28), and Annett (1). The detailed implications of the principle have been described by McManus and Mascie-Taylor (23). The only other genetic model compatible with Morgan's principle is Annett's second model (3), and I have argued elsewhere that it must be rejected on other grounds (21).

There are several problems with any genetic model of handedness:

1. Although almost all studies of handedness show significant familial trends, these are not as strong as might be expected under most simple Mendelian systems. Thus approximately 5% of the children of two right-handed parents are left-handed, and about 35% of the children of two left-handed parents are left-handed.

2. Studies of the handedness of twins show that while there is a slightly higher concordance for handedness in monozygotic (MZ) rather than in dizygotic (DZ)

twins, the overwhelming impression is of almost chance levels of concordance, for which conventional wisdom would suggest almost no genetic control of handedness. In passing, it should be noted that there is probably no clear evidence for twins having a higher incidence of sinistrality than singletons, or for the occurrence of the process known as mirror-imaging (for a review of handedness in twins, see ref. 16).

3. Many theorists have suggested that a relatively high proportion of left-handers may have become sinistral as a result of pathologic causes. Elsewhere, I have reviewed the historic basis and the evidence for the concept of pathologic left-handedness and have suggested that it has no empirical basis (20). In particular, I have found no evidence in two large studies for an association between left-handedness and birth trauma (18) or epilepsy (17). In fitting the genetic model, I have ignored such considerations, although it should be noted that the method of correction for differences in the incidence of left-handedness will generally take into account any such pathologic cases.

In this chapter, I present only the basic genetic model without providing the detailed justification for it, which may be found elsewhere (19). The model postulates that there are two alleles, D (for dextral) and C (for chance), which are at a single locus. Individuals who are of genotype DD are all right-handed [i.e., $p(L|DD) = 0$, a pure directional asymmetry]. Individuals who are of genotype CC exhibit pure fluctuating asymmetry; hence 50% of them are right-handed and 50% are left-handed [i.e., $p(L|CC) = 0.5$]. As such, the model is incompletely defined, since the phenotypic manifestation of the heterozygote, $p(L|DC)$, has not been given. This value cannot be defined *a priori* on theoretical grounds, however, and must be determined from the data. It is also necessary before fitting the model to know $p(C)$, the incidence of the C allele in the population [since this determines the true incidence of left-handedness, $p(L_t)$, which is assumed to be constant in all populations]. Neither $p(C)$ nor $p(L_t)$ can be determined in advance, and one or the other must be estimated from the data. Given particular values of $p(L_t)$ and $p(L|DC)$, any data set may be assessed for its fit to the model, a method of correcting for different manifest incidences of handedness having been described elsewhere (19). It is possible, therefore, by means of a computer, to assess the adequacy of fit of all data sets for all possible values of $p(L_t)$ and $p(L|DC)$ and hence to find the maximum likelihood estimates of those parameters. Such calculations suggest that $p(L|DC) = 0.25$ and $p(L_t) = 0.0775$. In other words, the genetic model is additive, rather than dominant or recessive, the heterozygous phenotype being midway between the two homozygotes. The true incidence of left-handedness is 7.75%; hence the frequency of the C allele is 0.155.

Table 1 summarizes the model which is a best fit to the data in the literature. Elsewhere (12), I have shown that this genetic model is a better fit to the available data than that of Rife (27) and Trankell (28) and is as good a fit as that of Annett (3), although that model fails on other grounds (21).

TABLE 1. *The proposed genetic model*[a]

Genotype	Phenotypes		Population frequency	
	Left	Right		
DD	0.0	1.0	0.714	Directional asymmetry
DC	0.25	0.75	0.262	Additivity of heterozygote
CC	0.5	0.5	0.024	Fluctuating asymmetry

[a] Alleles: D, dextral; C, chance. $p(L_t) = 0.0775$; $p(C) = 0.155$.

Consideration of Table 1 shows why left-handedness runs so poorly in families; even if two left-handers are both of the CC genotype, their offspring have only a 50% chance of being left-handed (although they are, as it were, breeding true for the phenotype of fluctuating asymmetry). Table 2 shows the expected distributions of left-handed offspring in families of one and two children according to the handedness of the two parents.

The relatively low concordance of MZ twins is more interesting. Consider the 2.4% of the population who are of genotype CC. Each has his handedness phenotype determined by a chance process *in utero*, resulting in 50% of individuals becoming left-handed. Exactly the same process is assumed to occur in MZ twins, except that the two chance processes are assumed to occur independently in the two twins. As a result, 25% of CC MZ twins are both right-handed, 25% are both left-handed, and 50% are discordant, one being right-handed and the other being left-handed. Similarly, 37.5% of MZ twin pairs of the DC genotype will be discordant for handedness (although all DD pairs will be concordant). The result, as shown in Table 3, is that for the population as a whole, the MZ concordance rate is only slightly higher than that in DZ twins.

To summarize thus far, it is suggested that the genetic model of Table 1 is capable of fitting all the available family and twin data on handedness, appropriate correction having been made for differences between studies in the manifest incidence of left-handedness.

TABLE 2. *Probability of parents of known handedness having particular combinations of right- or left-handed children*

Parents[a]	One child		Two children		
	R	L	RR	RL	LL
R × R	0.940	0.060	0.891	0.099	0.010
R × L	0.826	0.174	0.687	0.277	0.036
L × L	0.711	0.289	0.510	0.403	0.087

[a] R, right; L, left.

TABLE 3. *Predicted proportions of monozygotic (MZ) and diazygotic (DZ) twins of particular handedness combinations*[a]

Twins	R – R	R – L	L – L
MZ	0.867	0.110	0.022
DZ	0.859	0.127	0.014

[a] R, right; L, left.

A GENETIC MODEL OF LANGUAGE DOMINANCE AND APHASIA

A genetic model of language dominance must account for certain facts:

1. Tests of cerebral dominance by dichotic listening, unilateral electroconvulsive therapy (ECT), or intracarotid amytal suggest that between 8 and 20% of right-handers and 29 and 40% of left-handers show right-hemisphere dominance for language (7).
2. About 1 to 2% of right-handed aphasics and about 20 to 30% of left-handed aphasics have a right hemisphere lesion. The incidence of crossed dominance in right-handers, therefore, is estimated differently from clinical and laboratory data.
3. Left-handers are more likely to become aphasic after a cerebral lesion, are more likely to recover from that aphasia, and thus are less likely to suffer from aphasia (11,12).
4. Familial sinistrality seems to increase the likelihood of recovery from aphasia (12,32).

The following section outlines the extrapolation of the model of handedness to that of dominance and aphasia; once more, details of the statistical basis of the model fitting are omitted, and the interested reader is referred elsewhere (19).

Consider an individual of the CC genotype. During development, a random process determines whether or not he becomes right- or left-handed. If we allow the possibility in such individuals that a random process also determines whether or not they become right or left hemisphere dominant for language, and that the two random processes are statistically independent, then we would expect that 25% of CC individuals would be right-handed with right-language dominance, 25% right-handed with left language dominance, and so on.

Applying a similar process to individuals of DC and DD genotype (and of course all DD individuals must be right-handed and left-language dominant), then we can predict the incidence of right-hemisphere language dominance in right- and left-handers (Table 4). The incidence of right hemisphere dominance is compatible with that found in other studies, particularly after correction is made for differences in the manifest incidence of right dominance (due to nonspecificity or unreliability of the tests used). It is also clear from Table 4 that parental sinistrality relates to the likelihood of right hemisphere dominance, although the effect is far stronger in right-

TABLE 4. *Proportion of individuals who are expected to be right hemisphere dominant for language, as a function of handedness and parental handedness, under the simpler form of the model*

Parents[a]	Handedness	
	Right (%)	Left (%)
NK × NK	6.0	28.9
R × R	4.6	28.0
R × L	14.8	30.0
L × L	24.6	39.4

[a] NK, not known; R, right; L, left.

handers than left-handers. The only major problem with Table 4 is that the figure of 6.0% of right-handers showing crossed dominance is not compatible with the estimates of the incidence of crossed aphasia in the literature, which have an upper limit of about 2%.

Having allowed that two separate asymmetries may be independently controlled by the same gene, it is possible to extend the model in a purely formal manner by allowing three or more asymmetries to be determined by the same gene. Let there be two language dominances, which we may label LA and LB. We would then predict that one-eighth of CC individuals would be right-handed, with LA and LB both on the left, and so forth. At this point, it cannot be emphasized strongly enough that LA and LB do not correspond to Wernicke's and Broca's areas, or to any other established laterality system; they are merely a conceptual device for exploring the range of the model.

Considering LA and LB, it is clear that, since either may be right- or left-sided, three important groups of individuals can be distinguished: (a) LA and LB both left-sided, (b) LA and LB both right-sided, and (c) bilateral, in which case LA is on one side and LB on the other. If a lesion occurs in the right hemisphere, and if both LA and LB are right-sided, then the result will be an aphasia, since all linguistic capacity will have been removed. If LA and LB are both left sided, then a right-sided lesion will not cause any incapacity.

The interesting case arises if LA and LB are bilaterally distributed. A right-sided lesion will damage only one of the two language systems. Initially, we might expect this to cause aphasia or dysphasia, either because of the process of diaschisis or because in normal operation the two centers are integrated in their action and hence cannot function independently; the patient thus will be acutely aphasic. Since the patient still retains one language processing center, however, we may expect that in the long term he will recover functioning, using just his one remaining center. In the long run, the patient will not be permanently aphasic but will have recovered from his aphasia (Fig. 3).

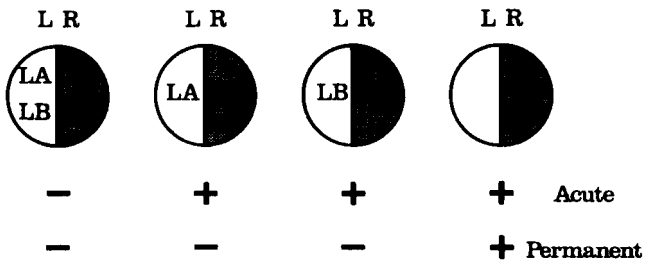

FIG. 3. Effects of a right-sided lesion on the four possible types of LA-LB organization, "+" indicating that a particular type of aphasia will occur and "−" indicating that it will not.

Under this extension of the model, it is necessary for a right hander to have LA and LB in the right hemisphere to suffer a permanent aphasia after a right-sided lesion. The usual estimates of the incidence of crossed aphasia are probably based on permanent aphasics, since these are the patients who are most likely to present for clinical psychologic assessment, and since studies of acute aphasia show a higher incidence of crossed aphasia than is usually reported.

Table 5 shows the expected incidence of the language dominance types by handedness. Table 6 shows the expected proportions of right- and left-handed aphasics who would be expected to have right-sided lesions, according to their parental handedness. It is clear that the incidence of permanent dextral crossed aphasia (1.8%) is now compatible with clinical data. It should also be noted from Table 6 that a parental history of sinistrality makes it more likely that a right-sided lesion has occurred. It might be thought at this point that the LA-LB model is incompatible with the simpler model described earlier (see Table 4), but this is not the case; if dominance as assessed by, e.g., dichotic tests is more dependent on one of LA and LB than on the other, then the LA-LB model reduces to the simpler model described above.

Table 7 shows the percentage of right- and left-handers who would be expected to be aphasic after a right- or left-sided lesion, or after a lesion on either side. Left-handers are more likely to be acutely aphasic after a cerebral lesion than are right-handers but are less likely to be permanently aphasic. Table 8 summarizes the probability of an acutely aphasic individual recovering language after a right- or left-sided lesion. Left-handers are more likely to recover than are right-handers, and the majority of this effect is with left-sided lesions. Parental handedness has an important

TABLE 5. *Expected proportions of right- and left-handers who would be expected to show right, left, or bilateral location of the LA and LB centers*

Handedness	Both left (%)	Bilateral (%)	Both right (%)
Right	89.7	8.6	1.7
Left	51.4	39.4	9.2

TABLE 6. *Expected proportion of aphasics who would have right-sided lesions by handedness and parental handedness*

Parental handedness[a]	Right-handers (%)		Left-handers (%)	
	Acute aphasia	Permanent aphasia	Acute aphasia	Permanent aphasia
R × R	7.4	1.3	34.2	13.9
R × L	20.9	5.3	35.7	16.6
L × L	30.6	13.2	42.7	30.9
NK × NK	9.5	1.8	34.8	15.1

[a] R, right; L, left; NK, now known.

influence on the likelihood of recovery, particularly in right-handers with left-sided lesions. A surprising prediction of the model is that a history of parental sinistrality makes recovery less likely after right-sided lesions, although the effect is small and primarily confined to those with two left-handed parents.

The above model has been fitted to a series of data sets from the literature and has generally been found to be compatible (see ref. 19). An interesting exception is that in fitting two series of data on aphasia after neurosurgical operation in patients with chronic epilepsy or temporal lobe glioma, it was found that the permanent model fitted the data better than the acute model, as was also the case in a series of non-operated patients with gliomata or meningiomata. It was argued that in both cases, the continual, chronic damage, be it recurrent ictal attacks or a slowly expanding tumor, had resulted in the abolition of diaschisis and the development of functional autonomy in the centers in the two hemispheres.

Once it has been suggested that several independent lateralized systems may be controlled by the same genetic mechanism, then the explanatory power of the model is rapidly increased; indeed, it rapidly exceeds that of the data available for testing it. Two possible cases can be briefly discussed. If the lateralization of visuospatial functions is also controlled by the same genetic locus as handedness and language dominance, then we might expect cases in which language and visuospatial ability are both in the same hemisphere. Such cases have been previously suggested, on

TABLE 7. *Expected incidence of aphasia after a unilateral lesion in right- and left-handers*

Lesion	Right-handers (%)		Left-handers (%)	
	Acute aphasia	Permanent aphasia	Acute aphasia	Permanent aphasia
Left	98.3	89.7	90.8	51.4
Right	10.3	1.7	48.6	9.2
All	54.3	45.7	69.7	30.3

TABLE 8. *Expected proportion of acutely aphasic individuals who would be expected to recover, as a function of handedness, lesion side, and parental handedness*

Parental handedness[a]	Right-handers (%)		Left-handers (%)	
	Left lesion	Right lesion	Left lesion	Right lesion
R × R	6.7	84.3	42.6	82.1
R × L	22.1	83.4	44.4	80.1
L × L	34.1	77.4	53.9	72.4
NK × NK	8.8	83.9	43.4	81.2

[a] R, right; L, left; NK, not known.

the basis of both tachistoscopic studies (14) and clinical cases (13). The number of such individuals is compatible with the predictions of the model. A more speculative possibility is that reading or writing skills may be lateralized independently, so that in some patients, a unilateral lesion will result in alexia without agraphia. As an example taken at random from the recent literature, consider the two cases described by Sevush et al. (29) of Wernicke's aphasia with intact oral reading after a left-sided lesion. The authors correctly point out the problems of interpreting these cases in conventional terms. If we suggest that some but not all of the typical processing of the left hemisphere is located in the right hemisphere in these individuals, however, then their defects become comprehensible.

Two other implications of the model are important for clinical and experimental purposes. First, if the present model is correct, then more than 10% of right-handed patients might have some linguistic ability in the right hemisphere, an incidence that is sufficiently high to make the interpretation of data from individual hemispherectomy, callosal section, or stroke patients difficult. Second, the present model allows for a certain rate of recovery from aphasia. No cognisance is taken in the model of the possible benefits of speech therapy, and it could be argued that the effects of therapy are primarily to speed recovery in those who would have recovered, rather than to change individuals from nonrecoverers to recoverers.

The model may also be extended to other areas in which laterality has been implicated. It is repeatedly claimed, both in stutterers and in those suffering from developmental dyslexia, that left-handedness is more common than in the general population [although McManus and Mascie-Taylor (24) failed to demonstrate any relationship between eye-hand dominance and a specific reading deficit, and Bishop (6) has found similar difficulties]. There are also suggestions that abnormal laterality is implicated in psychoses, particularly schizophrenia. If the present model is correct, then an implication is that all atypical forms of lateral organization (i.e., in which some or a majority of centers are not on their typical sides) should be more common in left-handers. If disorders such as dyslexia or stuttering involve failures of integration between, e.g., visuospatial and linguistic systems, because they are atypically arranged in the hemispheres, then the condition should be associated with sinistrality.

An atypical disposition of lateralized centers might require greater interhemispheric connections to produce functional integration and hence may explain the increased corpus callosum thickness that has been reported in schizophrenia (5).

CONCLUSIONS

This chapter briefly presents a genetic model that explains many of the features associated with the inheritance of handedness and of the association between handedness and language dominance and aphasia. Its major theoretical advantages are its conceptual simplicity (two alleles at a single locus and additivity) and its ability to be extended by the single gene controlling a number of independent lateralities. The resultant complexity is compatible with the complexity of the clinical phenomena. The major limitation of the model is a lack of adequate data for subjecting it to a rigorous test of its predictions.

REFERENCES

1. Annett, M. (1964): A model of the inheritance of handedness and cerebral dominance. *Nature*, 204:59–60.
2. Annett, M. (1972): The distribution of manual asymmetry. *Br. J. Psychol.*, 63:343–358.
3. Annett, M. (1978): *A Single Gene Explanation of Right and Left Handedness and Brainedness*. Lanchester Polytechnic, Coventry.
4. Annett, M., and Kilshaw, D. (1983): Right and left-hand skill. I: Estimating the parameters of the distribution of L-R differences in males and females. *Br. J. Psychol.*, 74:269–283.
5. Bigelow, L. B., Nasrallah, H. A., and Rauscher, F. P. (1983): Corpus callosum thickness in chronic schizophrenia. *Br. J. Psychiatry*, 142:284–287.
6. Bishop, D. V. M. (1983): How sinister is sinistrality? *J. R. Coll. Physicians Lond.*, 17:161–172.
7. Bradshaw, J. L., and Nettelton, N. C. (1983): *Human Cerebral Asymmetry*. Prentice Hall, New Jersey.
8. Chamberlain, H. D. (1928): The inheritance of left-handedness. *J. Hered.*, 19:557–559.
9. Dobbs, M. (1980): *The Sunday Times (London)*. November 9, p. 9.
10. Flew, A. (1975): *Thinking about Thinking*. Fontana, London.
11. Hécaen, H., and Albert, M. L. (1978): *Human Neuropsychology*. Wiley, New York.
12. Hicks, R. E., and Kinsbourne, M. (1978): Human handedness. In: *Asymmetrical Function of the Brain*, edited by M. Kinsbourne, pp. 523–549. Cambridge University Press, Cambridge.
13. Kreindler, A., Fradis, A., and Sevastopol, N. (1966): La réparation des dominance hémisphériques. *Neuropsychologia*, 4:143–149.
14. McGlone, J., and Davidson, W. (1973): The relationships between cerebral speech laterality and spatial ability with special reference to sex and hand preference. *Neuropsychologia*, 11:105–113.
15. McManus, I. C. (1979): *Determinants of Laterality in Man*. Doctoral dissertation, University of Cambridge.
16. McManus, I. C. (1980): Handedness in twins: A critical review. *Neuropsychologia*, 18:347–355.
17. McManus, I. C. (1980): Left-handedness and epilepsy. *Cortex*, 16:487–492.
18. McManus, I. C. (1981): Handedness and birth stress. *Psychol. Med.*, 11:485–496.
19. McManus, I. C. (1983): Handedness, language dominance and aphasia: A genetic model. *Psychol. Med.* [*Monogr. Suppl.*] (*in press*).
20. McManus, I. C. (1983): Pathologic left-handedness: Does it exist? *J. Commun. Disord.*, 16:315–344.
21. McManus, I. C. (1983): Right and left-hand skill: Failure of the right-shift model. *Br. J. Psychol.* (*in press*).
22. McManus, I. C. (1983): The interpretation of laterality. *Cortex*, 19:187–214.
23. McManus, I. C., and Mascie-Taylor, C. G. N. (1979): Hand-clasping and arm-folding: A review and a genetic model. *Ann. Hum. Biol.*, 6:527–558.
24. McManus, I. C., and Mascie-Taylor, C. G. N. (1983): Biosocial correlates of cognitive abilities. *J. Biosoc. Sci.*, 15:289–306.

25. Morgan, M. J. (1976): Embryology and inheritance of asymmetry. In: *Lateralisation in the Nervous System*, edited by S. R. Harnad, R. W. Doty, J. Jaynes, and G. Krauthamer, pp. 173–194. Academic Press, New York.
26. Ramaley, F. (1913): Inheritance of left-handedness. *Am. Naturalist*, 47:730–738.
27. Rife, D. C. (1950): Application of gene-frequency analysis to the interpretation of data from twins. *Hum. Biol.* 22:136–145.
28. Trankell, A. (1955): Aspects of genetics in psychology. *Am. J. Hum. Genet.*, 47:730–738.
29. Sevush, S., Roeltgen, D. P., Campanella, D. J., and Heilman, K. M. (1983): Preserved oral reading in Wernicke's aphasia. *Neurology*, 33:916–920.
30. van der Post, L. (1965): *Journey into Russia*. Penguin Books, Harmondsworth.
31. Woo, T. L., and Pearson, K. (1927): Dextrality and sinistrality of hand and eye. *Biometrika*, 19:165–199.
32. Zangwill, O. L. (1960): *Cerebral Dominance and its Relation to Psychological Function*. Oliver and Boyd, London.

Advances in Neurology

Volume 42

Progress in Aphasiology

Editor

F. Clifford Rose, F.R.C.P.

Physician in Charge
Department of Neurology
Charing Cross Hospital
London, England

Raven Press ■ New York

Raven Press, 1140 Avenue of the Americas, New York, New York 10036

Made in the United States of America

Library of Congress Cataloging in Publication Data
Main entry under title:

Progress in aphasiology.

(Advances in neurology; v. 42)
"Based on a Mansell Bequest Symposium of the Medical Society of London held in the summer of 1983"—Pref.
Includes bibliographies and index.
1. Aphasia—Congresses. I. Rose, F. Clifford (Frank Clifford) II. Mansell Bequest Symposium (1983 : London, England) III. Medical Society of London. IV. Series. [DNLM: 1. Aphasia—congress. W1 AD684H v. 42 / WL 340.5 P964 1983]
RC321.A276 vol. 42 616.8 s [616.85'52] 84-15979 [RC425]
ISBN 0-88167-023-5

The material contained in this volume was submitted as previously unpublished material, except in the instances in which credit has been given to the source from which some of the illustrative material was derived

Great care has been taken to maintain the accuracy of the information contained in the volume. However, Raven Press cannot be held responsible for errors or for any consequences arising from the use of the information contained herein.