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Hand-clasping and arm-folding: A review and a genetic model

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Summary. The literature on hand-clasping is reviewed and it is shown to be suitable for genetic analysis, there being no classification problems, sex differences, assortative mating or maternal inheritance. Further data from two studies of our own are reported.

Two-allele genetic models are discussed and shown to be inadequate at explaining the data. A simple three-allele model will, however, fit all the available data adequately, and will also account for the east–west cline in left hand-clasping. The limited data on arm-folding were also consistent with the three-allele model. Hand-clasping, arm-folding and handedness were shown not to be correlated one with another nor to show simple genetic linkage.

The genetic model is discussed in relation to the problem of asymmetry as a whole.

1. Introduction

Genetic models for human asymmetries are controversial. In the particular case of handedness, perhaps the most important asymmetry due to its intimate association with the specialization of the brain for language, there have been proponents of purely genetic models (e.g. Levy and Nagylaki 1972, Corballis and Beale 1976), and purely environmental models (Collins 1970). Handedness has been analysed in some detail elsewhere by one of us and thus will not be considered in this paper, it having been shown to be compatible with a simple genetic model (McManus 1978). In this paper we review and analyse similar data for two other asymmetries, hand-clasping and arm-folding, and conclude that both are comprehensible in terms of a simple genetic model.

2. Types of model of asymmetry inheritance

In asymmetric systems there are usually two phenotypes, right and left, R and L. These are mirror-images, or enantiomorphs. The assumption made in almost all previous models of handedness in particular, and asymmetries in general, has been that enantiomorphic phenotypes must be the product of alleles whose action is symmetric: we may name such alleles as *D* and *S*, Dextral and Sinistral. Morgan (1976) has challenged this view and suggested that there is not a single acceptable case in the literature in which enantiomorphic phenotypes can be shown to be derived from symmetric alleles. This forced him to conclude that genes *per se* were not of crucial importance in the inheritance of asymmetry, genes being 'left–right agnosic', and that instead some form of cytoplasmic inheritance was of importance. A further logical possibility, explored by one of us in the case of handedness (McManus 1978), is that enantiomorphic phenotypes may be produced by alleles whose actions are asymmetric. Specifically it was proposed that an allele *D* produced 100% right-handers in its homozygous form, while the other allele, *C*, for Chance, in its homozygous form

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produced exactly 50% left-handers and 50% right-handers (i.e. a racemic mixture). There are good biological precedents for proposing such a *C* allele (see McManus 1978).

Annett (1974) has already proposed such a model, although there are differences in detail between her model and the type being discussed here. The distinction between *D* and *C* alleles is equivalent to that proposed elsewhere between directional and fluctuating asymmetry (van Valen 1962).

Considering just two alleles, *D* and *C*, we may then consider a general asymmetric model (of the type proposed for handedness):

Model I	Genotype	Proportion of type L	Proportion of type R
	DD	$0{\cdot}5-a$	$0{\cdot}5+a$
	DC	$0{\cdot}5-ax$	$0{\cdot}5+ax$
	CC	0·5	0·5

a controls the rate of manifestation in *DD* genotypes, *x* controls the degree of dominance in the heterozygote. Clearly *a* has an upper limit of 0·5 (in which case *DD* produces 100% type R, and a lower limit of 0·0 (in which case there is no detectable inheritance of the asymmetry). The value of *x* can vary from 0·0 (in which case both *CC* and *DC* genotypes produce equal proportions of L and R) to 1·0 (where type R would show some degree of dominance). Handedness can be shown to fit a model in which $a=0{\cdot}5$, and $x=0{\cdot}5$ (i.e. an intermediate model, using the terminology of Wilson (1971 a, b)).

Models such as this one (type I) have very strong predictions and limitations. Let $p(\mathrm{L})$ be the proportion of type L in the population as a whole. Clearly $p(\mathrm{L})$ cannot be greater than 0·5 or less than $0{\cdot}5-a$. In particular, $p(\mathrm{L}|\mathrm{L}\times\mathrm{L})$, the proportion of type L progeny from a mating of two left-type parents, cannot exceed 0·5.

On grounds of symmetry it is also possible to conceive of a model which is the complement of that just described, viz:

Model II	Genotype	Proportion of type L	Proportion of type R
	SS	$0{\cdot}5+b$	$0{\cdot}5-b$
	SC	$0{\cdot}5+by$	$0{\cdot}5-by$
	CC	0·5	0·5

Using the nomenclature of Model I, *b* represents the rate of manifestation of the *SS* genotype and *y* represents the degree of dominance in the heterozygote. Note that in model II $p(\mathrm{L})$ must be *greater* than 0·5, and less than $0{\cdot}5+b$, for all possible sub-populations.

Considering models I and II together, it may be seen that, if for any sub-population $p(\mathrm{L})$ is greater than 0·5, then for all sub-populations $p(\mathrm{L})\geqslant 0{\cdot}5$ and vice-versa for $p(\mathrm{L})<0{\cdot}5$. This is a very strong constraint and provides a useful method for determining whether a model of type I or II will adequately explain a particular asymmetry. For the particular case of handedness, no single sub-population selected on grounds other than of its asymmetry *per se* has been shown to have a $p(\mathrm{L})$ greater than 0·5: the discovery of any such population would completely disprove the validity of a model of type I for handedness.

From a consideration of models I and II, it will be obvious that there is a third type of two-allele model: this is the case which Morgan (1976) has claimed has never been shown to exist, and which on theoretical grounds would seem to be highly unlikely, the alleles being symmetric in their effects. It will be presented here in its general form, the intention being to show on empirical grounds that it is indeed unsatisfactory for explaining the family data on hand-clasping and arm-folding.

Model III	Genotype	Proportion of type L	Proportion of type R
	DD	$0{\cdot}5-c$	$0{\cdot}5+c$
	DS	$0{\cdot}5+cz$	$0{\cdot}5-cz$
	SS	$0{\cdot}5+c$	$0{\cdot}5-c$

c is formally equivalent to a and b in Models I and II: z represents the dominance and takes values in the range -1 to $+1$. In this model p(L) can take any value in the range $0{\cdot}5+c$ to $0{\cdot}5-c$: the values of p(L) in sub-populations may thus 'straddle' the value of 0·5, an important distinction from models I and II.

Model III, as stated above, is completely symmetric. Clearly it is possible to have an extension of this model in which the penetrance in the genotypes *DD* and *SS* is not identical: this type of 'asymmetric–symmetric' model will not be considered in this paper, there being no evidence for even its possible existence. It will be briefly considered as a special sub-set of model IV (below).

For the rest of this paper, 'two-allele asymmetric model' will refer to models I and II, while 'two-allele symmetric model', will refer to model III.

Although thus far only two-allele models have been described, three alleles have been invoked, *D*, *C*, and *S*. It is conceivable that all three alleles exist. One may produce a general model of the form:

Model IV	Genotype	Proportion of type L	Proportion of type R
	DD	$0{\cdot}5-a$	$0{\cdot}5+a$
	SS	$0{\cdot}5+b$	$0{\cdot}5-b$
	CC	0·5	0·5
	DC	$0{\cdot}5-ax$	$0{\cdot}5+ax$
	SC	$0{\cdot}5+by$	$0{\cdot}5-by$
	DS	$0{\cdot}5-a+z(a+b)$	$0{\cdot}5+a-z(a+b)$

z represents the degree of dominance of *D* over *S*. By fixing the frequency of allele *S* at zero, this model becomes model I; by fixing allele *D* frequency at zero, it becomes model II; and by fixing allele *C* frequency at zero and making $a=b(\equiv c$ of model III), this becomes equivalent to model III. While this three-allele model is very general, it has several problems, one of which is that five independent parameters have to be estimated, i.e. a, b, x, y and z. Furthermore there are three alleles and yet only two phenotypes, and thus knowledge of p(L) alone is not sufficient to give the allele frequencies—one of them has to be independently estimated. It is thus probable that this model will be able to fit almost any data in view of the large number of free variables. In the first instance therefore, only one very restricted version of this model has been considered. Following on from the evidence that in the case of handedness the mode of inheritance fits a partially dominant model, the three-allele model has been

considered in which all the homozygotes show complete penetrance, and the heterozygotes always manifest mid-way between their respective homozygotes. This highly specific model is therefore:

Genotype	%L	%R
DD	0	100
CC	50	50
SS	100	0
DC	25	75
DS	50	50
SC	75	25

This model as such has no undefined parameters. There are, however, still only two phenotypes but three alleles, and thus one allele frequency has to be estimated. If one considers not just p(L) but the progeny of the three types of mating pair, then this degree of freedom can be removed, and for any particular population the optimal single set of values for allele frequencies may be estimated. This process will be discussed later. For the rest of this paper, this highly restricted version of Model IV will will be known as the 'three-allele model'.

3. Hand-clasping

Definition

When a person is asked to clasp his hands together, with the fingers interlocking, it is inevitable that one of the thumbs must be on top of the other. For any individual the particular thumb on top is constant, 'Right Hand-claspers' (RHC) having the right thumb on top. The only long-term follow-up of individuals in order to assess test–retest reliability was by Wiener (1932): in a limited sample of 22 he found 100% agreement after an interval of 18 months.

Although most easily measured by actual inspection, data from a survey by one of us (ICM), among graduates of the University of Cambridge, suggest that hand-clasping may accurately be assessed by questionnaire. The question was, "When you clasp your hands together with the fingers interlaced, which thumb is on the top? Right Left". Of 511 students only 5 (0·98%) did not answer this question satisfactorily. The incidence of left hand-clasping (LHC), as well as the family data so obtained, was statistically indistinguishable from that obtained by actual inspection of a group of persons living on a housing estate in Cambridge (CGNM-T); other aspects of the survey by CGNM-T have been considered elsewhere (Mascie-Taylor and Gibson 1978, 1979, Mascie-Taylor 1979, 1980).

Population incidence

Estimates of p(L) have been made for a total of 53 populations. These give a mean value of 43·85% (SE 1·09%), the distribution being shown in figure 1. It is clear that there are several populations in which p(L) is significantly greater than 0·50; Falk and Ayala's (1971) study of 'Caucasians' in New York, "more than 90% (of whom) are descendants of immigrants from Ireland, Italy and Portugal"; Ferronato, Thomas and Sadava's (1974) study of Californians; and our own studies in the United Kingdom, one of which (CGNM-T) was conducted among members of a Cambridge suburb (p(L) = 57·6%, n = 587) and the other of which (ICM) was conducted among graduates of the University of Cambridge, and their parents (p(L) = 56·32%, n = 1453). These data together

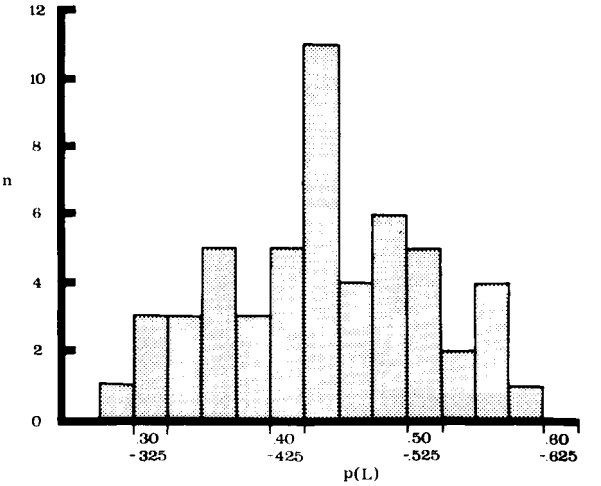

Figure 1. The incidence of left hand-clasping in 53 separate studies of different populations

completely exclude the possibility of a two-allele asymmetric genetic model (Mo and II).

One of the studies seems to give a value of p(L) which is strangely at variance the other studies: this is the study of Thessalonikan schoolchildren by Pelecanos (He found a p(L) of 18·7%, very different from the value of 47·6% found in Pat himself only a few years later (Pelecanos, Zacharopoulou and Yannopoulos 197 Thessalonikan data are difficult to explain and need to be independently repl before being treated seriously.

Figure 2 shows a map of the world on which are placed values of p(L) for di populations. Migrant populations are placed at their site of origin; thus emigrants to Brazil are placed in Holland, and so on. Americans are placed in A only if insufficient information is given to place them elsewhere. Incidences are in brackets if the original site cannot be adequately determined. It is apparent th is a cline extending across the whole of Eurasia and Australasia, from the Kingdom in the West (p(L) = 57·4%) to the Solomon Islands in the East (p(L) = Figure 3 shows the individual data points for Eurasia/Australasia plotted degrees of longitude east of Greenwich. The correlation is highly sig ($r = -0{\cdot}689$, $n = 40$, $p < 10^{-6}$). p(L) in migrants is closer to their sedentes t indigenous populations, suggesting that the cline might be due to genetic diffe Thus White Australians have p(L) values similar to those of Europe, and very d to those of the Aboriginal Australians.

Generational differences

Thirteen studies, including our own, present data which allows comparison incidence of left hand-clasping (LHC) in different generations, either parent–chi or a transverse study. In four studies the difference is significant with $p < 0{\cdot}05$, one study with $p < 0{\cdot}10$. In all these cases the younger individuals have the highe of p(L). The data points are shown in figure 4. While not conclusive, these data su secular trend towards a greater incidence of p(L).

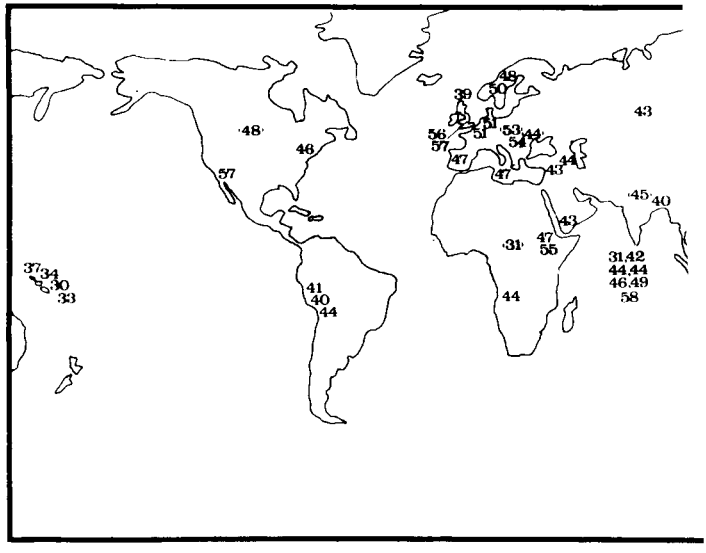

Figure 2. A map of the world on which are placed the individual incidences of left hand clasping in various populations. Values are expressed as percentages. All points are plotted as of the present day, rather than as of home, migrants being plotted as of their sedentes. In a few cases this was not possible for American studies and they have been plotted at their present homes. Data-points plotted in small brackets are those which cannot be accurately placed due to a poor description in the original papers. 'WA' refers to white Australians who could not more adequately be plotted elsewhere. The profusion of Indian studies has been plotted in the ocean beneath their country, due to lack of space.

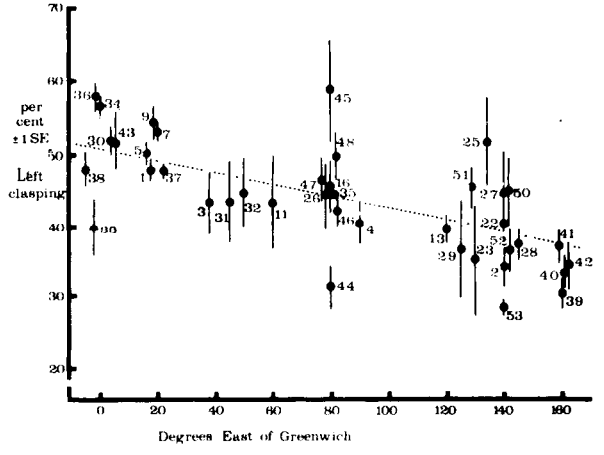

Figure 3. The incidence of left hand-clasping as a function of the degrees east of Greenwich, for all Eurasian and Australasian studies. Data points are plotted ± 1 standard error. Numbers alongside each point refer to the study from which it was taken (see Appendix A for key).

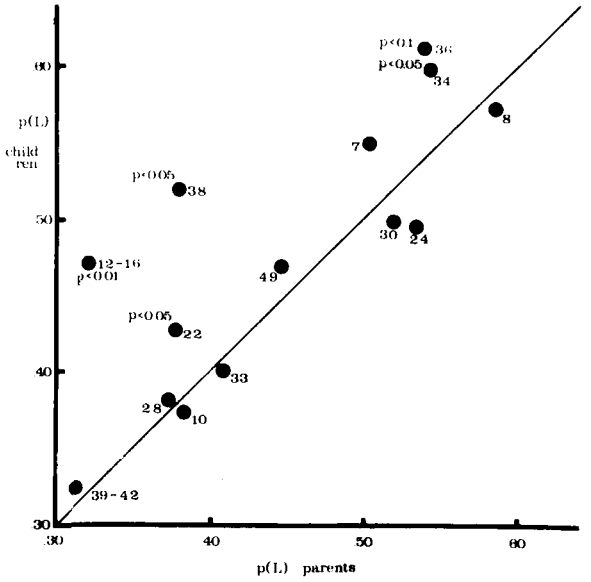

Figure 4. The incidence of left hand-clasping in children as a function of the incidence in parents, for 14 studies. Numbers alongside each point indicate the source (see Appendix A for key). Significance values are placed alongside points in which the incidence in parents and children differs with a p less than 0·10. For some of the studies, the incidences are not those in related individuals, but in generations of different age.

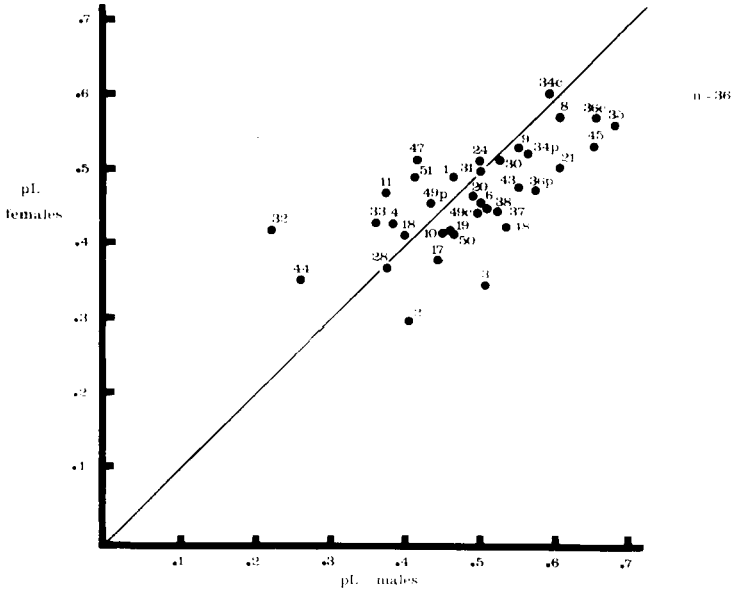

Figure 5. The incidence of left hand-clasping in females as a function of the incidence in males, for 36 separate populations. Numbers alongside data points indicate their source (see Appendix A for key); a suffix 'p' or 'c' indicates parental or child generation respectively. The diagonal line represents the line of equality for male and female incidences.

Sex differences

Many studies have suggested that there are sex differences in the incidence of LHC; however, others have failed to find such differences, or have found them in the reverse direction. Figure 5 summarizes the findings of studies on 36 populations. In only one study (Lourie (1972)—Kurds) is the incidence exactly the same in the two sexes. Of the

Table 1. Shows, for hand-clasping studies, the number of populations in which males have a higher incidence of LHC, by the population incidence of LHC (greater or less than 50%).

		Population incidence of LHC		
		<0·50	>0·50	
Sex differences in LHC	Male > Female	11	12	23
	Female > Male	3	9	12
		14	21	35

$\chi^2 = 0{\cdot}89$, 1 df, NS.

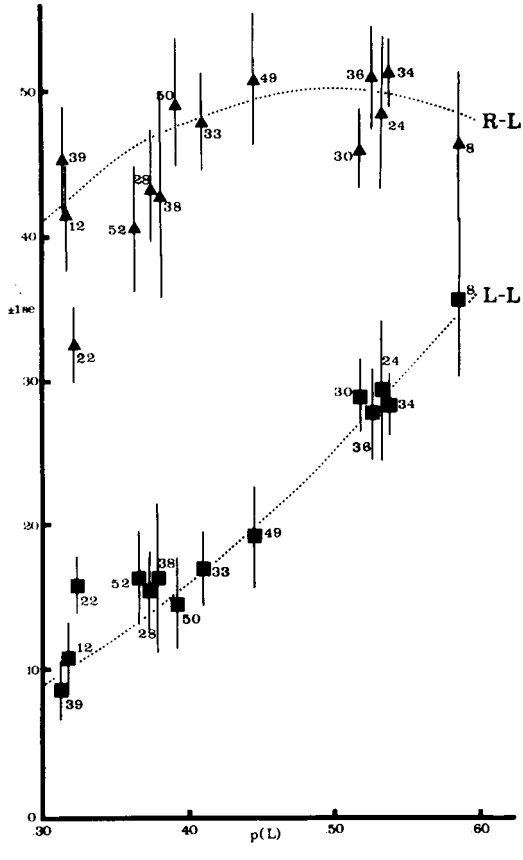

Figure 6. Data from fourteen studies on the patterns of parental mating, with respect to hand-clasping type. For clarity only R–L and L–L pairs are shown. Dotted lines indicate the expected incidences under a chance (binomial) hypothesis. Numbers alongside each point indicate the source (see Appendix A for key). Data points are plotted ±1 standard error. (■) L–L pairs; (▲) R–L pairs.

other studies, 12 have found females to have a higher incidence of LHC, while 24 have found males to have a higher incidence of LHC: this difference is not significant ($\chi^2 = 2{\cdot}85$, 1 df, NS). Neither is there a tendency for either sex to show more extreme lateralization of hand-clasping (i.e. to be further from the chance expectation of 50%) (see table 1, $\chi^2 = 0{\cdot}45$, 1 df, NS).

Mating patterns

Figure 6 summarizes data on mating patterns for hand-clasping. There is no evidence for assortative mating, proportions being accounted for almost entirely by binomial (chance) expectations. The data of Kawabe (1949) are slightly discrepant, but otherwise the fit of the binomial is adequate.

Familial data

Figure 7 shows that there is no evidence for a difference in the proportion of left hand-claspers among the progeny of L × R matings or of R × L matings. In conjunction with the earlier demonstration of a lack of difference of incidence of LHC in the two sexes, we may probably conclude that whatever the mode of inheritance of hand-clasping, it is unlikely to be sex-linked. For the rest of this paper R × L and L × R groups will be concatenated.

In the literature there are fourteen studies in incidence of LHC in the progeny of various mating types; to these may be added our own data, shown in table 2. If all these data are plotted as a function of p(L), then it becomes readily apparent that the 16 samples are not homogeneous; the four Japanese (J) studies show marked differences

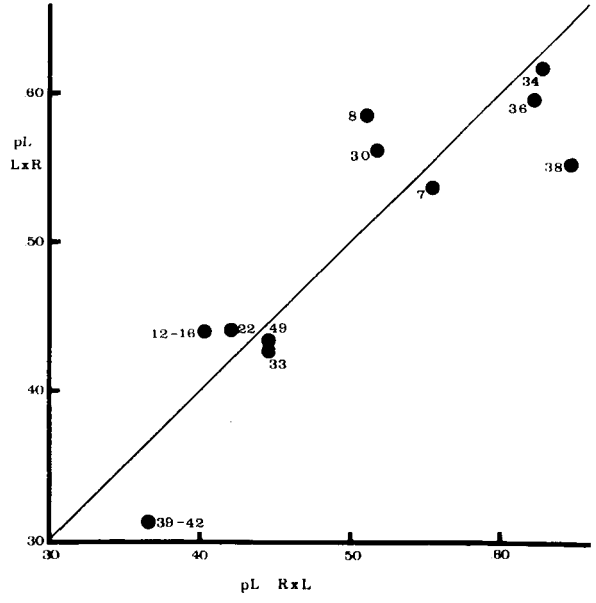

Figure 7. The incidence of left hand-clasping in the progeny of L × R matings as a function of the incidence of left hand-clasping in the progeny of R × L matings, for 11 studies. Numbers alongside data points indicate source (see Appendix A for key). None of the points are significantly different from equality, the line of equality being drawn as solid.

Table 2. Family tables for hand-clasping, by sex.

			Progeny			
			Right HC		Left HC	
	Father	Mother	Male	Female	Male	Female
(i) *ICM*	Right	Right	32	11	31	16
	Right	Left	27	11	44	20
	Left	Right	30	16	56	18
	Left	Left	28	22	54	24
(ii) *CGNM-T*	Right	Right	14	16	16	15
	Right	Left	10	13	21	17
	Left	Right	19	19	28	28
	Left	Left	8	18	31	28

For the ICM study only one child was analysed per parental pair, while for CGNM-T all children were included who were old enough for assessment.

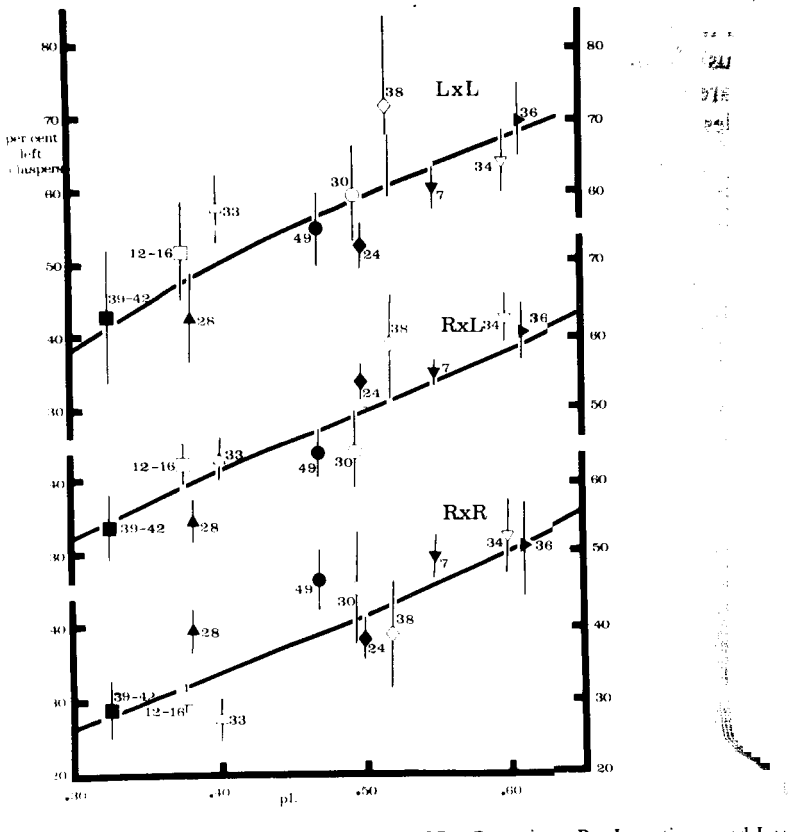

Figure 8. The incidence of left hand-clasping in the progeny of R × R matings, R × L matings, and L × L matings, for twelve non-Japanese studies. Numbers alongside data points indicate source (see Appendix A for key). Points are plotted ± 1 standard error. In order to avoid confusing overlap of standard error bars the data for R × L and L × L progeny have been displaced upwards by a scale value of 20%. Solid lines indicate predicted values using a two-allele symmetric model (see text for details). For a clearer perspective of the true relationship between R × R, R × L and L × L, see figure 9.

relative to the non-Japanese (NJ) studies. In figure 8 are shown the 12 non-Japanese studies: these show a regular relation between the incidence of progeny LHC in R × R, R × L, and L × L matings and the overall incidence of LHC. Figure 9 shows the four Japanese studies and fitted lines from the non-Japanese studies; clearly there is no overlap between the Japanese and non-Japanese groups.

The distinction between J and NJ studies is of great interest. Consider its implications for a two-allele symmetric model. Given values for c and z, then for any value of $p(\mathrm{L})$ the allele frequencies are completely defined. The model may then be used to predict the frequency of LHC in the three types of mating. A computer programme has been used to systematically vary possible values of c and z and then to fit the resulting values of $p(\mathrm{L}|\mathrm{R}\times\mathrm{R})$, $p(\mathrm{L}|\mathrm{R}\times\mathrm{L})$ and $p(\mathrm{L}|\mathrm{L}\times\mathrm{L})$ against empirically derived data, using a χ^2 test. There is no single pair of values for c and z for which an adequate fit may be found for all 16 data sets, i.e. J and NJ together. Specifically, for all values of $0{\cdot}5 \geqslant c \geqslant -0{\cdot}5, 0 \geqslant z \geqslant 1$, the minimum χ^2 value is 131·61, which is different from chance with a probability $<0{\cdot}001$; indeed, for a completely general two-allele model (i.e. $0 \geqslant p(\mathrm{L}|\mathrm{DD}) \geqslant 1{\cdot}0, 0 \geqslant p(\mathrm{L}|\mathrm{DS}) \geqslant 1{\cdot}0$ and $0 \geqslant p(\mathrm{L}|\mathrm{SS}) \geqslant 1{\cdot}0$, where $p(\mathrm{L}|\mathrm{DD})$, $p(\mathrm{L}|\mathrm{DS})$ and $p(\mathrm{L}|\mathrm{SS})$ were all varied independently), there was no adequate triplet of parameters

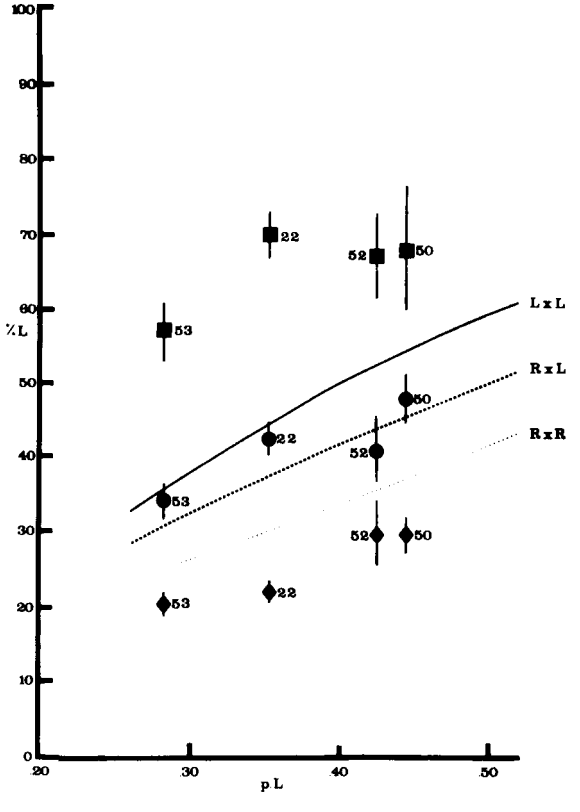

Figure 9. The proportion of left hand-clasping progeny from R × R, R × L and L × L mating pairs, for the four Japanese studies. Data points are plotted ±1 standard error. Numbers alongside data points indicate source (see Appendix A for key). (■) L × L; (●) R × L; (◆) R × R. Solid and dotted lines marked L × L, R × L, and R × R represent the fitted lines for *non*-Japanese studies shown in figure 8, and are present for comparison. Note: unlike figure 8, all data points in this figure are plotted on the same scale and axes.

which would fit all 16 sets of data. However, if the 4 J and 12 NJ studies are considered separately, then for each group a single c, z pair may be found which fits the data satisfactorily; this is shown graphically in figure 10. The implications of this analysis are clear. A two-allele symmetric model may only be used to fit hand-clasping family data if one is willing to accept that the degree of dominance and penetrance will be greater in Japanese than non-Japanese populations; that is, it is *not* allele frequencies alone which differ between populations. This conclusion is necessary for *any* two-allele model however constructed. While there are precedents for variations in the penetrance of a character between populations, a more parsimonious alternative for the differences between the J and NJ populations is that there are *three* alleles, and that changes in allele frequency alone determine differences between populations, the penetrance being identical in each case. As mentioned earlier, the generalized three-allele model (Model IV) has too many free-floating parameters for it to be useful. We therefore attempted to fit the simple, three-allele model described earlier; this model has *a priori* acceptability in view of its similarity to a model already shown to be useful in the case of handedness. The model has three alleles and only two phenotypes; a knowledge of $p(\mathrm{L})$ alone is therefore not sufficient for determining allele frequencies. However, the extra degree of freedom is lost if one considers the goodness of fit of family data. To do this, a computer program was used which systematically varied the frequency of one allele (say, C). For a particular value of C (where $D+C+S=1$) and a known value of $p(\mathrm{L})$, the values of L and S are uniquely defined. From these may be calculated the values of $p(\mathrm{L}|\mathrm{R}\times\mathrm{R})$, etc. and these values tested against empirical data. Clearly the accuracy of estimation of allele frequencies is entirely dependent upon the number of families included in the

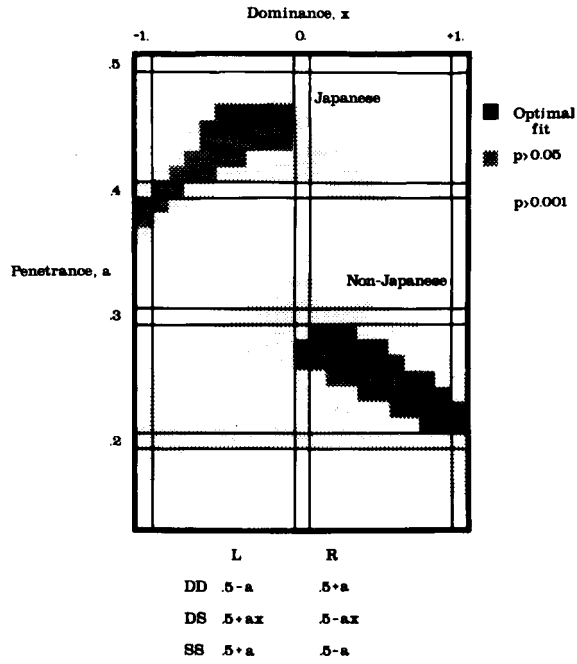

Figure 10. Shows, for Japanese and non-Japanese data analysed separately, the values of c and z in a two allele symmetric model which will satisfactorily fit the data. See text for further details.

data. The process of allele frequency estimation may be seen in figure 11 which shows the Japanese data only. The value of C is systematically altered and the χ^2 value calculated; the minimal χ^2 value may be found, as also may the 5% range of satisfactory C values. The same process for NJ populations is shown in figure 12. Note that in figure

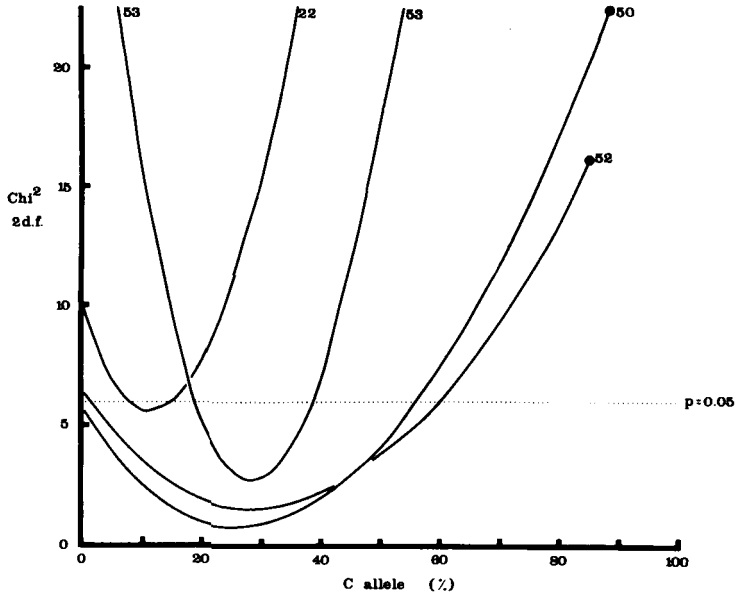

Figure 11. Goodness of fit of a three-allele model for the Japanese data with various values of C allele incidence. Numbers alongside curves indicate data source (see Appendix A for key). A solid circle at the end of a curve means that an absolute limit has been reached (see text for details).

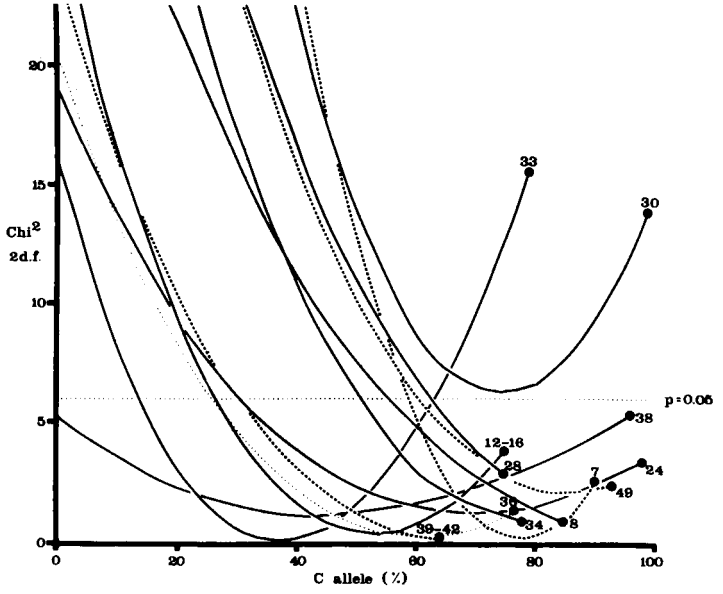

Figure 12. As for figure 11 but for non-Japanese studies.

Table 3. Shows optimal allele frequency estimates for 16 populations. Optima were estimated by observing goodness of fit while altering *C* allele frequency at intervals of 0·01. 5% ranges are also shown, except where a dagger is given, in which case the limit is absolute (see text). Data are arranged in order of ascending *D* allele estimates. All allele estimates are × 100.

	D			*S*			*C*			χ^2 Goodness of fit of optimal values		
		5% range			5% range			5% range				
Study	Optimal	Max	Min	Optimal	Max	Min	Optimal	Max	Min	(2 df)	*p*	*N* (children)
ICM (this study)	0	13·6	0†	21·8	35·3	21·8	78	78†	51	1·150	0·562	442
Ferronato *et al.* (1974)	0	14·9	0†	14·5	29·0	14·5†	85	85†	56	0·965	0·617	207
Falk and Ayala (1971)	6·0	15·5	0†	15·9	25·4	9·9†	78	90†	59	0·128	0·938	1187
CGNM-T (this study)	8·3	25·8	0†	30·6	48·1	22·6†	61	77†	26	0·224	0·894	301
Wiener (1932)	10·1	22·5	6·5†	3·9	16·4	0†	86	93†	61	2·037	0·361	469
Lai and Walsh (1965): White Australians	11·0	35·0	1·5†	9·9	33·9	0†	79	98†	31	1·229	0·540	198
Leguebe (1967)	17·7	‡	‡	17·2	‡	‡	65	‡	‡	6·311	0·042	912
Pons (1961)	24·1	48·1†	0†	27·8	51·8†	3·8†	48	96†	0†	0·991	0·609	106
Lai and Walsh (1965): New Guineans	23·9	23·9†	30·4	0	6·5	0†	76	76†	63	2·631	0·268	321
Rhoads and Damon (1973)	35·7	52·2	35·7†	0	16·7	0†	64	64†	31	0·099	0·951	257
Freire-Maia *et al.* (1958)	35·9	48·9	24·9†	11·0	24·0	0†	53	75†	27	0·363	0·834	466
Lutz (1908)	41·5	53·0	29·0	21·4	32·9	8·9	37	62	14	0·038	0·981	598
Yamaura (1940)	42·7	55·7†	28·2	31·2	44·2†	16·7	26	55	0	0·722	0·696	436
Yoshiwara (1957)	42·9	56·4	27·9	28·0	41·5	13·0	29	59	2	1·562	0·457	294
Kawabe (1953)	57·6	62·1	52·6	14·4	18·8	9·3	28	38	19	2·679	0·261	1498
Kawabe (1949)	59·1	60·6	57·1	29·8	31·3	27·8	11	15	8	5·526	0·063	1392

† Limits absolute rather than due to 5% values of χ^2.
‡ 5% limits not calculable due to no optimal fit with $P > 0{\cdot}05$
Note: C values are in integer units (see text). Values of *D* and *S* less than 1 have been rounded down to 0.

12 many of the lines terminate at relatively low values of χ^2; this is due to an absolute limitation on the frequency of the *C* allele, for clearly if *C* becomes too high, it is impossible to find a population of greater than a certain value of *p*(L). In the extreme case, if $C = 1{\cdot}0$, then all populations must have a *p*(L) of 0·50.

Table 3 shows, for all 16 groups of family data, the optimal values of *D*, *C* and *S*, and their 5% ranges, as well as the χ^2 value for the optimal fit, all but one of which are acceptable with $p > 0{\cdot}05$. Note that in many cases an absolute limit of allele frequency is more important than the 5% limits. The rows of table 3 are arranged in ascending order of optimal *D* estimates. It will be noted immediately that this shows a moderate correlation with the longitude of the population group; European ('Caucasian') groups have low *D* values, Japanese groups have high *D* values. This trend can probably explain the east–west cline demonstrated in figure 3.

A further problem of the two-allele symmetric model discussed earlier, and shown in figure 10, is that although there are several non-Japanese, non-European populations, these show penetrance values more akin to Europeans than to Japanese; this seems counter-intuitive in view of the east–west cline being shown identically for Japanese and for non-Japanese, non-European groups. Further study of table 3 provides an explanation of this anomaly. As the *D* values increase as one passes down through table 3, the *C* values show a concurrent decrease. However, the *S* values appear to show little relation to the *D* values. This may be demonstrated formally by a Pearsonian correlation analysis:

	C	*S*
D	$r = -0{\cdot}865$ $p = 1{\cdot}5 \times 10^{-5}$	$r = +0{\cdot}266$ $p = 0{\cdot}317$
C		$r = -0{\cdot}611$ $p = 0{\cdot}0118$

There is no correlation between the values of *D* and *S*, although highly significant negative correlations exist between *C* and *D*, and *C* and *S*. (The partial correlation of *D* and *S*, $r_{DS.C}$ is of course negative ($-0{\cdot}661$) and significant, since $D + C + S = 1$). From table 3 it is apparent that the *S* allele is more common in the Japanese populations than in the other Eastern groups. This can be seen more clearly in table 4. We can thus summarize. There is an east–west cline in LHC due to an increased proportion of *D* alleles in non-European populations. There is a higher frequency of *S* alleles in European than in non-Japanese, non-European populations, perhaps a reverse cline. However, as in some other ways the Japanese seem to resemble Europeans more than

Table 4. Mean allele frequency estimates for population groups. Analysis of variance after arcsin transformation ($p^1 = \sin^{-1}(\sqrt{p})$). Differences between geographical groups: *D Allele*: F(2, 13) = 7·79, $p = 0{\cdot}006$; E *vs* NJNE F(1, 10) = 4·28, $p = 0{\cdot}065$; E *vs* J F(1, 11) = 12·05, $p = 0{\cdot}0052$; J *vs* NJNE F(1, 5) = 3·87, $p = 0{\cdot}106$; *S Allele* F(2, 13) = 6·93, $p = 0{\cdot}0089$; E *vs* NJNE F(1, 10) = 7·16, $p = 0{\cdot}0232$; E *vs* J F(1, 11) = 2·35, $p = 0{\cdot}152$; **J *vs* NJNE F(1, 5) = 14·44, $p = 0{\cdot}0126$**; C *Allele* F(2, 13) = 13·63, $p = 0{\cdot}0006$; E *vs* NJNE F(1, 10) = 0·10, $p = 0{\cdot}75$; E *vs* J F(1, 11) = 24·40, $p = 0{\cdot}004$; J *vs* NJNE F(1, 5) = 24·76, $p = 0{\cdot}0042$.

Allele	European (E) $n = 9$	Non-Japanese Non-European (NJNE) $n = 3$	Japanese (J) $n = 4$
D	0·131	0·318	0·506
S	0·181	0·036	0·258
C	0·687	0·645	0·236

other Eastern groups, they also have high 'European', levels of *S*. The Japanese thus have high values of both *D* and *S* alleles; other groups have high values of either *D* or *S* alleles. Hence the apparent difference demonstrated in figures 8 and 9.

Twin data

Only two studies have provided data on hand-clasping in monozygotic and dizygotic twin pairs. In one study (Martin 1975), there is no indication of the population incidence of LHC. In neither study are there data from family studies, and thus it is not possible to estimate allele frequencies for the populations from which the twins were drawn.

For completeness, data from the studies are shown in table 5. It is worth noting that Martin's interpretation of a lack of genetic control, due to a failure to find an increased concordance in MZ rather than DZ twins, is erroneous; as shown in the case of handedness (McManus 1978), lack of concordance in genetic systems in which chance plays a large role is of little consequence. The three-allele model predicts expected frequencies in MZ twin pairs which are close to binomial frequencies.

Family size

There is no evidence for any differential fertility between right and left hand-claspers (Leguebe 1967); neither do our own data provide any evidence for such a possibility.

Table 5. Data on hand-clasping in twins.

		Pair type						
Twin type	Study	R–R		R–L		L–L		*n*
Monozygotic	Martin	9		12		7		28
	Dahlberg	18		34		17		69
	Total	27	(27·8%)	46	(47·4%)	24	(24·8%)	97
Dizygotic	Martin	1		11		7		19
	Dahlberg	34		56		33		123
	Total	35	(24·6%)	67	(47·2%)	40	(28·2%)	142

4. Arm-folding

Definition

If one asks someone to fold their arms, then one fore-arm crosses over the other. Left arm-folding (LAF) is defined as the left radius and ulna over the right radius and ulna. Test–retest reliability does not seem to be as high as for hand-clasping. Wiener (1932) found that 3 out of 22 individuals were inconsistent in their preference over an 18-month period. Assessment is usually by visual inspection, and a family study using this method has been carried out by one of us (CGNM-T). Unlike the case of hand-clasping, it was not felt practicable in a questionnaire study by the other author (ICM) to try to assess arm-folding, it being difficult to devise an unambiguous question.

Population incidence

The incidence of LAF has been determined in 23 population groups. The distribution, shown in figure 13, is very interesting. Unlike the case of hand-clasping,

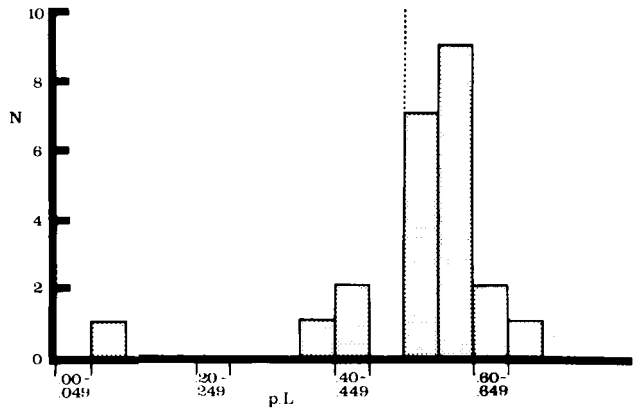

Figure 13. The incidence of left arm-folding, for 23 populations.

which shows an almost normal distribution, there is a very sharp cut-off, the majority having p(L) values of greater than 50%. However, there are four populations in which p(L) is less than 50%, all being significantly so ($p < 0{\cdot}001$ for all groups except the Fali, of Huizinga (1968), for whom $p < 0{\cdot}05$). Three of these groups (the Fali and Kurumba Huizinga 1968)), and the Angolan Negroes (Freire-Maia and de Almeida 1966) are African, while none of the groups with p(L) $> 0{\cdot}50$ are African. The fourth anomalous group is a group of Russian immigrants to Brazil, who have an extremely low incidence of LAF (8·8%) (Freire-Maia, Freire-Maia and Quelce-Salgado 1960). Although not explicitly stated, it is highly likely that this group is strongly inbred, possibly for several centuries.

Unless the data from these four groups with p(L) $< 0{\cdot}50$ can be shown to be methodologically inadequate, it is highly likely that a two-allele asymmetric model will be unable to cope with the arm-folding data.

Generational differences

There are insufficient data in the literature to investigate this phenomenon, but if effects exist they are almost certainly small.

Sex differences

Figure 14 shows data from 17 population groups for whom data are given on sex differences. There is no evidence for overall differences being significant.

Assortative mating

Table 6 shows data from five studies, two of which are our own. The incidences of LAF in the five studies do not differ significantly, and neither does the 5 × 3 table show any heterogeneity ($\chi^2 = 12{\cdot}87$, 8 df, NS). The data may thus be amalgamated. Of 879 mating pairs, 173 (19·68%) were both right arm-folding, 401 (45·62%) were of different arm-folding types, and 305 (34·69%) both showed LAF. Expected values under a binomial hypothesis are 158·7, 429·6 and 290·7, respectively, which do not differ significantly from observed values ($\chi^2 = 3{\cdot}89$, 2 df, NS).

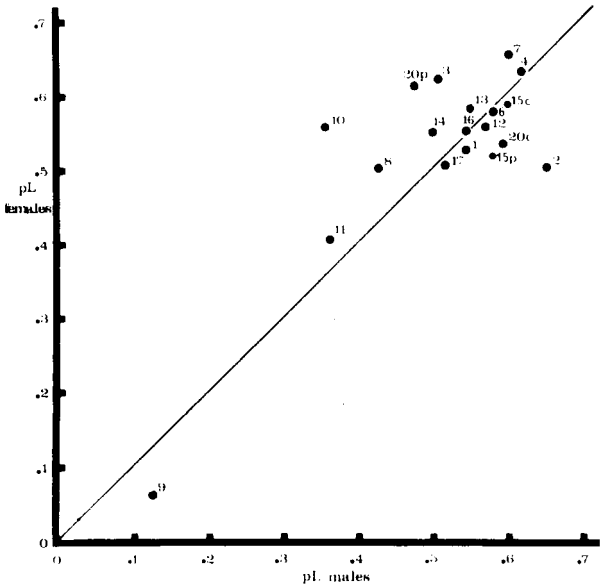

Figure 14. The incidence of left arm-folding in females as a function of the incidence of left arm-folding in males, for data from 19 populations. Numbers alongside data points indicate the source (for key see Appendix B).

Table 6. Mating patterns for arm-folding.

Mating type	Ferronato *et al.* (1974)	Leguebe and Martinez-Fuentes (1971)	CGNM-T (this study)	Rhoads and Damon (1973)	Wiener (1932)	Total
Right–Right	11	70	39	28	25	173
Right–Left	33	140	100	84	44	401
Left–Left	31	109	54	77	34	305
Total	75	319	193	189	103	879

Familial data

There are five studies in the literature which give family data on arm-folding. In addition one of us (CGNM-T) has carried out a survey of familial trends in arm-folding (see table 7 for data). The incidences of LAF in these six studies show no significant differences (CGNM-T, $n=301$, LAF = 58·47%; Falk and Ayala (1971), $n=1196$, LAF = 55·93%; Wiener (1932), $n=389$, LAF = 56·29%; Leguebe and Martinez-Fuentes (1971), $n=897$, LAF = 56·41%; Rhoads and Damon (1973), $n=260$, LAF = 58·84%; Ferronato *et al.* (1974), $n=204$, LAF = 56·86%: χ^2 for homogeneity = 1·64, 5 df, NS). It is thus possible, for the purposes of comparison, to combine the data from the six studies. Of 682 progeny of R × R matings, 51·31% show LAF; of 1553 progeny of R × L matings, 56·53% show LAF; and of 1013 progeny of L × L matings, 60·31% show LAF. These differences are significant overall ($\chi^2 = 13{\cdot}42$, 2 df, $p < 0{\cdot}005$). The difference between R × R and R × L is significant ($\chi^2 = 5{\cdot}03$, $p < 0{\cdot}05$) as is the difference between R × R and L × L ($\chi^2 = 13{\cdot}09$, 1 df, $p < 0{\cdot}001$). The difference between R × L and L × L is almost significant ($\chi^2 = 3{\cdot}45$, 1 df, $p < 0{\cdot}10$). This is strong evidence for a familial, and probably a genetic, component in the control of arm-folding.

Table 7. Arm-folding: family tables, by sex, of a study by CGNM-T.

		Right		Left	
Father	Mother	Male	Female	Male	Female
Right	Right	9	12	23	19
Right	Left	14	17	19	14
Left	Right	18	22	22	29
Left	Left	19	14	23	27

For each parental pair, all eligible children were included.

Although the proportions of LAF overall are the same in all six studies, the family
:a themselves are not homogeneous; thus if one considers the progeny of R × R
tings, then there are significant differences between the studies ($\chi^2 = 19{\cdot}78$, 5 df,
0·01). This situation is analogous to the differences between Japanese and non-
anese populations in the case of hand-clasping, and for identical reasons it means
: it will be possible to fit a two-allele model (of any type) only if one assumes that
etrance and/or dominance may differ between populations. This might be an
easonable assumption, and thus it was felt more reasonable, as with hand-clasping,
fit a three-allele model, and once again the simple three-allele model described earlier
as used. Table 8 shows, in an exactly analogous manner to that described earlier for
and-clasping, the optimal estimates and the 5% ranges of allele frequency estimates for
ie six studies. Five of the six studies are fitted satisfactorily by what is, in effect, a two-
.lele asymmetric model, the frequency of *D* being 0. The study of Falk and Ayala,
owever, requires a significant proportion of *D* alleles, which is difficult to explain. If the
opulation had contained a proportion of persons of African derivation, these *D* alleles
'ould have been comprehensible. According to Falk and Ayala their population
onsists of "all Caucasians, more than 90% are descendants of immigrants from
reland, Italy and Portugal"; possibly the Portuguese contingent contained a
roportion of 'African' *D* alleles derived perhaps from the Moorish invasions of the
berian peninsula.

In conclusion, the arm-folding data are satisfactorily fitted by a three-allele model
ith identical properties to that proposed for hand-clasping.

vin data

To our knowledge there are, in the literature, no arm-folding data from twins.

Inter-relations between asymmetries

There is strong evidence that three behavioural asymmetries, handedness, hand-
asping and arm-folding, are familial, and are probably genetic. For this conclusion to
of real worth, it is necessary to know whether there is any relationship between the
ymmetries; thus if hand-clasping were to be strongly correlated with handedness, the
milial trends in hand-clasping would be trivial.

landedness and hand-clasping

Seven studies have looked at this. Wiener (1932), Ferronato *et al.* (1974) and
Beckman and Elston (1962) all found no correlation between handedness and hand-
:lasping. One of our studies (ICM) found no correlation (see table 9(i)). The study by
CGNM-T found a significant correlation when all individuals were included in the

Table 8. As for table 3 but for arm-folding.

Study	*D*			*S*			*C*			χ^2 goodness of fit of optimal values	*p*
		5% limits			5% limits			5% limits			
	Optimal	Max	Min	Optimal	Max	Min	Optimal	Max	Min		
CGNM-T	0	2·5	0†	16·9	19·4	16·9†	83	83	78	4·84	0·088
Wiener (1932)	0	4·2	0†	12·7	16·7	12·7†	87	87†	79	3·43	0·179
Ferronato *et al.* (1974)	0	4·1	0†	13·8	17·8	13·8†	86	86†	78	4·11	0·128
Rhoads and Damon (1973)	1·1	17·6	0†	18·8	35·3	17·8†	80	82†	47	0·89	0·640
Leguebe and Martinez-Fuentes (1971)	1·1	9·5	0†	13·9	22·4	12·9†	85	87†	68	2·02	0·364
Falk and Ayala (1971)	18·1	27·1	8·1	29·9	38·9	19·9	52	72	34	0·16	0·918

† Limits absolute rather than due to 5% values of χ^2.
Note: *C* values are in integer units (see text). Values of *D* and *S* less than 1 have been rounded down to 0.

analysis (table 9 (ii *a*); $p<0{\cdot}005$). However, this seemed to be primarily due to a small number of left-handed families; when the data were re-analysed for the parental generation only (i.e. who were genetically unrelated) the relationship became non-significant (table 9(ii *b*)). The study of Pelecanos (1969) claimed to find a 'highly significant positive correlation'; however, no data are given, and, as explained earlier, this study is open to serious doubt in view of the very low incidence of LHC. Rhoads and Damon (1973) claimed to find a significant correlation ($p<0{\cdot}01$) but they give insufficient data to be able to assess this claim adequately. In summary, there is no adequate evidence for a correlation between handedness and hand-clasping.

Table 9. Relationship between handedness and hand-clasping. Including both parents and progeny.

		Hand-clasping			
		Right	Left	*n*	%LHC
(i) *ICM*					
Handedness	Right	545	688	1233	55·80
	Left	70	99	169	58·58
		$\chi^2=0{\cdot}36$, 1 df, NS.			
(ii) *CGNM-T*					
(*a*) *All individuals*					
Handedness	Right	290	342	632	54·1
	Left	13	42	55	76·3
		$\chi^2=9{\cdot}28$, 1 df, $p<0{\cdot}005$.			
(*b*) *Parents only*					
Handedness	Right	172	181	353	51·27
	Left	11	22	33	66·66
		$\chi^2=2{\cdot}28$, 1 df, NS.			

Handedness and arm-folding

Five studies have investigated this relationship. Data from a study by one of us are shown in table 10; no evidence was found for a correlation. Wiener (1932), Ferronato *et al.* (1974), Pelecanos (1969) and Beckman and Elston (1962) all found a similar lack of association. No studies have found a positive association, and thus this result seems fairly secure.

Table 10. Relationship between handedness and arm-folding (CGNM-T).

		Arm-folding			
		Right	Left	*n*	%LAF
Handedness	Right	276	357	633	56·39
	Left	29	25	54	46·29
		$\chi^2=1{\cdot}66$, 1 df, NS.			

Hand-clasping and arm-folding

Seven studies have investigated this relationship. Data from a study by one of us (CGNM-T) are shown in table 11; there is no significant association. A similar lack of correlation was found by Wiener (1932), Falk and Ayala (1971), Ferronato *et al.* (1974), Pelecanos (1969), Beckman and Elston (1962) and Rhoads and Damon (1973). No studies have found a significant correlation and thus the statistical independence of hand-clasping and arm-folding also seems fairly secure.

Table 11. Relationship between arm-folding and hand-clasping (CGNM-T).

		Arm-folding: Right	Left	*n*	%LHC
Hand-clasping	Right	130	171	301	56·81
	Left	174	212	386	54·92
		$\chi^2 = 0{\cdot}173$, 1 df, NS.			

Situs inversus

Situs inversus totalis (either with or without Kartagener's triad) is probably inherited in a similar manner to handedness, a two-allele asymmetric model describing the available data (McManus 1978). Situs is known to show no correlation with handedness (Torgerson 1950). No studies, to our knowledge, have looked at hand-clasping or arm-folding in situs inversus; it is conceivable but unlikely that one (or even both) of these behavioural asymmetries is associated with situs, and this topic clearly needs investigation.

Linkage

While there is no simple association between handedness, arm-folding and hand-clasping, it is possible that the genes for their control are located on the same chromosome, and thus would show genetic linkage. Without being able to accurately genotype individuals, it is not easy to say with certainty that linkage is or is not present. A crude analysis is, however, possible. Consider two parents, both of whom are right-handed and both of whom are right hand-claspers. If linkage occurs, then their children should tend to be either right-handed and right hand-clasping, or left-handed and left hand-clasping more often than chance would predict. And similarly for both parents right-handed and left hand-clasping, etc. Table 12 shows, for all pairs in which both parents are of the same handedness and hand-clasping, the proportion of progeny with the same or different hand-clasping and handedness. If linkage occurs, same–same and opposite–opposite should occur more frequently than same–opposite and opposite–

Table 12. Linkage of handedness and hand-clasping. The tables consider only mating pairs for which both parents are of the same handedness and of the same hand-clasping type. Table shows number of progeny in which hand-clasping and/or handedness are the same as the parents.

		Hand-clasping: Same	Opposite	
ICM				
Handedness	Same	79	63	142
	Opposite	16	7	23
		95	70	165
		$\chi^2 = 1{\cdot}047$, 1 df, NS.		
CGNM-T				
Handedness	Same	50	65	115
	Opposite	2	4	6
		52	69	121
		$\chi^2 = 0{\cdot}004$, 1 df, NS.		

For ICM data, only one progeny for each parental pair: for CGNM-T all progeny included.

Table 13. Linkage of handedness and arm-folding.

		Arm-folding		
		Same	Opposite	
Handedness	Same	37	72	109
	Opposite	2	4	6
		39	76	115
		$\chi^2 = 0{\cdot}17$, 1 df, NS.		

Data from CGNM-T only. As for table 11.

Table 14. Linkage of arm-folding and hand-clasping.

		Arm-folding		
		Same	Opposite	
Hand-clasping	Same	9	23	32
	Opposite	21	29	50
		30	52	82
		$\chi^2 = 1{\cdot}08$, 1 df, NS.		

Data from CGNM-T only; otherwise as for table 11.

ame; there is no evidence for this in the present analysis. Tables 13 and 14 show similar nalyses for handedness and arm-folding, and arm-folding and hand-clasping; again, here is no significant linkage.

5. Discussion and conclusions

It is beyond reasonable doubt that hand-clasping and arm-folding are familial, and n the absence of any other theory which can cope with the data, a genetic model seems easonable. If penetrance values are to remain constant within populations, a two-llele model is unacceptable, and it is necessary to postulate a three-allele model. This aises several questions which can perhaps be best discussed by considering the nature nd evolutionary development of asymmetries.

In the absence of genetic control, and due to the inevitable chance fluctuations that ne finds during development, one must expect that organisms will show a degree of symmetry, albeit often minimal. 50% of organisms will show one side as 'dominant' nd 50% the opposite side as 'dominant', i.e. a racemic mixture, due to *fluctuating symmetry*. *Directional asymmetry* is asymmetry in which a non-racemic mixture of the wo phenotypes is found, one enantiomorph predominating over the other. In its most xtreme form, all of a species are directional in the same way (e.g. in the fiddler crab and ther crustacea almost *all* organisms are born with the left claw larger (Vernberg and Costlow 1966, Hamilton, Nishimoto and Halusky 1976)). Directional asymmetry poses listinct problems for genetic systems. Consider the problem of an observer looking at a pair of gloves. He is in radio contact with another individual in a space-ship, who also has a pair of gloves in front of him. The problem for the observer is to tell his galactic colleague which of the two gloves he happens to be pointing at. This problem, first posed by Kant, is impossible to solve unless both the observer and his colleague have a known and conventional asymmetry. The problem would be insoluble if we were

communicating with a Martian of unknown asymmetry. (For an ingenious solution dependent upon the failure of sub-atomic parity conservation, see Gardner 1967.) The problem for genes determining asymmetries is essentially similar to that of the observer and the astronaut. The linearly sequenced gene-code (equivalent to the radio message) has to tell a three-dimensional system which of two possible enantiomorphic forms is the correct one. A possible, although highly unlikely, solution, lies in the asymmetry o the helix of the genetic DNA itself, or of the asymmetry of many other biological macro molecules; there is, however, no evidence that this information may be used by cells and it is extremely difficult to conceive of a possible mechanism.

The only clear, but somewhat unusual, example of the inheritance of an asymmetry i discussed by Morgan (1976) and found to be of particular interest. The snail, *Limnae peregra*, occurs in two forms, a common dextral, and a rare sinistral, form. Th asymmetry is determined by a recessive gene which has the strange property of workin one generation out of phase: the asymmetry of an individual depends upon th genotype of its 'mother' (they are hermaphroditic), and not at all upon its own genotyp (Boycott, Diver, Garstang and Turner 1920). The effect is due to cytoplasmi inheritance. Thus a gene produces cytoplasm of a particular asymmetric type However, the gene manifests after the asymmetry of the organism has actually beer determined, and the cytoplasm produced will therefore control the asymmetry of th individual's progeny, etc. Morgan proposed that cytoplasmic inheritance might be o importance in determining human asymmetries. Whilst it may have a role, the model clearly cannot cope with several asymmetries all of which appear to be statistically independent. Furthermore, evidence for maternal inheritance of these asymmetries is conspicuously lacking in most of the cases, and is, at best, dubious in the case o handedness. Whilst *all* of asymmetry cannot be explained by cytoplasmic inheritance, i is nevertheless quite possible that there is a cytoplasmic component in asymmetry inheritance, for it is still a problem that the genes are, except for their own helical asymmetry, essentially left–right agnosic.

Elsewhere (McManus 1978) a model has been proposed for the inheritance of handedness which depends upon two alleles, *D* and *C* (i.e. Model I). The *CC* genotype shows pure fluctuating asymmetry; the *DD* genotype is completely directional. The problem remains of how these genes are working. One solution is to suggest that some form of 'asymmetric signpost' is carried over from individual to individual by cytoplasmic inheritance. There is generally only one phenotype for this signpost and thus all organisms of the species possess it. The action of the *DD* genotype is to read this signpost and to go in the direction pointed. The *CC* genotype is effectively blind, and ignores the signpost. With such a system it is possible for the genotype for one asymmetry (say handedness) to be *DD*, and thus to read the signpost, and for another asymmetry (say *situs inversus*) to be *CC* (and thus to ignore the signpost), thereby producing statistical independence of *situs* and handedness.

Having proposed such a model, it is easy to postulate a mechanism for the *S* allele. The *D* allele is essentially left–right agnosic; it does not distinguish left and right, it merely goes in the direction pointed. The *SS* genotype can read the signpost, but has somehow misunderstood it, and always goes *consistently* in the *wrong* direction. The result would be phenotypes enantiomorphic to the *DD* genotype. It is worth stressing that the *D* and *S* alleles are still however left–right agnosic. Consider the rural recruits to the Imperial Russian Army who were supposedly taught to march in step by having a bundle of hay tied to their right foot and a bundle of straw tied to their left foot; the officer thus called out 'Straw —Hay—Straw—Hay' to make the men march in step. The

left–right agnosic recruits *appear* to be able to distinguish left from right. But an urban officer might well have confused the meaning of straw and hay, and consistently called straw 'hay', and vice-versa (a not unknown error among town dwellers). The result would be that the recruits would still act *consistently* but would now move in the exact mirror-image of the movements produced for the first officer, even though the intentions of the officers were identical. It is thus possible to have just a pair of left–right agnosic alleles, one of which produces the enantiomorph of the other.

The problem of the inheritance of asymmetry in *Limnaea* may now be seen in a fresh light. Two alternatives present. One is that the alleles might actually, as it were, turn the signpost around. The other is that they may reverse the reading of the signpost. While the first answer is possible, it does tend to produce an infinite regress, for how does the allele know which way to point the signpost? The only solution is by another signpost, and so on. And yet if the genes only alter the reading of the signpost, this also has problems, for the direction is determined before these genes have been read themselves; their action is to read the signpost on behalf of the next generation, and to leave some form of (analogue, presumably enantiomorphic) message to this effect. This latter alternative seems the more reasonable of the two possibilities. It does, however, leave us in the situation of having no definite example of the alteration of the cellular, cytoplasmic signpost, and hence having no definite evidence for its existence. It is therefore necessary to clearly describe the properties we would expect the signpost to have, so that it will be recognizable when it is found. We may distinguish lesions of the signpost from lesions of the signpost-reading system.

Let there be two statistically independent asymmetries producing phenotypes P, $\bar{P}$ and Q, $\bar{Q}$, where $\bar{P}$ is the mirror-image of P, etc. Consider a population of homozygous individuals of phenotype P, Q (i.e. homozygous at each of the loci, which show no linkage). There is also a cytoplasmic signpost, S, which can be reversed to produce its enantiomorph, $\bar{S}$. The signpost may also be 'blocked' or made illegible, symbolized s.

If the signpost is acting typically, i.e. S, then all progeny of the hypothesized population will be of form P, Q. If however the signpost should be reversed, $\bar{S}$, all progeny will be of form $\bar{P}$, $\bar{Q}$. If the signpost should be blocked, s, then $\bar{P}$ and P, and Q and $\bar{Q}$ will each appear randomly, and *independently*, neither the P nor the Q system having any chirality information of its own. We thus have the situation:

	Phenotype proportions			
Signpost	PQ	$\bar{P}$Q	P$\bar{Q}$	$\bar{P}\bar{Q}$
S	1·0	0·	0·	0·
$\bar{S}$	0·	0·	0·	1·0
s	0·25	0·25	0·25	0·25

Consider a mutation which meant that the P locus alone was unable to read the signpost, and that the population was homozygous for this mutant; we would then expect, with the three types of signpost:

Signpost	PQ	$\bar{P}$Q	P$\bar{Q}$	$\bar{P}\bar{Q}$
S	0·5	0·5	0·	0·
$\bar{S}$	0·	0·	0·5	0·5
s	0·25	0·25	0·25	0·25

It is thus possible, in principle, to distinguish lesions of the signpost from lesions of the reading system, by looking at the effects on two or more separate asymmetries. Note that in the above P and Q must both be primary asymmetries (i.e. be statistically independent).

Having discussed the implications of the *D* and *S* alleles of hand-clasping and arm-folding for theories of asymmetry inheritance *per se*, we may briefly discuss the details of hand-clasping and arm-folding.

Unlike handedness, there is no known anatomical or physiological basis for either hand-clasping or arm-folding; they appear to be simple behavioural preferences. There is no evidence, for either asymmetry, of any advantage, or any significant consequence upon behaviour in general. They are essentially trivial behaviours; and yet are controlled genetically. This does not appear to be due to their being 'correlated characters' (Huxley 1942), for no characters are known to correlate with either asymmetry; furthermore there is no evidence for any reproductive advantage associated with either hand-clasping (Leguebe 1967) or arm-folding (Leguebe and Martinez-Fuentes 1971). Analysis of the allele frequencies for hand-clasping shows that the frequency of *C* correlates inversely with both *D* and *S*, but *D* and *S* show no significant inter-correlation. A parsimonious explanation of this would be that initially all individuals were of *CC* genotype. The alleles *D* and *S* arose by mutation, and each spread by genetic drift and migration, producing the east-west cline demonstrated earlier. The importance of genetic drift in determining the allele frequency is shown by entering the estimated values into the model of Watterson and Perlow (1978).

Initially a model was tested in which only heterozygotes of intermediate penetrance were allowed. Later a further 26 models were tested in which various combinations of penetrance were allowed. Table 15 gives these results; it may be seen that the purely intermediate model described earlier is an excellent fit, with only one other model obtaining a slightly lower χ^2 value. It may also be seen that the heterozygote *SC must* show intermediate penetrance, while the degree of the dominances of *DC* or *DS* is not critical, all allowing good fits. Parsimony would suggest that in the first instance all three heterozygotes may be regarded as of intermediate penetrance.

Animal models of hand-clasping would be useful for testing the hypothesis proposed in this paper. Behavioural analogues might be the manner in which insects fold their wings, for if the wings are of sufficient length, it is inevitable that one wing must be laid over the other, and for many insects this would seem to be constant for any individual. 50% of *Drosophila melanogaster* fold left over right, and 50% vice-versa. An attempt has been made in this species to selectively breed for one of the two phenotypes; this was successful for a few generations and then the effect was lost (Purnell and Thompson 1973). Possibly the wild type is mostly *CC*. Selection might have encouraged lethals associated with homozygous *D* or *S*, and hence the loss of the character after a few generations. Not all insects are racemic for wing-folding; less than 0·1% of *Gryllotalpa gryllotalpa* and *Acheta domesticus* fold the left wing over the right, while this same phenotype is found in about 5–10% of *Acheta assimilis*, *A. pennsylvanicus*, *A. rubens*, and *Teleogryllus commodus* (Neville 1976). We may presume that the majority of these insects have *S* alleles.

Non-behavioural analogues of hand-clasping may be found in the fish. In teleost fish, the nerves from the eyes decussate completely at the optic chiasma, either the entire left nerve passing dorsal to the right nerve, or vice-versa. In the majority of teleosts, the chiasma shows a dimorphic racemic mixture (Parker 1903). The flatfish (*heterosomata*) represent an interesting group, for they have a gross asymmetry, one eye migrating

Table 15. Shows the goodness of fit of a group of three-allele models to the 16 sets of hand-clasping data. For all models penetrance of D and S is complete (i.e. $a=0{\cdot}5$ and $b=0{\cdot}5$ in Model IV). Each of the three heterozygotes can express in three different ways; as either of the relevant homozygotes (thus in a particular model DC might manifest as if it were DD (alternatively as if it were CC) or as an intermediate degree of penetrance (symbolized by I in the table). For intermediate penetrance $p(\text{RHC}|\text{DC})=\frac{1}{2}(p(\text{RHC}|\text{DD})+p(\text{RHC}|\text{CC}))$, etc. The next 16 columns show the χ^2 value of the optimal fit of the particular model for each of the data sets. The right-hand column gives the total of the 16 χ^2 values. Each individual data set has 2 d.f. and the total has 32 d.f. For the individual columns any χ^2 less than 5·99 is an adequate fit with $p<0{\cdot}05$, and for the total column a χ^2 of less than 56·66 is an adequate fit with $p<0{\cdot}05$. Optimal goodness of fit was determined by finding the minimum χ^2 value with the C allele frequency varied in 0·01 steps between 0·01 and 0·99.

Allele Pair Genotype Penetrance DC	SC	DS	Falk and Ayala (1971)	Ferronato *et al.* (1974)	Freire-Maia *et al.* (1958)	Kawabe (1949)	Kawabe (1953)	Lai and Walsh (1965): New Guineans	Lai and Walsh (1965): White Australians	Leguebe (1967)	Lutz (1908)	CGNM-T (this study)	ICM (this study)	Pons (1961)	Rhoads and Damon (1973)	Wiener (1932)	Yamaura (1940)	Yoshiwara (1957)	Total
		DD	278·9	53·8	58·4	65·3	87·2	87·6	35·0	160·5	50·0	64·7	116·1	16·2	35·1	112·4	34·1	23·9	1280·0
	SS	I	123·2	37·9	26·3	10·0	35·8	58·6	19·1	96·4	16·1	20·2	50·0	5·3	23·6	60·3	5·6	6·5	594·7
		SS	120·7	25·8	47·8	78·4	158·4	60·5	24·8	95·6	57·2	21·2	39·6	7·3	35·1	67·4	27·9	28·8	896·3
		DD	0·1	1·0	0·6	3·0	0·1	6·5	1·1	6·9	0·8	0·2	1·2	1·3	1·7	2·0	1·0	0·4	27·8
DD	I	I	0·1	1·0	0·5	5·0	0·7	6·5	1·2	6·3	0·1	0·2	1·1	1·1	1·7	2·0	0·7	1·4	29·9
		SS	0·2	1·0	0·4	12·0	1·8	6·5	1·4	5·6	0·1	0·2	1·1	1·0	1·7	2·0	1·5	3·0	39·5
		DD	28·2	8·3	20·0	34·3	42·4	33·4	2·0	9·1	19·0	16·0	28·0	4·1	17·2	4·0	7·2	7·2	280·4
	CC	I	41·7	9·9	16·6	10·0	30·0	30·9	6·3	21·7	6·8	7·4	13·5	4·5	14·2	27·6	4·7	5·0	250·8
		SS	147·1	12·1	33·7	59·9	116·8	40·1	7·7	89·2	32·9	13·4	36·3	5·3	13·3	50·0	14·9	13·1	685·7
		DD	278·9	63·7	58·4	65·3	87·2	87·6	35·0	160·5	50·0	64·7	116·1	17·9	35·8	112·4	34·1	23·9	1291·7
	SS	I	123·2	37·9	26·3	9·6	58·6	58·6	19·1	95·7	16·1	20·2	50·0	5·3	23·6	60·3	5·6	6·5	593·7
		SS	199·6	6·1	63·6	78·4	158·4	56·7	23·3	208·4	72·7	0·2	1·4	6·8	34·0	96·8	29·6	25·6	1061·6
		DD	0·1	1·0	0·6	3·3	0·2	2·6	0·9	7·3	1·0	0·3	1·2	1·4	0·1	1·9	1·3	0·2	23·5
I	I	I	0·1	1·0	0·4	5·5	2·7	2·6	1·2	6·3	0·0	0·2	1·1	1·1	0·1	2·0	0·7	1·6	26·7
		SS	0·2	1·0	0·3	14·2	6·5	2·6	1·5	5·1	0·3	0·2	1·1	1·1	0·1	2·1	1·8	3·5	41·7
		DD	0·3	0·4	18·0	24·1	24·4	20·5	2·0	9·1	12·6	12·7	17·2	0·3	10·3	4·0	7·2	4·3	167·4
	CC	I	26·3	6·5	9·9	10·0	20·3	18·2	4·2	14·9	3·7	5·1	9·5	3·5	8·0	17·5	3·5	3·8	164·7
		SS	93·0	14·5	44·0	78·4	158·4	42·8	9·0	49·0	44·5	15·9	25·1	4·5	21·4	54·8	20·4	15·9	691·6
		DD	278·9	63·7	58·4	65·3	87·2	87·6	35·0	160·5	50·0	64·7	116·1	18·6	35·8	112·4	34·1	23·9	1292·4
	SS	I	123·2	37·1	26·3	9·7	35·8	58·6	19·1	90·8	16·1	20·2	50·0	5·3	23·6	60·3	5·6	6·5	588·0
		SS	0·4	0·9	22·4	74·3	158·4	36·6	2·0	8·9	32·3	0·2	1·4	0·2	33·7	7·3	6·8	9·7	395·5
		DD	1·8	2·6	0·5	2·5	3·9	11·8	1·1	9·0	0·1	0·4	2·3	1·4	3·6	5·7	0·8	0·7	48·2
CC	I	I	0·1	0·9	0·4	6·6	6·5	4·6	1·4	5·6	0·0	0·2	1·2	1·1	1·2	2·3	0·9	2·1	35·0
		SS	0·2	0·9	0·4	17·4	9·8	4·6	1·7	4·4	0·7	0·2	1·1	1·2	1·3	2·4	2·2	4·3	52·8
		DD	0·3	0·4	13·5	19·3	19·3	17·0	2·0	9·1	9·2	0·3	1·9	0·3	8·0	4·0	7·2	3·2	115·1
	CC	I	20·0	5·1	8·3	9·9	19·9	16·5	3·6	11·7	2·9	3·9	7·3	2·9	7·4	14·7	3·1	3·6	141·0
		SS	76·2	16·4	36·6	78·4	132·0	36·8	10·6	37·9	36·1	10·2	19·9	4·5	15·9	45·5	19·4	17·2	599·7

round so that it is on the same side of the body as its opposite number. In the most primitive heterosomata, the *Psettodes*, the optic chiasma is also racemically dimorphic, and is unrelated to the eye which migrates. The other flatfish are classified into families according to whether the chiasma is racemically dimorphic (the soles, *Soleidae*) or is monomorphic, all species showing either right over left or left over right (the flounders, *Pleuronectidae*). The soles may be further divided into two types, according to whether the left or the right eye is the one that migrates (the tongue soles, *Cynoglossidae*, are all sinistral, while the true soles, the *Soleidae* are all dextral). The flounders may be similarly sub-divided according to the migrating eye (the turbots, *Bothidae*, are sinistral and have the right optic nerve over the left, while the flounders proper, the *Pleuronectidae*, are dextral, and have the chiasma with the left optic nerve over the right (Norman 1934)). While there are almost no known exceptions to the monomorphisms of the chiasma among the *Pleuronectidae*, the migrating eye is not quite so constant, and occasionally, in both flounders and soles, one finds reversed individuals, and in a few cases the majority are actually reversed. Thus it would seem that the chiasma and the migrating eye are independent asymmetries. There is sometimes a geographical variation in the migrating eye (but never in the chiasma). A Japanese flounder, *Kareius bicoloratis*, is typically dextral, all of 83 specimens from one series being so. The American flounder, *Platichthys stellatus*, although with a typically dextral chiasma, manifests with about 50% of reversal of the migrating eye, in those specimens found off the coast of California. As one travels up the Pacific coast of America, the proportion of eye reversal type increases until off the Alaskan peninsula, 68% are reversed, and those variants off the Japanese coast have 100% reversal (all of 476 specimens in one series). *K. bicoloratis* and *P. stellatus* are both 'dextral' flounders, although the latter species manifests, in the waters off Japan, as atypical reversed dextrals (i.e. sinistrals). We may suggest therefore that the Japanese variants of these species are all *DD* and *SS* genotypes respectively. The two species occasionally hybridize to produce the 'species', *Pseudoplatichthys oshorensis*; this is of particular interest as of 27 specimens caught, 14 were reversed and 13 non-reversed, i.e. exactly intermediate between their parental groups (Hubbs and Kuronuma 1941). This may be presumed to be formally equivalent to the genotype *DS* which, in the case of human hand-clasping and arm-folding, we have proposed forms a racemic mixture of the two phenotypes; the flatfish provide strong justification for this assumption.

A further example from the flatfish also helps in justifying our conceptual model of a cytoplasmic signpost. While reversed flounders are found fairly frequently in some species, these reversed individuals *always* show a typical arrangement of the chiasma, and a typical asymmetry of the viscera; it is *only* the eye migration which is reversed. Hubbs and Hubbs (1944) describe a single flatfish of species *Tanakius kitaharae* which is regarded as exceptionally rare, since not only was its eye-migration atypical for its species, but also its optic chiasma was reversed, as also was the asymmetry of the viscera, i.e. it was a *true* mirror-image of the typical form. It thus showed reversal of three independent asymmetries. This is the only reported case of a reversed chiasma in a flounder, and the only known case of situs inversus. The coincidence of all three asymmetries thus seems highly implausible in terms of mutation of three separate genetic loci. A more parsimonious explanation might be that the cytoplasmic signpost had been reversed, thus reversing all of the other asymmetries in one fell stroke. Certainly this remarkable case is otherwise very difficult to explain.

The heterosomata are therefore of exceptional interest. They show the manner in which the *CC* genotype can differentiate into both pure *DD* and pure *SS* genotypes, and

thereby produce completely different families of fish; they support the case for the *DS* genotype producing a racemic mixture of the phenotypes; and they provide a possible case of reversal of the 'cytoplasmic signpost'.

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Appendix A

Code numbers for data points on figures 3, 4, 5, 6, 7, 8, 9, 10 and 11.

Code	Study population	Code	Study population
1.	Beckman and Elston (1962).	28.	Lai and Walsh (1965) New Guinea.
2.	Beiguelmann (1964).	29.	Lai and Walsh (1965) Philippines.
3.	Bonné (1966).	30.	Leguebe (1967).
4.	Chattopadhyay (1968).	31.	Lourie (1972) Kurds.
5.	Dahlberg (1926).	32.	Lourie (1972) Yemenites.
6.	Downey (1926).	33.	Lutz (1908).
7.	Falk and Ayala (1971).	34.	McManus (1980, this study).
8.	Ferronato *et al.* (1974).	35.	Malhotra (1968).
9.	Forrai and Bánkövi (1969).	36.	Mascie-Taylor (1980, this study).
10.	Freire-Maia and de Almeida (1966).	37.	Pelecanos *et al.* (1974).
11.	Freire-Maia *et al.* (1960).	38.	Pons (1961).
12.	Freire-Maia *et al.* (1958) 'Caucasians'.	39.	Rhoads and Damon (1973) Baegu.
13.	Freire-Maia *et al.* (1958) 'Mongoloids'.	40.	Rhoads and Damon (1973) Kwaio.
14.	Freire-Maia *et al.* (1958) 'Mulattoes'.	41.	Rhoads and Damon (1973) Lau.
15.	Freire-Maia *et al.* (1958) 'Negroes'.	42.	Rhoads and Damon (1973) Nasioi.
16.	Freire-Maia *et al.* (1958) 'Indians'.	43.	Saldanha *et al.* (1960).
17.	Frisancho *et al.* (1977) Lowland Mestizos.	44.	Singh and Goel (1975) Khatri.
18.	Frisancho *et al.* (1977) Lowland Quechuas.	45.	Singh and Goel (1975) Bhaniya.
19.	Frisancho *et al.* (1977) Highland Quechuas.	46.	Srnivasan and Mukherjee (1975).
20.	Huizinga (1968) Fali.	47.	Tiwari and Bhasin (1969) Brahmin.
21.	Huizinga (1968) Kurumba.	48.	Tiwari and Bhasin (1969) Rajput.
22.	Kawabe (1949).	49.	Wiener (1932).
23.	Lai and Walsh (1965) Aborigines.	50.	Yamaura (1940) Japanese.
24.	Lai and Walsh (1965) White Australians.	51.	Yamaura (1940) Koreans.
25.	Lai and Walsh (1965) Chinese.	52.	Yoshiwara (1957).
26.	Lai and Walsh (1965) Indians.	53.	Kawabe (1953).
27.	Lai and Walsh (1965) Japanese.		

In the figures a letter 'p' after a number indicates parental population, and a letter 'c' means the child population.

Appendix B

As for Appendix A, but for arm-folding studies of figures 13 and 14.

Code	Study population	Code	Study population
1.	Beckman and Elston (1962).	11.	Huizinga (1968) Kurumba.
2.	Beiguelmann (1964).	12.	Leguebe and Martinez-Fuentes (1971).
3.	Bonné (1966).	13.	Lourie (1972) Kurds.
4.	Chattopadhyay (1968).	14.	Lourie (1972) Yemenites.
5.	Falk and Ayala (1971).	15.	Mascie-Taylor (1980, this study).
6.	Ferronato *et al.* (1974).	16.	Pelecanos (1969).
7.	Forrai and Bánkövi (1969).	17.	Pelecanos *et al.* (1974).
8.	Freire-Maia and de Almeida (1966).	18.	Rhoads and Damon (1973).
9.	Freire-Maia *et al.* (1960).	19.	Srnivasan and Mukherjee (1975).
10.	Huizinga (1968) Fali.	20.	Wiener (1932).

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Zusammenfassung. Es wird eine Literaturübersicht über das Handfalten gegeben, und es wird gezeigt, daß dies Merkmal für die genetische Analyse geeignet ist, da es keine Klassifikationsprobleme, Geschlechtsunterschiede, Partnersiebung oder geschlechtsgebundene Vererbung gibt. Weitere Daten von zwei eigenen Studien werden berichtet.

Es werden Zwei-Allelen-Modelle diskutiert und gezeigt, daß sie zur Erklärung der Daten ungeeignet sind. Ein einfaches Drei-Allelen-Modell paßt jedoch hinreichend gut auf alle erhältlichen Daten, und es kann auch den Ost–West-Gradienten des linkshändigen Faltens erklären. Die beschränkten Daten über Armfalten paßt

dann ebenfalls auf das Drei-Allelen-Modell. Es wird gezeigt, daß Handfalten, Armfalten und Händigkeit nicht miteinander korreliert oder einfach genetisch gekoppelt sind.

Das genetische Modell wird diskutiert im Zusammenhang mit dem Problem der Assymetrie allgemein.

Résumé. La littérature sur le joignement des mains est passée en revue et est montrée convenable pour une analyse génétique, en l'absence de problèmes de classification, de difference sexuelle, d'assortiment des unions ou d'hérédité maternelle. Des données supplémentaires sont fournies par deux études de notre fait.

Des modèles génétiques à deux allèles sont discutés et sont montrés inadéquats pour expliquer les données. Cependant, un modèle simple a trois allèles s'accorde correctement à toutes les données disponibles, et rend compte aussi du gradient est ouest du joignement des mains à gauche. Les données limitées sur le croisement des bras sont également cohérentes avec le modèle à trois allèles. Le joignement des mains, le croisement des bras et la latéralité manuelle ne représentent pas de corrélation entre eux ni ne montrent de linkage génétique simple.

Le modèle génétique est discuté en relation avec le problème de l'asymétrie en général.