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Genetics of Handedness in Relation to Language Disorder

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. . . 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

is in writing ability, the vast majority of individuals being unable to write at all well

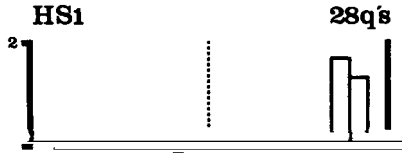


FIG. 1 Distribution of laterality

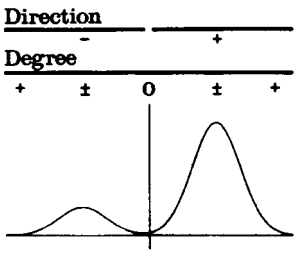
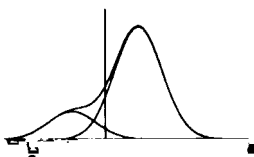


FIG. 2. The author's conception of the phenotypic description of handedness. Strongly lateralized scales (such as handedness inventories) show a bimodal distribution, in which the two modes are sufficiently far apart to mean that



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

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

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

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

handers and 29 and 40% of left-handers show right-hemisphere dominance for

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.

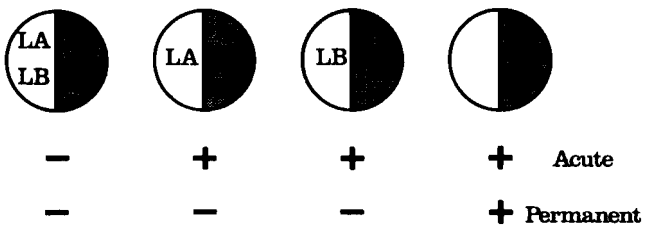


FIG. 3. Effects of a right-sided lesion on the four possible types of LA-LB organization,

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

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

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

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